ISSN 0971-8834 RNI-JK-ENG-1994-2673

JK-Practitioner

International Journal of Current Medical Science & Practice

April-June, 2026 Volume 31, Number 2 www.jkpractitioner.com

Reviews

OriginalArticles

IndexingandAbstracting
The Journal is indexed and abstracted inIndMed , EMBASE/Excerpta Medica ,INMED ,SCOPUS ,PUBMED (For Medline),Ulrich'sPeriodicalDirectoryUSA,IndexAcademicus(IA),IndexCoppernicus,EBSCO,GoogleScholarandislisted World Web in University and College Databases.Bibliographic details of the Journal are available in ICMR_NIC Centre's IndMED database (http://indmed.nic.in).Full text of the articles being made available in IndMED Database (http://indmed.nic.in) (2002-2015 issues only) and at www.jkpractitioner.com (1994 till date)

Volume 31, Number 2
April-June 2026

JK-Practitioner
ISSN 0971-8834

Volume 31, Number 2
RNI-JK-ENG-1994-2673

Aims and Scope

JK-Practitioner is a multidisciplinary Medical Journal Published Quarterly from Srinagar. Emphasis is placed on matters related to medicine in the country in particular, while articles from anywhere in the world are given due importance. Besides original articles and case reports the journal includes clinical reviews guest articles on current topics in medicine and therapeutics.

Information for Subscribers

The **JK-Practitioner**: **Frequency** Quarterly, one volume per year beginning January. Other issues in April, July and October. **Correspondence Regarding Editorial matters** should be addressed to the Chief Editor Prof.GM Malik, Srinagar Kashmir. **Annual Subscription rates**: **Personal** Rs.2000/-**Institutional Rs.1500/-.** Institutional rates apply to Libraries, Schools, Hospitals, Clinics, Commercial and Private Institutions and organisations. **Reduce rates** is available for medical students as well**. Rates outside India**: US $ 100.Gulf Countries SR 100.All other countries US $ 100 or equivalent. **Concessional rate for personal subscription** is available for three-year term at Rs.5000/-Outside India US $ 300 /-, Gulf Countries SR 300 /-.Postal charges within the country is Rs.150/- extra for each issue. **Single Current issue** Rs.1000/-Old issues of the journal can be purchased at 20% discount subject to availability. **Prices are subject to change**. **Change of Address**: The publisher must be notified 30 days in advance. Volume Index appears in October issue. **Indexing/Abstracting Services**: The journal is currently indexed and abstracted **EMBASE/Excerpta Medica database, IndMED PubMed (Medline -Specific Articles)** and listed in **Ulrich's Periodicals Directory** (USA),**INSDOC** (India) and **Index Academicus** (IA), **Scopus , SCI.**

 Information for authors submitting manuscripts: The manuscripts typed in MS Word (Font Times Roman, Size 12 , double spaced) and submitted electronically to zaffar.abbas@gmail.com in one file. Each table, Illustrations, photographs should be recorded on separate pages

The articles undergo a **standard review process**. Please allow **4-6 weeks** before information regarding acceptability, resubmission or rejection is conveyed to the authors. Once the article is cleared for publication the authors have to bear the publication cost. (APC-Article Processing Charge). Any communication (Acceptance to Printing) about the article will be with the **Corresponding Author**. A complete Address both institutional and Residential (If the Printed copy has to be delivered there). E mail, mobile No and Postal Pin is must for the Corresponding author). One Print Copy will be mailed to the Corresponding Author. Additional print copies if required will be charged at Rs.1000/-per copy. Once published the articles are available for free download from jkpractitioner.com
Website: - **http://www.jkpractitioner.com**

Design & Layout
Bhel Communication
Opp SBI, Near Gol Chakkar, Dhillon Marg Tagore Theatre Road, Model Town, Patiala-147001, India
(7009658583,7009004425). rajesh.behal@gmail.com

Review

Post-Traumatic Benign Paroxysmal Positional Vertigo: Diagnostic Challenges and Contemporary Management.

Shanib Ali, Seema Monga , Nasreen Begum.

Background: Post-traumatic benign paroxysmal positional vertigo (PT-BPPV) is a common but underdiagnosed cause of dizziness after head injury. It is distinct from idiopathic BPPV in its higher rates of bilateral and multicanal canal involvement, lower single-session repositioning success, and greater recurrence risk.

Objectives: To synthesise published evidence on the epidemiology, pathophysiology, diagnosis, and management of PT-BPPV, and to provide a clinician-oriented, decision-focused practice framework.

Methods: Structured narrative clinical review of PubMed/MEDLINE, Embase, Cochrane Library, IndMED, and IMSEAR, with the search updated through February 2026, restricted to English-language publications from January 2000 onwards. Studies were included if they reported original data on PT-BPPV epidemiology, diagnosis, management, or recurrence. Only studies with verifiable bibliographic details were cited with numerical data.

Results: Compared with idiopathic BPPV, PT-BPPV carries significantly higher odds of bilateral disease (OR 4.72), multicanal involvement (OR 4.67), and lower single-manoeuvre resolution (OR 0.56)[1,]. Head trauma is an independent recurrence risk factor[2].Vitamin D deficiency is modifiable and supplementation reduces recurrence in deficient patients[3,4]. Five evidence-based clinical steps are presented to guide diagnosis and management.

Conclusions: Bilateral positional testing, multi-session canalith repositioning, 25-hydroxyvitamin D evaluation, vestibular rehabilitation, and psychological screening form the evidence-based framework for PT-BPPV management. Prospectively registered multicentre trials are needed to strengthen the evidence base.

JK-Practitioner2026;31(2):1-6.

Author Affiliations:

Shanib Ali MS (ENT HNS),Senior resident , ENT HNS ,Shri siddhi Vinayak Medical College, UP, India **Seema Monga** MS (ENT HNS),Prof and Hod ,ENT HIMSR ,NEW DELHI , **Nasreen Begum** MS(ENT HNS) Assistant prof Shadan institute of medical sciences ,Hyderabad

Correspondence: Shanib Ali MS (ENT HNS),Senior resident , ENT HNS ,Shri Siddhi Vinayak Medical College, UP, India

Indexed: EMBASE ,SCOPUS , IndMED, ESBCO ,Google Scholar besides other national and international databases

Cite this article as: Ali S, Monga S,Begum N. Post-Traumatic Benign Paroxysmal Positional Vertigo: Diagnostic Challenges and Contemporary Management .JK Pract 2026;31(2):1-6.

Full length article available at **jkpractitioner.com** one month after publication

Keywords: post-traumatic BPPV; canalith repositioning; head injury; vestibular rehabilitation; vitamin D; Dix-Hallpike

Introduction

Post-traumatic benign paroxysmal positional vertigo (PT-BPPV) is the most common identifiable vestibular cause of dizziness after head injury. It is caused by displacement of otoconia into the semicircular canals and shares the clinical hallmark of idiopathic BPPV -- brief, position-triggered vertigo -- but differs in its epidemiology, canal involvement pattern, treatment response, and recurrence risk.

Despite its treatability, PT-BPPV is underrecognized in trauma and emergency settings. This structured narrative clinical review addresses a focused practice question: how should PT-BPPV be diagnosed and managed in trauma patients? The most relevant primary studies, meta-analyses, and clinical practice guidelines are synthesised, and a decision-oriented framework is provided. Where Indian-context data are available from published indexed studies, they are noted in a dedicated practice implications section.

Methods

A structured narrative clinical review was conducted using PubMed/MEDLINE, Embase, Cochrane Library, IndMED, and IMSEAR. The initial search covered publications from January 2000 to December 2024 and was subsequently updated through February 2026 to capture recent publications. Only English-language publications were included. Search terms included: post-traumatic BPPV, traumatic benign paroxysmal positional vertigo, head injury dizziness, canalith repositioning, and mild traumatic brain injury vestibular. The 2017 AAO-HNS clinical practice guideline[5] and Barany Society consensus documents were included regardless of publication date.

Studies were included if they reported original data on PT-BPPV epidemiology, diagnosis, management, or

recurrence, or if they provided evidence-based guidance directly applicable to clinical practice. Studies were excluded if their numerical data could not be traced to a confirmable primary source. This review did not apply formal PRISMA methodology or risk-of-bias assessment; it is a narrative clinical review intended to support clinical decision-making rather than provide a quantitative synthesis.

Epidemiology

PT-BPPV develops in a substantial proportion of patients after head injury. International trauma cohort data estimate prevalence at 15-28% of patients with closed head injury.[6,7]. Road traffic accidents are the dominant mechanism in working-age adults globally; falls are the leading cause in the elderly; and sports-related concussion increasingly accounts for cases in younger populations.

In the elderly, unrecognised PT-BPPV independently worsens fall risk, creating a cycle of vestibular dysfunction and recurrent injury. In athletes, undiagnosed BPPV prolongs return-to-sport timelines. Systematic positional assessment is absent from most trauma and sports medicine assessment protocols worldwide.

Practice implication (India): To our knowledge, one of the few indexed Indian studies reporting PT-BPPV incidence is a prospective cohort from a tertiary neurosurgery centre in Kerala, in which Haripriya et al. reported PT-BPPV in 17% of mild-to-moderate TBI patients[8].Systematic vestibular screening is not yet incorporated into standard post-trauma protocols at most Indian centres.

4. Pathophysiology

Otoconial Displacement

Traumatic shear forces detach calcium carbonate otoconia from the utricular macula. Displaced otoconia migrate into semicircular canal endolymph (canalolithiasis) or adhere to the cupula (cupulolithiasis), causing aberrant cupular deflection and positional vertigo. The greater mechanical forces involved in trauma explain the significantly higher rates of bilateral and multicanal involvement compared with idiopathic BPPV[1].

Vitamin D, Calcium Metabolism, and Otoconial Integrity

Vitamin D regulates calcium homeostasis and the structural integrity of otoconial matrix proteins. Meta-analytic data demonstrate a significant association between 25-hydroxyvitamin D deficiency (serum level <20 ng/mL) and BPPV recurrence, and show that supplementation in deficient patients reduces recurrence rates.[3,4].This is a modifiable risk factor and should be assessed in all patients with recurrent disease.

Coexisting Pathology

More severe injuries -- temporal bone fracture, perilymphatic fistula, intralabyrinthine haemorrhage -- produce distinct additional pathology requiring separate management. Cervicogenic dizziness, provoked by neck movement rather than head position, frequently coexists with PT-BPPV after whiplash and must be recognised as a separate entity.

5. Clinical Characteristics: PT-BPPV versus Idiopathic BPPV

PT-BPPV shares the core phenomenology of idiopathic BPPV but is distinguished by its epidemiology and treatment course. Table 1 summarises the key clinical differences based on the best available comparative evidence[1].

6. Diagnostic Evaluation

Diagnosis requires a structured approach covering clinical history, bedside positional testing, and targeted investigations. The diagnostic framework is summarised in Table 2.

Clinical History

The temporal relationship between head injury and dizziness onset (typically hours to days), the positional character of vertigo (rolling in bed, looking up, bending forward), episode duration under 60 seconds, nausea, and oscillopsia are cardinal features. Patients frequently do not volunteer positional triggers; direct questioning about specific head movements is essential.

Bedside Positional Testing

The Dix-Hallpike manoeuvre is the reference standard bedside test for posterior and anterior canal BPPV[1,5]. Cervical spine clearance must be confirmed before testing in the acute post-traumatic period. Bilateral testing is essential given the higher prevalence of bilateral disease in PT-BPPV[1]. The supine roll test is mandatory to detect horizontal canal BPPV.

Red Flags and Central Mimics

Central positional nystagmus -- caused by posterior fossa tumours, cerebellar degeneration, or demyelination -- lacks the latency and fatigability of peripheral BPPV and mandates neuroimaging before repositioning. Persistent postural-perceptual dizziness (PPPD) -- chronic non-spinning dizziness worsened by movement -- frequently develops after BPPV, particularly in anxious patients, and requires distinct management[9].Red flags mandating MRI are listed in Table 3.

7. Contemporary Management

The management algorithm for PT-BPPV is presented in Table 4. Multiple canalith repositioning procedure (CRP) sessions are the norm in PT-BPPV rather than the exception.

Table 1. Clinical comparison of idiopathic and post-traumatic BPPV.

Feature	Idiopathic BPPV	Post-Traumatic BPPV
Age at onset	Peak 50-70 years	Broader range; younger patients predominate (TBI demographics)
Sex	Female > Male (~2:1)	Male predominance in road trauma cohorts
Canal involved	Posterior (~80-90%)	Posterior canal predominates; bilateral and multicanal involvement significantly more frequent[1]
Bilateral disease	~10% of cases	Significantly higher (OR 4.72; p=0.006 vs idiopathic)[1]
Multicanal disease	Uncommon (~12%)	Significantly higher (OR 4.67; p=0.018 vs idiopathic)[1]
Single-session CRP success	>80%	Lower (OR 0.56 vs idiopathic; p=0.013)[1]; multiple sessions expected
Recurrence	~15-20% at 1 year	Head trauma is a significant independent recurrence risk factor[3]
Vitamin D association	Deficiency linked to recurrence	Same; supplementation reduces recurrence in deficient patients[3,4]
Psychological comorbidity	Anxiety common	Anxiety, depression, and PPPD frequent after traumatic vestibular injury[9]

CRP = canalith repositioning procedure; OR = odds ratio; PPPD = persistent postural-perceptual dizziness. Superscript numbers refer to Vancouver-format references.

Table 2. Diagnostic approach to post-traumatic dizziness.

Clinical Step	Key Actions	Pitfalls / Red Flags
History	Confirm onset relative to injury; characterise positional triggers; record episode duration (<60 s = BPPV); note nausea, oscillopsia, auditory symptoms	Patients may not report positional triggers spontaneously; brief consultations miss the diagnosis
Cervical clearance	Confirm no unstable cervical fracture before positional testing in acute trauma	Omitting clearance in acute trauma is unsafe
Dix-Hallpike test	Bilateral testing essential; 45-degree head rotation, rapid head-hang; observe for upbeat-torsional nystagmus with latency and fatigability	Central nystagmus lacks latency and fatigability and does not suppress with fixation
Supine roll test	Head elevated 30 degrees, brisk 90-degree rotation each side; geotropic = canalolithiasis; apogeotropic = cupulolithiasis	Horizontal canal BPPV missed if roll test omitted
Red flag screen	Seek: pure vertical nystagmus, direction-changing nystagmus, gait ataxia, cranial nerve signs, diplopia, failure to respond to CRP, new vertigo in known malignancy	Any red flag requires MRI brain (posterior fossa sequences) before repositioning
Vitamin D	Measure serum 25-hydroxyvitamin D in recurrent BPPV; target >30 ng/mL	Deficiency is modifiable; missed opportunity in recurrent cases[3,4].
Imaging	MRI if atypical nystagmus, CRP failure, or neurological signs; CT temporal bone if fracture or perilymphatic fistula suspected	Imaging not required for classic presentation

CRP = canalith repositioning procedure; MRI = magnetic resonance imaging.

Table 3. Red flags mandating neuroimaging before canalith repositioning.

No.	Red Flag Feature
1	Nystagmus without latency or fatigability on repeat testing
2	Pure vertical nystagmus (downbeat or upbeat)
3	Direction-changing nystagmus with fixed head position
4	Nystagmus pattern inconsistent with a single semicircular canal
5	Severe gait ataxia disproportionate to vestibular complaint
6	Acute hearing loss with severe vertigo (consider AICA territory ischaemia)
7	Diplopia, dysarthria, dysphagia, or cranial nerve palsy
8	Failure to respond to 4-5 well-performed canalith repositioning procedures
9	New positional vertigo in a patient with known malignancy
10	Headache onset coinciding with onset of positional vertigo

Any of these features mandates MRI brain with posterior fossa sequences before repositioning manoeuvres are performed.

Table 4. Management algorithm for post-traumatic BPPV.

Step	Intervention	Evidence / Notes
1. Canal identification	Dix-Hallpike (posterior/anterior canal) + supine roll test (horizontal canal) at every visit; treat dominant canal first	Bilateral and multicanal disease significantly more prevalent in PT-BPPV[1]
2. CRP - posterior canal	Epley manoeuvre first-line; Semont manoeuvre if cervical extension is limited	Level A evidence (AAO-HNS 2017)[2]; OR 0.56 vs idiopathic for single-session success[1]
3. CRP - horizontal canal	Barbecue roll (Lempert) for canalolithiasis; Gufoni manoeuvre for cupulolithiasis	Multiple manoeuvre sessions expected in PT-BPPV
4. Reassess after each manoeuvre	Repeat positional testing to detect canal conversion before ending the session	Canal conversion well documented; missed if reassessment omitted
5. Number of sessions	Do not label refractory until 4-5 well-performed sessions completed	Lower single-session resolution is the rule in PT-BPPV[1]
6. Post-procedure restrictions	No post-procedure positional restrictions recommended	AAO-HNS 2017 guideline, Level A[2]
7. Vitamin D supplementation	Measure 25-hydroxyvitamin D; supplement if <20 ng/mL; target >30 ng/mL (oral cholecalciferol)	Meta-analysis supports recurrence reduction[4]; Indian pilot RCT corroborates[9]
8. Vestibular rehabilitation	Prescribe for residual imbalance; gaze stabilisation plus balance training	Evidence-based adjunct; task-shift to trained physiotherapists where specialists unavailable
9. Refractory BPPV	Re-characterise all canals; exclude multicanal disease, central pathology, vitamin D deficiency, vestibular hypofunction	Systematic reassessment before specialist referral
10. Psychological screen	PHQ-9 and GAD-7; refer for CBT if PPPD or significant anxiety/depression	PPPD requires distinct management from BPPV[6]

CRP = canalith repositioning procedure; CBT = cognitive-behavioural therapy; PPPD = persistent postural-perceptual dizziness.

7.1 Canalith Repositioning Procedures

The Epley manoeuvre is first-line for posterior canal BPPV[5]. The Semont manoeuvre offers equivalent efficacy and may be preferred when cervical extension is limited. The barbecue roll (Lempert) is preferred for horizontal canal canalolithiasis. Given

the high prevalence of bilateral and multicanal disease[1],all canals must be assessed bilaterally, the dominant canal treated first, and reassessment performed after each manoeuvre to detect canal conversion. Post-procedure positional restrictions are not recommended[5]. A case should not be designated refractory until at least four to five well-performed sessions have been completed.

7.2 Vitamin D Supplementation

Serum 25-hydroxyvitamin D should be measured in all patients with recurrent BPPV. Documented deficiency should be corrected with oral cholecalciferol to a target level above 30 ng/mL. Meta-analytic evidence supports a reduction in recurrence with supplementation in deficient patients[3,4].

7.3 Vestibular Rehabilitation

Supervised vestibular physical therapy -- gaze stabilisation exercises, habituation, and balance training -- is an evidence-based adjunct to CRPs for residual disequilibrium. Home exercise programmes with written and video-based instruction are appropriate where specialist physiotherapy is unavailable.

7.4 Psychological Comorbidity

Anxiety and depressive symptoms are common after traumatic vestibular dysfunction, and PPPD may develop in patients with high anxiety burden[9].Screening using PHQ-9 and GAD-7, and referral for cognitive-behavioural therapy, are appropriate components of comprehensive care.

8. Evidence Summary

The principal studies informing this review are summarised in Table 5.

Table 5. Evidence summary of included studies.

Study	Country	Design	n	Key Finding
Di Cesare T et al., 2021[1] Int J Audiol 60(5):393-7	Italy	Retrospective cohort	795	Bilateral OR 4.72; multicanal OR 4.67; single-manoeuvre success OR 0.56; all p<0.05 vs idiopathic BPPV
Bhattacharyya N et al., 2017[5] Otolaryngol HNS 156(3S):S1-47	USA	CPG (AAO-HNS)	N/A	Level A: Epley manoeuvre; Dix-Hallpike reference standard bedside test; no post-procedure restrictions
Li X et al., 2025[2] Eur J Med Res 30(1)	China	Meta-analysis	Pooled	Head trauma: independent recurrence predictor after CRP (SMD 0.48; 95% CI 0.34-0.63)
Jeong SH et al., 2022[3] J Neurol 269(2):619-26	Korea	Meta-analysis	Pooled	Vitamin D supplementation significantly reduces BPPV recurrence; most pronounced in deficient patients
Rhim G & Kim MJ, 2024[4] Nutrients 16(5):689	Korea	Literature review	Pooled	Recurrence 13-65% by follow-up duration; vitamin D deficiency significant risk factor
Staab JP et al., 2017[9] J Vestib Res 27(4):191-208	Int'l	Consensus criteria	N/A	Diagnostic criteria for PPPD; CBT and serotonergic therapy recommended
Hilton M & Pinder D, 2014[11] Cochrane Database Syst Rev	Int'l	Cochrane SR	Pooled	Epley superior to sham for posterior canal BPPV
Haripriya GR et al., 2018[8] Indian J Otolaryngol HNS 70(3):337-41 PMID 30211085	India	Prospective cohort	128	PT-BPPV reported in 17% of mild-to-moderate TBI patients at tertiary centre in Kerala
Sharma K et al., 2022[10] Indian J Otolaryngol HNS 74(Suppl 3):4405-8	India	Pilot RCT	40	Vitamin D3 plus CRP reduced recurrence vs CRP alone in posterior canal BPPV
Kong TH & Seo YJ, 2026[6] Front Neurol 17:1784282 DOI: 10.3389/fneur.2026.1784282	Korea	Background review	N/A	Structured clinical pathway proposed for PT-BPPV; heterogeneous canal involvement noted; repeated CRPs often required

CBT = cognitive-behavioural therapy; CPG = clinical practice guideline; CRP = canalith repositioning procedure; OR = odds ratio; PPPD = persistent postural-perceptual dizziness; RCT = randomised controlled trial; SMD = standardised mean difference; SR = systematic review. Only studies with confirmable bibliographic details are included.

9. Practice Implications

The following recommendations apply across clinical settings.

- Bilateral Dix-Hallpike and supine roll testing should be performed in all patients presenting with post-traumatic dizziness. The assessment requires no specialist equipment and takes under three minutes.
- Cervical spine clearance must be confirmed before positional testing in the acute post-traumatic period.
- Bilateral and multicanal BPPV are significantly more prevalent in PT-BPPV[1].Assessment of all canals bilaterally is required at each visit.
- Multiple CRP sessions should be anticipated. A case should not be designated refractory until at least four to five well-performed sessions have been completed.
- Serum 25-hydroxyvitamin D should be measured in recurrent BPPV. Documented deficiency should be corrected targeting a level above 30 ng/mL.[3,4].
- Vestibular rehabilitation should be prescribed for residual imbalance.
- Psychological comorbidity (anxiety, depression, PPPD) should be screened and addressed[9].
- Red flags for central pathology (Table 3) should be sought at every assessment; their presence mandates neuroimaging before repositioning.
- **India-specific note:** Task-shifting to trained physiotherapists using standardised protocols is appropriate given limited specialist availability outside metropolitan centres. India's high background prevalence of vitamin D deficiency makes routine 25-hydroxyvitamin D assessment particularly relevant in recurrent PT-BPPV.

10. Limitations

- This is a structured narrative clinical review without a pre-registered protocol, formal risk-of-bias assessment, or duplicate independent reviewer selection. Publication bias cannot be excluded.
- Indian-specific epidemiological data are limited. To our knowledge, the Haripriya et al. cohort is one of the few indexed Indian studies reporting PT-BPPV incidence with a traceable primary source[8].Multicentre Indian studies with pre-specified outcomes are needed.
- Several numerical claims from earlier manuscript versions were removed because they could not be traced to indexed primary sources. Only figures from verifiable publications are reported here.
- Evidence on emerging technologies (smartphone nystagmus analysis, telemedicine-guided repositioning) is descriptive; prospective validation is awaited.

11. Conclusion

PT-BPPV is a common, treatable, and underdiagnosed condition in trauma care settings worldwide. It is clinically and prognostically distinct from idiopathic BPPV: bilateral and multicanal canal involvement are more prevalent, single-session repositioning success is lower, and recurrence risk is higher. Vitamin D deficiency is a modifiable contributor to recurrence.

Five evidence-based take-home points for clinicians: (1) perform routine bilateral positional testing in all post-trauma dizziness; (2) plan for multiple CRP sessions; (3) measure and correct 25-hydroxyvitamin D deficiency in recurrent disease; (4) prescribe vestibular rehabilitation for residual imbalance; and (5) screen for PPPD and psychological comorbidity. Prospectively registered multicentre trials are needed to strengthen the evidence base.

References

1**. Di Cesare T, Tricarico L, Passali GC, et al**. Traumatic benign paroxysmal positional vertigo: personal experience and comparison with idiopathic BPPV. Int J Audiol. 2021;60(5):393-397. PMID: 32959692.

2**. Li X, Chen Y, Wang H, et al.** Risk factors for recurrence of benign paroxysmal positional vertigo following canalith repositioning procedures: a meta-analysis. Eur J Med Res. 2025;30(1). DOI: 10.1186/s40001-025-02482-x.

3**. Jeong SH, Lee SU, Kim JS**. Prevention of benign paroxysmal positional vertigo with vitamin D supplementation: a meta-analysis. J Neurol. 2022;269(2):619-626. PMID: 34312735.

4. **Rhim G, Kim MJ.** Vitamin D supplementation and recurrence of benign paroxysmal positional vertigo. Nutrients. 2024;16(5):689. PMID: 38474817.

5**. Bhattacharyya N, Gubbels SP, Schwartz SR, et al.** Clinical practice guideline: benign paroxysmal positional vertigo (update). Otolaryngol Head Neck Surg. 2017;156(3 Suppl):S1-S47. PMID: 28248609.

6. **Kong TH, Seo YJ.** Post-traumatic benign paroxysmal positional vertigo: mechanisms, clinical phenotypes, and a structured clinical pathway for management. Front Neurol. 2026;17:1784282. DOI: 10.3389/fneur.2026.1784282. Published 3 February 2026. PMC12909193.

7**. Castillo-Bustamante M, Ramos BF, Whitney S, et al.** Exploring the link between traumatic brain injury and benign paroxysmal positional vertigo. Cureus. 2025;17(4):e81847. DOI: 10.7759/cureus.81847. PMID: 40196155.

8. **Haripriya GR, Mary P, Dominic M, Goyal R, Sahadevan A.** Incidence and treatment outcomes of post traumatic BPPV in traumatic brain injury patients. Indian J Otolaryngol Head Neck Surg. 2018;70(3):337-341. DOI: 10.1007/s12070-018-1329-0. PMID: 30211085.

9. **Staab JP, Eckhardt-Henn A, Horii A, et al**. Diagnostic criteria for persistent postural-perceptual dizziness (PPPD): consensus document of the committee for the classification of vestibular disorders of the Barany Society. J Vestib Res. 2017;27(4):191-208. PMID: 28508853.

10. **Sharma K, Ojha T, Dabaria R, et al.** Relation between posterior canal benign paroxysmal positional vertigo and vitamin D deficiency. Indian J Otolaryngol Head Neck Surg. 2022;74(Suppl 3):4405-4408.

11**. Hilton MP, Pinder DK**. The Epley (canalith repositioning) manoeuvre for benign paroxysmal positional vertigo. Cochrane Database Syst Rev. 2014;(12):CD003162. PMID: 25485940.

Review

Surface Electromyography as a Remote Monitoring and Biofeedback Tool for Quadriceps Reactivation Post Robot-Assisted Total Knee Arthroplasty: A Scoping Review of Protocols, Technologies, and Outcomes

Dheeraj Kumar, Jasmine Kaur Chawla

Abstract

Background: Use of robotics in total knee arthroplasty (raTKA) is well known, and has shown better results. However, neurogenic muscle inhibition and inability to voluntarily activate the quadriceps muscle are the key challenges of raTKA, leading to functional disability. Furthermore, the use of surface electromyography (sEMG) helps quantify the neuromuscular activity of muscles in real time. sEMG, when coupled with biofeedback and remote monitoring technologies, can efficiently predict quadriceps reactivation during home-based rehabilitation.

Objective: To identify and map existing evidence on the role of sEMG in remote monitoring and biofeedback for quadriceps activation following total knee arthroplasty (TKA) and robotic-assisted total knee arthroplasty (raTKA), and to generate protocols for future trials.

Methods: The scoping review was framed following the JBI methodology (Peters et al., 2021) and foundational guidelines given by Arksey and O'Malley and Levac et al. (2005). Screening of records and data charting was done by two reviewers using a piloted charting form. The scoping review is narrative and descriptive in nature. No critical appraisal was performed.

Results: The review consists of four parallel domains: (i) quadriceps dysfunction and AMI after TKA; (ii) early functional improvements with raTKA (including earlier straight-leg raise and earlier quadriceps reactivation); (iii) sEMG-based biofeedback training to improve quadriceps strength, activation, and functionality after all forms of knee surgery, with varied outcomes for TKA; and (iv) telerehabilitation and remote-monitoring systems with high engagement and non-inferior outcomes after TKA. There is an adjunct to literature where researchers has combined raTKA patients, sEMG monitoring at home, and quadriceps-specific outcomes within a closed-loop biofeedback protocol.

Conclusions: Converging but fragmented evidence supports the use of sEMG for remote monitoring and biofeedback of quadriceps reactivation following TKA, and raTKA provides a platform for standardised TKA surgery to test these digital innovations. Standardised protocol (including SENIAM; normalised amplitudes; defined feedback targets and a core outcome set) is needed to inform pragmatic randomised trials that determine efficacy, cost-effectiveness and scalability.

JK-Practitioner2026;31(2):7-15.

Author Affiliations:

Dheeraj Kumar , Research Scholar, **Jasmine Kaur Chawla ,**Associate Professor **:**Department of Physiotherapy, School of Allied Health Sciences, Manav Rachna International Institute of Research and Studies, Faridabad.

Correspondence: Jasmine Kaur Chawla, Department of Physiotherapy SAHS, MRIIRS.E mail *jasminekaurchawla.sahs@mriu.edu.in*

Indexed: EMBASE ,SCOPUS , IndMED, ESBCO ,Google Scholar besides other national and international databases

Cite this article as: Kumar D, Chawla JK. Surface Electromyography as a Remote Monitoring and Biofeedback Tool for Quadriceps Reactivation Post Robot-Assisted Total Knee Arthroplasty: A Scoping Review of Protocols, Technologies, and Outcomes.JK Pract 2026;31(2):7-15.

Full length article available at **jkpractitioner.com** one month after publication

Keywords. Surface electromyography; biofeedback; telerehabilitation; remote monitoring; robot-assisted total knee arthroplasty.

Introduction

The end-stage knee osteoarthritis is one of the major causes of disability worldwide, with a projected rate of 60%- 100% by 2050 (Courties et al. 2024). Total knee arthroplasty (TKA) is the preferred approach to treat end-stage OA (Inacio et al. 2017). Though TKA is a reliable way to reduce pain, not all patients achieve the desired levels of strength and functional performance.(Inacio et al. 2017). According to Mizner et al. (2003), quadriceps femoris weakness is a chronic issue following TKA, characterized by reduced voluntary activation, which can further induce arthrokinematic changes in the joint and lead to implant failure. Not only quadriceps strength but the synchronized activation of quadriceps and hamstrings is also altered which further delays patient's recovery (Stevens-Lapsley et al., 2010). Body mass index, age and gender can also plays a critical role in early gain of functionality post operation (Paravlic et al., 2022). Arthrogenic muscle inhibition (AMI) is due to altered afferent input

from damaged and inflamed peri-articular tissues, but also involves spinal reflex pathways, and in the chronic phase, motor cortex excitability continuous reflex that impairs the central nervous system's ability to fully activate the quadriceps(Rice et al., 2014).

Robotics is used for pre-operative imaging, dynamic soft-tissue balancing, and haptic or semi-autonomous bone resection are in robot-Assisted TKA systems (e.g., Mako, ROSA Knee, NAVIO/CORI) to enhance the positioning of the implants (Held et al., 2023; Kayani et al., 2018. A prospective cohort study by Kayani et al. (2018) showed that raTKA, compared to traditional TKA, was linked to less pain, reduced opioid usage, early mobilization and discharge with better ROM of the Knee. Lesser post-operative swelling, quicker quadriceps reactivation, and earlier discharge were reported in the major platforms with raTKA compared to conventional robotic systems (Held et al., 2023).

Surface EMG is non-invasive procedure which provide real-time muscle activation (Yang et al., 2023). It can also be used as biofeedback device to re-train muscle activation, simultaneously can measure side-to-side asymmetry, and provide quantified progress to patient and clinician (Wasielewski et al., 2011). Karaborklu Argut et al. (2022) has summarised eight randomised controlled trials and found that EMG biofeedback was better than standard care at enhancing quadriceps strength and activation. One study has compared sEMG biofeedback to NMES post-TKA and found it is more superior in improving strength, range of motion, function, and quality of life (Armshaw et al., 2024).

In this technological era, rehabilitation is also utilising digitalisation and advanced hyper-sensitive modalities. Telerehabilitation and remote-monitoring programs using smartphone, inertial measurement unit (IMU) and wearable sEMG technologies have been shown to be feasible with high patient adherence (Mobbs et al., 2023; Nuevo et al., 2024).

Review Questions

This scoping review sought to comprehensively map the evidence on the use of sEMG as a tool for remote monitoring and biofeedback to target quadriceps reactivation following TKA and raTKA. In particular, we wanted to answer four questions:

1.What sEMG protocols (electrodes, signal processing, normalisation, feedback thresholds) have been applied to reactivate the quadriceps after TKA or other type of knee surgery?

2.What hardware (form factors), software and connectivity have been used to provide sEMG biofeedback and remote monitoring, and how will they be used in raTKA?

3.What clinical, neuromuscular and patient-reported outcomes, as well as the feasibility, adherence and safety have been reported?

4.What research and implementation challenges need to be resolved to incorporate sEMG biofeedback for remote monitoring in raTKA?

Methodological justification

An overview of reviews would focus on effects in systematic reviews but would not include relevant primary evidence of device validation, engineering and feasibility studies, which is critical to our questions (Munn et al., 2018). So we chose a scoping review because we aim to systematically map and describe the extent of the evidence (regardless of the source), to clarify definitions of concepts, to describe different methods and technologies, and to identify gaps to guide a future trial (Peters et al., 2021). This choice was informed by the Right Review tool (Amog et al., 2022; Clyne et al., 2024) and is in accordance with the JBI definition of a scoping review (Munn et al., 2022; Peters et al., 2021).

Methods

Protocol, registration, and reporting

This scoping review was conducted using the JBI methodology for scoping reviews outlined by Peters et al. (2021), building on the work of Arksey and O'Malley (2005) and Levac et al. (2010).

Review team and knowledge users

In line with current JBI recommendations (Peters et al., 2021; Pollock et al., 2022), the review team consist of two reviewers from physiotherapy background. Knowledge users (two practicing physiotherapists were involved in refining the review questions, inclusion criteria, data-charting form, and the interpretation of the findings, consistent with the JBI co-creation approach (Pollock et al., 2022). This was to help ensure that the review's development, findings and implications were relevant to clinical practice and patients and that conclusions were not overreached (a known threat to conclusions derived from scoping reviews (Pollock et al., 2024)).

Eligibility criteria (PCC)

Inclusion criteria were based on the JBI Population, Concept, Context (PCC) model (Peters et al., 2021) and questions.

Population: primary TKA (including raTKA) in adults aged ≥18. We included studies of unicompartmental arthroplasty or other (non-arthroplasty) knee surgery (for example, anterior cruciate ligament reconstruction or meniscectomy) as methodological references for sEMG protocols as few TKA-specific studies were available.

Concept: use of sEMG of quadriceps muscles (rectus femoris, vastus medialis, vastus lateralis, vastus intermedius) for monitoring, biofeedback or closed-loop sEMG-triggered NMES. Invasive EMG and diagnostic-only studies were excluded.

Setting: any rehabilitation setting (inpatient, outpatient, home, hybrid) and pre-operative, early (≤6

weeks), subacute (6 weeks - 6 months) and late (>6 months) post-operative time points.

Type of studies: randomised and non-randomised controlled, cohort, feasibility, registered protocol, systematic reviews, narrative reviews, engineering/device development/validation studies, and peer-reviewed conference proceedings. Editorials, case reports (n < 3) and non-English sources without abstracts in English were excluded. Studies of last 10-15 years were included.

Information sources

We searched nine literature databases from their inception to the date of the search: PubMed/MEDLINE, Embase, Scopus, Web of Science Core Collection, Cochrane Central Register of Controlled Trials (CENTRAL), CINAHL, IEEE Xplore, PEDro and Google Scholar (first 200 hits). We also searched two trial registries for ongoing trials: ClinicalTrials.gov and the WHO International Clinical Trials Registry Platform. All included studies and reviews were also checked for relevant references and forward citation searches were conducted in Web of Science and Google Scholar.

Search strategy

The information specialist developed the search strategy, with systematic consultation from content and methodological experts, and peer reviewed it using the Peer Review of Electronic Search Strategies (PRESS) checklist prior to use. The search strategy included four blocks, connected by Boolean operators: (i) surgical population (TKA, raTKA, synonyms), (ii) sEMG (synonyms), (iii) intervention or delivery (biofeedback, telerehabilitation, remote monitoring, wearables), and (iv) Quadriceps target. Searches included both controlled vocabulary (MeSH, Emtree, CINAHL Subject Headings) and free text with suitable truncation. The complete search strategies for each database, including a breakdown of syntax and numbers of hits, can be found in Supplement S2.

Source of evidence selection

Two reviewers independently screened the titles and abstracts of all records in duplicate against the eligibility criteria. The full texts of potentially relevant records were then screened in duplicate. Conflicts were resolved through discussion. Cohen's κ was calculated to determine inter-rater agreement at both steps, aiming for a κ of at least 0.70. Reasons for excluding full texts were documented and presented in the PRISMA-ScR flow diagram (Figure 1; Supplement S5).

Data charting

A structured data-charting form was developed a priori and synchronised with the review questions and the JBI charting recommendations (Peters et al., 2021; Pollock et al., 2023). The form was pilot-tested independently by two reviewers on a 10% random sample of sources, and refined to enhance reliability. Details extracted from the studies were: authors, publication year, country, and study design; age, sex, type of surgery and time since surgery; sEMG device and placement (with reference to SENIAM compliance (Hermens et al., 2000)); processing and normalisation method; biofeedback mode, dose, and frequency; remote-monitoring features and connectivity; comparators; outcome domains and outcome measures; duration of follow-up; and study limitations. The completed form is available in Supplement S3.

Critical appraisal of individual sources

In line with PRISMA-ScR (Tricco et al., 2018) and scoping reviews more generally (Peters et al., 2021) we did not conduct a formal risk-of-bias assessment. This choice is in line with the mapping nature of the review and the variety of sources included (such as engineering and device-validation studies, for which existing risk-of-bias tools are not tailored). However, we did describe characteristics important to interpretation (e.g., randomisation, blinding, sample size, reporting) to guide readers' assessment of the evidence base.

Synthesis and presentation

We have used narrative synthesis (Pollock et al., 2023). Data were tabulated, counted and frequencies calculated. Inductive qualitative content analysis was used to describe the components of sEMG protocols, types of biofeedback, and remote-monitoring functions. Evidences were gathered from four review questions (protocols, technologies, outcomes and implementation) in an evidence map (Table 1; Figure 2). Research findings are summarised with reference to the review purpose, with implications for future research and policies, in line with current guidance to prevent overstretched conclusions in scoping reviews (Pollock et al., 2024).

Results

Selection of evidence and characteristics of sources

Figure 1 illustrates the flow of records through the processes of identification, screening, eligibility and inclusion (PRISMA-ScR flow diagram, Supplement S5). The included sources were published mostly since 2010 and were diverse, including randomised controlled trials, prospective cohort studies, feasibility and pilot studies, engineering and methodological studies, systematic reviews and meta-analyses. They were predominantly published from Europe, North America and East Asia. Most TKA-specific sources reported cohorts of traditional approach; few reported raTKA-specific cohorts. See Supplement S4 for the characteristics-of-sources table.

Evidence map

The sources identified in the charted into four parallel streams of evidence that reflected the conceptual building blocks of the review (Figure 2):

Stream A -quadriceps dysfunction and AMI post TKA;

Stream B - raTKA and early functional recovery;
Stream C - sEMG biofeedback of quadriceps reactivation; and strea
Stream D - remote monitoring and telerehabilitation following TKA.

Importantly, none of the sources included talks about all four streams of research in the same integrated study - i.e. there was no study found that offered a combination of a raTKA cohort, home-based remote sEMG monitoring, closed-loop biofeedback, and quadriceps-specific neuromuscular outcomes. This is the main evidence gap the review has identified, summarised in Table 1.

Stream A - Quadriceps dysfunction and AMI after TKA

Mizner et al. (2003) have shown that muscle atrophy and reduced mobility are the drivers of quadriceps weakness following TKA and a follow up quantitative study has shown that atrophy and delayed early mobilization are responsible of about 85 percent of the quadriceps strength loss in one month post-surger. The review by Rice and McNair (2010) placed AMI as a presynaptic reflex inhibition caused by changes in afferent input due to damaged peri-articular tissue, followed by other modifications in motor cortex excitability due to post- op knee effusion (Rice et al., 2014). The recent meta-analysis by Paravlic et al. (2022) established that reduced quadriceps activity is the primary cause of weakness after a surgical procedure, rather than atrophy, BMI, sex, and age can increase the course of recovery. Stevens-Lapsley et al. (2010) stressed that it is vital to attenuate quadriceps activation deficits early to improve longer-term functionality, and the recent narrative review by Churchill et al. (2024) listed perioperative interventions to decrease AMI with NMES and voluntary contraction training becoming the most promising options.

Stream B -Robot-assisted TKA and early recovery

The prospective cohort study by Kayani et al. (2018) (n = 80; 40 raTKA v/s 40 conventional TKA) reported decreased post-operative pain, decreased opioid use, decreased time-to-first-straight-leg-raise, increased knee flexion at discharge, reduced inpatient physiotherapy sessions, and reduced . An identical group had a subsequent five-year follow-up study which demonstrated similar functional outcomes between groups in favour of raTKA (Kayani et al., 2023). The comparative analysis by Held et al. (2023) summarised the evidence in the existing robotic systems, outlining the accuracy of implant positioning improvement, the decreased swelling of the soft tissues, the time spent on quadriceps reactivation, and the discharge with raTKA. A study of NAVIO robot-assisted TKA with intensive rehabilitation programme was an observational study that reported improvements in flexion recovery and functional capacity at the short and medium term (Scaturro et al., 2023).

Stream C - sEMG biofeedback for quadriceps reactivation

Evidence base

The systematic review of EMG biofeedback of the quadriceps between orthopaedic knee populations by Wasielewski et al. (2011) indicated that EMG biofeedback resulted in clinically meaningful effects on the quadriceps activation and functioning after arthroscopic meniscectomy and reconstruction of the anterior cruciate ligament. Karaborklu Argut et al. (2022) studied eight randomised trials of EMG biofeedback following orthopaedic knee surgeries (PEDro scores 68) and found that most studies reported EMG biofeedback to be better than usual care, home exercises or electrical stimulation in terms of quadriceps strength and activation with inconsistent effects on range of motion. The meta-analysis of six trials conducted by Xie et al. (2021) on knee surgery surgeries indicated statistically significant positive effect on knee range of motion in favour of EMG biofeedback (SMD -0.48; 95% CI -0.82 to -0.14) and suggested the use of EMG biofeedback as an addition to standard rehabilitation following knee surgery.

Armshaw et al. (2024) found that sEMG biofeedback has advantages over NMES after TKA with individualized adaptive thresholds and that the dose and individualization of biofeedback were at least as important as its presence. Christanell et al. (2012), randomised 16 patients with or without EMG biofeedback to vastus medialis in the early post-operative period and found better knee extension and integrated EMG (iEMG) in the biofeedback group, demonstrating that biofeedback can modify the neuromuscular recovery trajectories in small samples.

Protocol characteristics

There was convergence and heterogeneity of protocols across included biofeedback sources. The most frequently used target muscles were vastus medialis (often the vastus medialis oblique), then vastus lateralis and rectus femoris; multi-channel experiments used all three heads. Electrode positions were done according to SENIAM protocols where they were reported (Hermens et al., 2000) but methodological studies by Finni and Cheng using MRI-verified locations revealed significant variability in lateral electrode positioning over vastus medialis and rectus femoris despite SENIAM being used, with cross-talk being especially susceptible in vastus lateralis when the electrodes Sampling rates were usually 1000-2000Hz, band-pass filters 20-450Hz + a 50/60Hz notch, and root-mean-square (RMS) windows 50-250ms. normalisation methods used included maximum voluntary isometric contraction (MVIC), sub-maximal isometric reference contractions (favored

Table 1. Evidence map across four streams: representative findings, key gaps, and implications for an integrated raTKA + remote sEMG biofeedback pathway.

Evidence stream	Representative findings	Key gaps	Implication for raTKA + remote sEMG
A. AMI after TKA	Activation failure ~2× atrophy contribution at one month; AMI persists for months to years (Mizner et al., 2003, 2005; Paravlic et al., 2022; Rice & McNair, 2010; Rice et al., 2014).	Rarely captured with sEMG in the home setting; limited biofeedback integration.	Remote sEMG can objectively track AMI trajectory in the early post-operative window.
B. raTKA early recovery	↓Pain, ↓opioids, shorter time to straight-leg raise, earlier discharge (Held et al., 2023; Kayani et al., 2018); medium-term differences attenuate (Kayani et al., 2023).	Rehabilitation protocols not yet tailored to the earlier reactivation window.	raTKA provides a standardised surgical platform for testing digital rehabilitation.
C. sEMG biofeedback	Reviews support improved strength, activation, and ROM (Giggins et al., 2013; Karaborklu Argut et al., 2022; Wasielewski et al., 2011; Xie et al., 2021); TKA RCTs mixed (Armshaw et al., 2024; Sklempe Kokic et al., 2022).	Protocol heterogeneity; mostly clinic-based; few raTKA-specific studies.	Harmonised protocols required before remote, raTKA-specific implementation.
D. Telerehabilitation	Non-inferior outcomes vs usual care; high adherence (Bolam et al., 2021; Correia et al., 2018; Mobbs et al., 2023; Nuevo et al., 2024; Ramkumar et al., 2019; Small et al., 2019; Zhai et al., 2025); wearable sEMG emerging (Hu et al., 2025; Plavoukou et al., 2025).	IMU-dominant; sEMG integration early; data-security and regulatory frameworks still maturing.	Technical substrate for the proposed integrated pathway exists and is maturing.

AMI, arthrogenic muscle inhibition; IMU, inertial measurement unit; raTKA, robot-assisted total knee arthroplasty; ROM, range of motion; sEMG, surface electromyography; TKA, total knee arthroplasty.

inferior or superior to standard care (Yang et al., 2023; Zhai et al., 2025).

The nearest analogue to the suggested integrated pathway is the KneE-PAD randomised controlled trial by Plavoukou et al. (2025), which implemented eight Delsys Trigno Avanti wireless wearable sensors that combine sEMG and IMU with avatar-based feedback to provide telerehabilitation to knee osteoarthritis patients, which showed significant differences between-group in quadriceps. It has also been reported that multimodal knee-osteoarthritis platforms with sEMG, pressure, and IMU signals have been used to provide intelligent home rehabilitation (Hu et al., 2025).

Outcomes reported across streams

The outcomes were reported in five categories, (a) neuromuscular (sEMG RMS, peak-to-peak, integrated EMG, median frequency, symmetry index, voluntary activation ratio); (b) strength and performance (isometric peak torque, rate of torque development Table 1 shows the mapping of the representative findings, gaps, and implications of the integrated raTKA + remote sEMG pathway.

management[9].Red flags mandating MRI are listed in Table 3.

7. Contemporary Management

The management algorithm for PT-BPPV is presented in Table 4. Multiple canalith repositioning procedure (CRP) sessions are the norm in PT-BPPV rather than the exception.

Discussion

Summary of evidence

This scoping review was a systematic mapping of the evidence of sEMG-based remote-monitoring and biofeedback techniques with regard to the quadriceps reactivation following TKA and raTKA. There are four findings that are outstanding. To start with, the neural problem with quadriceps weakness in the initial post-operative weeks is the only type of problem that can be quantified and re-trained non-invasively consecutively at the bedside or even at home with the use of sEMG (Mizner et al., 2003, 2005; Paravlic et al., 2022; Rice and McNair, Second, raTKA is linked to a

faster quadriceps reactivation, a reduced time to straight-leg raise, and an early discharge in relation to conventional TKA (Held et al., 2023; Kayani et al.,
), which forms a specific period of 2-6 post-operative weeks when a specific reactivation intervention can be particularly effective. Third, sEMG biofeedback has a credible evidence base including knee surgeries (Armshaw et al., 2024; Christanell et al., 2012; Giggins et al., 2013; Karaborklu Argut et al., 2022; Wasielewski et al., 2011; Xie et al., 2021). Fourth, clinical effectiveness and technical viability of telerehabilitation in the post-TKA period is well-established (Bolam et al., 2021; Correia et al., 2018; Mobbs et al., 2023; Nuevo et al., 2024; Ramkumar et al., 2019; Small et al., 2019; Zhai et al., 2025), existing platforms have revolutionized sensor based signal acquisition. The development of wearable multimodal systems that can record sEMG and IMU and pressure data concurrently in AI-enabled feedback platform can empower and facilitates the conceptual and practical design of an advanced remote rehabilitation paradigm (Hu et al., 2025; Plavoukou et al., 2025).

None of the studies has combined raTKA cohort with home-based wearable (like sEMG) and closed-loop biofeedback and structured quadriceps activation. The nearest existing analogue is the KneE-PAD pilot in knee osteoarthritis (Plavoukou et al., 2025), which demonstrates that wearable sEMG-based telerehabilitation can potentially lead to measurable improvements in the neuromuscular activity and functional mobility when compared to conventional physiotherapy. The logical next step is to translate this approach into the context of the raTKA post-operative environment, but is currently limited by the heterogeneity of the protocols and the lack of a central outcome set.

A three-layer integration framework

To transform this evidence into testable practice, we suggest an inductive three-layer framework of measurement, feedback, and monitoring which is the result of inductive analysis of the charted protocol and technology characteristics.

Measurement: Bipolar electrodes in compliance with SENIAM guidelines (at least 1 kHz sample, Bluetooth Low Energy to smartphone). Normalisation should be done with a sub-maximal reference contraction, during the first two weeks and during MVIC in the fourth to sixth weeks and semi-permanent skin marking should be used to preserve inter-session reproducibility (Finni and Cheng, 2009 Hermens et al., 2000).

Feedback: Real-time visual and optional auditory feedback with adaptive thresholds (baseline and progressive increments), reflected to a clinician dashboard. Avatar-guided or gamified interfaces will probably be able to maintain engagement (Plavoukou et al., 2025; Wasielewski et al., 2011). A fallback should be a closed-loop sEMG-triggered NMES that patients should be able to cross an activation threshold.

Monitoring and adaptation: Cloud synchronisation, compliance analytics, safety- alerting rules and scheduled synchronous video review at 2-, 6-, and 12-week milestones preserve the therapeutic alliance and provide objective remote oversight.

Methodological and implementation considerations

There are several factors that early post-operative sEMG acquisition must contend with, including oedema, dressings, haematoma and incision orientation, each of which influences placement and signal quality. Although the strict compliance with the MRI-proven data, substantial variability in lateral positioning over vastus medialis and rectus femoris was observed with the potential of cross-talk over vastus lateralis when electrodes were placed too close to the rectus femoris (Finni and Cheng, 2009; Hermens et al., 2000). Practical solutions such as palpation-assisted placement, semi-permanent skin marking, per-session reference contractions, and automated signal-quality checks (signal-to-noise ratio, electrode impedance, cross-talk indices) emerged in the patient on the paired smartphone prior to the start of recording.

The harmonised core outcome set, which spans neuromuscular, strength, functional, patient-reported and implementation domains is a precursor to pooled evidence. Proposed minimum components include quadriceps sEMG amplitude (RMS) and side-to-side symmetry index; isometric peak torque and rate of torque development; knee extension lag and straight-leg raise time (which can be directly compared with the raTKA cohort data (Kayani et al., 2018)); timed up-and-go and 30-second chair stand; KOOS, Forgotten Joint Score-12, and EQ-5D-5L; and adherence and time-on-device. Equity considerations - digital literacy, connectivity, visual or auditory impairment, and multilingual interfaces - and data-governance pathways under GDPR / HIPAA-equivalent frameworks must also be designed in as much as possible, and the feedback mechanisms of AI will also require explicit attention under Software-as-a-Medical-Device pathways.

Implications for practice and research

According to the present-day guidelines on the scoping review (Pollock et al., 2024), the implications below are presented as a research and policy direction and as descriptive observations that are relevant to the current practice; they cannot be read as recommendations of the clinical effectiveness.

Three observations are formed by the mapped evidence to clinicians. Following both TKA and raTKA,

early mobilization, quantified quadriceps activation, range of motion and VAS, are initial goal of rehabilitation, although reactivation window appears to be considerably more permissive in the case of raTKA (Held et al., 2023; Kayani et al., 2018). Several systematic reviews support the use of sEMG biofeedback as an adjunct to standard rehabilitation (Giggins et al., 2013; Karaborklu Argut et al., 2022; Wasielewski et al., 2011; Xie et al., 2021). Adequately documented feasibility and compliance data in commercially available telerehabilitation platforms can be reasonable options when patients are unable to attend in-person physiotherapy (Bolam et al., 2021; Correia et al., 2018; Mobbs et al., 2023; Nuevo et al., 2024; Ramkumar et al., 2019; Small et al., 2019; Zhai et al., 2025).

In the case of researchers, a pragmatic, adequately powered randomised controlled trial comparing an integrated raTKA + wearable sEMG-biofeedback-enabled telerehabilitation against a standardised telerehabilitation comparator (IMU only) and a usual-care comparator. Design priorities consist of: stratification by baseline activation deficit; an intervention window of 12 weeks with at least 12-month follow-up; pre-registration; blinded outcome assessors of strength and performance measures; and embedded health-economic and implementation-science evaluation. Qualitative research within a group of patients and therapists will help to identify facilitators and barriers to continuous engagement with wearable sEMG at scale.

Strengths and limitations

The strengths of this review are that it uses a modern methodological framework (JBI, Peters et al. 2021), was peer-reviewed, and searched nine databases and two trial registries, which is also a JBI-recommended practice that we found valuable in framing review questions and avoiding overstretched conclusions (Peters et al., 2021; Pollock et al., 2022, 2024).

There are a number of restrictions that must be mentioned. First, it was not done as a formal critical review of risk of bias, and the certainty of evidence cannot therefore be inferred, and no quantitative pooling was done. Second, the literature on digital-health and raTKA is rapidly changing, and this review will need periodic updating; readers should consult registered trials to access the most up-to-date evidence. Third, without raTKA-specific sEMG research, there are a number of inferences made based on traditional TKA and adjacent knee-surgical cohorts; although these inferences are explicit and traceable, they remain inferences. Fourth, the presence of non-English sources with no abstracts that could be translated was used to filter out grey literature, thus potentially under-sampling regional evidence. Lastly, the fact that the pace of regulatory and connectivity standards applicable to medical wearable devices is so fast means that the considerations of implementation that have been summarised here need to be re-assessed against jurisdictional rules at the time of any future trial.

Conclusions

Post TKA and raTKA, quadriceps activation is a key for functionality and less post op complications. The objective way to measure the neuromuscular deficit is through surface EMG and it can help clinician achieve quantifiable results . The elements of an integrated sEMG-guided remote biofeedback pathway of quadriceps reactivation after raTKA are in isolation and have not been synthesised as a single pathway of care. The next steps that are required are harmonised protocols, a core outcome set, and pragmatic randomised controlled trials. This scoping review can help in creating framework for future studies.

Declarations

Ethics approval. Not applicable -this study used only published and registered evidence.

Funding. Partially funded by De Luca foundation and Delsys (equipment)

Conflicts of interest. The authors declare no conflicts of interest .

Data availability. Search strategies, the charting form, and the extracted data will be made publicly available on acceptance.

Reporting standards. PRISMA-ScR 20-item checklist (Supplement S1).

Supplements

- S1 -PRISMA-ScR 20-item checklist.
- S2 -Full search strategies (all databases and registries).
- S3 -Data-charting form.
- S4 -Characteristics of included sources.

References

Courties, A., Kouki, I., Soliman, N., Mathieu, S., & Sellam, J. (2024). Osteoarthritis year in review 2024: Epidemiology and therapy. Osteoarthritis and Cartilage, 32(11), 1397-1404. https://doi.org/10.1016/j.joca.2024.07.014

Amog, K., Pham, B., Courvoisier, M., Mak, M., Booth, A., Godfrey, C., Hofmeyer, A., Kirkpatrick, P., Munn, Z., Tricco, A. C., & Langlois, E. V. (2022). The web-based 'Right Review' tool asks reviewers simple questions to suggest methods from 41 knowledge synthesis methods. Journal of Clinical Epidemiology, 147, 42-51. https://doi.org/10.1016/j.jclinepi.2022.03.004

Arksey, H., & O'Malley, L. (2005). Scoping studies: Towards a methodological framework. International Journal of Social Research Methodology, 8(1), 19-32. https://doi.org/10.1080/1364557032000119616

Armshaw, B., Vaidya, M., & Mehta, S. (2024). Surface electromyography-based biofeedback can facilitate recovery from total knee arthroplasty. Journal of Applied Behavior Analysis, 57(3), 540-562. https://doi.org/10.1002/jaba.1076

Bolam, S. M., Batinica, B., Yeung, T. C., Weaver, S., Cantamessa, A., Vanderboor, T. C., Yeung, S., Munro, J. T., Fernandez, J. W., Besier, T. F., & Monk, A. P. (2021). Remote

patient monitoring with wearable sensors following knee arthroplasty. Sensors, 21(15), 5143. https://doi.org/10.3390/s21155143

Campbell, F., Tricco, A. C., Munn, Z., Pollock, D., Saran, A., Sutton, A., Aromataris, E., & Khalil, H. (2023). Mapping reviews, scoping reviews, and evidence and gap maps (EGMs): The same but different -the 'Big Picture' review family. Systematic Reviews, 12(1), 45. https://doi.org/10.1186/s13643-023-02166-9

Christanell, F., Hoser, C., Huber, R., Fink, C., & Luomajoki, H. (2012). The influence of electromyographic biofeedback therapy on knee extension following anterior cruciate ligament reconstruction: A randomized controlled trial. Sports Medicine, Arthroscopy, Rehabilitation, Therapy & Technology, 4(1), 41. https://doi.org/10.1186/1758-2555-4-41

Churchill, L., Bade, M. J., Koonce, R. C., Stevens-Lapsley, J. E., & Bandholm, T. (2024). The past and future of peri-operative interventions to reduce arthrogenic quadriceps muscle inhibition after total knee arthroplasty: A narrative review. Osteoarthritis and Cartilage Open, 6(1), 100429. https://doi.org/10.1016/j.ocarto.2023.100429

Clyne, B., Sharp, M. K., O'Neill, M., Pollock, D., Lynch, R., Amog, K., Booth, A., Godfrey, C., Hofmeyer, A., Tricco, A. C., & Langlois, E. V. (2024). An international modified Delphi process supported updating the web-based 'Right Review' tool. Journal of Clinical Epidemiology, 170, 111333. https://doi.org/10.1016/j.jclinepi.2024.111333

Correia, F. D., Nogueira, A., Magalhães, I., Guimarães, J., Moreira, M., Barradas, I., Teixeira, L., Tulha, J., Seabra, R., Lains, J., & Bento, V. (2018). Home-based rehabilitation with a novel digital biofeedback system versus conventional in-person rehabilitation after total knee replacement: A feasibility study. Scientific Reports, 8(1), 11299. https://doi.org/10.1038/s41598-018-29668-0

Finni, T., & Cheng, S. (2009). Variability in lateral positioning of surface EMG electrodes. Journal of Applied Biomechanics, 25(4), 396-400. https://doi.org/10.1123/jab.25.4.396

Giggins, O. M., Persson, U. M., & Caulfield, B. (2013). Biofeedback in rehabilitation. Journal of NeuroEngineering and Rehabilitation, 10, 60. https://doi.org/10.1186/1743-0003-10-60

Held, M. B., Grosso, M. J., Gazgalis, A., Sarpong, N. O., Neuwirth, A. L., Cooper, H. J., & Shah, R. P. (2023). Comparative assessment of current robotic-assisted systems in primary total knee arthroplasty. Bone & Joint Open, 4(1), 3-12. https://doi.org/10.1302/2633-1462.41.BJO-2022-0070.R1

Hermens, H. J., Freriks, B., Disselhorst-Klug, C., & Rau, G. (2000). Development of recommendations for SEMG sensors and sensor placement procedures. Journal of Electromyography and Kinesiology, 10(5), 361-374. https://doi.org/10.1016/S1050-6411(00)00027-4

Hu, J., Wang, S., Shen, Y., & Miao, X. (2025). A wearable system for knee osteoarthritis based on multimodal physiological signal assessment and intelligent rehabilitation. Sensors, 25(23), 7334. https://doi.org/10.3390/s25237334

Inacio, M., Paxton, E., & Graves, S. (2017). Projected increase in total knee arthroplasty in the United States - an alternative projection model. Osteoarthritis and Cartilage, 25(11), 1797-1803.

Karaborklu Argut, S., Celik, D., & Yasacı, Z. (2022). Effectiveness of therapeutic electromyographic biofeedback after orthopedic knee surgeries: A systematic review. Disability and Rehabilitation, 44(14), 3364-3372. https://doi.org/10.1080/09638288.2020.1867904

Kayani, B., Konan, S., Tahmassebi, J., Pietrzak, J. R. T., & Haddad, F. S. (2018). Robotic-arm assisted total knee arthroplasty is associated with improved early functional recovery and reduced time to hospital discharge compared with conventional jig-based total knee arthroplasty: A prospective cohort study. The Bone & Joint Journal, 100-B(7), 930-937. https://doi.org/10.1302/0301-620X.100B7.BJJ-2017-1449.R1

Kayani, B., Fontalis, A., Haddad, I. C., Donovan, C., Rajput, V., & Haddad, F. S. (2023). Robotic-arm assisted total knee arthroplasty is associated with comparable functional outcomes but improved forgotten joint scores compared with conventional manual total knee arthroplasty at five-year follow-up. Knee Surgery, Sports Traumatology, Arthroscopy, 31(12), 5453-5462. https://doi.org/10.1007/s00167-023-07578-7

Levac, D., Colquhoun, H., & O'Brien, K. K. (2010). Scoping studies: Advancing the methodology. Implementation Science, 5, 69. https://doi.org/10.1186/1748-5908-5-69

Mizner, R. L., Stevens, J. E., & Snyder-Mackler, L. (2003). Voluntary activation and decreased force production of the quadriceps femoris muscle after total knee arthroplasty. Physical Therapy, 83(4), 359-365. https://doi.org/10.1093/ptj/83.4.359

Mizner, R. L., Petterson, S. C., Stevens, J. E., Vandenborne, K., & Snyder-Mackler, L. (2005). Early quadriceps strength loss after total knee arthroplasty: The contributions of muscle atrophy and failure of voluntary muscle activation. The Journal of Bone and Joint Surgery. American Volume, 87(5), 1047-1053. https://doi.org/10.2106/JBJS.D.01992

Mobbs, R. J., Amin, T., Stulberg, S. D., Kerina, J. M., Hernandez, V., & Bolander, R. (2023). Remote monitoring using wearable technology after knee arthroplasty using a joint-specific wearable device: A prospective cohort study of 435 patients with 6-week follow-up. Journal of Orthopaedic Experience & Innovation, 4(1). https://doi.org/10.60118/001c.72644

Munn, Z., Peters, M. D. J., Stern, C., Tufanaru, C., McArthur, A., & Aromataris, E. (2018). Systematic review or scoping review? Guidance for authors when choosing between a systematic or scoping review approach. BMC Medical Research Methodology, 18(1), 143. https://doi.org/10.1186/s12874-018-0611-x

Munn, Z., Pollock, D., Khalil, H., Alexander, L., Mclnerney, P., Godfrey, C. M., Peters, M., & Tricco, A. C. (2022). What are scoping reviews? Providing a formal definition of scoping reviews as a type of evidence synthesis. JBI Evidence Synthesis, 20(4), 950-952. https://doi.org/10.11124/JBIES-21-00483

Nuevo, M., Rodríguez-Rodríguez, D., Jauregui, R., Fabrellas, N., Zabalegui, A., Conti, M., & Prat-Fabregat, S. (2024). Telerehabilitation following fast-track total knee arthroplasty is effective and safe: A randomized controlled trial with the ReHub® platform. Disability and Rehabilitation, 46(12), 2629-2639. https://doi.org/10.1080/09638288.2023.2228689

Paravlic, A. H., Meulenberg, C. J. W., & Drole, K. (2022). The time course of quadriceps strength recovery after total knee arthroplasty is influenced by body mass index, sex, and age of patients: Systematic review and meta-analysis. Frontiers in

Medicine, 9, 865412. https://doi.org/10.3389/fmed.2022.865412

scoping reviews. JBI Evidence Implementation, 19(1), 3-10. https://doi.org/10.1097/XEB.0000000000000277

Plavoukou, T., Kasnesis, P., Contiero Syropoulou, A., Papagiannis, G., Stasinopoulos, D., & Georgoudis, G. (2025). A sensor-augmented telerehabilitation system for knee osteoarthritis: A randomized controlled trial of neuromuscular, functional, and psychosocial outcomes. Sensors, 25(23), 7113. https://doi.org/10.3390/s25237113

Pollock, D., Alexander, L., Munn, Z., Peters, M. D. J., Khalil, H., **Godfrey, C. M., McInerney, P., Synnot, A., & Tricco, A. C. (2022).** Moving from consultation to co-creation with knowledge users in scoping reviews: Guidance from the JBI Scoping Review Methodology Group. JBI Evidence Synthesis, 20(4), 969-979. https://doi.org/10.11124/JBIES-21-00416

Pollock, D., Peters, M. D. J., Khalil, H., McInerney, P., Alexander, L., Tricco, A. C., Evans, C., de Moraes, É. B., Godfrey, C. M., Pieper, D., Saran, A., Stern, C., & Munn, Z. (2023). Recommendations for the extraction, analysis, and presentation of results in scoping reviews. JBI Evidence Synthesis, 21(3), 520-532. https://doi.org/10.11124/JBIES-22-00123

Pollock, D., Evans, C., Jia, R. M., Alexander, L., Pieper, D., Brandão de Moraes, E., Peters, M. D. J., Tricco, A. C., Khalil, H., Godfrey, C. M., Saran, A., Campbell, F., & Munn, Z. (2024). 'How-to': Scoping review? Journal of Clinical Epidemiology, 176, 111572. https://doi.org/10.1016/j.jclinepi.2024.111572

Ramkumar, P. N., Haeberle, H. S., Ramanathan, D., Cantrell, W. A., Navarro, S. M., Mont, M. A., Bloomfield, M., & Patterson, B. M. (2019). Remote patient monitoring using mobile health for total knee arthroplasty: Validation of a wearable and machine learning-based surveillance platform. The Journal of Arthroplasty, 34(10), 2253-2259. https://doi.org/10.1016/j.arth.2019.05.021

Rice, D. A., & McNair, P. J. (2010). Quadriceps arthrogenic muscle inhibition: Neural mechanisms and treatment perspectives. Seminars in Arthritis and Rheumatism, 40(3), 250-266. https://doi.org/10.1016/j.semarthrit.2009.10.001

Rice, D. A., McNair, P. J., Lewis, G. N., & Dalbeth, N. (2014). Quadriceps arthrogenic muscle inhibition: The effects of experimental knee joint effusion on motor cortex excitability. Arthritis Research & Therapy, 16(6), 502. https://doi.org/10.1186/s13075-014-0502-4

Scaturro, D., Vitagliani, F., Caracappa, D., Tomasello, S., Chiaramonte, R., Vecchio, M., Camarda, L., & Letizia Mauro, G. (2023). Rehabilitation approach in robot-assisted total knee arthroplasty: An observational study. BMC Musculoskeletal Disorders, 24(1), 140. https://doi.org/10.1186/s12891-023-06230-2

Sklempe Kokic, I., Vuksanic, M., Kokic, T., Peric, I., & Duvnjak, I. (2022). Effects of electromyographic biofeedback on functional recovery of patients two months after total knee arthroplasty: A randomized controlled trial. Journal of Clinical Medicine, 11(11), 3182. https://doi.org/10.3390/jcm11113182

Small, S. R., Bullock, G. S., Khalid, S., Barker, K., Trivella, M., & Price, A. J. (2019). Current clinical utilisation of wearable motion sensors for the assessment of outcome following knee arthroplasty: A scoping review. BMJ Open, 9(12), e033832. https://doi.org/10.1136/bmjopen-2019-033832

Stevens-Lapsley, J. E., Balter, J. E., Kohrt, W. M., & Eckhoff, D. G. (2010). Quadriceps and hamstrings muscle dysfunction after total knee arthroplasty. Clinical Orthopaedics and Related Research, 468(9), 2460-2468. https://doi.org/10.1007/s11999-009-1219-6

Tricco, A. C., Lillie, E., Zarin, W., O'Brien, K. K., Colquhoun, H., Levac, D., Moher, D., Peters, M. D. J., Horsley, T., Weeks, L., Hempel, S., Akl, E. A., Chang, C., McGowan, J., Stewart, L., Hartling, L., Aldcroft, A., Wilson, M. G., Garritty, C., … Straus, S. E. (2018). PRISMA Extension for Scoping Reviews (PRISMA-ScR): Checklist and explanation. Annals of Internal Medicine, 169(7), 467-473. https://doi.org/10.7326/M18-0850

Wasielewski, N. J., Parker, T. M., & Kotsko, K. M. (2011). Evaluation of electromyographic biofeedback for the quadriceps femoris: A systematic review. Journal of Athletic Training, 46(5), 543-554. https://doi.org/10.4085/1062-6050-46.5.543

Xie, Y. J., Wang, S., Gong, Q. J., Wang, J. X., Sun, F. H., Miyamoto, A., Ou, X., Wang, L., Wang, S. Q., & Zhang, C. (2021). Effects of electromyography biofeedback for patients after knee surgery: A systematic review and meta-analysis. Journal of Biomechanics, 120, 110386. https://doi.org/10.1016/j.jbiomech.2021.110386

Yang, C., Shang, L., Yao, S., Ma, J., & Xu, C. (2023). Cost, time savings and effectiveness of wearable devices for remote monitoring of patient rehabilitation after total knee arthroplasty: Study protocol for a randomized controlled trial. Journal of Orthopaedic Surgery and Research, 18(1), 454. https://doi.org/10.1186/s13018-023-03898-z

Zhai, S., Wu, R., Du, G., Xu, J., Zhou, H., Wang, Y., & Liu, Z. (2025). Smart device-assisted telerehabilitation versus conventional rehabilitation after total knee arthroplasty: A systematic review and meta-analysis. Journal of Orthopaedic Surgery and Research, 20, 954. https://doi.org/10.1186/s13018-025-06393-9

Profile of Solitary Plasmacytoma-A Ten Year Data on Outcomes From A Tertiary Care Cancer Centre of Kashmir (India) .

Mushtaq Ahmad Sofi , Mohmad Hussain Mir , Farhana Siraj , Shahida Nasreen , Asifa Andleeb , Kaneez Fatima , Nazir Ahmad Dar , Musaib Mushtaq .

Abstract

Background

Solitary plasmacytoma, comprising solitary bone plasmacytoma (SBP) and extramedullary plasmacytoma (EMP), is a rare plasma cell neoplasm with a significant risk of progression to multiple myeloma (MM). This study evaluates clinicodemographic characteristics, treatment outcomes, and prognostic factors influencing disease progression.

Methods

A retrospective analysis of 24 patients diagnosed with plasmacytoma (SBP = 18; EMP = 6) was performed at SKIMS Srinagar. Clinical features, treatment modalities, response rates, and survival outcomes were assessed. Event-free survival (EFS), overall survival (OS), and progression to MM were analyzed, along with potential prognostic factors.

Results

The median age was 48 years with a male predominance (70.8%). Radiotherapy was the primary treatment modality. Local control was achieved in 87.5% of patients. During a median follow-up of 32 months, 33% of patients progressed to multiple myeloma (MM), predominantly in the SBP subgroup. Median time to progression was 21 months. Median EFS and OS were 38 and 122 months, respectively, with 5-year EFS of 45.5%. Local control was significantly associated with improved EFS (p = 0.001), while vertebral involvement showed a borderline association with progression (p = 0.057). Serum M protein status did not significantly impact outcomes.

Conclusion

Radiotherapy achieves high local control in solitary plasmacytoma; however, progression to multiple myeloma remains a significant risk, especially in patients with bone involvement and larger tumor burden. Optimal local control is a key prognostic determinant, emphasizing the importance of effective initial therapy and close long-term surveillance.

JK-Practitioner2026;31(2):16-20.

Introduction

Solitary plasmacytoma is a rare and distinct plasma cell neoplasm characterized by a localized proliferation of monoclonal plasma cells in the absence of systemic manifestations of multiple myeloma. It is broadly categorized into solitary bone plasmacytoma (SBP) and extramedullary plasmacytoma (EMP), depending on whether the lesion arises within bone or soft tissue. Although these entities share a common clonal origin, they differ in anatomical distribution, biological behavior, and risk of progression to systemic disease[1].Solitary Plasmacytomas account for approximately 3–5% of all plasma cell dyscrasias. SBP is more frequently encountered and predominantly involves the axial skeleton, particularly vertebrae, pelvis, and ribs, reflecting areas of active hematopoiesis. In contrast, EMP most commonly arises in the head and neck region, especially within the upper aerodigestive tract, including the nasal cavity, nasopharynx, and oropharynx. The disease typically presents in the fifth to seventh decade of life, with a clear male predominance[2]. The pathogenesis of solitary plasmacytoma involves clonal expansion of plasma cells, often associated with cytogenetic abnormalities similar to those seen in multiple myeloma, such as immunoglobulin heavy chain translocations. However, unlike multiple myeloma,solitary plasmacytoma

Author Affiliations

Mushtaq Ahmad Sofi ,Shahida Nasreen, Asifa Andleeb , Kaneez Fatima , Nazir Ahmad Dar , Musaib Mushtaq ;
Department of Radiation Oncology:
Mohmad Hussain Mir; Department of Medical Oncology: DR **Farhana Siraj** :
Department of General Medicine
SKIMS SOURA

Correspondence

Dr. Mohmad Hussain Mir
Assistant Professor
Medical Oncology SKIMS Soura
Contact: 9419023962
Email: mhussainmir@gmail.com

Indexed

EMBASE, SCOPUS, IndMED , ESBCO, Google Scholar besides other national and international databases.

Cite this article as

Sofi MA , Mir MH , Siraj F , Nasreen S , Andleeb A , Fatima K, Dar NA , Mushtaq M. Profile of Solitary Plasmacytoma-A Ten Year Data on Outcomes From a Tertiary Care Cancer Centre of Kashmir (India). JK Pract2026;31(2):16-20.

Full length article available at **jkpractitioner.com** one month after publication

Keywords

Solitary plasmacytoma, radiotherapy, multiple myeloma, event-free survival, prognostic factors

lacks diffuse bone marrow involvement and does not fulfill the diagnostic criteria of systemic disease, including CRAB features like hypercalcemia, renal impairment, anemia, or multiple osteolytic lesions. The distinction between localized plasmacytoma and early multiple myeloma is critical, as it directly influences therapeutic strategy and prognosis3.Accurate diagnosis requires a comprehensive workup, including histopathological confirmation of monoclonal plasma cell infiltration and thorough systemic evaluation to exclude disseminated disease. Bone marrow examination typically reveals minimal or absent clonal plasma cells (<10%). Advanced imaging modalities such as magnetic resonance imaging (MRI) and fluorodeoxyglucose positron emission tomography/computed tomography (FDG PET/CT)or low dose whole body computed tomography have become integral in staging, as they improve detection of occult lesions and reduce the risk of understaging. Radiotherapy remains the cornerstone of treatment for both SBP and EMP due to the intrinsic radiosensitivity of plasma cells. Definitive radiotherapy with doses ranging from 40 to 50 Gy achieves excellent local control rates, often exceeding 80–90%. Surgical intervention may be considered in selected cases, particularly for structural stabilization or complete excision of localized extramedullary lesions. The role of systemic therapy remains limited and is generally reserved for patients with high-risk features or disease progression4. Despite favorable local control, solitary plasmacytoma carries a significant risk of progression to multiple myeloma, particularly in patients with SBP. Long-term follow-up studies have demonstrated progression rates of up to 50–70% over a decade. Several prognostic factors have been identified, including tumor size, persistence of monoclonal protein after treatment, and minimal bone marrow involvement at diagnosis. These factors underscore the importance of vigilant surveillance even after successful local therapy5.Given the rarity of this disease, most available evidence is derived from retrospective analyses and institutional experiences, resulting in variability in diagnostic approaches and treatment strategies. There remains a need for further studies to better delineate prognostic factors, optimize therapeutic protocols, and improve long-term outcomes.In this context, the present study aims to evaluate the Clinicopathological characteristics, treatment patterns, and outcomes of patients with solitary plasmacytoma treated at a tertiary care center, thereby contributing to the existing body of evidence and providing insights into real-world clinical practice.

Study Design and Methodology

This retrospective observational clinicodemographic study was conducted at SKIMS Soura (a tertiary care oncology center) to evaluate the demographic profile, clinical characteristics, treatment patterns, and outcomes of patients with solitary plasmacytoma. All consecutive cases diagnosed between January 2016-Dec 2025 were identified from state cancer institutional medical records. Ethical approval was obtained from the Institutional Ethics Committee, and the study was conducted in accordance with the Declaration of Helsinki. Adult patients (≥18 years) with histologically confirmed solitary plasmacytoma, including solitary bone plasmacytoma and extramedullary plasmacytoma, were included. Diagnosis required exclusion of multiple myeloma, defined by bone marrow plasma cells <10%, absence of CRAB features (hypercalcemia, renal dysfunction, anemia, lytic bone lesions), and no additional lesions on imaging (skeletal survey/MRI/PET-CT). Only patients with complete baseline data and who received definitive treatment (radiotherapy ± surgery) were analyzed. Patients with multiple myeloma at presentation, multiple lesions, prior plasma cell dyscrasia, incomplete records, or inadequate follow-up were excluded. Data were extracted using a structured proforma, including demographic variables (age, gender), clinical features (symptoms, ECOG performance status, disease site), diagnostic findings (imaging, laboratory parameters including serum protein electrophoresis and immunofixation, bone marrow biopsy), and treatment details (radiotherapy dose, fractionation, technique, and surgical intervention). Outcome measures included local control, progression to multiple myeloma, progression-free survival (PFS), and overall survival (OS). Statistical analysis was performed using SPSS software. Continuous variables were expressed as mean ± standard deviation or median (range), and categorical variables as frequencies and percentages. Survival outcomes were estimated using the Kaplan–Meier method, with subgroup comparisons performed using the log-rank test. A p-value <0.05 was considered statistically significant. Patient confidentiality was maintained through data anonymization.

Results

A total of 24 patients with plasmacytoma were analyzed, comprising 18 cases of solitary bone plasmacytoma (SBP) and 6 cases of extramedullary plasmacytoma (EMP). The mean age at presentation was 48 years, with a male predominance (70.8%). The majority of patients belonged to rural areas (60.0%). Pain was the most common presenting symptom (54.2%), followed by swelling/mass and neurological weakness. No statistically significant differences were

observed between SBP and EMP groups in baseline characteristics.Radiotherapy (RT) alone was the predominant treatment modality (79.16%), while a minority underwent surgery with or without adjuvant radiotherapy (Table 1). Overall, local control was achieved in 21 patients (87.5%), whereas 3 patients (12.5%) experienced local failure. All patients with local failure had bone plasmacytoma and tumor size ≥5 cm, and 66.7% of these patients progressed to multiple myeloma (Table 2).

Table 1. Baseline and Clinical Characteristics of Plasmacytoma Patients (N = 24)

Variable	Overall (N=24)	SBP (n=18)	EMP (n=6)	p-value*
Age (years), mean (SD)	48	48	50	0.68
Gender, n (%)				0.65
- Male	17 (70.8)	13 (72.2)	4 (66.7)	
- Female	7 (29.2)	5 (27.8)	2 (33.3)	
Residence, n (%)				0.99
- Rural	14 (60.0)	10 (55.5)	4 (66.6)	
- Urban	10 (40.0)	8 (44.4)	2 (33.3)	
Presenting symptom, n (%)				0.46
- Pain	13 (54.2)	11 (61.1)	2 (33.3)	
- Swelling/mass	3 (12.5)	1 (5.6)	2 (33.3)	
- Weakness	3 (12.5)	3 (16.7)	0 (0.0)	
- Nasal bleeding	2 (8.3)	0 (0.0)	2 (33.3)	
- Other / unknown	3 (12.5)	3 (16.7)	0 (0.0)	
Treatment Given, n				
- RT alone	19(79.16)	15(83.33)	4(66.6)	<0.001
- Surgery ± RT	5(20.83)	3 (16.66)	2(40.0)	

(12.5%) were recorded, of which 2 were disease-related (Table 7).

Table 2: Treatment Response and Local Control (n = 24)

Parameter	Number (n)	Percentage (%)
Achieved Local Control	21	87.5
Failed Local Control	3	12.5
Bone Plasmacytoma among failures	3	100
Large Mass (≥5 cm) among failures	3	100
Progression to MM (among failures)	2	66.7

Table 3: Serum M Protein Response

Parameter	Number (n)	Percentage (%)
Patients evaluated	14	—
Undetectable post-treatment	8	57
Detectable post-treatment	6	43

Table 4: Disease Progression & Patterns

Parameter	Number (n)	Percentage (%)
Total progressed to MM	8	33
SBP progressing to MM	7	—
EMP progressing to MM	1	—
Median time to MM (months)	21	Range: 5–96

Table 5: Survival Outcomes

Parameter	Value
Median Follow-up (months)	32 (3–133)
Median EFS (months)	38
Median OS (months)	122
5-year EFS (%)	45.5

Table 6: Prognostic Factors for Progression to MM

Factor	p-value	Significance
Local Control vs No Local Control	0.001	Significant
Vertebral Lesions	0.057	Borderline
Presence of M Protein	0.69	Not Significant
Value of M Protein	0.44	Not Significant
Disappearance of M Protein	0.40	Not Significant

Table 7: Treatment & Outcomes

Parameter	Number (n)	Percentage (%)
Underwent HD Melphalan + Stem Cell Rescue	3	12.5
Total Deaths	3	12.5
Deaths due to disease	2	—
Deaths due to other causes	1	—

Serum M protein was evaluable in 14 patients, of whom 57% achieved complete disappearance following treatment. However, persistence of M protein did not demonstrate prognostic significance (Table 3).During a median follow-up of 32 months (range 3–133 months), 8 patients (33%) progressed to MM, with the majority belonging to the SBP subgroup. The median time to progression was 21 months (Table 4). Survival analysis revealed a median event-free survival (EFS) of 38 months and median overall survival (OS) of 122 months. The 5-year EFS rate was 45.5%, while OS remained favorable. Local control was significantly associated with improved EFS (p = 0.001), though no statistically significant difference in OS was observed (Table 5). Vertebral involvement showed a trend towards increased progression risk (p = 0.057). Neither the presence nor the dynamics of serum M protein had a significant impact on progression (Table 6).Three patients (12.5%) required high-dose melphalan followed by autologous stem cell transplantation upon progression to MM. At the end of follow-up, 3 deaths (12.5%) were recorded, of which 2 were disease-related (Table 7).

Discussion

This study provides insight into the clinicodemographic characteristics, treatment outcomes, and prognostic factors in patients with solitary plasmacytoma. The observed median age of 48 years and male predominance are consistent with previously reported literature, which describes plasmacytoma as a disease of middle-aged individuals with a slight male preponderance[6,7]. The predominance of SBP over EMP in our cohort aligns with established epidemiological patterns[8].Radiotherapy remains the cornerstone of treatment for solitary plasmacytoma, achieving excellent local control rates ranging from 80–95% in multiple series[9,10]. In the present study, the local control rate of 87.5% is comparable to these outcomes, reaffirming the efficacy of RT as definitive therapy. Notably, all cases of local failure were associated with bone lesions and tumor size ≥5 cm, highlighting tumor burden as a critical determinant of treatment outcome. This observation is supported by prior studies identifying lesion size >5 cm as an adverse prognostic factor[11].Progression to multiple myeloma remains the most significant clinical challenge in plasmacytoma management. In our cohort, 33% of patients progressed to MM, which falls within the reported range of 30–60% for SBP and lower rates for EMP[12, 13]. The predominance of progression in SBP compared to EMP further corroborates existing evidence suggesting a more indolent course for EMP[14]. The median time to progression (21 months) is also consistent with previously reported intervals, indicating that the highest risk period lies within the first 2–3 years post-treatment[15].A key finding of this study is the strong association between local control and improved event-free survival. Patients achieving local control demonstrated significantly better EFS, emphasizing the importance of optimal primary treatment. However, no statistically significant difference in overall survival was observed, likely reflecting the effectiveness of salvage therapies and the relatively long natural history of the disease.The prognostic role of serum M protein remains controversial. While some studies suggest persistence of M protein as a predictor of progression, our analysis did not demonstrate a significant association[16, 17]. This may be attributed to the limited number of patients in whom M protein was evaluable or the heterogeneity in disease biology. Similarly, vertebral involvement showed only a borderline association with progression, warranting further investigation in larger cohorts.High-dose chemotherapy with autologous stem cell transplantation was utilized in selected patients who progressed to MM, reflecting current standard practice for eligible patients[18]. The relatively low mortality rate observed in this study underscores the favorable prognosis of plasmacytoma when appropriately managed.Overall, the findings of this study reinforce the importance of early diagnosis, adequate radiotherapy, and close follow-up, particularly in patients with high-risk features such as large tumor size and bone involvement. Future research should focus on molecular and imaging biomarkers to better stratify patients at risk of progression.

Conclusion

Solitary plasmacytoma demonstrates excellent local control with radiotherapy; however, progression to multiple myeloma remains a significant concern, particularly in patients with bone lesions and large tumor size. Achieving local control is a critical determinant of improved event-free survival. Long-term surveillance is essential for early detection of systemic progression.

Funding : Nil

Conflict of Interest : Nil

References

1.**Caers J, Paiva B, Zamagni E, Leleu X, Bladé J, Kristinsson SY, et al.** Diagnosis, treatment, and response assessment in solitary plasmacytoma: updated recommendations. Haematologica. 2018; 103(11):1778–90.

2.**Ozsahin M, Tsang RW, Poortmans P, Belkacémi Y, Bolla M, Dinshaw KA, et al.** Outcomes and patterns of failure in solitary plasmacytoma. RadiotherOncol. 2006;80(2):207–12.

.**Rajkumar SV, Dimopoulos MA, Palumbo A, Blade J, Merlini G, Mateos MV, et al.** International Myeloma Working Group updated diagnostic criteria. Lancet Oncol. 2014;15(12):e538–48.

4.**Tsang RW, Campbell BA, Goda JS, Kelsey CR, Kirova YM, Parikh RR, et al**. Radiation therapy for solitary plasmacytoma. Int J RadiatOncolBiol Phys. 2018;101(4):794–808.

5.**Reed V, Shah J, Medeiros LJ, Ha CS, Mazloom A, Weber DM, et al**. Solitary plasmacytomas: outcome and prognostic factors. Cancer. 2011;117(19):4468–74.

6.**Dimopoulos MA, Moulopoulos LA, Maniatis A, Alexanian R**. Solitary plasmacytoma of bone and asymptomatic multiple myeloma. *Blood*. 2000;96(6):2037–2044.

7.**Soutar R, Lucraft H, Jackson G, Reece A, Bird J, Low E, et al**. Guidelines on the diagnosis and management of solitary plasmacytoma of bone and solitary extramedullary plasmacytoma. *Br J Haematol*. 2004;124(6):717–726.

8.**Kilciksiz S, Karakoyun-Celik O, Agaoglu FY, Haydaroglu A**. A review for solitary plasmacytoma of bone and extramedullary plasmacytoma. *RadiotherOncol*. 2008;87(3):309–317.

9.**Tsang RW, Campbell BA, Goda JS, Kelsey CR, Kirova YM, Parikh RR, et al.** Radiation therapy for solitary plasmacytoma and multiple myeloma: Guidelines from the International Lymphoma Radiation Oncology Group. *Int J RadiatOncolBiol Phys*. 2018;101(4):794–808.

10.**Ozsahin M, Tsang RW, Poortmans P, Belkacemi Y, Bolla M, Dinshaw KA, et al**. Outcomes and patterns of failure in solitary plasmacytoma: A multicenter Rare Cancer Network study of 258 patients. *RadiotherOncol*. 2006;80(2):186–191.

11.**Liebross RH, Ha CS, Cox JD, Weber D, Delasalle K, Alexanian R.** Solitary bone plasmacytoma: Outcome and prognostic factors following radiotherapy. *J ClinOncol*. 1998;16(4):1573–1577.

12.**Knobel D, Zouhair A, Tsang RW, Poortmans P, Belkacemi Y, Bolla M, et al**. Prognostic factors in solitary plasmacytoma of the bone: A multicenter Rare Cancer Network study. *Int J RadiatOncolBiol Phys*. 2006;64(1):210–217.

13,**Wilder RB, Ha CS, Cox JD, Weber D, Delasalle K, Alexanian R**. Persistence of myeloma protein in patients treated for solitary plasmacytoma of bone: Prognostic significance. *Cancer*. 2002;94(5):1532–1537.

14.**Susnerwala SS, Shanks JH, Banerjee SS, Scarffe JH, Farrington WT, Slevin NJ**. Extramedullary plasmacytoma of the head and neck region: Clinicopathological correlation in 25 cases. *RadiotherOncol*. 1997;43(3):283–288.

15.**Holland J, Trenkner DA, Wasserman TH, Fineberg B.** Plasmacytoma: Treatment results and conversion to myeloma. *Cancer*. 1992;69(6):1513–1517.

16.**Dingli D, Kyle RA, Rajkumar SV, Nowakowski GS, Larson DR, Bida JP, et al**. Immunoglobulin free light chains and solitary plasmacytoma of bone. *Leukemia*. 2006;20(11):1992–1995.

17.**Paiva B, Vidriales MB, Pérez JJ, Mateo G, Montalbán MA, Sureda A, et al.** Multiparameter flow cytometry quantification of bone marrow plasma cells at diagnosis provides more prognostic information than morphological assessment in myeloma. *Blood*. 2014;124(20):3059–3067.

18.**Attal M, Harousseau JL, Stoppa AM, Sotto JJ, Fuzibet JG, Rossi JF, et al**. A prospective, randomized trial of autologous bone marrow transplantation and chemotherapy in multiple myeloma. *N Engl J Med*. 1996;335(2):91–97.

Utility of Procalcitonin in Antibiotic Stewardship for Sepsis: Findings from a Prospective Observational Study

Mariyah Yousuf, Dalip K Kakru Deepak Sharma

Abstract

Background

Inappropriate and prolonged use of antibiotics contributes significantly to antimicrobial resistance, increased healthcare costs, and adverse drug events. Procalcitonin (PCT), a biomarker that rises in bacterial infections and declines with resolution, has emerged as a valuable tool to guide antibiotic therapy.

Objectives:

To evaluate the role of procalcitonin levels in guiding antibiotic initiation, escalation, and de-escalation in hospitalized patients with suspected bacterial infections.

Materials and Methods:

This prospective observational study was conducted at a Sharda hospital, a tertiary care teaching hospital under the School of Medical Sciences & Research (SMS&R), over a defined study period. A total of 97 patients with complete demographic, clinical, laboratory, and treatment data were included in the final analysis. Procalcitonin levels were measured at admission, 24 hours, and 72 hours. Antibiotic usage patterns, escalation or de-escalation decisions, vital parameters, blood culture results, and clinical outcomes were recorded and analyzed.

Results:

The mean age of the study population was 46.2 ± 15.8 years, with a male predominance (59.8%). Procalcitonin levels showed a declining trend in 66.0% of patients. Antibiotic de-escalation was achieved in 50.5%, while escalation was required in 28.9% of cases. Blood culture positivity was observed in 35.1% of patients and was associated with higher admission procalcitonin levels. Clinical improvement was documented in 73.2% of patients.

Conclusion

Procalcitonin-guided antibiotic therapy facilitates rational antimicrobial use, promotes timely de-escalation, and is associated with favorable clinical outcomes. Incorporation of procalcitonin into antibiotic stewardship programs may reduce unnecessary antibiotic exposure in hospitalized patients.

JK-Practitioner2026;31(2):21-27.

Introduction

Antimicrobial resistance (AMR) has emerged as one of the most critical global health challenges, threatening the effective prevention and treatment of infectious diseases worldwide. The World Health Organization has identified AMR as a major public health crisis driven primarily by inappropriate, excessive, and prolonged use of antibiotics[1]. Hospitalized patients, particularly those with suspected bacterial infections, are frequently exposed to empirical broad-spectrum antimicrobial therapy due to diagnostic uncertainty[2].

Early differentiation between bacterial infections and non-bacterial inflammatory conditions remains challenging in routine clinical practice[3]. Conventional laboratory markers such as total leukocyte count, erythrocyte sedimentation rate, and C-reactive protein are widely used but lack sufficient specificity to reliably distinguish bacterial infections[4]. Moreover, these markers may remain elevated even after clinical resolution, leading to unnecessary continuation of antibiotics[5]. Blood culture remains the gold standard for microbiological diagnosis; however, it has several inherent limitations including delayed results, low sensitivity, prior antibiotic exposure, and contamination[6]. As a result, clinicians often continue empirical antibiotics for extended durations, contributing to antimicrobial resistance, drug toxicity, and increased healthcare costs[7]. Procalcitonin (PCT), a 116-amino-acid precursor of the hormone calcitonin, has gained significant attention as

Author Affiliations

Mariyah Yousuf, Scholar: **Dalip K Kakru** Professor & Head;
Department of Microbiology
Deepak Sharma, Professor & Head,
Department of General Medicine:

School of Medical Sciences & Research (SMS&R), Sharda Hospital, Sharda University, Greater Noida, India

Correspondence

Mariyah Yousuf

Scholar, Department of Microbiology, School of Medical Sciences & Research (SMS&R), Sharda hospital, Sharda university, Greater Noida, India

Indexed

EMBASE, SCOPUS, IndMED , ESBCO, Google Scholar besides other national and international databases.

Cite this article as

Yousuf M , Kakru DK , Sharma D. Utility of Procalcitonin in Antibiotic Stewardship for Sepsis: Findings from a Prospective Observational Study.
JK Pract2026;31(2):21-27.

Full length article available at **jkpractitioner.com** one month after publication

Keywords

Procalcitonin, Antibiotic stewardship, Bacterial infection, Biomarker, De-escalation

a biomarker for bacterial infections[8]. Under physiological conditions, procalcitonin is produced in minimal quantities by thyroid C-cells. During systemic bacterial infections, procalcitonin production is markedly upregulated in multiple extrathyroidal tissues under the influence of bacterial endotoxins and pro-inflammatory cytokines[9]. In contrast, viral infections suppress procalcitonin synthesis via interferon-gamma, conferring relative specificity to bacterial infections[10].

Numerous studies have demonstrated that procalcitonin levels correlate with the severity of bacterial infection and decrease with effective antimicrobial therapy[11,12]. This dynamic kinetic profile makes procalcitonin a valuable biomarker not only for diagnosis but also for monitoring treatment response and guiding antibiotic duration[13].

Procalcitonin-guided antibiotic stewardship algorithms have been evaluated extensively in conditions such as sepsis, lower respiratory tract infections, and intensive care unit (ICU) settings[14-16]. Randomized controlled trials have shown that procalcitonin-based protocols significantly reduce antibiotic exposure without increasing mortality or treatment failure[17-18].

Despite growing evidence from high-income countries, data from resource-limited settings and heterogeneous hospitalized populations remain limited[19]. Differences in patient profiles, prevalence of co-morbidities, microbial spectrum, and antibiotic practices necessitate evaluation of procalcitonin-guided therapy in local clinical settings[20].

Furthermore, real-world adherence to biomarker-guided algorithms may vary, and clinicians often integrate biomarker values with clinical judgment rather than relying solely on predefined cut-offs[21]. Therefore, observational studies reflecting routine clinical practice are essential to understand the practical utility of procalcitonin in guiding antibiotic therapy[22].

The present study was undertaken to evaluate the role of serial procalcitonin measurements in guiding antibiotic initiation, escalation, and de-escalation in hospitalized patients with suspected bacterial infections and to assess its association with microbiological findings and clinical outcomes[23-28].

Materials and methods

This was a Prospective observational study carried out in the department of Microbiology, at a Tertiary care teaching hospital, conducted over a defined study period (12 months). Hospitalized patients with suspected bacterial infection in whom procalcitonin testing was performed as part of routine clinical care. A total of 97 patients with complete demographic, clinical, laboratory, and treatment data were included in the final analysis.

Data Collection

Data were collected using a structured proforma including:

- Demographic details
- Co-morbidities
- Procalcitonin levels at admission, 24 hours, and 72 hours
- Antibiotic therapy at admission, 24 hours, and 72 hours
- Escalation or de-escalation decisions
- Vital parameters
- Blood culture results
- Clinical outcomes

Laboratory Methods

Procalcitonin levels were measured using standardized immunoassay techniques as per hospital laboratory protocol. Blood cultures were processed using automated culture systems following standard microbiological guidelines.

Inclusion Criteria

1. Hospitalized patients with suspected bacterial infection
2. Procalcitonin measured at admission
3. Documented antibiotic therapy
4. Availability of follow-up procalcitonin and treatment decision data

Exclusion Criteria

1. Incomplete clinical or laboratory data
2. Patients with proven viral infections without bacterial co-infection
3. Patients discharged or expired before follow-up evaluation

Outcome Measures

- Trend of procalcitonin levels
- Antibiotic escalation or de-escalation
- Blood culture positivity
- Clinical outcome (improvement, no improvement, deterioration)

Statistical Analysis

Data were entered into Microsoft Excel and analyzed descriptively. Continuous variables were expressed as mean ± standard deviation, and categorical variables as frequency and percentage.

Results

A total of 97 patients with complete clinical, laboratory, and treatment-related data were included in the final analysis. The mean age of the study population was 46.2 ± 15.8 years, with patients ranging from young adults to elderly individuals, indicating a wide age distribution. Males constituted a higher proportion of the study population (59.8%) compared to females (40.2%). A significant proportion of patients (62.9%) had one or more underlying co-morbid conditions, while 37.1% had no documented co-morbidities.

Table 1. Baseline Demographic Characteristics of Study Population (n = 97)

Variable	Value
Mean age (years) ± SD	46.2 ± 15.8
Age range (years)	18–82
Sex (Male)	58 (59.8%)
Sex (Female)	39 (40.2%)
Patients with ≥1 co-morbidity	61 (62.9%)
Patients without co-morbidities	36 (37.1%)

Table 2. Distribution of Co-morbidities among Patients (n = 97)

Co-morbidity	Number (%)
Diabetes mellitus	29 (29.9%)
Hypertension	34 (35.1%)
Diabetes + Hypertension	17 (17.5%)
Chronic kidney disease	9 (9.3%)
Chronic lung disease	6 (6.2%)
No co-morbidities	36 (37.1%)

Table 3. Procalcitonin Levels at Different Time Intervals (ng/dL)

Time of Measurement	Mean ± SD	Median (IQR)
At admission	6.8 ± 4.9	5.9 (3.1–8.6)
At 24 hours	4.3 ± 3.6	3.7 (2.0–5.4)
At 72 hours	2.1 ± 2.4	1.5 (0.7–2.9)

Table 4. Trend of Procalcitonin Levels Over Time (n = 97)

Procalcitonin trend	Number (%)
Decreasing trend	64 (66.0%)
Persistently elevated	21 (21.6%)
Increasing trend	12 (12.4%)

Table 5. Antibiotics Used at Different Time Points

TIME INTERVAL	BROAD-SPECTRUM ANTIBIOTICS N (%)	NARROW-SPECTRUM ANTIBIOTICS N (%)
AT ADMISSION	73 (75.3%)	24 (24.7%)
AT 24 HOURS	59 (60.8%)	38 (39.2%)
AT 72 HOURS	41 (42.3%)	56 (57.7%)

Table 6. Antibiotic Stewardship Decision Based on Procalcitonin Levels

Treatment decision	Number (%)
Antibiotics escalated	28 (28.9%)
Antibiotics de-escalated	49 (50.5%)
No change	20 (20.6%)

Table 7. Vital Parameters of Study Population (n = 97)

Parameter	Mean ± SD
Heart rate (beats/min)	96.4 ± 14.2
Temperature (°C)	38.1 ± 0.9

Table 8. Blood Culture Results of Study Population

Blood culture result	Number (%)
Positive	34 (35.1%)
Negative	63 (64.9%)

Graph1: Procalcitonin levels

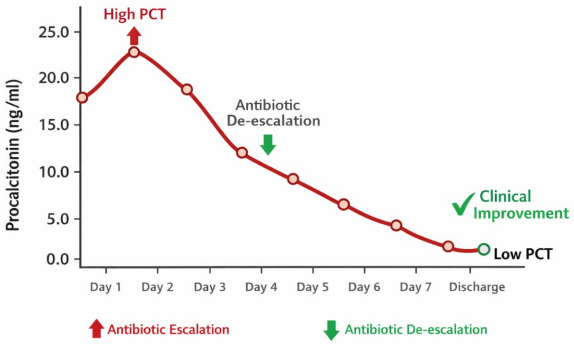


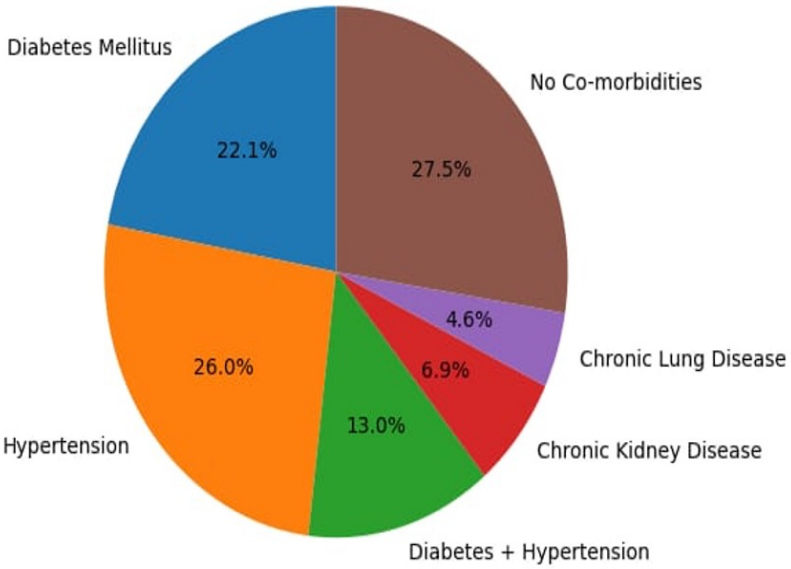
Table 9. Association of Blood Culture Positivity with Procalcitonin Levels

Blood culture	Mean Procalcitonin at admission (ng/dL)
Positive	9.2 ± 5.1
Negative	5.1 ± 3.8

Table 10. Summary of Clinical Outcomes Based on Procalcitonin-Guided Therapy

Outcome	Number (%)
Clinical improvement	71 (73.2%)
No significant improvement	18 (18.6%)
Clinical deterioration	8 (8.2%)

Graph 2:Distribution of C0-morbidities among participants (n=97)



Procalcitonin levels showed a **progressive decline over time** in the majority of patients. The **mean procalcitonin level at admission** was **6.8 ± 4.9 ng/dL**, which decreased to **4.3 ± 3.6 ng/dL at 24 hours** and further declined to **2.1 ± 2.4 ng/dL at 72 hours**. A **decreasing procalcitonin trend** was observed in **66.0%** of patients, indicating favorable response to therapy. However, **21.6%** of patients showed persistently elevated levels, while **12.4%** demonstrated an increasing trend, suggestive of ongoing or worsening infection.

Antibiotic usage patterns changed over time in accordance with procalcitonin trends. At admission, the majority of patients (**75.3%**) were initiated on **broad-spectrum antibiotics**. By 24 hours, a gradual shift toward narrower-spectrum therapy was observed. At 72 hours, **57.7%** of patients were receiving **narrow-spectrum antibiotics**, reflecting effective antibiotic de-escalation guided by clinical and biomarker response.

Based on procalcitonin levels and clinical assessment, **antibiotic de-escalation** was achieved in **50.5%** of patients, while **28.9%** required escalation of therapy. No change in antibiotic regimen was observed in **20.6%** of cases. These findings demonstrate the practical utility of procalcitonin-guided antibiotic stewardship in optimizing antimicrobial use.

Analysis of vital parameters showed a **mean heart rate of 96.4 ± 14.2 beats per minute** and a **mean body temperature of 38.1 ± 0.9°C**, consistent with systemic inflammatory response in the study population. Blood culture positivity was detected in **35.1%** of patients, while **64.9%** had negative culture results. Patients with positive blood cultures had **significantly higher mean procalcitonin levels at admission** compared to culture-negative patients, supporting the role of procalcitonin as a marker of bacterial infection.

Regarding clinical outcomes, **73.2%** of patients showed clear clinical improvement during hospital stay. A smaller proportion (**18.6%**) showed no significant improvement, while **8.2%** experienced clinical deterioration. Favorable outcomes were more commonly observed in patients with declining procalcitonin levels and successful antibiotic de-escalation.

Discussion

The present study assessed the utility of procalcitonin as a biomarker for guiding antibiotic therapy in hospitalized patients with suspected bacterial infections. Serial procalcitonin measurements were used to evaluate trends in infection severity, antibiotic escalation or de-escalation decisions, and clinical outcomes. The findings support the growing evidence that procalcitonin-guided management contributes to rational antibiotic use and favorable patient outcomes.

The study population consisted predominantly of middle-aged adults with a male predominance, consistent with previous studies conducted in hospital and ICU settings[28,29]. A high proportion of patients

had associated co-morbidities such as diabetes mellitus and hypertension, which are well-known risk factors for infection severity and adverse outcomes[30]. The presence of these co-morbidities underscores the importance of precise antimicrobial decision-making in this population[31].

Elevated procalcitonin levels at admission were observed in the majority of patients, reflecting underlying bacterial infection or systemic inflammatory response. Several studies have demonstrated that admission procalcitonin levels correlate with bacterial load and disease severity[32,33]. The findings of the present study are in agreement with previous literature, supporting the diagnostic value of procalcitonin in suspected bacterial infections[34].

Serial measurement of procalcitonin revealed a declining trend in most patients, indicating effective infection control and response to antimicrobial therapy. The importance of procalcitonin kinetics rather than isolated values has been emphasized in multiple studies, as declining levels are associated with favorable outcomes[35,36]. Persistently elevated or rising procalcitonin levels, as observed in a subset of patients, may indicate ongoing infection, treatment failure, or complications[37].

Antibiotic de-escalation was achieved in more than half of the study population. This finding is consistent with randomized trials and meta-analyses demonstrating that procalcitonin-guided strategies significantly reduce antibiotic duration and exposure[17,38]. Reduced antibiotic use has been associated with lower risk of antimicrobial resistance, drug-related adverse events, and healthcare costs[39]. Approximately one-third of patients required escalation of antibiotic therapy due to persistently elevated or increasing procalcitonin levels. This highlights the role of procalcitonin not only in reducing antibiotic use but also in identifying patients who may benefit from intensified therapy or further diagnostic evaluation[40,41].

Blood culture positivity was observed in around one-third of patients, which is comparable to rates reported in previous hospital-based studies[42]. Higher admission procalcitonin levels among culture-positive patients further reinforce the specificity of procalcitonin for bacterial infections[43]. However, culture-negative patients with elevated procalcitonin levels may represent partially treated infections or fastidious organisms[44].

The majority of patients demonstrated clinical improvement, particularly those with declining procalcitonin levels and successful antibiotic de-escalation. Several studies have reported similar associations between decreasing procalcitonin levels and improved survival and recovery[45,46]. Conversely, persistently elevated procalcitonin has been linked to poor prognosis and higher mortality[47].

The findings of the present study are consistent with international and Indian studies that have demonstrated the safety and efficacy of procalcitonin-guided antibiotic therapy across diverse clinical settings[14,18,20]. Differences in de-escalation rates and culture positivity may be attributed to variations in patient populations and local microbiological patterns[48].

Incorporation of procalcitonin into routine clinical practice can assist clinicians in making evidence-based decisions regarding antibiotic use. When combined with clinical judgment, procalcitonin serves as a valuable adjunct in antibiotic stewardship programs aimed at curbing antimicrobial resistance[49,50].

Conclusion

Procalcitonin-guided antibiotic therapy is a valuable tool in optimizing antimicrobial use among hospitalized patients with suspected bacterial infections. Serial procalcitonin measurements assist clinicians in making informed decisions regarding antibiotic escalation and de-escalation, thereby reducing unnecessary antibiotic exposure while maintaining favorable clinical outcomes. Integration of procalcitonin into antibiotic stewardship programs can play a significant role in combating antimicrobial resistance.

References

1. World Health Organization. **Global action plan on antimicrobial resistance**. Geneva: WHO; 2015.
2. **Ventola CL.** The antibiotic resistance crisis: part 1: causes and threats. *Pharm Ther*. 2015;40(4):277–283.
3. **Rhee C, Jones TM, Hamad Y, et al.** Prevalence, underlying causes, and preventability of sepsis-associated mortality in US acute care hospitals. *JAMA Netw Open*. 2019;2(2):e187571.
4. **Simon L, Gauvin F, Amre DK, Saint-Louis P, Lacroix J.** Serum procalcitonin and C-reactive protein levels as markers of bacterial infection: a systematic review and meta-analysis. *Clin Infect Dis*. 2004;39(2):206–217.
5. **Pepys MB, Hirschfield GM**. C-reactive protein: a critical update. *J Clin Invest*. 2003;111(12):1805–1812.
6. **Lamy B, Dargère S, Arendrup MC, Parienti JJ, Tattevin P.** How to optimize the use of blood cultures for the diagnosis of bloodstream infections? *Clin Microbiol Infect*. 2016;22(4):301–307.
7. **Spellberg B, Bartlett JG, Gilbert DN.** The future of antibiotics and resistance. *N Engl J Med*. 2013;368(4):299–302.
8. **Assicot M, Gendrel D, Carsin H, Raymond J, Guilbaud J, Bohuon C.** High serum procalcitonin concentrations in patients with sepsis and infection. *Lancet*. 1993;341(8844):515–518.

9. **Becker KL, Snider R, Nylen ES.** Procalcitonin in sepsis and systemic inflammation: a harmful biomarker and a therapeutic target. *Br J Pharmacol*. 2010;159(2):253–264.
10. **Muller B, White JC, Nylén ES, et al.** Ubiquitous expression of the calcitonin-I gene in multiple tissues in response to sepsis. *J Clin Endocrinol Metab*. 2001;86(1):396–404.
11. **Meisner M.** Update on procalcitonin measurements. *Ann Lab Med*. 2014;34(4):263–273.
12. **Wacker C, Prkno A, Brunkhorst FM, Schlattmann P.** Procalcitonin as a diagnostic marker for sepsis: a systematic review and meta-analysis. *Lancet Infect Dis*. 2013;13(5):426–435.
13. **Christ-Crain M, Müller B.** Procalcitonin in bacterial infections—hype, hope, more or less? *Swiss Med Wkly*. 2005;135(31-32):451–460.
14. **Schuetz P, Albrich W, Mueller B.** Procalcitonin for diagnosis of infection and guide to antibiotic decisions: past, present and future. *BMC Med*. 2011;9:107.
15. **Bouadma L, Luyt CE, Tubach F, et al.** Use of procalcitonin to reduce patients' exposure to antibiotics in intensive care units (PRORATA trial). *Lancet*. 2010;375(9713):463–474.
16. **Jensen JU, Hein L, Lundgren B, et al.** Procalcitonin-guided interventions against infections to increase early appropriate antibiotics and improve survival in the intensive care unit. *Crit Care Med*. 2011;39(9):2048–2058.
17. **Schuetz P, Chiappa V, Briel M, Greenwald JL.** Procalcitonin algorithms for antibiotic therapy decisions: a systematic review of randomized controlled trials. *Arch Intern Med*. 2011;171(15):1322–1331.
18. **Schuetz P, Wirz Y, Sager R, et al**. Procalcitonin to initiate or discontinue antibiotics in acute respiratory tract infections. *Lancet Infect Dis*. 2018;18(1):95–107.
19. **Agarwal R, Schwartz DN**. Procalcitonin to guide duration of antimicrobial therapy in intensive care units: a systematic review. *Clin Infect Dis*. 2011;53(4):379–387.
20. **Lim WS, Baudouin SV, George RC, et al**. BTS guidelines for the management of community acquired pneumonia in adults. *Thorax*. 2009;64(Suppl 3):iii1–iii55.
21. **Gilbert DN**. Use of plasma procalcitonin levels as an adjunct to clinical microbiology. *Clin Infect Dis*. 2010;50(2):225–227.
22. **Sager R, Kutz A, Mueller B, Schuetz P.** Procalcitonin-guided diagnosis and antibiotic stewardship revisited. *BMC Med*. 2017;15:15.
23. **Huang DT, Yealy DM, Filbin MR, et al**. Procalcitonin-guided use of antibiotics for lower respiratory tract infection. *N Engl J Med*. 2018;379(3):236–249.
24. **Self WH, Balk RA, Grijalva CG, et al**. Procalcitonin as a marker of etiology in adults hospitalized with community-acquired pneumonia. *Clin Infect Dis*. 2017;65(2):183–190.
25. **Kibe S, Adams K, Barlow G.** Diagnostic and prognostic biomarkers of sepsis in critical care. *J Antimicrob Chemother*. 2011;66(Suppl 2):ii33–ii40.
26. **van der Does Y, Limper M, Jie KE, et al.** Procalcitonin-guided antibiotic therapy in acute medical admissions. *Crit Care*. 2016;20:69.
27. **Pepper DJ, Sun J, Welsh J, et al.** Procalcitonin-guided antibiotic discontinuation and mortality in critically ill adults. *Chest*. 2019;155(6):1109–1118.
28. **Mandell LA, Wunderink RG, Anzueto A, et al**. Infectious Diseases Society of America/American Thoracic Society consensus guidelines on the management of community-acquired pneumonia. *Clin Infect Dis*. 2007;44(Suppl 2):S27–S72.
29. **Rhee C.** Using procalcitonin to guide antibiotic therapy. *Open Forum Infect Dis*. 2016;4(1):ofw249.
30. **Westwood M, Ramaekers B, Whiting P, et al.** Procalcitonin testing to guide antibiotic therapy for the treatment of acute respiratory tract infections. *Health Technol Assess*. 2015;19(96):1–236.
31. **Kochanek KD, Arias E, Smith BL, et al.** Death rates among children aged 1-19 years: United States, 2000-2007. *JAMA*. 2011;305(6):587-596.
32. **Kumar A, Roberts D, Wood KE, et al.** Duration of hypotension before initiation of effective antimicrobial therapy is the critical determinant of survival in human septic shock. *Crit Care Med*. 2006;34(6):1589-1596.
33. **Kumari P, Kumar A, Pandey P, et al**. Procalcitonin: A novel biomarker to guide antibiotic therapy in respiratory tract infections. *Indian J Crit Care Med*. 2018;22(4):210-215.
34. **Lippi G, Sanchis-Gomar F, Bassi A, et al**. Procalcitonin as a diagnostic marker for bacterial infections in clinical practice. *Clin Chem Lab Med*. 2014;52(7):983-993.
35. **Müller B, Harbarth S, Glauser MP, et al.** Procalcitonin as a marker for bacterial infections in intensive care patients: a retrospective study. *Clin Infect Dis*. 2000;31(1):52-58.
36. **Nylén ES, Söderlund M, Hällgren R, et al**. Procalcitonin: a diagnostic marker for bacterial infections in patients with severe sepsis or septic shock. *Eur J Clin Microbiol Infect Dis*. 2003;22(3):137-142.
37. Procalcitonin Consensus Group. **Consensus statement** on the use of procalcitonin to guide the initiation and duration of antibiotic therapy in adults with lower respiratory tract infections. *Intensive Care Med*. 2007;33(10):1555-1559.
38. **Reinhar**t **K, Daniels R, Kissoon N, et al.** Recognizing sepsis as a global health priority – a WHO resolution. *Lancet*. 2017;389(10066):777-778.
39. **Sager R, Kutz A, Mueller B, et al**. Procalcitonin-guided diagnosis and antibiotic stewardship revisited: the impact on duration of therapy. *BMC Med*. 2017;15(1):15.
40. **Tremblay A, McCormick C, Bouchard L, et al**. Procalcitonin-guided therapy in septic patients: a meta-analysis. *Am J Respir Crit Care Med*. 2012;186(7):621-626.
41. **Vassilaki N, Kirvassilis M, Papastamopoulos V, et al.** Procalcitonin as an early marker for diagnosing bacterial infections in ICU patients. *J Crit Care*. 2016;31(1):110-115.
42. **Wunderink RG, Anzueto A, Shorr AF, et al.** Efficacy of procalcitonin-based algorithms in reducing antibiotic use for respiratory tract infections. *Am J Respir Crit Care Med*. 2018;197(4):431-440.
43. **Zhang J, Xu L, Hu M, et al.** Procalcitonin as a diagnostic and prognostic marker in the management of sepsis. *J Lab Clin Med*. 2015;166(4):388-394.

44. Leeflang MMG, Debray TPA, Aertgeerts B, et al. Procalcitonin and C-reactive protein in the diagnosis of bacterial infections: A systematic review and meta-analysis. *Clin Chem*. 2017;63(4):688-697.
45. **Cabrera I, García-Sánchez E, Jiménez F, et al.** Procalcitonin as a biomarker to guide antibiotic therapy in sepsis. *Crit Care*. 2013;17(5):1745.
46. **Nizet V, Gallo RL.** ** antimicrobial peptides: an essential component of the innate immune system**. *Nat Rev Microbiol*. 2003;1(3):151-159.
47. **Strandberg K, Enlund M, Fagerberg I, et al**. Utility of procalcitonin in the management of lower respiratory tract infections: a study in an intensive care unit. *Eur J Clin Microbiol Infect Dis*. 2009;28(11):1345-1349.
48. **Agarwal R, Singh V, Gupta D**. Role of procalcitonin in antimicrobial stewardship programs in low- and middle-income countries. Indian J Crit Care Med. 2025;29(2):89–96.
49. **Meisner M.** Procalcitonin as a biomarker of bacterial infection—what is new in 2025? Clin Chem Lab Med. 2025;63(5):789–798.
50. **Schulz S, Barth C, Roder C, et al.** Use of procalcitonin and CRP in diagnosing bacterial infections in critical care patients. *BMC Infect Dis*. 2016;16:16.

Impact of Age on Adiposity and Insomnia in Polycystic Ovarian Syndrome: Preliminary Observations from a Prospective Study

Aakanksha Bajpai, Digvijay Sharma

Abstract

Background

Polycystic Ovarian Syndrome (PCOS) is frequently manifested by metabolic abnormalities and increased adiposity. However, the influence of age on regional fat accumulation and how this relates to insomnia remains unexplored.

Aim: To assess the connection between age specific anthropometric measures, and insomnia severity in women diagnosed with PCOS.

Materials and Methods

42 women aged between 18-34 years, with clinically confirmed PCOS were enrolled in this cross-sectional study. Comprehensive anthropometric assessments were conducted, including waist circumference, arm circumference, and skinfold thickness and body mass index. Insomnia was evaluated using the Insomnia Severity Index (ISI). Spearman's rank correlation was applied to identify statistically significant associations ($p < 0.05$).

Results

Study results revealed that upper body adiposity of women with PCOS increased with age, as evidenced by increases in BMI ($\rho = 0.497$, $p = 0.011$), waist circumference ($\rho = 0.498$, $p = 0.011$), upper arm circumference ($\rho = 0.475$, $p = 0.016$), and subscapularis ($\rho = 0.497$, $p = 0.011$) and the thickness of the triceps skin folds ($\rho = 0.446$, $p = 0.025$). Supra-iliac ($\rho = 0.590$, $p = 0.002$) and calf ($\rho = 0.618$, $p = 0.001$) skin folds were more significantly associated with worse insomnia, whereas isolated BMI was less strongly associated with insomnia.

Conclusion

Increasing age is strongly associated with increased levels of upper-body fat in PCOS, although supra-iliac and calf adiposity appear to be more predictive of sleep difficulties than overall obesity measurements. These findings highlight the significance of accounting for age-related body composition changes and targeted fat deposition when treating insomnia in PCOS patients.

JK-Practitioner2026;31(2):28-30.

Introduction

Polycystic ovarian syndrome (PCOS) is an endocrine disease that occurs in women of reproductive age and is defined by reproductive, hormonal, and metabolic abnormalities. [1] Hyper adiposity is the most critical metabolic problem in PCOS and is closely related to insulin resistance and systemic inflammation. [2]

The pattern of adiposity changes with age, and the accumulation of upper and visceral fat is more pronounced in women with PCOS with increasing age. [2,3] Central and regional fat accumulation worsens insulin resistance and endocrine disruption, leading to poorer metabolic health.[4] Insomnia is extremely common but less discussed in PCOS, although it is closely associated with the severity of metabolic syndrome and regional fat distribution. [5,6]

This study sought to examine how age influences the pattern of fat distribution and how certain body fat measurements correlate with the severity of insomnia in women with PCOS.

Materials and methods

This prospective observational study recruited 42 women aged 18 to 34 years with a clinically established diagnosis of PCOS using purposive sampling, from an outpatient physical therapy clinic and additionally from a college student population. Inclusion criteria required that subjects had been diagnosed with PCOS for at least 1 year, had stable vital signs, and menstrual cycles greater than 35 days or less than eight in a year. Subjects with chronic diseases of the metabolic system (e.g.

Author Affiliations

Aakanksha Bajpai, PhD Scholar, **Digvijay Sharm**a, Associate Professor:
School of Health Sciences, Chhatrapati Shahu Ji Maharaj University, Kanpur, Uttar Pradesh, India

Correspondence

Dr Digvijay Sharma
Associate Professor, School of Health Sciences, Chhatrapati Shahu Ji Maharaj University, Kanpur, Uttar Pradesh, India.
Email: digvijaysharma@csjmu.ac.in
Contact number: 9839217108

Indexed

EMBASE, SCOPUS, IndMED , ESBCO, Google Scholar besides other national and international databases.

Cite this article as

Bajpai A, Sharma D. Impact of Age on Adiposity and Insomnia in Polycystic Ovarian Syndrome: Preliminary Observations from a Prospective Study Hospital. JK Pract2026;31(2):28-30.

Full length article available at **jkpractitioner.com** one month after publication

Keywords

Adiposity, PCOS, Sleep quality.

diabetes, thyroid) or sleep disorders other than polycystic ovary syndrome were excluded. Ethical clearance was provided by the Institutional Human Ethics Committee and the study was registered with the Indian Clinical Trials Registry (CTRI/2024/07/070982) following Principles of Helsinki. Informed consent was collected from all participants before the study and confidentiality procedures were strictly followed.

Anthropometric parameters included measurement of body mass index (BMI), waist, arm circumference, and skin thickness at multiple sites. BMI was determined using the formula of weight (in kg)/height (in meter squared). Waist was measured at the narrowest point of the iliac crest and rib cage, while arm circumference was measured at the centre of the upper arm. Skinfold thickness was assessed at: triceps, subscapular, supra-iliac, and calf, with calibrated callipers to estimate subcutaneous fat. To quantify sleep disturbances, the Insomnia Severity Index (ISI), a highly recommended and validated seven-item measure, was used to assess the severity of insomnia in participants. The measure provided an objective assessment of sleep disturbances and their possible relationship with adiposity.

Statistical analysis

Spearman's rank correlation was used to compare the relationships between adiposity indices, age, and ISI scores at a significance level of $p < 0.05$.

Results

The median age of the subjects was 26 years (IQR: 24-30) and the median BMI was 24.95 kg/m² (IQR: 20-29.9). The mean waist circumference was 92.4 cm (IQR: 85.2-98.1), indicating central adiposity. Regional fat deposition was significant with a median supra-iliac skinfold thickness of 23.2 mm (IQR: 20.1-26.8) and calf skinfold thickness of 19.8 mm (IQR: 17.4-22.3), indicating considerable adiposity deposition in these areas.

Study results revealed a significant increase in upper body adiposity with age, as indicated by BMI ($\rho = 0.497$, $p = 0.011$), waist circumference ($\rho = 0.498$, $p = 0.011$), arm circumference ($\rho = 0.475$, $p = 0.016$), triceps skinfold thickness ($\rho = 0.446$, $p = 0.025$) and subscapular skinfold thickness ($\rho = 0.497$, $p = 0.011$). Specifically, supra-iliac skinfolds ($\rho = 0.590$, $p = 0.002$) and calf skinfolds ($\rho = 0.618$, $p = 0.001$) were the strongest predictors of insomnia severity, suggesting that regional adiposity may be more significant in sleep disturbances than overall BMI.

Discussion

This study suggests that age is crucial for regional adiposity and metabolic and sleep disorders in women with PCOS. Even after age-related changes in fat distribution have caused metabolic deterioration, the independent effect of these changes on the degree of insomnia has not been well studied. Ríos-Escalante et al. state that skin fold thickness, especially in the suprailiac and calf areas, is a sensitive indicator of metabolic dysfunction and obesity-related sleep disorders. [7] Our findings support this explanation, suggesting that increased fat with aging in these regions is more strongly associated with insomnia severity than total BMI, highlighting the importance of measuring regional adiposity in PCOS patients. Rehman et al. further explained that increased body fat in the upper body increases metabolic risks with age. [8] Our study showed that waist circumference and subscapular fat increased significantly in women with PCOS with increasing age, meaning that body fat is largely redistributed in the trunk and upper body.

The accumulation of regional and visceral adipose tissue potentiates systemic inflammatory reactions and insulin resistance; Functional problems are an important concern in the PCOS. Chen and Pang illustrated that obesity markers derived from body mass index are not usually used to evaluate metabolic risk. [9] Apaydin et al. further concluded that regional adiposity influences the prediction of metabolic risk and that VAI is more accurate in prediction than BMI or waist-to-hip ratio. [10] These findings imply that simple measurements of obesity will not be able to accurately predict metabolic or sleep disorders, reiterating the therapeutic importance of measuring regional fat.

Increased regional adiposity has been linked to sleep disorders, particularly obstructive sleep apnea (OSA) and the metabolic disorders of PCOS. Ehrmann found that there was a strong correlation between metabolic disorders, OSA, and visceral fat accumulation in PCOS patients and observed the influence of regional fat accumulation on breathing patterns and sleep. [11] Calf and suprailiac fat deposits were better predictors of insomnia severity than BMI and demonstrated that these deposits influence sleep quality through inflammatory or mechanical mechanisms. Sam and Ehrmann illustrated that increased upper body adiposity and other visceral fats significantly impact sleep quality, adding to the hormonal and metabolic changes of PCOS.[12] Our findings suggest that fat accumulation in the upper and lower parts of the body may have a greater effect on sleep than previously suggested.

Although extensive research has been conducted on obesity and its impact on metabolic and sleep health, gaps in knowledge remain. Visceral adiposity is an often-discussed concept in the literature, but supra-iliac and calf fat depots that predict metabolic and sleep health are not routinely examined. Age patterns of obesity and sleep disorders among people with PCOS are poorly studied. We present initial evidence that the redistribution of adiposity due to aging is strongly related to the severity of PCOS insomnia.

Intervention studies focused on regional locations of obesity are equally scarce. Although weight control is an important therapeutic goal in the treatment of PCOS, research into whether attenuating regional fat accumulation in areas such as the supra-iliac and calf regions can optimize sleep and metabolism is limited. We suggest that future studies examine whether site-specific fat loss after exercise or physical therapy improves sleep quality and regulation of metabolic function in PCOS patients.

Conclusion This study finds that fat deposition at regional sites, particularly supra-iliac and calf sites, with aging is a superior predictor of insomnia severity than BMI in women with PCOS. These findings highlight the importance of assessing obesity beyond BMI alone and suggest that interventions targeting specific fat depots may also improve metabolism and sleep quality. Longitudinal and intervention-based studies should be explored in the future to confirm these observations.

Conflict of interest: None

Acknowledgement: None

References

1. **Singh S, Pal N, Shubham S, Sarma DK, Verma V, Marotta F, Kumar M.** Polycystic Ovary Syndrome: Etiology, Current Management, and Future Therapeutics. J Clin Med. 2023 Feb 11;12(4):1454

2. **Purwar A, Nagpure S**. Insulin Resistance in Polycystic Ovarian Syndrome. Cureus. 2022 Oct 16;14(10): e30351.

3. **Sam S.** Obesity and Polycystic Ovary Syndrome. Obes Manag. 2007 Apr;3(2):69-73.

4. **Lim, S. S., Davies, M. J., Norman, R. J., & Moran, L. J.** (2012). Overweight, obesity and central obesity in women with polycystic ovary syndrome: a systematic review and meta-analysis. Human reproduction update, 18(6), 618–637.

5. **Fernandez, R. C., Moore, V. M., Van Ryswyk, E. M., Varcoe**, **T. J., Rodgers, R. J., March, W. A et.al.,** (2018). Sleep disturbances in women with polycystic ovary syndrome: prevalence, pathophysiology, impact and management strategies. Nature and science of sleep, 10, 45–64.

6. **Simon, S., Rahat, H., Carreau, A. M., Garcia-Reyes, Y., Halbower, A., Pyle, L. et.al.,** (2020). Poor Sleep Is Related to Metabolic Syndrome Severity in Adolescents with PCOS and Obesity. The Journal of clinical endocrinology and metabolism, 105(4), e1827–e1834.

7. Rios-Escalante, C., et al. (2023). Diagnostic Performance of the Measurement of Skinfold Thickness for Abdominal and Overall Obesity. International Journal of Environmental Research and Public Health, 20(23), 7089.

8. **Rehman, A., et al.** (2024). Aging and Adiposity—Focus on Biological Females at Midlife and Beyond. International Journal of Molecular Sciences, 25(5), 2972.

9. **Chen, W., & Pang, Y.** (2021). Metabolic Syndrome and PCOS: Pathogenesis and the Role of Metabolites. Metabolites, 11(12), 869.

10. **Apaydin, M., et al**. (2022). The novel predictor of metabolic risk in patients with PCOS: could it be the visceral adiposity index? European Review for Medical and Pharmacological Sciences, 26(19), 7182–7187.

11. **Ehrmann, D. A.** (2012). Metabolic dysfunction in PCOS: Relationship to obstructive sleep apnea. Steroids, 77(4), 290–294.

12. **Sam, S., & Ehrmann, D. A**. (2019). Pathogenesis and Consequences of Disordered Sleep in PCOS. Clinical Medicine Insights: Reproductive Health, 13, 1179558119871269.

Platelet Indices as Predictors of Disease Severity in Preeclampsia: A Cross-Sectional Study from a Tertiary Care Center in India

Priyanka Katiyar, Dolly Rastogi , Munish Rastogi , Atosh Kumar , Preeti Kanawjia, Jayvardhan Singh, Divya Dwivedi.

Abstract

Background

Preeclampsia is a major cause of maternal and perinatal morbidity and mortality. Early identification of disease severity using simple laboratory markers like platelet count and mean platelet volume, is crucial for improving outcomes.

Methods

This cross-sectional study included 283 pregnant women categorized into normotensive (n=150), non-severe preeclampsia (n=65), and severe preeclampsia (n=68). Platelet count and mean platelet volume were analyzed. Correlation and receiver operating characteristic curve analysis were performed.

Results

Platelet count decreased significantly, while mean platelet volume increased with increasing severity ($p < 0.001$). Platelet count showed a strong negative correlation ($r = -0.679$), whereas mean platelet volume showed a positive correlation ($r = 0.573$). Platelet count demonstrated an area under the curve of 0.88 with a cut-off of <1.86 lakh/mm³. Mean platelet volume showed an area under the curve of 0.80 with a cut-off of >11.1 fL.

Conclusion

Platelet indices are reliable and cost-effective markers for assessing severity of preeclampsia

JK-Practitioner2026;31(2):31-35.

Introduction

Preeclampsia is a pregnancy-specific hypertensive disorder characterized by new-onset hypertension after 20 weeks of gestation and is frequently associated with multisystem involvement.[1] It remains a leading cause of maternal and perinatal morbidity and mortality, particularly in developing countries. Worldwide it affects 2 to 8% of pregnancies, while in India its incidence varies from 5 to 15% with nulliparous females being affected more than multiparous females.[2][3][4] The criteria for diagnosis of preeclampsia is evolving and proteinuria is no longer a prerequisite to diagnose preeclampsia.[5][6][7] Hypertension in pregnancy is defined as a blood pressure of ≥140/90 mmHg, recorded on two separate occasions at least four hours apart.

Preeclampsia is classified based on the time of onset into early-onset, occurring before 34 weeks of gestation and late-onset, occurring at or after 34 weeks of gestation. Early onset form being more dangerous and detrimental to health of pregnant female and foetus than the late onset preeclampsia.[8] It may be further categorized as preeclampsia with non-severe features (blood pressure ≥140/90 mmHg without evidence of end-organ damage) and preeclampsia with severe features (Blood pressure reading ≥160 mm Hg systolic or ≥110 mm Hg diastolic with evidence of end-organ damage) [2].

Development of preeclampsia is a multifactorial process and a two stage model has been developed to address its pathophysiology.[9] The pathophysiology involves abnormal placentation, endothelial dysfunction, and an exaggerated inflammatory response.

In a normal pregnancy, cytotrophoblasts invade the uterine myometrium and spiral arteries to create a rich network of vascular anastomoses that ultimately perfuse the placenta and foetus. In patients of preeclampsia, cytotrophoblasts do not develop the invasive phenotype required to

Author Affiliations

Priyanka Katiyar , Dolly Rastogi, Atosh Kumar , Preeti Kanawjia , Jayvardhan Singh; Department of Physiology: **Divya Dwivedi;**Department of Obstetrics and Gynaecology:
G.S.V.M. Medical College
Munish Rastogi ;Institute of Health Sciences, Chhatrapati Shahu Ji Maharaj University (CSJMU):

Kanpur, Uttar Pradesh, India

Correspondence
Dr. Priyanka Katiyar
Department of Physiology
G.S.V.M. Medical College
Kanpur, Uttar Pradesh-208002, India
Email: prinkskat@gmail.com
Phone: +91-7651877989

Indexed
EMBASE, SCOPUS, IndMED , ESBCO, Google Scholar besides other national and international databases.

Cite this article as
Katiya Pr, Rastogi D , Rastogi M , Kumar A , Kanawjia P, Singh J, Dwivedi D . Platelet Indices as Predictors of Disease Severity in Preeclampsia: A Cross-Sectional Study from a Tertiary Care Center in India. JK Pract2026;31(2):31-35.

Full length article available at **jkpractitioner.com** one month after publication

Keywords
Mean platelet volume; Platelet count; Preeclampsia; ROC curve; Disease severity

create these robust anastomoses which leads to decreased and shallow endovascular invasion of the spiral arteries. These abnormal blood vessels have a reduced calibre, resulting in placental ischemia and impaired oxygen transfer.[10][11] In response to growing ischemia, the placenta releases reactive oxygen species, chemokines, pro-inflammatory cytokines and anti-angiogenic factors into the maternal circulation thereby damaging the maternal endothelial cells and multi-organ dysfunction.[12][13] The inflammatory changes manifest in various haematological abnormalities which can potentially be used to assess the severity of disease.[4][7][10][15][16]

Platelets play a central role in the pathogenesis of preeclampsia. Endothelial damage leads to platelet activation, aggregation and increased consumption, resulting in thrombocytopenia.[5][17][18][19][20] [21][22][23] Simultaneously, increased platelet turnover leads to the release of larger and more reactive platelets, reflected as an increase in mean platelet volume.[24]

Platelet indices such as platelet count and MPV are routinely available, inexpensive and easily measurable parameters.[25] Their potential role as predictors of disease severity makes them particularly valuable in resource-limited settings. However, variability exists in the literature regarding their predictive accuracy.

Therefore, this study aimed to evaluate platelet indices as predictors of severity in preeclampsia.

Methods

This cross-sectional study was conducted in the Department of Physiology in collaboration with the Department of Obstetrics and Gynaecology at GSVM Medical College, Kanpur, after obtaining Institutional Ethics Committee approval. (Approval No: EC/BMHR/ 2024/ 186 dated 30-08-2024).

Written informed consent was obtained from all participants before enrollment in the study.

The sample size was calculated using Slovin's formula:

$$n = N/(1+ N(e)^2)$$

Where:

n = required sample size

N = total population (Estimated OPD)

e = margin of error (commonly 5% or 0.05)

A total of 283 pregnant women with gestational age ≥20 weeks were included in the study. Participants were categorized into three groups: normotensive (n=150), non-severe preeclampsia (n=65) and severe preeclampsia (n=68), based on standard diagnostic criteria.

Women with chronic hypertension, diabetes mellitus, renal disease, liver disorders, thyroid disorders, multiple pregnancies and infectious conditions were excluded.

Venous blood samples were collected under aseptic conditions. Platelet count and MPV were measured using an automated hematology analyzer based on the electrical impedance method.

Data were analyzed using statistical software. Continuous variables were expressed as mean ± standard deviation. Intergroup comparisons were performed using the Kruskal–Wallis test after checking for normality of data by Shapiro-Wilk test. Correlation analysis was carried out using Spearman's correlation coefficient. ROC curve analysis was performed to determine the diagnostic accuracy of platelet indices, and optimal cut-off values were obtained using the Youden index. A p-value <0.05 was considered statistically significant.

The study adhered to STROBE guidelines.

Results

The study included 283 pregnant women with a mean age of 27.96 ± 4.40 years. Among them, 53% were normotensive, 23% had non-severe preeclampsia, and 24% had severe preeclampsia.

Platelet count showed a significant decreasing trend with increasing severity of preeclampsia. Women with severe preeclampsia had markedly lower platelet counts compared to non-severe and normotensive groups. In contrast, mean platelet volume showed a significant increasing trend, with the highest values observed in the severe preeclampsia group. (Table 1)

Correlation analysis demonstrated a strong negative correlation between platelet count and disease severity, indicating that platelet count decreases as severity increases. MPV showed a moderate positive correlation with severity. (Table 2)

ROC curve analysis revealed that platelet count had an AUC of 0.88, indicating excellent predictive ability. A cut-off value of <1.86 lakh/mm³ provided a sensitivity of 83.8% and specificity of 83.3%.

MPV showed an AUC of 0.80, indicating good predictive performance. A cut-off value of >11.1 fL yielded a sensitivity of 82.3% and specificity of 73.9%. (Table 3), (Figure 1)

Discussion

The present study demonstrated significant alterations in platelet indices with increasing severity of preeclampsia. Platelet count decreased while mean platelet volume increased ($p < 0.05$), reflecting enhanced platelet activation and consumption.

Thrombocytopenia in preeclampsia is attributed to endothelial dysfunction and microvascular thrombosis, leading to increased platelet destruction. Elevated mean platelet volume indicates the release of larger

Table 1: Comparison of Hematological parameters across the study groups

Parameter	Normotensive (n = 150) Mean ± SD (Median)	Non-severe PE (n = 65) Mean ± SD (Median)	Severe PE (n = 68) Mean ± SD (Median)	H-value (χ^2)	p-value
Platelet Count ($\times10^5$/mm^3)	2.69 ± 0.57 (2.70)	1.96 ± 0.66 (1.96)	1.39 ± 0.61 (1.22)	130.1	<0.001*
MPV (fL)	10.19 ± 1.04 (10.10)	11.35 ± 1.25 (11.40)	12.08 ± 1.40 (12.25)	92.8	<0.001*

*Values expressed as mean ± standard deviation (SD) with median in parentheses.
Kruskal–Wallis test applied.

Table 2: Spearman's correlation between hematological parameters and severity of preeclampsia

Parameter	r-value (Spearman's)	p-value	Interpretation
Platelet Count ($\times10^5$/mm^3)	-0.679***	<0.001*	Strong negative correlation
Mean Platelet Volume (MPV) (fL)	0.573***	<0.001*	Moderate positive correlation

Spearman's correlation coefficient (r) used for analysis.
***p < 0.001, statistically highly significant.*

Table 3: ROC curve analysis of platelet count and mean platelet volume (MPV) for prediction of severe preeclampsia

Parameter	Cut-off	Sensitivity (%)	Specificity (%)	AUC (95% CI)
Platelet Count ($\times10^5$/mm^3)	1.86	83.8	83.3	0.88 (0.83–0.93)
MPV (fL)	11.10	82.4	74.0	0.80 (0.74–0.86)

ROC: Receiver operating characteristic; AUC: Area under the curve; CI: Confidence interval. Cut-off values derived using Youden index.

and more reactive platelets from the bone marrow.

These findings are consistent with previous studies of Salvi et al. (2022) [26] ($p < 0.001$), Tesfay et al. (2019) [27] ($p < 0.05$) and Dadhich et al. (2012) [28], all of whom reported similar trends. Walle et al. (2022) [29] and Sachan et al. (2021) [30] also highlighted MPV as a significant diagnostic marker. However, Mandala et al. (2024) [31] did not find a statistically significant difference in platelet indices ($p > 0.05$) and Umezuluike et al. (2021) [17] also reported non-significant differences between mild and severe groups, indicating variability across studies.

In contrast to studies where MPV showed superior predictive ability, the present study found that platelet count had better diagnostic performance than MPV, as reflected by a higher AUC. This difference may be due to variations in population characteristics, disease severity distribution and methodological differences.

These findings support the use of platelet indices as simple screening tools in routine antenatal care, especially in low-source settings.

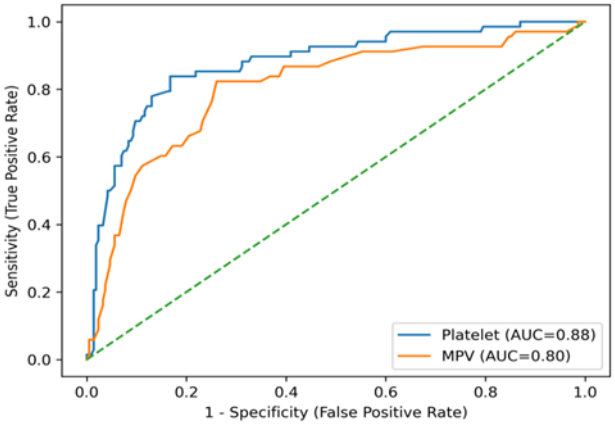


Figure 1: Receiver operating characteristic (ROC) curves of platelet count and mean platelet volume (MPV) for prediction of severe preeclampsia.

Strengths and limitations:

The major strength of this study lies in the use of simple, routinely available hematological parameters and the application of ROC analysis to derive clinically useful cut-off values. All laboratory investigations were performed using standardized and validated methods with automated analyzers, ensuring accuracy,

consistency and reproducibility of results. Additionally, the study design incorporated appropriate statistical analysis, including non-parametric tests and correlation analysis, which strengthened the validity of associations observed.

Despite its strengths, the present study has certain limitations. Being a cross-sectional study, it does not allow for establishing a causal relationship between the investigated parameters and the progression of preeclampsia. The study was conducted at a single tertiary care centre, which may limit the generalizability of the findings to the broader population. Additionally, the use of a non-probability convenience sampling technique may introduce selection bias. The study did not include longitudinal follow-up, which could have provided better insight into temporal changes in parameters and their predictive value for outcomes.

Conclusion

Platelet indices, particularly platelet count, are routinely available, cost-effective markers for assessing severity of preeclampsia and can aid in early identification of high-risk patients, particularly in resource-limited settings and facilitate timely intervention.

Acknowledgements: Nil

Declarations: NIL

Conflict of Interest: None

Funding: None

Ethical approval: Institutional Ethics Committee approval. (Approval No: EC/BMHR/ 2024/ 186 dated 30-08-2024).

References:

1.**Cunningham, F. G., Spong, C. Y., Casey, B. M., & Sheffield, J. S.** (2014). *Williams obstetrics* (24th ed.). McGraw Hill Education.

2.**American College of Obstetricians and Gynecologists**. (2020). Gestational hypertension and preeclampsia: ACOG practice bulletin No. 222. *Obstetrics & Gynecology, 135*(6), e237–e260.

3.**Jindal, M., Jindal, D., Naik, V., Sahasrabhojanee, M., & Pednekar, G.** (2021). Epidemiology and fetomaternal outcomes in cases of imminent eclampsia and eclampsia: A retrospective study. *Indian Journal of Obstetrics and Gynecology Research, 8*(1), 39–48.

4.**Kang, A., & Struben, H.** (2008). Pre-eclampsia screening in the first and second trimester. *Therapeutische Umschau, 65*(11), 663–666.

5.**Magee, L. A., Brown, M. A., Hall, D. R., Gupte, S., Hennessy, A., Karumanchi, S. A., et al.** (2022). The 2021 international society for the study of hypertension in pregnancy classification, diagnosis & management recommendations. *Pregnancy Hypertension, 27*, 148–169.

6.**Hurrell, A., Duhig, K., Vandermolen, B., & Shennan, A. H.** (2020). Recent advances in the diagnosis and management of pre-eclampsia. *Faculty Reviews, 9*, 10.

7.**Lambert, G., Brichant, J. F., Hartstein, G., Bonhomme, V., & Dewandre, P. Y.** (2014). Preeclampsia: An update. *Acta Anaesthesiologica Belgica, 65*(4), 137–149.

8.**Than, N. G., Romero, R., Györffy, D., Posta, M., Bhatti, G., Done, B., et al**. (2022). Molecular subclasses of preeclampsia characterized by longitudinal maternal proteomics study. *Journal of Perinatal Medicine, 51*(1), 51–68.

9.**Walle, M., Getu, F., Gelaw, Y., & Getaneh, Z.** (2022). Diagnostic value of hepatic and renal biochemical tests for detection of preeclampsia. *International Journal of General Medicine, 15*, 7761–7771.

10.**Bisson, C., Dautel, S., Patel, E., Suresh, S., Dauer, P., & Rana, S.** (2023). Preeclampsia pathophysiology and adverse outcomes. *Frontiers in Medicine, 10*, 1144170.

11.**Ridder, A., Giorgione, V., Khalil, A., & Thilaganathan, B**. (2019). Preeclampsia and uterine artery blood flow. *International Journal of Molecular Sciences, 20*(13), 3263.

12**,Phipps, E., Prasanna, D., Brima, W., & Jim, B.** (2016). Preeclampsia: Updates in pathogenesis and guidelines. *Clinical Journal of the American Society of Nephrology, 11*(6), 1102–1113.

13.**Mannaerts, D., Heyvaert, S., De Cordt, C., Macken, C., Loos, C., & Jacquemyn, Y.** (2019). Predictive parameters for preeclampsia. *Journal of Maternal-Fetal & Neonatal Medicine, 32*(9), 1412–1419.

14,**LaMarca, B., Cornelius, D. C., Harmon, A. C., Amaral, L. M., Cunningham, M. W., Faulkner, J. L., & Wallace, K.** (2016). Immune mechanisms in hypertension during preeclampsia. *American Journal of Physiology-Regulatory, Integrative and Comparative Physiology, 311*(1), R1–R9.

15.**Redman, C. W., & Sargent, I. L.** (2009). Placental stress and pre-eclampsia. *Placenta, 30*(Suppl A), S38–S42.

16.**Ives, C., Sinkey, R., Rajapreyar, I., et al**. (2020). Preeclampsia—Pathophysiology and clinical presentations. *Journal of the American College of Cardiology, 76*(14), 1690–1702.

17.**Umezuluike, B. S., Anikwe, C. C., Nnachi, O. C., Iwe, B. C. A., Ifemelumma, C. C., & Dimejesi, I. B. O.** (2021). Platelet parameters and outcomes in severe preeclampsia. *Heliyon, 7*(12), e08484.

18.**Mohammed, B. A. B. B.** (2017). Platelet indices among Sudanese pregnant women. *International Journal of Sciences, 6*, 71–75.

19.**Ciobanu, A. M., Colibaba, S., Cimpoca, B., Peltecu, G., & Panaitescu, A. M.** (2016). Thrombocytopenia in pregnancy. *Medica – Journal of Clinical Medicine, 11*(5), 55–60.

20.**Gernsheimer, T., James, A. H., & Stasi, R. (**2013). Thrombocytopenia in pregnancy. *Blood, 121*, 38–47.

21.**Freitas, L. G., Alpoim, P. N., Komatsuzaki, F., Carvalho, M. D., & Dusse, L. M.** (2013). Platelet indices in preeclampsia. *Hematology, 18*(6), 360–364.

22.**Bowersox, N. A., Talavera, F., Ramus, R. M., & Vera, E. V.** (2016). Thrombocytopenia in pregnancy. Medscape. Retrieved from https://emedicine.medscape.com/article/27867-overview#a1

23.**Annam, V., Srinivasa, K., Yatnatti, S., & Suresh, R.** (2011). Evaluation of platelet indices in preeclampsia and eclampsia.

24.**Mohapatra, S., Pradhan, B. B., Satpathy, U. K., Mohanty, A**., **& Pattnaik, J. R**. (2007). Platelet estimation and prognostic value. *Indian Journal of Physiology and Pharmacology, 51*(2), 160–164.

25.**Bellos, I., Fitrou, G., Pergialiotis, V., & Daskalakis, G.** (2018). Mean platelet volume in pre-eclampsia. *Hypertension in Pregnancy, 37*(4), 174–180.

26.**Salvi, P., Gaikwad, V., & Ali, R**. (2022). Platelet indices in preeclamptic patients. *New Indian Journal of Obstetrics and Gynecology, 9*(1), 69–73.

27.**Tesfay, F., Negash, M., Alemu, J., Yahya, M., Teklu, G., Yibrah, M., et al.** (2019). Platelet parameters in prediction of preeclampsia. *PLoS One, 14*(11), e0225536.

28**.Dadhich, S., Agrawal, S., Soni, M., Choudhary, R., Jain, R., Sharma, S., & Saini, S. L**. (2012). Predictive value of platelet indices. *Journal of South Asian Federation of Obstetrics and Gynaecology, 4*(1), 17–21.

29.**Walle, M., Asrie, F., Gelaw, Y., & Getaneh, Z.** (2022). Role of platelet parameters in diagnosis of preeclampsia. *Journal of Clinical Laboratory Analysis, 36*(4), e24305.

30.**Sachan, R., Patel, M. L., Vandana, Sachan, P., & Shyam, R.** (2021). Platelet indices and prediction of preeclampsia. *Journal of Family Medicine and Primary Care, 10*(2), 838–843.

Mandala, D., Puligujja, P., & Kumar, K. H. (2024). Haematological parameters in preeclampsia. *Journal of Academic Medicine and Pharmacy, 7*(2), 1186–1190

Measurement of Radiological Cardiothoracic Ratio: Acentric Study

Dheeraj Kumar, Abhishek Kumar Chaudhary

Abstract

Background: The Cardiothoracic Ratio (CTR) is a critical diagnostic parameter derived from chest X-rays, used to assess cardiac enlargement or cardiomegaly. While CTR is influenced by factors such as age and gender, population-specific studies are limited, particularly in the Indian context. This study explores the relationship between CTR, age, and gender using retrospective data from digital X-ray imaging.

Aim: The study aimed to evaluate the influence of age and gender on CTR and establish baseline CTR values for the studied population. It also sought to determine whether age and gender significantly predict CTR using statistical analyses.

Materials and Methods: Data were collected from 26 patients using the Kiran Medical Systems Ultisys-40 digital X-ray machine. CTR was calculated as the ratio of the maximum heart diameter to the maximum thoracic diameter. Descriptive statistics, paired t-tests, correlation analysis, and linear regression were employed to analyze the data. The sample was stratified by gender and age groups (18-25, 26-35, and 36-75 years) to assess variations in CTR.

Results: Descriptive analysis showed slight variations in CTR across age groups and genders. Females aged 26-35 had the highest median CTR (0.45), while males aged 36-75 exhibited the highest average CTR (0.46). The paired t-test revealed no significant difference in mean CTR between genders (p-value = 0.666). Regression analysis indicated a weak relationship between age and CTR, with non-significant coefficients for both males (p-value = 0.980) and females (p-value = 0.149). The models explained minimal variance (R^2 = 0.000025 for males and 0.0848 for females).

Conclusion: Age and gender do not significantly influence CTR in the studied population. The findings provide baseline CTR values for the Indian population, contributing to improved diagnostic accuracy. However, the weak predictive power of the models suggests the need for further research incorporating additional variables such as BMI and health conditions.

JK-Practitioner2026;31(2):36-40.

Introduction

Advances in medical imaging technology have revolutionized the field of diagnostic medicine. X-ray imaging has evolved as one of the most important imaging modalities used for the evaluation of structural anatomy and assessment of pathological changes.[1] The most common measurement obtained from chest X-rays PA View is the cardiothoracic ratio, which is defined as the maximal horizontal diameter of the heart divided by the maximal horizontal diameter of the thorax.[2] In clinical practice, CTR values help in the diagnosis and follow-up of cardiac enlargement or cardiomegaly, conditions typically linked with cardiovascular diseases. The normal range of CTR in adults is 0.42 to 0.50.[3] If the CTR is above 0.50, it is termed cardiomegaly, while values below 0.42 are indicative of small heart syndrome.[4] Various studies have found that CTR differs across groups, and such differences appear to be due in part to age, gender, and general health condition. Therefore, knowledge about the distribution of CTR values within a particular group is paramount for establishing baseline values as well as finding variations, which may indicate some kind of disease or health condition. In the case of CTR and its relationship to age and gender, we are interested in investigating if there is any association as well as how these aspects may influence cardiac and thoracic dimensions.

In addition, we perform a variety of statistical tests, for example, mean and median calculation, p-value test, t-tests, correlation coefficients, and regression analyses for further quantification of relationships

Author Affiliations

Dheeraj Kumar , Assistant Professor;
Abhishek Kumar Chaudhary, BMRIT Student.
School of Health Sciences, Chhatrapati Shahu Ji Maharaj University, Kanpur, Uttar-Pradesh, India 208024

Correspondence

Dheeraj Kumar
Assistant Professor
MRIT, Ph.D. (Radiology and Imaging) Pursing
School of Health Sciences, Chhatrapati Shahu Ji Maharaj University, Kanpur, Uttar-Pradesh, India 208024
Email Id: dheeraj199494@gmail.com

Indexed

EMBASE, SCOPUS, IndMED , ESBCO, Google Scholar besides other national and international databases.

Cite this article as

Kumar D, Chaudhary AK. Measurement of Radiological Cardiothoracic Ratio: Acentric Study.JK Pract2026;31(2):36-40.

Full length article available at **jkpractitioner.com** one month after publication

Keywords

Cardiothoracic Ratio (CTR), age, gender, digital X-ray, regression analysis.

between age, gender, and CTR[5] Such analyses are fundamental in medical research, because they provide a quantitative basis for determining normal ranges and potential risk factors. This study is important for several reasons. First, the establishment of baseline CTR values that are specific to the population studied can provide useful references for healthcare providers who are using similar equipment. Second, understanding the impact age and gender may have over a CTR value further increases the clinician's capacity to better interpret them, especially in regions where advanced healthcare facilities and testing apparatuses are scarce or difficult to access.

Materials and methods

Study Design: This was a retrospective, cross-sectional study conducted to observe CTR in a sample population, hence, based on digital radiographs. The study aims at measuring the relationship between CTR, age, and gender. Data collection was performed from a single digital X-ray machine: Ultisys-40 model from Kiran Medical Systems (under Trivitron Healthcare Pvt. Ltd.) equipment's ID: G-XR-208243.

Methodology: Data for the current paper were sourced from X ray reports over the time span of August 2024 to December 2024. All the radiographs were routine clinical reports. Ethical approval had taken place through the IRB, HEC Reference No. 2025 Jan 022, and patient data had been fully anonymized so as not to release any information that may be perceived to infringe on patients' rights for privacy concerns.

Eligibility criteria: The inclusion criteria for the study encompassed patients aged over 18 years, including both males and females, who had complete and reliable records of cardiothoracic ratio (CTR) measurements. In contrast, the exclusion criteria ruled out cases lacking CTR details and those with underlying cardiopulmonary diseases that could influence the accuracy of CTR measurement.

Cardiothoracic Ratio (CTR): The measurement of CTR was performed from the digital radiographs and recorded in mm[6].

Cardiothoracic Ratio (CTR) Measurement Formula:

$$CTR = \frac{\text{Maximum Heart Diameter}}{\text{Maximum Thoracic Diameter}}$$

The CTR was measured using the X-ray images from the Kiran Medical Systems Ultisys-40 digital X-ray machine software.

T-test: T-test is independent sample; it tested to see the difference in the mean CTR between the subjects of each gender. At a p-value of 0.05, with a two tailed test that was required to find whether there occurred any difference to be reckoned with that was apparent between the two genders concerned.

Correlation Analysis: To calculate the relationship of age with CTR among all participants as well as in each group of gender, Pearson's correlation coefficient was computed showing in table 2. It provides the linear association of age with CTR. The correlation analysis found a statistically significant positive correlation between the male's age and female's CTR ($r = 0.813$, $p < 0.01$) and between the male's age and female's age ($r = 0.571$, $p < 0.01$). A moderate, non-significant positive correlation was identified between female's age and female's CTR ($r = 0.291$, $p = 0.149$). There was

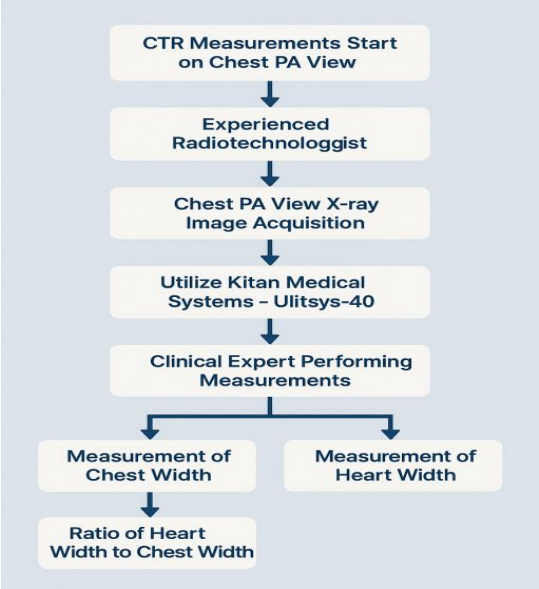


Figure 2 Showing CTR measurement flow chart

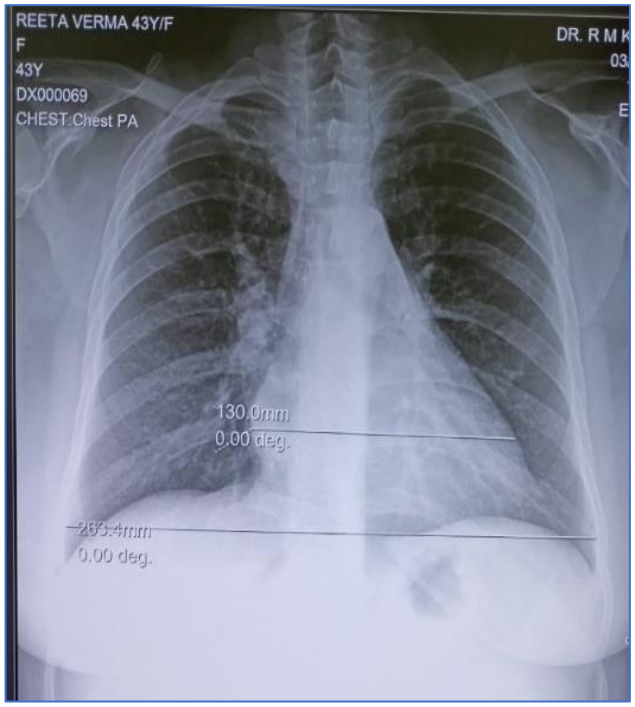


Figure 1 Chest PA View showing the CTR measurement Protocol by the digital radiography software.

Table1: Cardiothoracic Ratio (CTR) Distribution by Age Group and GenderMean, median, and standard deviation of CTR values across different age groups for male and female participants.

Group		
Gender	**Male**	**Female**
18-25 years:		
Mean	0.433	0.419
Standard Deviation	0.052	0.042
26-35 years:		
Mean	0.436	0.413
Standard Deviation	0.058	0.005
36-75 years:		
Mean	0.42	0.463
Standard Deviation	0.035	0.075

Table 1 Pearson Correlation Between Age and CTR in Male and Female Subjects

Correlations		AGE of Female	CTR of Female	AGE of Male	CTR of male
AGE of Female	Pearson Correlation	1	.291	.571**	-.067
	Sig. (2-tailed)		.149	.002	.743
CTR of Female	Pearson Correlation	.291	1	.813**	.113
	Sig. (2-tailed)	.149		.000	.583
	N	26	26	26	26
AGE of Male	Pearson Correlation	.571**	.813**	1	-.005
	Sig. (2-tailed)	.002	.000		.980
CTR of male	Pearson Correlation	-.067	.113	-.005	1
	Sig. (2-tailed)	.743	.583	.980	
	N	26	26	26	26

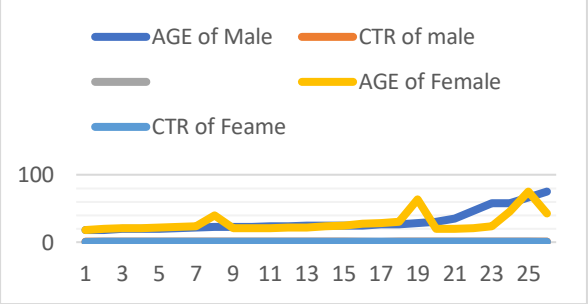
**. Correlation is significant at the 0.01 level (2-tailed).

no high correlation between CTR of male and any other variable, having very low correlation values with age and CTR of females or age of males ($p > 0.05$).

Stratified analysis

Data are stratified by gender and by three age groups (18-25, 26-35, 36-75) for the calculation of average, median, and total CTR within each age group, so that some insight might be gained as to CTR distribution by defined demographic segments.

Figure 3 Comparison of Female and Male CTR *With Age*.



Results

Descriptive Statistics

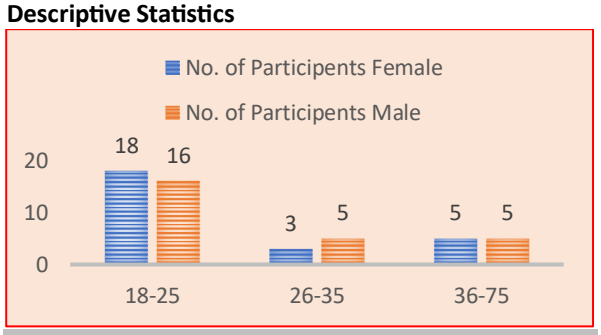


Figure 4 Bar graph showing gender wise participants with age range

The descriptive analysis of the Cardiothoracic Ratio (CTR) for both male and female participants across different age groups (18-25, 26-35, and 36-75) is summarized in bar graph below.

Overall, females aged 26-36 exhibited the highest median CTR (0.45), while males aged 36-75 exhibited the highest average CTR (0.46). The CTR values for both genders were similar in the 18-25 age range, with an average CTR of 0.43 for females and 0.41 for males. The bar graph above illustrates the number distribution for CTR for both genders, which were classified under Small Heart Syndrome, Normal, and Cardiomegaly. For females, there were 13 cases classified under Small Heart Syndrome, 12 under Normal, and 1 cardiomegaly. For the males, 11 were classified under Small Heart Syndrome, 14 normal, and 1 cardiomegaly. A similar pattern emerged between genders. Small Heart Syndrome and Normal predominated, and the rest included Cardiomegaly by only 1 case for both. This shows that most subjects had CTR values within or below the normal range, and cases of cardiac enlargement were rare.

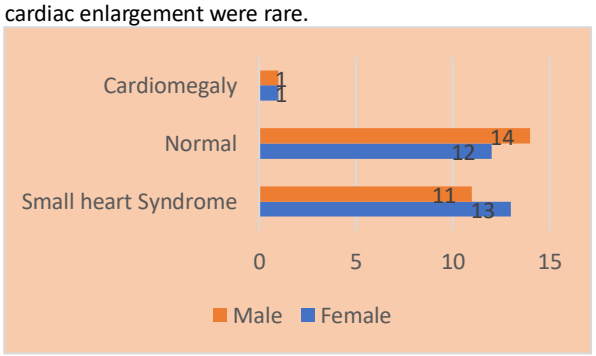


Figure 5 Cardiothoracic Ratio (CTR) for both male and female participants across different age groups

The paired two-sample t-test was conducted to compare the mean Cardiothoracic ratio (CTR) of females and males. The results show that the mean CTR for females is 0.4265, while the mean CTR for

males is 0.4319, indicating a slight difference between the two groups. The variances for females and males are 0.0021 and 0.0023, respectively, suggesting similar variability in CTR for both groups. The Pearson correlation coefficient is 0.113, indicating a weak positive relationship between the paired observations. The t-statistic is -0.437, and the p-value for the two-tailed test is 0.666, which is much greater than the common significance level of 0.05. Therefore, the mean CTR of females is not significantly different from that of males. Moreover, t-statistic is less than the critical t-value of 2.060 (two-tailed). As such, the conclusion should be that there is not any significant difference between means. Therefore, we fail to reject the null hypothesis that there is no difference in CTR between females and males. Variance is the measure that quantifies the spread or dispersion of the CTR values within a group. In this case, variances are pretty low; that is, the measurement of CTR within both groups is pretty tightly scattered around the respective means.

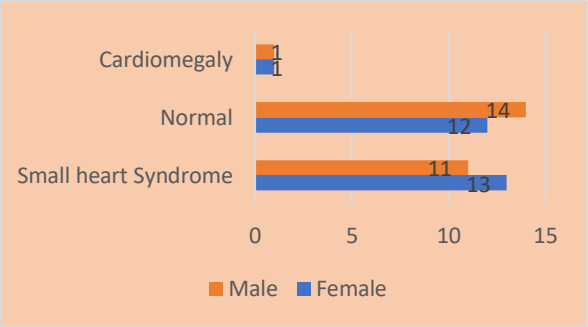


Figure 6 Bar graph showing the CTR of Participants, Cardiomegaly, normal, and small heart syndrome.

Discussion

The study explored the relationship between demographic factors (age and gender) and Cardiothoracic Ratio (CTR) using retrospective digital X-ray data. Establishing baseline CTR values for the Indian population is a key contribution, aiding in improved diagnostic accuracy[4]. The findings revealed that females aged 26–35 years had the highest median CTR (0.45), while males aged 36–75 years had the highest average CTR (0.46), aligning with global standards. However, statistical analysis (paired t-test, p = 0.666) showed no significant CTR difference between genders, and regression analysis confirmed that age was not a significant predictor for either gender (p = 0.980 for males, p = 0.149 for females). These results indicate that CTR remains consistent across age and gender groups, reinforcing its reliability as a diagnostic tool without the need for demographic adjustments.[7] This is particularly valuable in resource-limited settings where advanced diagnostic tools are scarce. The study also identified a low prevalence of cardiomegaly, suggesting a relatively healthy sample or exclusion of individuals with cardiopulmonary diseases. While CTR is useful for identifying individuals at cardiovascular risk, further research with a larger and more diverse sample is necessary for broader applicability.

Limitations include the small sample size (26 participants), single geographic region, and reliance on one imaging system (Kiran Medical Systems Ultisys-40). Future studies should incorporate larger samples, multiple imaging systems, and additional factors such as BMI, lifestyle, and comorbidities.

Conclusion

This study established the baseline Cardiothoracic Ratio values for the Indian population and demonstrated that age and gender have minimal influence on CTR. The findings reinforce CTR as a stable and reliable diagnostic parameter particularly in resource-limited settings where standardized reference values are essential. The study revealed slight variations in CTR across age and gender groups with Females aged 26–35 years showing the highest median CTR (0.45) and Males aged 36–75 years the highest average C However statistical analyses, including t-tests and regression models confirmed that neither gender nor age significantly affect CTR. These findings suggest that clinicians can use a standard CTR reference range without demographic adjustments and simplify cardiovascular assessments. While the study provides valuable insights its limitations, including a small sample size and single geographic focus, highlight the need for further research. Future studies should integrate more diverse populations and additional variables such as BMI, lifestyle and comorbidities to enhance the understanding of CTR's clinical utility.

By contributing to the growing body of literature on CTR in cardiovascular diagnosis, this study paves the way for more comprehensive research. Expanding the scope of analysis through advanced statistical modeling and multi-center studies could improve CTR diagnostic accuracy further improving patient outcomes in cardiovascular disease management.

Conflict of Interest: The authors declare no conflict of interest related to this study.

Acknowledgment: The authors express gratitude to School of Health Sciences for providing access to the digital X-ray imaging data. Special thanks to the radiology department staff for their support in data collection

References

1. **E. Bercovich and M. C. Javitt,** "Medical Imaging: From Roentgen to the Digital Revolution, and Beyond," Rambam Maimonides Med J, vol. 9, no. 4, p. e0034, Oct. 2018, doi: 10.5041/RMMJ.10355.

2. **C. Saul Danzer,** "DANZER : THE CARDIOTHORACIC RATIO 513 Absence of any characteristic blood-pressure phenomena. THE CARDIOTHORACIC RATIO : AN INDEX OF CARDIAC ENLARGEMENT."

3. **K. Truszkiewicz, R. Poręba, and P. Gać**, "Radiological cardiothoracic ratio in evidence-based medicine," May 01, 2021, MDPI. doi: 10.3390/jcm10092016.

4. **D. Bell and Y. Weerakkody**, "Differential diagnosis for a small cardiothoracic ratio," in Radiopaedia.org, Radiopaedia.org, 2010. doi: 10.53347/rID-9884.

5. **J.-B. du Prel, B. Röhrig, G. Hommel, and M. Blettner**, "Choosing Statistical Tests," Dtsch Arztebl Int, May 2010, doi: 10.3238/arztebl.2010.0343.

6. **F. S. Torres, D. A. Eifer, F. Sanchez Tijmes, E. T. Nguyen, and K. Hanneman,** "Diagnostic performance of chest radiography measurements for the assessment of cardiac chamber enlargement," Can Med Assoc J, vol. 193, no. 44, pp. E1683–E1692, Nov. 2021, doi: 10.1503/cmaj.210083.

7. **J. Irvin et al.,** "CheXpert: A large chest radiograph dataset with uncertainty labels and expert comparison," 33rd AAAI Conference on Artificial Intelligence, AAAI 2019, 31st Innovative Applications of Artificial Intelligence Conference, IAAI 2019 and the 9th AAAI Symposium on Educational Advances in Artificial Intelligence, EAAI 2019, pp. 590–597, 2019, doi: 10.1609/aaai.v33i01.3301590.

Prevalence of 'Serologic Weak D' among blood donors at a tertiary care hospital: An Observational study from North India.

Rubiya Ryhan , Hilal Ahmad Tali, Saima Muzaffer , Rumana H Makhdoomi, Kaneez Fatima, Shagufta Roohi

Abstract

Background: Weak D refers to reduced expression of D antigen on the red blood cells that require an extended testing to get detected. The most important risk with this phenotype is alloimmunization among the recipients.

Materials and Methods: It is a hospital based observational study conducted over a period of 3 years. During this period all blood donor samples were tested for ABO and Rh D typing by routine serologic methods. Samples which were initially negative were further tested for weak D antigen using IgG Anti-D in the indirect antiglobulin test phase.

Results: A total of 37334 blood donor samples were analysed for ABO & Rh grouping. Out of the total samples 34037 (91.2 %) were Rh-D positive & 3297 (8.8 %) were Rh-D negative. Of all the test samples, 09/37334 (0.03 %) turned out to be weak D-antigen positive. And of all the Rh-D negative samples 09 /3297 (0.3 %) were weak D- antigen positive.

Conclusion: This study stresses the need to identify individuals with weak D and other D variants and to inform them about their status as donors and/ or recipients of blood or blood products

JK-Practitioner2026;31(2):41-44.

Introduction

Rh system is the 2nd most immunogenic and complex blood group system after ABO with more than 58 antigens included in it. [1] Rh D antigen is considered the major antigen of this blood group system. [2] The Rh D antigen is encoded by the RH D gene which is highly polymorphic and any mutation in this gene may lead to different phenotypic expressions of the D antigen known as D-Variants (including Weak D, Partial D, and Del, etc). [3,4]

The term "Weak D" Phenotype was 1st described by Stratton in 1946 as "Du". [5,6] It corresponds to a quantitative decrease in the expression of normal D antigens on RBCs. [7] As a consequence of decreased number of D antigens, weak or no agglutination reaction is demonstrated by these RBCs with the anti D reagent by conventional tube technique, [8,9] and require an extended testing with indirect antiglobulin test or molecular tests to get detected. So any test sample which may be labeled as Rh D negative by routine serologic methods may be found Rh D positive on further confirmation.

The clinical significance of detecting weak D and other D variants of the Rh (D) system lies in the fact that even lessthan 0.5 ml of Rh D antigen exposure in Rh negative recipient can induce antibody response. [8] So The most important risk with this phenotype is alloimmunization among the recipients. Individuals with weak D phenotype are typed depending upon whether the person is donor or the recipient; the recipients with weak D are considered D negative and must be transfused with D negative blood and the donors are considered as D positive. Mothers with weak D fetus must receive Rh immunoprophylaxis as passage of weak D red cells from fetus to mother may result in sensitization. [1]

This study aims to estimate prevalence of weak D antigen among D-negative blood donors and to provide data for improving blood transfusion protocols, especially for recipients prone to alloimmunization.

Materials and Methods

Study Design: This is an observational, hospital-based study

Study Population: The study was conducted in the Department of Blood Transfusion and Immunohematology, SKIMS Soura, Srinagar,

Author Affiliations

Rubiya Ryhan, Associate Professor, **Saima Muzaffer ,** Senior Resident, **Rumana H Makhdoomi** , Professor: Department of Blood Transfusion and Immunohematology; **Kaneez Fatima,** Department of Radiotherapy **:Shagufta Roohi ,**Department of Microbiology: SKIMS Soura -Srinagar: **Hilal Ahmad Tali,** Consultant - Department of Gastroenterology & Hepatology, GMC -Srinagar

Correspondence

Dr.Rubiya Ryhan
Associate Professor Department of Blood Transfusion and Immunohematology SKIMS Soura - Srinagar
Email: rubiyaryhan38@gmail.com

Indexed

EMBASE, SCOPUS, IndMED , ESBCO, Google Scholar besides other national and international databases.

Cite this article as

Ryhan R , Tali HA , Muzaffer S, Makhdoomi RH, Fatima K, Roohi S. Prevalence of 'Serologic Weak D' among blood donors at a tertiary care hospital: An Observational study from North India.JK Pract2026;31(2):41-44.

Full length article available at **jkpractitioner.com** one month after publication

Keywords

Rh D-positive, Rh D- negative, Weak D antigen.

over a three-year period from March 2021 to April 2024. Blood donors who met the criteria for donation and were deemed fit during this period were included in the study. Donors who were deferred or found unfit for donation were excluded.

Sampling Procedure: The study encompassed all blood donor samples collected during the specified period, with no additional sampling procedure required.

Sample Size: The sample size was determined by the total number of donors (n = 37,334) who donated blood during this specified period.

Human Subjects Protection: Informed consent was obtained from all blood donors prior to blood donation and sample collection, and proper ethical approval for the study was secured from the institutional ethics committee.

Blood Group Typing: A total of 37334 blood donor samples were tested routinely for ABO and Rh blood groups. Rh blood group typing was done by conventional tube method using two anti D reagents; monoclonal IgM anti-D (Tulip Diagnostics Private Limited, Verna, Goa, India) and polyclonal IgM + IgG anti-D blend (Tulip Diagnostics Private Limited, Verna, Goa, India). Blood samples that were negative for Rh D by conventional tube technique (CCT) were further tested by Indirect Antiglobulin Test (IAT) and Gel Card System (GCS) (ID Diaclon Anti-D, ID Microtyping System) for weak D antigen.

A 5% suspension of the cells to be tested was made. Equal volumes (2 drops) of each of anti-D serum and 5% red cell suspension were placed in a clean glass tube, mixed well, and incubated at RT for 45- 60 minutes (as advised by the manufacturer)

(Sedimentation method) or the tube was incubated at 37^{O}C for 10-15 minutes and centrifuged at 1000× g for one minute (spin tube method). The tube was gently resuspended and the cell button was observed for agglutination. If the test red cells were agglutinated, the conventional tube test (CCT) result was recorded as D antigen positive. If the test red cells were not agglutinated, the test was recorded as D antigen negative. Further, the D negative cells were washed 3-4 times with large volumes of normal saline. After the final wash, the saline was decanted and two drops of antihuman globulin serum was added and the tube centrifuged at 1000× g for 1 minute. The cell button was resuspended and examined for agglutination. All negative results were confirmed by microscope. The samples showing agglutination after addition of Antihuman globulin (AHG) serum (J. Mitra & Co. Pvt. Ltd) were considered weak D positive. Parallel positive and negative controls were set up to rule out any DAT Positive sample. For testing of weak D by gel card method, 1% red cell suspension was prepared in LISS. 50 µL of 1% RBC suspension was dispensed in microtube of IgG card followed by the addition of 50 µL of monoclonal anti-D IgG (Diamed ID Microtyping System). This was followed by incubation at 37^{o}C for 15 minutes and centrifugation. All the results were read and interpreted by two observers independently

Statistical Analysis; Descriptive statistics was performed for categorical variables in the form of numbers (n) and percentages (%), using Microsoft Excel and Statistical Package for Social Sciences (SPSS) version 29.

Results:

A total of 37334 donor blood samples were analysed for ABO & Rh blood grouping. Out of these total 37334 samples 34037 (91.2 %) were Rh-D positive & 3292 (8.8 %) were Rh-D negative. Among the all total 37334 samples 8850 (**23.7 %**) were of group A {8129 (28.1%) A positive and 721 (1.9 %) A negative], 12463 (**33.3 %**) were of group B {11433 (30.6 %) B positive and 1030 (2.7 %) B negative}, 13243 (**35.5 %**) of group O {11866 (31.8 %) O positive and 1377 (3.7 %) O negative} & 2778 (**7.44 %**) were of AB group {2609 (6.9 %) AB positive and 7.7 (0.5 %) AB negative} (Table 1)

Table 1: Showing Blood Group Distribution and Weak D Positivity Among Blood Donors

Blood Group	Rh Positives n (%)	Rh Negatives n (%)	Total (%) n (%)	Weak D positives n (%)
A	8129 (21.8 %)	721 (1.9 %)	8850 (23.7 %)	2 (0.01 %)
B	11433(30.6 %)	1030 (2.7 %)	12463(33.3 %)	3(0.01 %)
O	11866 (31.8 %)	1377 (3.7 %)	13243(35.5%)	4 (0.01%)
AB	2609(6.9%)	169 (0.5%)	2778(7.44%)	00
Total	34037(91.2%)	3297(8.8%)	37334(100 %)	9 (0.03 %)

All the Rh-D negative (3297) samples were subjected to weak D testing. Of all the test samples 09/37334 (0.03 %) turned out to be weak D positive. **Table 1**. And of all the Rh-D negative samples 09 /3297 (0.3 %) were weak D positive.

Table 2: Showing Frequency of Weak D Positivity Among Rh Negative Blood Donors

Blood Group	Number	Weak D positives	%
A Negative	721	02	0.3 %
B Negative	1030	03	0.3 %
O Negative	1377	04	0.3 %
AB Negative	169	00	00 %
Total	3297	09	0.3 %

Discussion

For safety of blood transfusion, determination of Serologic Weak D (and other D variants) is of utmost importance. In literature more than 100 variant of D phenotypes have been reported. [3] The incidence of weak D (& other D variants) varies worldwide. Although various authors have given the prevalence of serologic weak D (& other D variants) in their populations, the comparative analysis becomes difficult due to the lack of set standards for performing the tests, type of reagents used (monoclonal / polyclonal, single / blended), objective and subjective variation in interpretation of test results. [10] Further, it has been adequately documented that D epitopes distribution differs with different geographic locales & ethnicities of the populations. [11] Kulkarni et al highlighted this fact by testing 42 confirmed D variants with 7 commercially available anti-D reagents in India. [12]

In our study, the incidence of weak D comprised 0.03 % of all study samples & 0.3 % of all Rh - negative samples. Comparable results were obtained by Makroo RN et al, 2010, [8] Devi G et al 2016 [13] and Srivastava RK et al (2018). [14] Agarwal N et al 2013 [15] observed a lower prevalence of weak D in their study (0.005 % of total donors and 0.09 % of Rh negative donors). However, higher results were obtained by Deepthi Krishna G et al 2015 [1] (0.6 % of total individuals and 1.04 % of Rh negative individuals screened), Lamba HS et al 2017, [16] Rehmani MT et al 2017, [17] and Brar RK et al (2020). [18] Table 3. The occurrence of weak D antigen is about 0.23 % to 0.5 % in Europe and 3 % in USA, [19,20] while in India, the frequency of weak D antigen turned out to be 0.3 to 0.5 %. [21,22]

This study highlights the prevalence of serologic weak D in our blood donor population who are prototype representatives of this region. It also stresses the need to identify individuals with variant D (rather than weak D or partial D) and to inform them about their status as donors and recipients of blood and blood products. The current opinion is that weak D & other D variant subjects should be treated as D positive as donors to prevent alloimmunization if accidently transfused to D negative recipients. Partial D recipients should be considered as D negative, else they will form antibodies against the missing epitopes of the D antigen when transfused with D positive blood. [23] Alloimmunization of D negatives can occur with weak D, while in childbearing age can be disastrous and can lead to hemolytic disease of newborn. Newborns of D negative mother should be tested for weak D and Rh

Table 3: Comparison of prevalence of Weak-D in blood donors from different regions of the world

Sr. no.	Author	Total Study participants/ donors	Weak D Prevalence	
			% among Rh D negative donors	% among total donors
1.	Makroo RN et al, 2010 [8]	184072	0.12	0.01
2.	Devi G et al, 2016 [13]	7019	0.43	0.01
3.	Srivastava RK et al, 2018 [14]	1,66,338	0.35	0.004
4.	Agarwal N et al, 2013 [15]	58,614	0.09	0.005
5.	Deepthi K G et al, 2015 [1]	46654	1.04	0.0.06
6.	Lamba HS et al, 2017 [16]	13043	0.94	0.06
7.	Rehmani MT et al 2017 [17]	55874	0.98	0.08
8	Brar RK et al 2020 [18]	6415	1.51	0.07
9	Our Study	37334	0.3	0.03

immunoglobulin is recommended for mothers of weak D positive infants in order to prevent immunization. [23] The study, despite successfully highlighting the prevalence of serological weak D in the blood donor's population has certain limitations, as it doesn't distinguish between weak D & other D variants (like partial D & Del types) in our study.

Conclusion

The present study provides us the information regarding prevalence of serologic weak D antigen in our blood donor population. Not testing for the weak D antigen in the blood group may have devastating immunological and medical consequences especially in chronically transfused patients like thalassemia, sickle cell anemia, chronic renal failure and in pregnant women. Alloimmunization (with weak D) in such patients especially women in the child bearing age is catastrophic. It also stresses the need to identify individuals with other D variants (rather than weak D or partial D) and to inform them about their status as donors and recipients of blood and blood products.

References

1.**Deepthi KG, Sreedhar KV, Arun R, Jothibai DS**. A study on Rh incompatibility and frequency of weak D among blood donors and patients at a tertiary care referral teaching hospital in Tirupati, Andhra Pradesh. J Clin Sci Res 2015; 4:281-4.

2.**Westhoff MC.** The structure and function of Rh complex. Semin Hematol 2007;44(1):42–50.

3.**Wafi ME, Housse HE, Nourichafi N, Bouisk K, Benajiba M, Habti N.** Prevalence of weak D phenotype among D Negative C/E + blood donors in Morocco. Int. J Blood Transfus Immunohematol 2016; 6; 3- 7

4.**Subramaniyan R**. Prevalence of D variants in the Indian donor population. Hematol Transfus Cell Ther. 2019;41(2):190–3.

5.**Sandler S.G**. Serological weak D phenotypes: a review and guidance for interpreting the RhD blood type using the RHD genotype. *Br J Haematol.* 2017;179(1):10–19.

6.**Garratty G**. Do we need to be more concerned about weak D antigens? Transfusion. 2005; 45:1547-51.

7.**Wagner FF, Frohmajer A, Ladewig B, Eicher NI, Lonicer CB, Müller TH, et al**. Weak D alleles express distinct phenotypes. Blood 2000 Apr 15;95(8):2699–708.

8.**Makroo RN, Raina V, Chowdhry M, Bhatia A, Gupta R, Rosamma NL**. Weak D prevalence among Indian blood donors. Asian J Transfus Sci 2010;4(2):137–9.

9.**Sing A, Solanki A, Agarwal D, Chandra T**. Prevalence of serologic weak D in Rh D negative blood donors in India: Immunohematological problems & recommendations for donors. Panacea Journal of Medical Sciences. 2022; 12(3): 686–689.

10.**Mamatha M, Vani BS, Akarapu J, Enumula D.** A Study to Determine the Prevalence of Weak D in Donors and Patients at a Tertiary Care Teaching Hospital in Telangana J. Adv. Med. Med. Res., 2022 vol. 34, no. 24, pp. 17-21,

11.**Kulkarni SS, Gupte SC, Vasantha K, Mohanty D, Ghosh K.** Varied distribution of RhD epitopes in the Indian population. National Medical Journal of India. 2007 Jul 1;20(4):169.

12.**Kulkarni SS, Vasantha K, Gupte SC, Mohanty D, Ghosh K.** Potential of commercial anti-D reagents in the identification of partial D variants in Indian population. Indian J Med Res 2007; 125:641-4.

13.**Devi G, Singh UR, Garg A.** Detection of weak Rh D (Du) phenotype among blood donors. Indian Journal of Basic and Applied Medical Research. 2016: 5 (4):135-38.

14.**Srivastava RK, Prasad D, Halder D.** Study of Prevalence of Weak D Antigen (Du) Amongst Supposed Rh Negative Blood Donors in a Tertiary Care Hospital of Jharkhand. Int J Scientific Res. 2018;7(8):69–70.

15.Agarwal N, Agarwal A, Chandola I. Prevalence of weak D in northern hilly areas of Uttarakhand, India. Asian Journal of Transfusion Science, Vol. 7, No. 1, January-June, 2013, pp. 90-91.

16.Lamba HS, Kaur K, Kaur K, Vij AS. Prevalence of Weak D (Du) in Blood Donors in a Referral Teaching Hospital. Int J Med Dent Sci. 2017;6(2):1509–12.

17.Rahmani MT, Ahmad M, Saeed M, Hussain S, Hanif A, Rashid N. Deter mination of Weak "D" Antigen among Rhesus Negative Pakistani Blood Do nors. Ann Pak Inst Med Sci. 2016; 12(3):131-35.

18.Brar RK, Shaiji PS, Sehgal S. Testing for weak D Antigen: Spectrum and its applied role in rhesus-negative transfusions in Andaman and Nicobar Islands. Tzu Chi Med J. 2020;32(2):167–70.

19.**Race RR, Taylor GI, Capell DF, Mcfarlan MN.** Recognition of further common Rh genotypes in man. Nature 1944; 153:52-3.

20.**Trup J, Kornstand L**. Presence of anti-c in the serum of 42 women giving birth to c positive babies: serological and clinical findings. Acta Obstet Gynecol Scand 1977; 56: 185-8.

21.**Reid ME, Lomas FC, Olsson ML.** The Blood Group Antigen Facts Book, 3rd ed. New York: Elsevier Academic Press; 2012. P. 27-52.

22.**Ballas SK, Clark MR, Mohandas N, Colfer HF, Caswell MS, Bergren MO, et al.** Red cell membranes and cation deficiency in Rh Null Syndrome. Blood 1984; 63:1046-55.

23.**Manjula Jain, Ritu Kumari, Shivani Kushwaha, Sangeeta Pahuja, Meenu Pujani, Neha Sethi**. Frequency of variant D in Delhi, Indaia. Asian Journal of Transfusion Science, Vol. 8, No. 2, July-December, 2014, pp. 142-143.

Efficacy of *Salvia hispanica* (chia seeds) supplementation in promoting weight loss in obese individuals

Aamena Zaidi, Divya Singh

Abstract

Background

Excess fat accumulation that poses a health risk to people is known as obesity. The World Health Organization estimates that over 650 million individuals worldwide suffer from obesity. Risk factors of most chronic diseases, including diabetes, heart disease, metabolic disorders, osteoarthritis, and cancer, are increased by obesity. Because of the correlation between increased body mass index (BMI) and ovarian morphological abnormalities and follicular developmental issues, infertility is highly prevalent in obese and overweight The herbaceous species Salvia hispanica, commonly referred to as chia seeds, is a member of the Lamiaceae family and used both as food and medicine Research has recently focused on its speckled chemical composition, which may help those who consume it. In addition to being a high source of soluble and insoluble dietary fibres, Salvia hispanica may also have anti-inflammatory and antioxidant properties that help people lose weight and reduce the negative effects of obesity.

Aim

More focus has been directed to the possible positive health effects of omega-3 and fiber-rich seeds in the combating over weight and obesity. According to preliminary research, eating white Salvia hispanica L. (Salba chia) seeds may prolong feelings of fullness and might aid in weight loss. Thus the objective of this study to explore the effects of consuming Salvia hispanica on body weight, BMI and waist circumference in participants who were overweight or obese, as well as its safety.

Materials and methods

50 over weight and obese subjects were included in the study. Their diet was supplemented with 30 gm of Salvia hispanica daily for 12 weeks and weight, BMI and waist circumference were documented and analysed before and after study period.

Results

A total of 50 over weight and obese subjects aged between 25-50 years were recruited in the study. Of the fifty subjects in our study, 35 (70%) were females and 15 (30%).

Conclusion

It showed decreases in both weight and waist circumference among the participants. These findings suggest that Salvia hispanica seeds could assist in weight management for overweight and obese individuals. Additional studies are necessary to determine if these effects are sustained over time.

JK-Practitioner2026;31(2):45-47.

Introduction

Salvia hispanica L., an oilseed utilized by the Aztecs for nutritional and medicinal purposes, was dubbed "running food" due to its energy-providing properties during extended trade journeys. [1] With over 80 varieties, commonly known as Salba chia, this plant exhibits varying nutritional profiles. Initial clinical studies indicate that Salba may enhance feelings of fullness, decrease waist size, reduce post-meal blood sugar levels, and positively impact other cardiovascular disease risk factors, suggesting its potential in weight control strategies. Both forms of dietary fibre, which are found in abundance in Salba, are associated with a lower risk of obesity and a greater sense of satiety. It also contains a complete protein profile with all essential amino acids. Comprising 21% protein, Salba offers one of the most filling macronutrients. The seed's 28% fat content includes 65% alpha-linolenic acid, which is an omega-3 fatty acid and is known for its satiating properties. Furthermore, Salba is high in minerals like calcium, which may help in maintaining weight. [2] The seed also contains antioxidants that could help reduce inflammation. [3] Given its

Author Affiliations

Aamena Zaidi, Assistant Professor; **Divya Singh,** M. Sc.:
Department of Human Nutrition, School of Health Sciences, Chhatrapati Shahu Ji Maharaj University, Kanpur

Correspondence

Aamena Zaidi
Assistant Professor, Department of Human Nutrition, School of Health Sciences, Chhatrapati Shahu Ji Maharaj University, Kanpur.
Email: aamenazaidi@gmail.com
Contact No: +919807524958

Indexed

EMBASE, SCOPUS, IndMED , ESBCO, Google Scholar besides other national and international databases.

Cite this article as

Zaidi A, Singh D. Efficacy of *Salvia hispanica (*chia seeds) supplementation in promoting weight loss in obese individuals. JK Pract2026;31(2):45-47.

Full length article available at **jkpractitioner.com** one month after publication

Keywords

Salba chia, overweight, obese, weight, waist circumference.

nutritional composition, Salba may promote satiety, potentially supporting weight loss and consequently improving type 2 diabetes management and cardiovascular disease risk factors.

Materials and Methods

Research Design

Research design of the current investigation was based on pre-post intervention study. In this research design, a variable of interest is measured before and after an intervention in the same participants. The study recruited 50 participants from the local area, consisting of 35 women and 15 men between 16 and 50 years old. Potential subjects were evaluated for eligibility using predetermined inclusion and exclusion criteria.

Ethics Approval and Participant Recruitment

The institutional ethics committee granted approval for the study (letter no. CSJMU/R&D/1454/2023, dated 19/09/2023). After obtaining informed consent, participants were provided with Salba chia seed supplements to add to their regular diet. The supplement was administered at a rate of 30 gm per 1000 Kcal intake. This dosage was selected because it was similar to that used in a prior long-term randomized controlled trial by Vertommen and associates [4], which found no negative side effects.

Supplement Administration

Weekly doses of Salba chia supplements were pre-packaged in pouches for each participant. Subjects were given instructions regarding the incorporation of supplement into their daily diets and were also advised to follow a reduced-calorie diet to achieve a weight loss of 0.5-1 kg per week.

Table 1: Nutritive value of 30 g study supplement

Nutritional composition	Chia seeds powder
Energy	115 Kcal
Protein	6.9 gm
Carbohydrate	11.2 gm
Fat	10.4 gm
Dietary fibre	10.5 gm

Study Protocol

The data gathering process included taking body measurements and evaluating dietary habits at the start and at regular points throughout the 3-month study duration. Body measurements which were measured before and after study included Height, Weight, Body Mass Index (BMI) as well as Waist circumference, while dietary evaluations involved collecting food intake information using proven methods such as food frequency surveys and diet recall interviews.

Table 2: Effect of Salba Chia seeds supplementation on anthropometric parameters

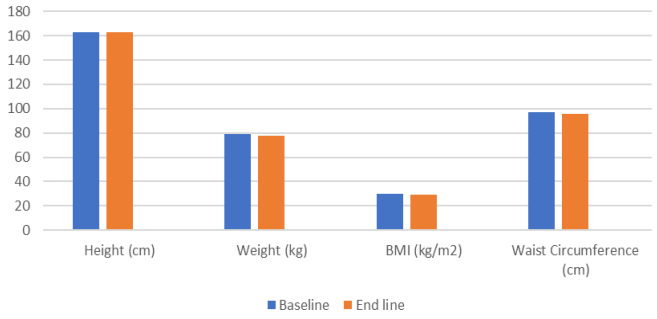
Variables	Baseline	End line	Mean reduction	Mean reduction (%)	p-value
Height (cm)	163±00.00	163±00.00	0	0%	NS
Weight (kg)	79.3±13.20	77.4±12.52	1.9	-2.45%	S
BMI (kg/m^2)	29.80±4.58	29.02±4.15	0.78	-0.29%	S
Waist Circumference (cm)	97.12±3.54	96±3.27	1.12	-0.11%	S

Values denoted as mean +SD.
p-value <0.05=NS Not significant, >0.05=S Significant

Results

Examination of Salba chia supplementation's impact on weight reduction showed decreases in body weight, BMI, and waist size among study participants. The average weight loss was 1.9 kg, equating to a 2.45%
decrease from initial measurements. Comparable patterns were seen for BMI, with a reduction of 0.78 kg/m2 (0.29% loss), and waist size, with a decrease of 1.12 cm (0.11% reduction). A statistically meaningful difference was observed between the final measurements and the initial values.

Fig 1: **Effect of Salba Chia seeds supplementation on anthropometric parameters**



Discussion

Given the circumstances, it's not surprising that the trial only resulted in minor, clinically insignificant weight reduction. These findings contradict those

from Nieman's [5] study on black chia seed weight loss. Participants were asked to add 30 g of chia daily to their daily diet. 30 g of Salba chia provided approximately 115 Kcal and participants may have consumed more calories than normal. Our study aimed to facilitate weight loss by substituting dietary calories with Salba, rather than simply adding supplement calories to daily energy consumption. 5–10% reduction in body weight leads to improved overall health outcomes as indicated by several studies. [6] The current investigation did not achieve this range, indicating that participants did not experience clinically significant weight loss.

Nevertheless, current guidelines for managing overweight and obesity suggest that gradual and consistent weight reduction is considered the most beneficial and long-lasting approach for patients. Medical professionals are advised to consider any weight reduction as a positive outcome. [7] Therefore, even the minimal weight loss observed among study participants could have positive implications for their health.

Conclusion

The study findings supported the hypothesis that supplementation with Salba chia would not trigger unwanted side effects or induce any unfavourable reactions, which is consistent with earlier studies on this seed. Given the escalating rates and healthcare expenses linked to obesity, it is crucial to conduct additional research on Salba chia as a potential complement to calorie-restricted diets for weight reduction. In particular, future investigations should focus on whether Salba chia can contribute to sustaining weight loss over extended periods, as this area requires further exploration.

Conflict Of Interest

Declared none.

Acknowledgement

The authors would like to thank Department of Human Nutrition, School of Health Sciences, CSJM University, Kanpur for providing the facilities for conducting the research work.

References

1. **Sellman S.** The Return of the Ancient Seed. THC Inc Pub; 2008; ISBN-13 :978-0979397936

2. **Zemel MB, Thompson W, Milstead A, et al**. Calcium and dairy acceleration of weight and fat loss during energy restriction in obese adults. Obesity Research. 2012; 12(4):582-90.

3. **Asplund K.** Antioxidant vitamins in the prevention of cardiovascular disease: a systematic review. J of Int Med. 2002; 251(5):372-92.

4. **Vertommen J.** Efficacy and Safety of 1 Month Supplementation of SALBA (Salvia Hispanica Alba) Grain to Diet of Normal Adults on Body Parameters, Blood Pressure, Serum Lipids, Minerals Status and Haematological Parameters. Results of a Pilot Study. 2005

5. **Nieman DC, Cayea EJ, Austin MD, et al**. Chia seed does not promote weight loss or alter disease risk factors in overweight adults. Nutr Res. 2009 Jun;29(6):414-8

6. **Goldstein DJ**. Beneficial health effects of modest weight loss. Int J Obes Relat Metab Disord. 1992 Jun; 16(6):397-415.

7. **Lau DC, Douketis JD, Morrison KM, et al**. Obesity Canada Clinical Practice Guidelines Expert Panel. 2006 Canadian clinical practice guidelines on the management and prevention of obesity in adults and children [summary]. CMAJ 2007;176(8 Suppl):S1-13

Dynamic Neuromuscular versus Lumbopelvic Stabilization Exercises for the Management of Chronic Lumbopelvic Pain in Menopausal Women: A Pilot Randomized Controlled Trial

Sadaf Khan , Jasmine Kaur Chawla.

Abstract

Background- Chronic lumbopelvic pain is more prevalent in the female population worldwide. It is mostly attributed to the physiological changes induced in the woman's body during pregnancy and childbirth, which produce biomechanical alterations. Later in the menopausal stage, women tend to have impaired neuromuscular control and pelvic instability, leading to Lumbopelvic pain. Both Dynamic Neuromuscular Stabilization (DNS) and Lumbopelvic Stabilization exercises have demonstrated their therapeutic effects in alleviating pain, but their comparative effectiveness for managing chronic lumbopelvic pain is unknown.

Aims: The study aims to compare the therapeutic effects of lumbopelvic stabilization exercises and the Dynamic Neuromuscular Stabilization exercise protocol in alleviating chronic lumbopelvic pain in postmenopausal women.

Materials and Methods- A pilot randomized controlled trial was conducted on 30 postmenopausal women diagnosed with chronic lumbopelvic pain. Participants were randomly assigned to three groups (n = 10 each): dynamic neuromuscular stabilization (DNS), lumbopelvic stabilization (LPS), and control. The intervention groups received their respective protocols for 20 minutes, 3 times per week, for 4 weeks, while the control group received advice on healthy lifestyle modification. Pre- and post-intervention values of BMI, pain (NPRS), and muscle strength (pressure biofeedback) were analyzed to find a better approach for chronic lumbopelvic pain.

Results: The results showed that both groups are effective for alleviating the chronic lumbopelvic pain, but the DNS had a better prognosis in terms of decrease in Pain (DNS- mean change≈ -5.10, $p < 0.001$ vs LPS - mean change≈ -4.50, $p < 0.001$) and increased muscle strength (Pressure biofeedback DNS- mean change≈ +8.60, $p<0.001$ vs LPS - mean change≈ +5.90, $p < 0.001$).

Conclusion: DNS has more promising therapeutic effects and may offer additional benefits by restoring optimal neuromuscular control, lumbopelvic alignment, and postural alignment compared to the LPS group. The findings of this study suggest that DNS can be incorporated into rehabilitation programs for this high-risk population for long-term management.

JK-Practitioner2026;31(2):48-53.

Introduction

Lumbopelvic pain is defined as pain in the lumbar spine and pelvic region, which includes the lumbar spine, sacroiliac joints, pelvis, or surrounding soft tissues [1,2]. Postpartum lumbopelvic pain (LBPP) is the most common occurrence in women [3], primarily due to ligament laxity and postural changes [4]. During pregnancy, hormonal shifts cause ligament laxity and structural misalignment in the lumbar spine, increasing lordosis as a result of weight gain [5,6]. These factors eventually lead to instability in the lumbopelvic region [7,8]. All these changes predispose women to decreased core muscle function, ultimately resulting in lumbopelvic pain[9].

The prevalence of musculoskeletal pain in women worldwide is 35%, out of which 29% is chronic lumbopelvic pain [2,3,10,11,] .77.8% of postmenopausal women suffer from LPP, and in the Indian population itself, it has been found to affect 70.4% of post- menopausal women [2,12,].

The exact pathological cause of lumbopelvic pain remains unknown [2,]as it's considered multifactorial because it is affected by biomechanical, hormonal, and neuromuscular factors.[13]. It is often attributed to the pregnancy and childbirth-induced physiological changes in the body of the woman over the years [8,14,]. Ultimately leading to impaired core

Author Affiliations

Sadaf Khan , Research Scholar ,**Jasmine Kaur Chawla,** Associate Professor Department of Physiotherapy, School of Allied Health Sciences, Manav Rachna International Institute of Research and Studies, Faridabad

Correspondence

Dr.Jasmine Kaur Chawla
Associate Professor,
Department of Physiotherapy
SAHS ,MRIIRS.
E mail
jasminekaurchawla.sahs@mriu.edu.in

Indexed

EMBASE, SCOPUS, IndMED , ESBCO, Google Scholar besides other national and international databases.

Cite this article as

Khan Sadaf , Chawla JK. Title- Dynamic Neuromuscular versus Lumbopelvic Stabilization Exercises for the Management of Chronic Lumbopelvic Pain in Menopausal Women: A Pilot Randomized Controlled Trial
JK Pract2026;31(2):48-53.

Full length article available at **jkpractitioner.com** one month after publication

Keywords

chronic lumbopelvic pain, lumbopelvic stabilization exercise, Dynamic Neuromuscular Stabilization, obesity, postmenopausal women.

stability, altered neuromuscular control, and lumbopelvic asymmetry [1,15,16] . Despite high prevalence, there is a lack of evidence-based, effective therapeutic interventions that may provide long-term symptomatic relief. Current studies conducted on LPP reveal a gap in the literature in terms of management strategies for the menopausal women.

Dynamic Neuromuscular Stabilization exercises is based on developmental kinesiology as well as reflexive core activation[17], while Lumbopelvic Stabilization emphasizes strengthening of the core muscle group[18], both have proved to be beneficial for many musculoskeletal ailments. Studies are suggestive of the beneficial effects of targeted stabilization approaches, such as lumbopelvic Stabilization and Dynamic Neuromuscular stabilization, which have shown improvements in pain alleviation, posture control, and functional control, but their comparative efficacy as a therapeutic remedy for chronic lumbopelvic pain in menopausal women remains undiscovered.

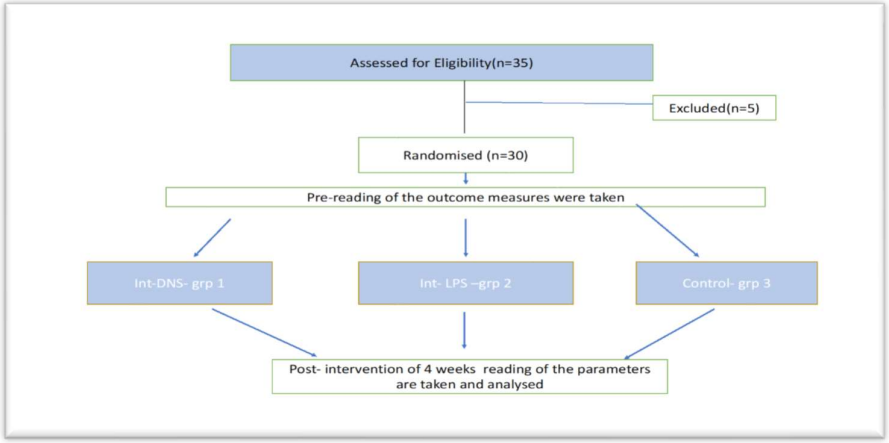
This study aims to interpret the effectiveness of the menopause population-specific exercise protocol of the interventions for the alleviation of chronic lumbopelvic pain. The objective is to find the effectiveness of the menopause population-specific exercise protocols, compare them, and validate the protocol for the alleviation of chronic lumbopelvic pain, its effect on obesity, and abdominal muscle strength. Hence, the experimental hypothesis is that there is a significant difference in the pre-post intervention values of LPP, obesity, and abdominal muscle strength.

Materials and Methods-

This pilot study is a Pilot randomized controlled trial adhering to the CONSORT guidelines. It is conducted to evaluate the effectiveness of the Dynamic neuromuscular stabilization exercise versus the Lumbopelvic stabilization exercise protocol for menopausal women. The research is approved by the Institutional Ethical Committee of Manav Rachna International University and is conducted on the campus. A convenient sampling method is opted for sample population selection. The inclusion criteria comprise menopausal women with cessation of menses for a year, who are suffering from chronic lumbopelvic pain for more than three months. Women of the age group 40-55 years having a BMI more than 23 (classified as overweight/obese) [19]. Informed consent was obtained from the 24 women who fulfilled the inclusion criteria and were willing to finish the protocol. The women with a history of recent trauma or any surgery, mainly fractures, abdominal surgery, or hysterectomy, any spinal injury or deformity, radiculopathy or inflammatory pathology of the spine, or neurological or systemic conditions (e.g., rheumatoid arthritis, fibromyalgia) are excluded. Screening of participants was conducted as per the inclusion criteria, and the women who were willing to finish the protocol duration were only recruited.

Protocol- A four-week exercise protocol was demonstrated to participants in the respective intervention groups, with the same dosing schedule of 3 times per week for 20 minutes. Demographic data and informed consent were obtained from the sample population. Pre-intervention readings were taken of the outcome measures, which include Pain (Numeric Pain Rating Scale that is calibrated from 1-10,

Fig. 1 CONSORT Flow Diagram



Abdominal Muscle strength assessed through the isometric contraction of the Transverse abdominis muscle through the Pressure Biofeedback unit, and also obesity, by measuring the weight changes in kg that may develop and be evaluated in terms of BMI (in kg/m^2). The BMI was recorded as a secondary anthropometric variable. Both the intervention exercises were designed according to menopausal population-specific requirements to target the Lumbopelvic musculature, which may help restore biomechanical alignment for the treatment and prevention of chronic lumbopelvic pain. Both exercise protocols are designed in a progressive manner, with initial contraction and later progressive strengthening of the musculature.

Interventions

Group one was demonstrated dynamic neuromuscular stabilization exercises protocol for eight weeks with the progression for the dosimetry of 20 min per day for 3 days per week. Week1&2 exercises lay emphasis on Foundational Stabilization and Breathing Retraining. It includes 3 exercises, namely the Supine 3-Month-Old Position, Diaphragmatic Breathing with Abdominal Wall Activation, and Side-Lying Position with Ribcage Expansion. Week 3&4 exercises were based on Core Activation and Dynamic Stabilization. It includes three exercises namely Quadruped Position with Diaphragmatic Breathing, Quadruped Rocking and Wall-Supported Squat with DNS Breathing. Week 5&6 e &r6 exercises e for Advanced Stability and Functional Movements, which include Modified Bear Position (Kneeling on All Fours), Rolling Pattern Activation, and Bird Dog with DNS Breathing. Week 7&8 exercises were for functional Integration and Stability in Dynamic Tasks. It includes Dead Bug in DNS 3-Month-Old Position, lunge with DNS core engagement and DNS Plank with Shoulder Taps**.**

Group two demonstrated the Lumbopelvic Stabilization exercises for the same dosimetry of 8 weeks, 3 days per week. Week 1&2 exercises were included to activate the muscles and achieve stability. They include Abdominal hollowing (Drawing-in Maneuver), Alternate Leg Raise with core stabilization (Marching), Isometric Gluteal Squeezes, and the Clamshell. Week 3-6 exercises were incorporated for achieving stability and building endurance through exercise progression. The exercises included are Clamshell progression, Gluteal Bridge progression, Pelvic tilts- (antero-posterior), Wall-supported Mini squats, Modified Dead Bug (with single limb). The week 7&8 exercises are to bring the dynamic control with strengthening and Functional movements. They are inclusive of Dead Bug (with alternate limbs), Side-lying hip abduction (Clamshell progression), Bridging Progression - Single-leg Bridge (Gluteal bridge progression), Side plank with knees supported, and Bird Dog- (Quadruped Arm and leg reach).

Group three comprises the control group that continued with their daily routine. All the groups were sensitized for healthy lifestyle modifications for the menopausal age like instructions for a proper, balanced diet rich in proteins and nutrients. Additionally, certain precautions while lifting anything heavy, wearing the proper footwear, and using proper ergonomics for daily life activities, such as sitting with a straight back or avoiding standing in one place for too long. After the four weeks, post-intervention reading was taken from all the groups for the statistical evaluation.

Statistical Analysis

The data were collected and analyzed through IBM SPSS software (Statistical Package of Social Science). A p-value of less than 0.05 was considered significant, and a parametric test was applied for analysis. For the within-group Paired t-test was performed, while for the pre-post values analysis, One-way ANOVA is used.

Results

The sample population of obese post-menopausal women with chronic lumbopelvic pain was allocated to three groups: Control (lifestyle advice), DNS, and LPS. The Shapiro-Wilk test was used to assess the normality of the data, and the baseline characteristics were analyzed.

Table 1. Within-group paired t-tests for BMI, pain, pressure biofeedback (n = 10 per group)

Variable	Group	t statistic	p value	Mean change (Post − Pre)
BMI	Control	—	—	0.00
BMI	DNS	13.37	<0.001	−0.87
BMI	LPS	7.65	<0.001	−0.52
Pain	Control	1.50	0.168	−0.20
Pain	DNS	21.86	<0.001	−5.10
Pain	LPS	16.74	<0.001	−4.50
Pressure biofeedback	Control	−1.00	0.343	0.10
Pressure biofeedback	DNS	−25.30	<0.001	8.60
Pressure biofeedback	LPS	−32.87	<0.001	5.90

Note:

- Abbreviations: BMI – Body Mass Index; DNS – Dynamic Neuromuscular Stabilization; LPS – Lumbopelvic Stabilization exercises.
- 'Pressure biofeedback' refers to pressure readings obtained using a pressure biofeedback unit as an indirect measure of Transversus abdominis isometric activation. Mean change refers to the difference between post-intervention and pre-intervention values.
- '—' indicates no variability in change scores, hence no t-test computed

Within-group changes in pain, muscle activation, and BMI (Table-1): In the control group, there was no meaningful improvement in any primary outcome across the intervention period. Pain scores showed only a small, non-significant reduction (mean change ≈ −0.20, $p = 0.168$), while BMI also remained unchanged (mean change = 0.00 for both outcomes), and pressure biofeedback values demonstrated a negligible, non-significant increase (mean change ≈ 0.10, $p = 0.343$). These findings indicate that lifestyle advice alone was insufficient to alter the chronic lumbopelvic pain, trunk muscle performance, as well as weight or BMI over the four weeks.

Although in contrast, both active interventions demonstrated statistically and clinically relevant improvements in all primary outcomes. In the DNS group, pain decreased substantially (mean change ≈ −5.10, $p < 0.001$), accompanied by a large increase in pressure biofeedback values (mean change ≈ +8.60, $p < 0.001$) alongside a modest but significant reduction In BMI (mean change≈ −0.87, $p < 0.001$).Similarly the LPS group exhibited pronounced reduction in pain

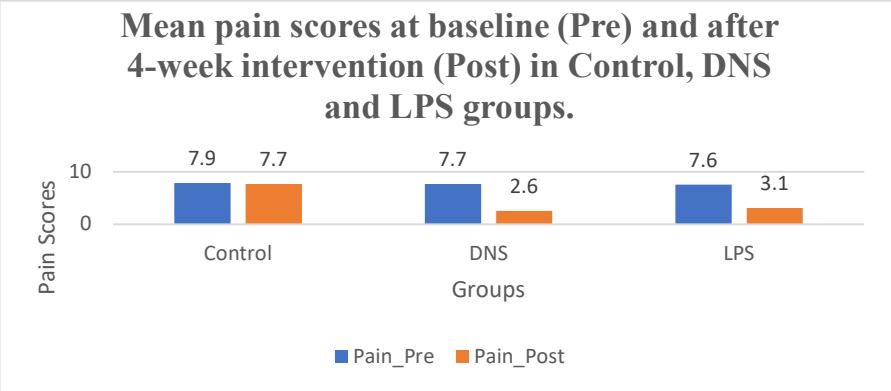


Figure 2: Mean pain scores at baseline (Pre) and after 4-week intervention (Post) in Control, DNS, and LPS groups. DNS and LPS show marked reductions in chronic lumbopelvic pain compared with the control group.

(mean change ≈ −4.50, p < 0.001), robust gains in pressure biofeedback (mean change ≈ +5.90, p < 0.001), with a smaller yet significant BMI decrease (mean change ≈ −0.52, p < 0.001). The direction and magnitude of these within-group changes are visually evident in the pre–post pain trajectories shown in Figure 2

Between-group differences in change scores of (Pre-Post) values (ANOVA) (Table-2): One-way ANOVA on change scores (Pre-Post) revealed significant main effects of group for all variables. For pain, the effect of group on change was highly significant (F ≈ 148.36, p < 0.001), with mean pain reductions of −5.10 in DNS and −4.50 in LPS compared to only −0.20 in the control group.

Table 2. One-way ANOVA on change scores (Post – Pre) for BMI, pain, pressure biofeedback

Variable	F statistic	p value	Mean Δ Control	Mean Δ DNS	Mean Δ LPS
BMI	64.92	<0.001	0.00	−0.87	−0.52
Pain	148.36	<0.001	−0.20	−5.10	−4.50
Pressure biofeedback	358.67	<0.001	0.10	8.60	5.90

Note:

- Abbreviations: F statistic – value from the one-way analysis of variance (ANOVA) on change scores
- Δ – Change score, calculated as post-intervention minus pre-intervention for each participant.
- p value – probability associated with the F statistic, with values < 0.05 considered statistically significant.

Post-hoc pairwise comparisons of change scores between groups (Table 3): Post-hoc pairwise comparisons (Bonferroni-adjusted) of change scores further clarified which groups differed significantly from one another (Table 3). For all variables, DNS and LPS each differed significantly from the control group (adjusted p < 0.001 for Control vs DNS and Control vs LPS across BMI, pain, pressure biofeedback, and pelvic asymmetry), confirming that both exercise-based interventions were superior to lifestyle advice alone (Table 3). When comparing DNS and LPS directly, the magnitude of pain reduction did not differ significantly after correction (adjusted p ≈ 0.327), despite DNS showing a slightly larger mean decrease, indicating that both protocols produced comparable analgesic effects within the limits of this pilot sample. In contrast, DNS outperformed LPS for BMI and pressure biofeedback, with Bonferroni-adjusted p values indicating significant differences in change scores for these outcomes (e.g., BMI: adjusted p ≈ 0.005; pressure biofeedback and pelvic asymmetry: adjusted p < 0.001). These post-hoc findings have concluded that DNS has consistently exhibited the largest mean improvements, LPS showing intermediate gains, and the control group remains essentially minimally changed.

Conclusion

This Pilot randomized controlled trial has finally concluded the effectiveness of both the Dynamic neuromuscular Stabilization exercise protocol as well as Lumbopelvic Stabilization exercise protocol, which are specifically designed for post-menopausal women to manage chronic lumbopelvic pain for a longer time. Along with the effectiveness of these exercises, the results indicate that the exercise protocol designs are completely safe and effective for menopausal women. Thus, the findings of this study validate both the

exercise protocol design. All the exercises are meticulously designed by considering the joint health as well as muscle laxity and weakness,

along with decreased bone strength produced in this population due to the hormonal influence of decreased estrogen [], ultimately deteriorating the musculoskeletal health. Even the mental health and Psychological well-being were taken into consideration to ensure that women can perform all the exercises with ease as well as enable them to perform the loads of responsibilities that a woman has to endure. The prerequisites for any exercise demonstration in this high-risk population is safety, as well as ease of performing it, without any financial overload or any instrumental need in the comfort of home as per their own convenience.

Both interventions improved the key clinical outcomes while control group did not show significant improvement is suggestive of that exercise-based rehabilitation can play a crucial role in the alleviation of chronic lumbopelvic pain in this population. DNS performed better in terms of pain than LPS group due to the emphasis on neuromuscular control and reflexive stabilization. DNS improved the coordination of deep trunk muscles as well as enhanced the central motor control eventually contributing to spinal stability and nociceptive input reduction.

Table 3. Post-hoc pairwise comparisons (Bonferroni-adjusted p values) for change scores (Pre– Post treatment)

Variable	Group means (Δ)	Comparison	'p' value	Bonferroni-adjusted p value
BMI	Control: 0.00 DNS: −0.87 LPS: −0.52	Control vs DNS	<0.001	<0.001
		Control vs LPS	<0.001	<0.001
		DNS vs LPS	0.002	0.005
Pain	Control: −0.20 DNS: −5.10 LPS: −4.50	Control vs DNS	<0.001	<0.001
		Control vs LPS	<0.001	<0.001
		DNS vs LPS	0.109	0.327
Pressure biofeedback	Control: 0.10 DNS: 8.60 LPS: 5.90	Control vs DNS	<0.001	<0.001
		Control vs LPS	<0.001	<0.001
		DNS vs LPS	<0.001	<0.001

Note:

- Abbreviations: Δ denotes the change score calculated as post-intervention minus pre-intervention for each participant.
- Group means (Δ) are reported for descriptive purposes.
- Post-hoc p values based on pairwise comparisons of change scores between groups using independent-samples t-tests, with Bonferroni adjustment applied for three comparisons per variable (Control vs DNS, Control vs LPS, DNS vs LPS).
- Adjusted p values:
  For Control vs DNS and Control vs LPS (all variables), Bonferroni-adjusted p values < 0.001.
  For DNS vs LPS, the adjusted p values by variable were:
  BMI: raw p ≈ 0.0016 → Bonferroni-adjusted p ≈ 0.0047 (still significant).
  Pain: raw p ≈ 0.1091 → Bonferroni-adjusted p ≈ 0.3272 (not significant).
  Pres. Biofeedback: raw p so small that even after ×3 it remains < 0.001.

BMI on the other hand did not demonstrated any statistical difference as the exercise protocol were designed to induce weight loss but rather improve the neuromuscular function and stabilization. But the exercises were meant to restore the proper biomechanical alignment of the spine by restoring the lumbopelvic rhythm. The increased abdominal muscle strength is indicative of its implication in the long-term use for regaining the proper lumbopelvic mechanical structure by strengthening the weakened musculature.

To conclude, both exercises are effective for chronic lumbopelvic pain management in menopausal women with DNS, demonstrating superior outcomes and results, as a better long-term rehabilitation approach for the chronic lumbopelvic pain in post-menopausal women.

Limitation- The study needs to be conducted with a large sample population for a longer duration of exercise protocol to verify its effectiveness.

Conflict of interest- There is no conflict of interest between authors or any third party.

Funding- No funding

References-

1-**Sushil P, Chawla JK, Parasher RK.** Content Validation of Lumbopelvic Pain Risk Factors Questionnaire among Women: A Modified Delphi Consensus Study. The Open Public Health Journal. 2023 Sep 4;16(1).

2-**Chawla JK, Sushil P, Kumar P, Singh M, Sharma R.** Development of prediction model for risks of musculoskeletal chronic lumbopelvic pain in Indian women. Scientific Reports. 2024 Sep 29;14(1):22566.

3-**Bergström C, Persson M, Nergård KA, Mogren** I. Prevalence and predictors of persistent pelvic girdle pain 12 years postpartum. BMC musculoskeletal disorders. 2017 Sep 16;18(1):399.

4- **Ehsani F, Sahebi N, Shanbehzadeh S, Arab AM, ShahAli S.** Stabilization exercise affects function of transverse abdominis and pelvic floor muscles in women with

postpartum lumbo-pelvic pain: a double-blinded randomized clinical trial study. International urogynecology journal. 2020 Jan;31(1):197-204.

5- **Yoseph ET, Taiwo R, Kiapour A, Touponse G, Massaad E, Theologitis M, Wu JY, Williamson T, Zygourakis CC.** Pregnancy-related spinal biomechanics: a review of low back pain and degenerative spine disease. Bioengineering. 2025 Aug 10;12(8):858.

6- **Casagrande D, Gugala Z, Clark SM, Lindsey RW**. Low back pain and pelvic girdle pain in pregnancy. JAAOS-Journal of the American Academy of Orthopaedic Surgeons. 2015 Sep 1;23(9):539-49.

7-**Daneau C, Abboud J, Marchand AA, Houle M, Pasquier M, Ruchat SM, Descarreaux M.** Mechanisms underlying lumbopelvic pain during pregnancy: a proposed model. Frontiers in Pain Research. 2021 Dec 2;2:773988.

8-**Vleeming A, Albert HB, Östgaard HC, Sturesson B, Stuge B**. European guidelines for the diagnosis and treatment of pelvic girdle pain. European Spine Journal. 2008 Jun;17(6):794-819.

9- **Skundric G, Vukicevic V, Lukic N.** Effects of core stability exercises, lumbar lordosis and low-back pain: A systematic review. Journal of Anthropology of Sport and Physical Education. 2021;5(1):17-23.

10. **Wong WS, Fielding R**. Prevalence and characteristics of chronic pain in the general population of Hong Kong. The Journal of Pain. 2011 Feb 1;12(2):236-45.

11. **Hoy D, Bain C, Williams G, March L, Brooks P, Blyth F, Woolf A, Vos T, Buchbinder R.** A systematic review of the global prevalence of low back pain. Arthritis & rheumatism. 2012 Jun;64(6):2028-37.

12. **Nageswari C, Meena N, Thillaieaswaran B.** Enhancing well-being: investigating the role of Pilates in alleviating low back pain among postmenopausal women–a scoping review. Physical Therapy Reviews. 2025 Jul 4;30(4):296-307.

13. **Sushil P, Chawla JK, Parasher RK.** Impact of Socioeconomic Disparities, General Health, Reproductive, and Exercise Status in Indian Women with Lumbopelvic Pain. The Open Public Health Journal. 2023 Jul 10;16(1).

14. **Ehsani F, Sahebi N, Shanbehzadeh S, Arab AM, ShahAli S**. Stabilization exercise affects function of transverse abdominis and pelvic floor muscles in women with postpartum lumbo-pelvic pain: a double-blinded randomized clinical trial study. International urogynecology journal. 2020 Jan;31(1):197-204.

15 **Moheboleslam Z, Mohammad Rahimi N, Aminzadeh R. A** systematic review and meta-analysis of randomized controlled trials of stabilizing exercises for lumbopelvic region impact in postpartum women with low back and pelvic pain. Biological Research For Nursing. 2022 Jul;24(3):338-49.

16. **Sherburn M, Guthrie JR, Dudley EC, O'Connell HE, Dennerstein L**. Is incontinence associated with menopause?. Obstetrics & gynecology. 2001 Oct 1;98(4):628-33.

17**. Kobesova A, Valouchova P, Kolar P.** Dynamic Neuromuscular Stabilization: Exercises based on developmental kinesiology models. Functional Training Handbook. 2014;25:51.

18. **Skundric G, Vukicevic V, Lukic N.** Effects of core stability exercises, lumbar lordosis and low-back pain: A systematic review. Journal of Anthropology of Sport and Physical Education. 2021;5(1):17-23.

19. **Misra A**. Ethnic-specific criteria for classification of body mass index: a perspective for Asian Indians and American Diabetes Association position statement. Diabetes technology & therapeutics. 2015 Sep;17(9):667-71.

Effect of Whey Protein Supplementation on Performance Determinants and Inflammatory Biomarkers of Male Football Players: A Randomized Parallel-Group Placebo-Controlled Trial

Kingshuk Ghosh , Jasmine Kaur Chawla , Indranil Manna .

Abstract

Introduction: Resistance training (RT) is beneficial in developing strength, power, and speed in footballers. Taking protein supplements combined with RT is expected to cause a more significant muscular hypertrophy over time. Also, the intermittent high-intensity sprints, rapid changes of direction, and powerful eccentric decelerations, the biomechanical demands of football induce significant micro-trauma to the skeletal muscle fibres. Choice of Whey-Protein supplementation combined with RT can significantly dictate adaptations to resistance training and the rate of post-match physiological restoration. But the practice of periodized resistance training and intake of protein supplements is rare in non-elite football players in rural areas.

Objectives: The present study was designed to find out the impacts of resistance training combined with whey protein supplementation on different performance determinants and inflammatory biomarkers of non-elite football players from rural areas.

Methods: A total of 96 football players who have been playing football at the district level for more than 2 years were selected for the study. Then they were subdivided into two groups: (i) Whey Protein Supplement Group (WPSG, n=48), where a supplement of whey protein was provided in 1.00 gm/ kg BM/day, and (ii) Placebo Control Group (PCG, n=48), which received a maltodextrin placebo. Both groups followed a resistance training schedule for two mesocycles of 6 weeks (2 sessions/wk, for 12 weeks). Assessment of body composition, physical fitness, physiological variables, and inflammatory biomarkers was measured at 0 weeks, 6 weeks, and 12 weeks**.**

Results:Significantly greater improvements in lean body mass, muscular strength, vertical jump, anaerobic power, sprint speed, agility, and anaerobic capacity were established in WPSG compared with the PCG ($p \leq 0.05$). Whey protein supplementation also significantly diminished the increases in inflammatory biomarkers, including CRP, CK, and LDH.

Conclusion: Whey protein supplementation in combination with resistance training effectively enhances performance determinants, lean body mass, and recovery while reducing exercise-induced inflammation in non-elite male football players.

JK-Practitioner2026;31(2):54-62.

Introduction

A complex physiological profile, including high aerobic capacity, muscular strength, anaerobic power, and metabolic capacities, is the main endeavour of playing football. Consequently, one of the most important objectives in preparing football players for competition is developing high levels of muscular strength. Resistance training (RT) is known to improve performance-enhancing qualities like strength, power, speed, agility, and endurance, reducing the risk of injury. It has been demonstrated that prolonged periodized RT could be effective for increasing the size of skeletal muscles and, eventually, their ability to generate force. Therefore, resistance training must be incorporated into the training regimens of Football [Tabassum et al.,2023].

RT is only maximized when accompanied by an adequate supply of essential amino acids (EAAs) to stimulate muscle protein synthesis (MPS). The intermittent high-intensity sprints, rapid changes of direction, and powerful eccentric decelerations, the biomechanical demands of football induce significant micro-trauma to the skeletal muscle fibres. The modern football athlete operates within a narrow recovery window where the choice of protein supplementation can significantly dictate adaptations to resistance training and the rate of

Author Affiliations

Kingshuk Ghosh , Research Scholar**,** Department of Physiology **,Jasmine Kaur Chawla ,**Associate Professor Department of Physiotherapy:
Manav Rachna International Institute of Research and Studies, Faridabad, Haryana, India. **Indranil Manna,** Associate Professor, Department of Physiology, Midnapore College (Autonomous), Midnapore, West Bengal, India.

Correspondence

Dr. Indranil Manna
Associate Professor, Department of Physiology, Midnapore College (Autonomous), Midnapore, West Bengal, India.

Indexed

EMBASE, SCOPUS, IndMED , ESBCO, Google Scholar besides other national and international databases.

Cite this article as

Ghosh K , Chawla JK , Manna I . Effect of Whey Protein Supplementation on Performance Determinants and Inflammatory Biomarkers of Male Football Players: A Randomized Parallel-Group Placebo-Controlled Trial. **J**K Pract2026;31(2):54-62.

Full length article available at **jkpractitioner.com** one month after publication

Keywords

Resistance training, Strength, Power, Speed, Whey Protein, Inflammatory biomarkers. Football players.

post-match physiological restoration. The Endurance athletes involved in various team sports have to maintain a positive nitrogen balance, so the recent recommendation for football players indicates potential benefits from higher amounts, specifically 1.6–2.2 g/kg body mass/day [Williamson et al., 2019]. High-quality protein sources are crucial, as they contain a high proportion of essential amino acids (EAAs) necessary for MPS [Sousa et al., 2022; Jacinto et al., 2022]. Recent evidence suggests that Leucine may reverse the net negative whole-body protein balance and can improve the recovery of inflammatory biomarkers like Creatine Kinase (CK), Lactate dehydrogenase (LDH), and C-Reactive protein (CRP) [Jackman et al., 2017; Wang & Wu, 2023]. Among the protein sources available, whey protein (WP) is considered the most popular one. Whey protein is a byproduct of cheese production. Due to its high concentration of branched-chain amino acids (BCAAs), particularly leucine, whey protein has been the gold standard for athletes for a long time [Griffen et al., 2022].

Indian football players have lower muscle mass than Europeans [Pomeroy et al., 2019]; therefore, they lack in muscular strength and power and cannot keep up with the physical and physiological capabilities of other international players [Kulkarni et al., 2013]. Lack of proper resistance training and protein supplementation also aids in this problem. But the practice of periodized resistance training and intake of protein supplements is rare in non-elite football players in rural areas. However, in the case of the availability of resistance training, there is no adequate weight selection through the 1RM test for athletes and no proper training load monitoring, which ultimately leads to overtraining associated with reduced muscular strength and sometimes injury. Dietary protein supplementation should be given scientifically and periodically. In this regard, the present study was designed to find out the impacts of resistance training and whey protein supplementation on different performance determinants and inflammatory biomarkers of non-elite football players from rural areas.

Even though whey protein supplementation has been widely studied in athletes, limited research has directly examined its impact on both performance determinants and inflammatory biomarkers among male football players. The previous studies mainly focused on strength and recovery outcomes, with incomplete information about biomarkers such as LDH, CRP, and CK. Additionally, several studies lack randomized placebo-controlled designs and fail to assess the sports-specific physiological demands of football. Research on South Asian or Indian football players is also equally limited. Therefore, the current study aims to investigate the effect of whey protein supplementation combined with RT on performance determinants and inflammatory biomarkers among male football players using a randomized parallel-group placebo-controlled design.

II. METHODS

A. Participants

This study was conducted by the Department of Physiology, Midnapore College Research Centre, Vidyasagar University, W.B., India. Male football players (age: 18–20 years) who participated in district-level competitions for the last two years were randomly selected for this study. G*power software [Kang, 2021] was used to compute the sample size. A minimum of 52 subjects was required to carry out the study, as computed from the software. A total of 96 football players were selected for the study. Then they were subdivided into two groups as (i) Placebo Control Group (PCG, n=48) and (ii) Whey Protein Supplement Group (WPSG, n=48). Participants unwilling to take part in the study, had a record of illness or disease for at least three months before the research study started, were excluded from the study.

B. Ethical Consideration, CTRI Registration and Informed Consent Statement

Written information was given to all the participants about the objectives and possible complications of the study, and written consent was taken from them. The Institutional Ethics Committee approval was obtained for the present study [Registration No – CE/2023-24/05; Date: 15/10/24]. The study is also registered in the Clinical Trials Registry India [CTRI No-CTRI/2025/08/092533, Register on 06/08/25].

C. Resistance Training Protocol

Participants of both groups followed a periodization of resistance training schedule. The total training program was divided into two different 6-week mesocycles: (i) 1st mesocycle of Muscular Endurance Phase (MEP, 1-6 weeks, 1hr. 30mins. /session), and (ii) 2nd mesocycle of Maximal Strength Phase (MSP, 6-12 weeks, 1hr.15mins. /session) [Bompa & Buzzichelli, 2021]. Other football-specific training will continue in the remaining days for 2 hours/day, 4 days/week. The first mesocycle focused mainly on muscular endurance, characterized by short rest periods between sets and high training volume (3 sets of 10 repetitions – 2 minutes of rest), designed to help the body adjust anatomically and become ready for a more demanding training session during the maximal strength phase. Subjects were engaged in resistance training at a moderate level (65-75% of 1RM) during this period. The second mesocycle, to acquire maximum strength, training volume (4 sets of 5 repetitions – 3 minutes of rest) was reduced, and training load increased (85-95% of 1RM) during this phase [Barjaste & Mirzaei, 2017]. 1-repetition

maximum (1-RM) was used to monitor training intensity [Kim et al., 2023]. The training programme was followed according to the suggestions of the qualified coaches.

D. Nutritional Intervention:

Participants were advised to stick to their regular nutritional routine and avoid taking any extra supplements beyond those that were supplied throughout the intervention to reduce the possibility of dietary confounding outcomes. Participants of the Whey Protein Supplement Group (WPSG) were supplied with a Whey protein (NUTRABAY PURE, India: contain 77.9 gm of protein/ 100 gm) supplement (1.00 g/kg BM whey protein powder + 0.2 g/kg BM sucrose powder). The Placebo Control Group (PCG) was provided Maltodextrin supplement (1.00 g/kg BM Maltodextrin powder + 0.2 g/kg BM sucrose powder). The composition of the two supplements is given in Table 02. The subject consumed two doses of the supplement (0.6 g/kg BM mixed supplement powder) dissolved in water after their morning and afternoon training sessions. Sucrose powder was mixed with protein supplement to ensure that the supplements and placebo would be comparable in energy content, appearance, taste, and texture. The supplement will be taken by the subjects around half an hour after each training session [Macnaughton et al., 2016].

E. Study Design

Nutritional status of the subjects was assessed at baseline (0 week). The selected Body composition, Strength, Power, Speed, Physiological, and Biochemical variables were measured in each group at the beginning of the Muscular Endurance Phase (0 week) and the end of the Muscular Endurance Phase (MEP, 6 weeks), and the Maximal Strength Phase (MSP, 12 weeks).

F. Assessments of Nutritional Status:

The nutritional status of the subjects was assessed by questionnaire method using the 24-hour recall method, at baseline (0week). An attempt was made to find out whether the subjects consume protein as per the recommendation of the Indian Council of Medical Research (ICMR) [Castell et al., 2015].

F. Measurement of body composition variables

Height (stature) and body mass were measured, and body mass index (BMI) and body surface area (BSA) were determined [Chandrasekar et al., 2023]. A skinfold caliper (Cescorf, USA) was used to determine percent body fat, total fat mass, and lean body mass (LBM) following standard methods [Siri, 1961]. The mid-upper arm circumference (MUAC) was measured using standard procedures [Chandrasekar et al., 2023].

G. Measurements of physical fitness variables

A hand grip dynamometer, back and leg dynamometer (Baseline, USA) were used for measurement of hand grip strength, back, and leg strength following standard methods. The vertical jump (VJ), standing broad jump (SBJ), agility and sit-up (abdominal strength), and agility tests were performed using standard procedures. The 30-m sprint times were taken, and speed was determined [Chandrasekar et al., 2023]. The running-based anaerobic sprint test (RAST) was performed to determine the anaerobic power [Burgess et al., 2016].

H. Measurements of physiological variables

The resting heart rate of the subjects was measured after fifteen minutes of rest [McArdle et al., 2015]. The Yo-Yo Intermittent Recovery Test 1 (YYR1) was used for the determination of maximum oxygen uptake (VO2max) of the subjects [Bangsbo et al., 2008].

I. Measurement of Inflammatory Biomarkers

A 5 ml of 12-hour fasting blood sample was drawn from an antecubital vein 24 hours after the last exercise bout for the determination of selected biochemical parameters such as Creatine Kinase (CK), C-Reactive protein (CRP), Lactate Dehydrogenase (LDH), Aspartate Aminotransferase (AST), Alanine Aminotransferase (ALT), Urea, and Uric Acid [Manna, Khanna, & Dhara, 2010].

J. Statistical Analysis

Descriptive statistics were computed for all variables. A two-way ANOVA analysis was performed to find out the effects of group, training, and their interaction (group × training) on all the variables. Bonferroni post-hoc multiple comparison tests were used to detect within-group differences. An independent samples t-test was used to determine between-group differences. For each dependent variable, the main effects of training and group, as well as the interaction effect (group × training), were reported along with their corresponding F-values, degrees of freedom, and partial eta squared (η2p) as a measure of effect size. Pearson's correlation analysis was used to investigate the relationship between variables. The statistical significance was chosen at $p<0.05$ [Banerjee, 2018]. All statistical analyses were carried out using SPSS software (version 27.0; IBM Corp., Armonk, NY, USA).

III. RESULT

A. Nutritional Assessment of Two Groups at Baseline

The nutritional assessment of both the Placebo control group (PCG) and the Whey protein supplement group (WPSG) showed no significant differences ($p\leq0.05$) between the groups. But both PCG (53.33 ± 7.39) and WPSG (54.01 ± 6.40) consumed less protein, as per the recommendation of the Indian Council of Medical Research (ICMR) [Castell et al., 2015].

B. Effect of Whey Protein Supplementation Combined with Resistance Training on Body Composition

After a 12-week intervention, both the Placebo Control (PCG) and the Whey Protein Supplement Group (WPSG) showed changes in body composition. The WPSG demonstrated a significant increase in total body weight. After 12 weeks, it rose to 59.30 ± 4.02 kg from the baseline of 54.80 ± 4.28 kg, which was

significantly higher than the PCG. Simultaneously, Lean Body Mass (LBM) in the WPSG increased considerably from 50.49 ± 4.10 kg to 55.92 ± 3.78 kg. Body fat percentage (BF%) significantly reduced in both groups. WPSG also achieved a superior increment in mid-upper arm circumference (MUAC), concluding the study at 22.27 ± 2.85 cm.

C. Effect of Whey Protein Supplementation Combined with Resistance Training on Physical and Physiological Determinants

The strength adaptation in both groups yielded resistance training, but the WPSG consistently achieved a greater magnitude of improvement. Both right and left grip strength increased after the intervention. In PCG, the Right grip strength (GSTR) increased from 30.41 ± 3.15 kg to 40.47 ± 3.48 kg, whereas after 12 weeks, WPSG achieved a significantly higher GSTR, increasing from 31.30 ± 2.95 kg to 42.83 ± 2.20 kg. GSTL in WPSG increased from 29.72 ± 3.09 kg to 40.41± 2.15 kg, which is significantly higher than the PCG, which rose from 29.16 ± 3.37 kg to 36.57 ± 3.16 kg. After 12 weeks of training, leg strength reached 141.35 ± 7.11 kg, and the back strength increased up to 116.64 ± 9.91 kg in WPSG when compared to PCG. Abdominal strength (repetitions/minute) increased significantly in both groups. After 12 weeks, the abdominal strength of PCG improved from 40.29 ± 4.16 rep/min at baseline to 43.97 ± 2.96 rep/min at week 12. While the WPSG recorded a significantly greater improvement, rising from 40.52 ± 4.78 rep/min to 45.85 ± 1.96 rep/min.

Both groups resulted in significant improvements in jumping performance after a 12-week resistance training intervention. Vertical Jump (VJ/CMJ) in the Placebo Control Group (PCG) increased from a baseline of 39.12 ± 2.78 cm to 43.56 ± 2.89 cm at 12 weeks. A significantly greater enhancement in VJ/CMJ, rising from 39.20 ± 2.87 cm to 46.55 ± 1.88 cm, could be seen in WPSG, statistically outperforming the PCG. In addition, the Standing Broad Jump Test (SBJT) was enhanced in the PCG from 6.26 ± 0.72 ft. to 7.55 ± 0.84 ft. The WPSG exhibited a superior increase in SBJT, progressing from 6.30 ± 0.70 ft. to 7.90 ± 0.69 ft. by the conclusion of the trial.

Both groups showed improvements in their anaerobic power outputs after 12 weeks. But average power (A-Power) improved significantly in WPSG (183.18 ± 22.11 W to 269.56 ± 35.10 W) compared to PCG (190.79 ± 38.17 W to 243.50 ± 69.99 W). At 12 weeks, WPSG showed an increase in their Fatigue Index, concluding

Table 01. Changes in body composition variables among the Football players

Parameters	Placebo Control Group (n=48)			Whey Protein Supplement Group (n=48)			Two-Way ANOVA		
	0 Wk	6 Wk	12Wk	0 Wk	6 Wk	12 Wk	Group F1 (ηp^2) [p value]	Training F2(ηp^2) [p value]	Interactio F3(ηp^2) [p value]
BMI (kg/m^2)	20.03 ± 1.19	20.08 ± 1.17	19.97 ± 1.00	19.91 ± 0.40	20.43* ± 0.45	21.72* #$ ± 0.46	36.9 (0.116) [p≤0.001]	17.81 (0.11) [p≤0.001]	35.22 (0.20) [p≤0.001]
BF%	7.18 ± 1.59	7.12 ± 1.61	5.62*# ± 1.29	7.87 ± 1.52	6.47$* ± 1.26	5.69*# ± 1.24	0.298 (0.001) [0.585]	51.61 (0.268) [p≤0.001]	3.08 (0.21) [0.047]
LBM (kg)	51.67 ± 4.86	51.19 ± 4.74	51.72 ± 4.43	50.49 ± 4.10	52.18* ± 3.98	55.92* $ ± 3.78	7.52 (0.26) [0.006]	11.14 (0.07) [p≤0.001]	8.67 (0.058) [p≤0.001]
MUAC (cm)	20.04 ± 2.95	20.04 ± 2.94	21.01 ± 3.06	20.50 ± 2.84	21.43 ± 2.88	22.27* $ ± 2.85	9.03 (0.003) [0.03]	5.41 (0.03) [0.05]	0.71 (0.005) [0.49]

[Data are presented as Mean ± SD. Two-way ANOVA was performed to examine the effects of Group, Training, and Group × Training interaction. The Bonferroni post-hoc test was applied for within-group comparisons. * Indicates significant difference compared with 0 week (p≤0.05), # indicates significant difference compared with 6 weeks (p≤0.05). F values, p values, and partial eta squared (ηp^2) are reported. An independent samples t-test was used to determine between-group differences. $ indicates a significant difference compared with the PCG (p≤0.05). Degrees of freedom for the independent t-test = 94; critical t = 1.986. SD = standard deviation, ANOVA= Analysis of Variance, PCG= placebo control group, WPSG= whey protein supplement group; BMI= body mass index, BF%= body fat percentage, LBM = lean body mass, MUAC = mid upper arm circumference,]

Table 02. Changes in Physical and Physiological determinants among the football players

Parameters	Placebo Control Group (n=48)			Whey Protein Supplement Group (n=48)			Two-Way ANOVA		
	0 Wk	6 Wk	12Wk	0 Wk	6 Wk	12 Wk	Group F1 (ηp^2) [p value]	Training F2(ηp^2) [p value]	Interaction F3(ηp^2) [p value]
GSR (kg)	30.41 ± 3.15	38.75* ± 3.07	40.47*# ± 3.48	31.30 ± 2.95	38.64* ± 2.78	42.83*#$ ± 2.20	8.91 (0.003) [0.03]	338.70 (0.706) [p≤0.001]	4.21 (0.016) [0.029]
GSL (kg)	29.16 ± 3.37	34.69* ± 2.77	36.57*# ± 3.16	29.72 ± 3.09	36.08*$ ± 2.38	40.41*#$ ± 2.15	32.83 (0.104) [p≤0.001]	248.71 (0.638) [p≤0.001]	8.56 (0.057) [p≤0.001]
Back Str. (kg)	94.97 ± 10.45	107.81* ± 9.84	111.72* ± 8.77	95.47 ± 10.46	108.54* ± 10.42	116.64*#$ ± 9.91	3.02 (0.11) [0.08]	90.14 (0.39) [p≤0.001]	1.48 (0.01) [0.23]
Leg Str. (kg)	113.35 ± 12.42	129.62* ± 9.86	134.10* ± 9.81	112.06 ± 11.94	128.52* ± 11.90	141.35*#$ ± 7.11	1.65 (0.006) [0.199]	136.19 (0.491) [p≤0.001]	5.01 (0.034) [0.00]
VJ / CMJ (cm)	39.12 ± 2.78	43.15* ± 2.91	43.56* ± 2.89	39.20 ± 2.87	44.06* ± 1.97	46.55*#$ ± 1.88	18.77 (0.062) [p≤0.001]	134.52 (0.48) [p≤0.001]	8.06 (0.054) [p≤0.001]
SBJT (f.inch)	6.26 ± 0.72	7.21* ± 0.83	7.55* ± 0.84	6.30 ± 0.70	7.26* ± 0.69	7.90*#$ ± 0.69	2.91 (0.10) [0.89]	91.64 (0.39) [p≤0.001]	1.36 (0.010) [p≤0.001]
Abdominal Strength (rep/min)	40.29 ± 4.16	42.39* ± 3.60	43.97* ± 2.96	40.52 ± 4.78	43.33* ± 3.21	45.85*#$ ± 1.96	5.82 (0.02) [0.016]	38.56 (0.21) [p≤0.001]	1.28 (0.009) [0.277]
Agility (sec.)	19.06 ± 1.13	17.80* ± 1.19	16.62*# ± 1.04	19.35 ± 1.29	17.35* ± 1.19	15.65*#$ ± 0.65	8.45 (0.029) [0.004]	186.05 (0.56) [p≤0.001]	7.77 (0.52) [p≤0.001]
Speed (sec)	6.49 ± 0.33	6.21* ± 0.44	6.10* ± 0.43	6.40 ± 0.34	5.79*$ ± 0.31	5.61*#$ ± 0.31	57.25 (0.16) [p≤0.001]	66.01 (0.31) [p≤0.001]	7.55 (0.051) [p≤0.001]
AP (watt)	190.79 ± 38.17	224.77* ± 55.70	243.50* ± 69.99	183.18 ± 22.11	239.04* ± 29.97	269.56*#$ ± 35.10	4.29 (0.015) [0.04]	59.86 (0.29) [p≤0.001]	3.51 (0.024) [0.03]
FI (watt/sec)	1.87 ± 0.72	2.15 ± 1.11	1.67# ± 0.88	2.12 ± 0.68	2.57*$ ± 0.77	2.52*$ ± 0.43	29.09 (0.094) [p≤0.001]	5.37 (0.03) [0.005]	3.51 (0.024) [0.03]
VO_{2max} (ml/kg/min)	43.54 ± 2.38	43.62 ± 2.22	44.70*# ± 1.55	43.52 ± 6.35	43.60* ± 2.47	44.80 ± 2.30	0.005 (0.00) [0.944]	8.75 (0.05) [p≤0.001]	0.23 (0.00) [0.97]

Data are presented as Mean ± SD. Two-way ANOVA was performed to examine the effects of Group, Training, and Group × Training interaction. The Bonferroni post-hoc test was applied for within-group comparisons. * Indicates significant difference compared with 0 week (p≤0.05), # indicates significant difference compared with 6 weeks (p≤0.05). F values, p values, and partial eta squared (ηp^2) are reported. An independent samples t-test was used to determine between-group differences. $ indicates a significant difference compared with the PCG (p≤0.05). Degrees of freedom for the independent t-test = 94; critical t = 1.986. SD = standard deviation, ANOVA= Analysis of Variance, PCG= placebo control group, WPSG= whey protein supplement group; GSR= grip strength in right hand, GSL= grip strength in left hand, SBJ= standing broad jump, VJ= vertical jump, AP= average power, FI= fatigue index, VO_{2max} = maximum oxygen consumption]

Maximal sprint speed, reflecting on Sprint time, significantly decreased in the WPSG after 12 weeks, from a baseline of 6.40 ± 0.34 seconds to 5.61 ± 0.31 seconds. The decreased sprint time indicates a significant improvement in sprinting speed compared to the PCG. A similar pattern followed by the agility performance, WPSG completed the agility test in 15.65 ± 0.65 seconds by the conclusion of the study, indicating superior neuromuscular adaptation.

After the 12-week resistance training intervention, VO2max (Aerobic capacity) resulted in a positive from 43.54 ± 2.38 ml/kg/min at baseline to 44.70 ± 1.55 ml/kg/min. The WPSG also demonstrated an increase, shifting from 43.52 ± 6.35 ml/kg/min at baseline to 44.80 ± 2.30 ml/kg/min at 12 weeks. However, a direct comparison between the groups showed no significant difference in the magnitude of increased VO2max; the whey protein supplementation did not yield a statistically superior aerobic outcome compared to the placebo.

D. Effect of Whey Protein Supplementation Combined with Resistance Training on Inflammatory Biomarkers

Table 03. Changes in Inflammatory biomarkers among the football players

Parameters	Placebo Control Group (n=48)			Whey Protein Supplement Group (n=48)			Two-Way ANOVA		
	0 Wk	6 Wk	12Wk	0 Wk	6 Wk	12 Wk	Group F1 (ηp^2) [p value]	Training F2(ηp^2) [p value]	Interaction F3(ηp^2) [p value]
CRP (mg/l)	1.57 ± 0.62	1.64 ± 0.58	2.62*# ± 0.68	1.63 ± 0.63	1.68 ± 0.29	1.82$ ± 0.52	12.125 (0.041) [p≤0.001]	34.83 (0.19) [p≤0.001]	17.77 (0.11) [p≤0.001]
CK (U/L)	310.64 ±112.69	322.81 ± 113.38	392.04*# ± 89.80	315.20 ± 113.20	319.95 ± 108.93	340.97$ ± 94.26	1.74 (0.188) [0.06]	7.11 (0.04) [p≤0.001]	1.95 (0.014) [0.143]
LDH (U/L)	349.08 ± 87.46	364.68 ± 84.42	444.02*# ± 83.46	349.97 ± 94.95	361.00 ± 100.99	396.54$ ± 96.34	2.41 (0.008) [0.121]	16.20 (0.10) [p≤0.001]	02.04 (0.014) [0.13]
Total protein (gm/dl)	6.92 ± 0.75	7.10 ± 0.87	7.67*# ± 0.83	6.86 ± 0.74	7.92*$ ±0.84	8.47*# $ ± 0.95	27.34 (0.088) [p≤0.001]	47.54 (0.25) [p≤0.001]	8.56 (0.057) [p≤0.001]
Urea (mg/dl)	25.86 ± 7.53	34.13* ± 8.40	37.78* ± 7.77	25.33 ± 7.43	25.86$ ± 7.53	44.93*#$ ± 9.37	0.33 (0.001) [0.565]	98.27 (0.41) [p≤0.001]	22.07 (0.135) [p≤0.001]
Uric Acid (mg/dl)	6.34 ± 1.16	6.75 ± 1.09	7.90*# ± 0.87	6.23 ± 1.26	6.34 ± 1.16	7.67*# ± 1.11	3.57 (0.013) [0.060]	48.91 (0.25) [p≤0.001]	0.452 (0.637) [0.00]
AST (U/L)	35.84 ± 10.64	35.90 ± 10.51	35.87 ± 10.55	35.60 ± 10.87	35.84 ± 10.64	35.81 ± 10.33	0.009 (0.00) [0.924]	0.00 (0.00) [0.99]	0,00 (0.00) [0.99]
ALT (U/L)	39.63 ± 10.57	39.68 ± 11.04	48.06*# ± 10.34	39.51 ± 10.80	39.63 ± 10.57	45.38*# ± 9.53	0.592 (0.002) [0.442]	14.67 (0.94) [p≤0.001]	0.48 (0.00) [0.614]

[Data are presented as Mean ± SD. Two-way ANOVA was performed to examine the effects of Group, Training, and Group × Training interaction. The Bonferroni post-hoc test was applied for within-group comparisons. * Indicates significant difference compared with 0 week (p≤0.05), # indicates significant difference compared with 6 weeks (p≤0.05). F values, p values, and partial eta squared (ηp^2) are reported. An independent samples t-test was used to determine between-group differences. $ indicates a significant difference compared with the PCG (p≤0.05). Degrees of freedom for the independent t-test = 94; critical t = 1.986. SD = standard deviation, ANOVA = Analysis of Variance, PCG = placebo control group, WPSG = whey protein supplement group; CK=creatine kinase, CRP = C-Reactive Protein, LDH=Lactate Dehydrogenase, AST=Aspartate Aminotransferase, ALT=Alanine Aminotransferase]

Aspartate Aminotransferase (AST) levels showed no significant changes across the 12 weeks for either the PCG (35.84 ± 10.64 to 35.87 ± 10.55 U/L) or the WPSG (35.60 ± 10.87 to 35.81 ± 10.33 U/L). While the Alanine Aminotransferase (ALT) increased significantly from baseline to week 12 in both groups (PCG: 39.63 ± 10.57 to 48.06 ± 10.34 U/L; WPSG: 39.51 ± 10.80 to 45.38 ± 9.53 U/L), there was no significant difference found between the two groups.

In PCG, the systemic inflammation increased significantly as the C-Reactive Protein (CRP) of PCG increased to 2.62 ± 0.68 mg/l at week 12 from 1.57 ± 0.62 mg/l at baseline. On the contrary, the WPSG maintained significantly lower levels, ending the trial at 1.82 ± 0.52 mg/l. After 12 weeks, a significant elevation was shown in CK levels of PCG (310.64 ± 112.69 U/L to 392.04 ± 89.80 U/L). Whereas the WPSG showed an inhibited response (315.20 ± 113.20 U/L to 340.97 ± 94.26 U/L), which indicates significantly lower CK levels compared to the placebo group. A similar pattern was also observed in Lactate Dehydrogenase (LDH). A significant increase, rising from 349.08 ± 87.46 U/L to 444.02 ± 83.46 U/L, can be shown in PCG. The WPSG reduced the increase from 349.08 ± 87.46 U/L to 444.02 ± 83.46 U/L, which was significantly lower than the PCG.

A significant increase in total protein was found after 12 weeks among the WPSG (6.86 ± 0.74 gm/dl to 8.47 ± 0.95 gm/dl) compared to PCG (6.92 ± 0.75 to 7.67 ± 0.83). Compared to the PCG (25.86 ± 7.53 to 37.78 ± 7.77), urea levels also exhibited a significant increase in the WPSG, rising from 25.33 ± 7.43 mg/dl to 44.93 ± 9.37 mg/dl at week 12. Uric acid increased significantly after only 3rd phage in both groups, but between groups, there was no difference.

III. DISCUSSION

The study's objective was primarily fixed to evaluate the synergistic effect of a 12-week structured resistance training program and whey protein supplementation on body composition, performance determinants, and inflammatory biomarkers in male football players. Given the critical role of physical fitness and recovery in elite football, understanding the specific benefits of protein sources is of paramount importance. The data obtained from the present study exhibit that the strategic integration of whey protein significantly enhanced the growth and increase in lean body mass, maximal strength, speed, and power, while modulating systemic inflammation and cellular turnover. A notable increased Boby Weight (BW), LBM, and MUAC observed among the WPSG demonstrates the efficacy of protein supplementation in optimizing

the anabolic environment. Performance-enhancing qualities can be improved by RT; however, protein is crucial for boosting muscle protein kinetics and promoting muscle protein synthesis (MPS). A total daily protein intake of approximately 1.6 to 2.2 g/kg body mass/day seems to be the most important factor for optimizing muscle mass accretion [Kerksick et al.,2018]. Whey protein is well known for its high leucine content, a primary amino acid agonist that stimulates MPS [Joy et al., 2013; Park et al., 2019; Wang & Wu, 2023].

The grip strength (GSR and GSL) is considered a reliable proxy of total peripheral strength. On the other hand, the abdominal strength indicates core muscular endurance. Both types of strength are important for maintaining balance, succeeding in physical duals, and avoiding injuries in football. In the current study, a significantly greater gain in GSR and GSL, along with abdominal strength, demonstrates the whey protein's potent anabolic efficiency when it is combined with structured muscular overload. Both the leg and back strength of PCG and WPSG had improved significantly and progressively. However, the strength adaptation of the lower-body and posterior chain was significantly improved by Whey protein ingestion. Previous studies also observed these types of localized strength improvements due to the effects of whey protein ingestion. Obradović et al. (2020) demonstrated that male college athletes taking whey protein supplementation whey protein supplementation in male college athletes obtained significantly greater increases in localized strength measures compared to a placebo [Obradović et al., 2020].

The integration of whey protein alongside a 12-week structured resistance training program successfully maximized the explosive capabilities, evidenced by the significantly superior Highest Power, Average Power, and Anaerobic Capacity observed in the whey-supplemented footballer. Vertical and horizontal jumping parameters (CMJ and SBJ) are critical indicators of lower-body explosive power and force production. The current data demonstrate that while resistance training alone enhances explosive power, the addition of whey protein significantly magnifies these adaptations. In the present study, performance variables critical to football match-play, such as sprint speed, agility, and anaerobic power, responded highly favourably to the combined intervention of RT and whey protein supplementation. Sprint time significantly decreased, and agility test times improved in the whey group. Interestingly, these robust improvements contrast with some previous literature.

Previous studies also indicated that protein supplementation (whey, soy, or plant-derived) frequently has little to no significant impact on indirect measures of explosive strength, such as countermovement jump (CMJ) or 30-meter sprint timings, specifically when overall dietary protein intake is already sufficient [Kritikos et al., 2021; Teixeira et al., 2022]. In another comparative study between whey protein and rice protein, in combination with structured RT, whey protein significantly increases the strength performance in college-aged males [Joy et al., 2013]. The greater adaptability in power and speed performance observed in the current study could be linked to the restricted 12-week RT protocol. Previous studies suggested that the prolonged structured RT significantly affects the force-generating ability of skeletal muscle. The combination of essential amino acids from whey is likely to be beneficial for athletes to maximize their mechanical output during training, because it enhances inter-session recovery. This greater recovery helps athletes to generate higher anaerobic capacity and force [Kritikos et al., 2021; Teixeira et al., 2022].

A central finding of this study is the divergent inflammatory and physiological responses between the two groups. Disruption or damage to muscle fibres is consistently caused by rigorous resistance training. In the present study, this was evidenced by the significant rise in C-Reactive Protein (CRP) in the placebo group. The whey protein group, however, successfully blunted this systemic inflammatory spike. This supports the concept that adequate protein intake is crucial for decreasing undesirable inflammatory reactions and aiding in the recovery of strength.

Rigorous resistance training causes structural disruption and damage to muscle fibres, elevating circulating systemic markers [Morton et al.,2018]. A critical and highly functional outcome of this study is the protective effect of whey protein against this cellular damage. The placebo group experienced massive, compounding elevations in Creatine Kinase (CK) and Lactate Dehydrogenase (LDH) by week 12. In stark contrast, the whey protein group successfully attenuated these markers, maintaining significantly lower levels of both CK and LDH. These findings strongly align with established sports nutrition literature. As Nieman et al. (2020) demonstrated, whey protein supplementation effectively decreases the rise of muscle damage biomarkers like CK and myoglobin compared to a control following intense exercise [Nieman et al., 2020]. Similarly, Lollo et al. (2014) established that whey protein administration significantly decreases both CK and LDH, preserving normal muscle function during the intense training regimes of a championship season [Lollo et al.,2014]. While some acute trials, such as Mor & Ipekoglu (2018), suggested that a single dose of whey protein might not immediately lower muscle damage values (including LDH and CK) post-exercise, our 12-week longitudinal data confirms that chronic, consistent whey supplementation provides a powerful, cumulative protective effect, reducing undesirable

structural disruption and expediting muscle recovery in football players [Mor & Ipekoglu, 2018].

Finally, hepatic and systemic stress markers (AST and ALT) provided further insight into the metabolic toll of the intervention. While AST levels remained stable, ALT levels increased significantly over the 12 weeks for both the whey and placebo cohorts, with no statistical difference between them. Previous research by Mor & Ipekoglu (2018) noted that acute whey protein consumption does not produce any immediate positive effect on measured muscle damage values, including ALT and AST [Mor & Ipekoglu, 2018]. Furthermore, recent comparative trials by Loureiro et al. (2023) demonstrated that pea protein actually resulted in significantly lower ALT and CK levels post-game compared to whey protein, suggesting plant proteins might offer unique systemic recovery advantages. However, our data indicates that over a 12-week chronic resistance training block, whey protein does not disproportionately elevate liver transaminases (ALT) compared to a carbohydrate placebo, affirming its safety and metabolic viability alongside its superior anabolic benefits [Loureiro et al.,2023].

IV.CONCLUSSION

The present study concludes that the supplementation of whey protein with a combination of RT can significantly promote the development of body composition, strength, speed, power, and agility among male football players. Whey protein enhanced the training-induced adaptation by improving the LBM, compared to resistance training alone. The supplementation also effectively supports improved recovery and reduced physiological stress by considerably attenuating exercise-induced inflammatory responses and muscle damage markers such as CRP, CK, and LDH. Despite the ALT levels increasing in both groups, the whey protein did not show any adverse hepatic effect, confirming it is safe during the prolonged training period. Broadly, the findings of the current study suggest that the chronic supplementation of whey protein combined with periodized resistance training is a highly efficient nutritional strategy for improving performance determinants and recovery of non-elite football players, specifically from rural areas with limited access to nutritional strategies and scientific training.

References

1. **Kulkarni, K., Levin, G. T., Penailillo, L., & Singh, A. (**2013). Physical and physiological characteristics of elite Indian national footballers.

2. **Pomeroy, E., Mushrif-Tripathy, V., Cole, T. J., Wells, J. C., & Stock, J. T.** (2019). Ancient origins of low lean mass among South Asians and implications for modern type 2 diabetes susceptibility. Scientific Reports, 9(1), 10515.

3.**Kang, H**. (2021). Sample size determination and power analysis using the G*Power software. Journal of Educational Evaluation for Health Professions, 18, 17. https://doi.org/10.3352/jeehp.2021.18.17

4.**Castell, G. S., Serra-Majem, L., &Ribas-Barba, L**. (2015). What and how much do we eat? 24-hour dietary recall method. Nutricion Hospitalaria, 31(3), 46-48.

5.**Chandrasekar, S. J. A., Govindasamy, K., & Govarthanan, K**. (2023). Test and measurement in physical education (8th ed.). AkiNik Publications. https://doi.org/10.22271/ed.book.2008

6.**Siri, W. E.** (1961). Body composition from fluid spaces and density: Analysis of methods. Nutrition, 9, 480–491. PMID: 8286893

7.**Burgess, K., Holt, T., Munro, S., & Swinton, P.** (2016). Reliability and validity of the Running Anaerobic Sprint Test (RAST) in soccer players. Journal of Trainology, 5(2), 24–29. https://doi.org/10.17338/trainology.5.2_24

8.**McArdle, W. D., Katch, F. I., & Katch, V. L**. (2015). Essentials of exercise physiology (5th ed.). Lippincott Williams & Wilkins.

9.**Bangsbo, J., Iaia, F. M., & Krustrup, P.** (2008). The Yo-Yo intermittent recovery test. Sports Medicine, 38(1), 37–51. https://doi.org/10.2165/00007256- 200838010-00004

10.**Tabassum, Y., Amjad, S., & ud Din, B. M.** (2023). Investigating the Effects of High Intensity Resistance Training on Physical Fitness of University Male Football Players. THE SKY-International Journal of Physical Education and Sports Sciences (IJPESS), 7, 27-34.

11.**Williamson, E., Kato, H., Volterman, K. A., Suzuki, K., & Moore, D. R.** (2019). The effect of dietary protein on protein metabolism and performance in endurance-trained males. Medicine Science and Sports Exercise, 51(2), 352-60.

12.**Jacinto, J. L., Nunes, J. P., Gorissen, S. H., Capel, D. M., Bernardes, A. G., Ribeiro, A. S., ... & Aguiar, A. F.** (2022). Whey protein supplementation is superior to leucine-matched collagen peptides to increase muscle thickness during a 10-week resistance training program in untrained young adults. International Journal of Sport Nutrition and Exercise Metabolism, 32(3), 133-143.

13.**Kerksick, C. M., Wilborn, C. D., Roberts, M. D., Smith-Ryan, A., Kleiner, S. M., Jäger, R., ... & Kreider, R. B. (**2018). ISSN exercise & sports nutrition review update: research & recommendations. Journal of the international society of sports nutrition, 15(1), 38.

14.**Joy, Jordan M., Ryan P. Lowery, Jacob M. Wilson, Martin Purpura, Eduardo O. De Souza, Stephanie MC Wilson, Douglas S. Kalman, Joshua E. Dudeck, and Ralf Jäger**. "The effects of 8 weeks of whey or rice protein supplementation on body composition and exercise performance." Nutrition journal 12, no. 1 (2013): 86.

15.**Park, Y., Park, H. Y., Kim, J., Hwang, H., Jung, Y., Kreider, R., & Lim, K.** (2019). Effects of whey protein supplementation prior to, and following, resistance exercise on body composition and training responses: A randomized double-blind placebo-controlled study. Journal of exercise nutrition & biochemistry, 23(2), 34-44.

16.**Lollo, P. C. B., Amaya-Farfan, J., Faria, I. C., Salgado, J. V. V., Chacon-Mikahil, M. P. T., Cruz, A. G., ... & Arruda, M. (**2014). Hydrolysed whey protein reduces muscle damage markers in Brazilian elite soccer players compared with whey protein and maltodextrin. A twelve-week in-championship intervention. International dairy journal, 34(1), 19-24.

17.**Mor, A., & Ipekoglu, G**. (2018). The effect of whey protein supplementation on exercise-induced muscle damage. Int J Med Sci Public Health, 7(7), 132-137.

18.**Nieman, D. C., Zwetsloot, K. A., Simonson, A. J., Hoyle, A. T., Wang, X., Nelson, H. K., ... & Guérin-Deremaux, L**. (2020). Effects of whey and pea protein supplementation on post-eccentric exercise muscle damage: a randomized trial. Nutrients, 12(8), 2382.

19.**Obradović, J., Vukadinović Jurišić, M., & Rakonjac, D.** (2020). The effects of leucine and whey protein supplementation with eight weeks of resistance training on strength and body composition. J Sports Med Phys Fitness, 60(6), 864-869.

20.**Teixeira, F. J., Matias, C. N., Faleiro, J., Giro, R., Pires, J., Figueiredo, H., ... & Schoenfeld, B. J.** (2022). A novel plant-based protein has similar effects compared to whey protein on body composition, strength, power, and aerobic performance in professional and semi-professional futsal players. Frontiers in Nutrition, 9, 934438.

21.**Loureiro, L. L., Ferreira, T. J., Cahuê, F. L. C., Bittencourt, V. Z., Valente, A. P., & Pierucci, A. P. T. R.** (2023). Comparison of the effects of pea protein and whey protein on the metabolic profile of soccer athletes: a randomized, double-blind, crossover trial. Frontiers in Nutrition, 10, 1210215.

22.**Kritikos, S., Papanikolaou, K., Draganidis, D., Poulios, A., Georgakouli, K., Tsimeas, P., ... & Fatouros, I. G**. (2021). Effect of whey vs. soy protein supplementation on recovery kinetics following speed endurance training in competitive male soccer players: a randomized controlled trial. Journal of the International Society of Sports Nutrition, 18(1), 23.

23.**Banerjee, B. (2018).** Mahajan's methods in biostatistics for medical students and research workers (9th ed.). Jaypee.

24.**Manna, I., Khanna, G. L., & Dhara, P. C. (**2010). Effect of training on physiological and biochemical variables of soccer players of different age groups. Asian journal of sports medicine, 1(1), 5.

25.**Jackman, S. R., Witard, O. C., Philp, A., Wallis, G. A., Baar, K., & Tipton, K. D**. (2017). Branched-chain amino acid ingestion stimulates muscle myofibrillar protein synthesis following resistance exercise in humans. Frontiers in physiology, 8, 266695.

26.**Sousa, M., Teixeira, V. H., & Soares, J.** (2014). Dietary strategies to recover from exercise-induced muscle damage. International journal of food sciences and nutrition, 65(2), 151-163.

27.**Wang B & Wu G** (2023). Dietary leucine and muscle health: Molecular mechanisms and clinical implications. Frontiers in Nutrition, 10, 1184456.

28.**Morton, R. W., Murphy, K. T., McKellar, S. R., Schoenfeld, B. J., Henselmans, M., Helms, E., ... & Phillips, S. M**. (2018). A systematic review, meta-analysis and meta-regression of the effect of protein supplementation on resistance training-induced gains in muscle mass and strength in healthy adults. British journal of sports medicine, 52(6), 376-384.

Original Article

An institutional study on determining the optimal minimum segment width for Intensity Modulated Radiation Therapy plans in base-of-tongue cancer

Priya Saini, Mary Joan, Anirudh Pradhan

Abstract

Purpose: The aim of this study was to determine the optimal minimum segment width (MSW) for intensity-modulated radiotherapy (IMRT) planning in base-of-tongue (BOT) cancer, with a focus on balancing dosimetric quality and delivery efficiency.

Materials and Methods: A retrospective analysis was performed on 30 BOT cancer patients treated with IMRT. For every patient, four distinct treatment plans were created, corresponding to MSW settings of 0.5 cm, 1.0 cm, 1.5 cm, and 2.0 cm, while all other optimization and constraint parameters remained constant throughout. Dosimetric evaluation included target coverage metrics (D95, Dmean, Dmax), conformity index (CI), homogeneity index (HI), and doses to organs at risk (OARs). Delivery efficiency was assessed using monitor units (MUs) and treatment time. Statistical differences across MSW settings were analyzed using the Wilcoxon signed-rank test.

Results: Increasing MSW was associated with a progressive reduction in CI and decreased PTV dose coverage (D95) for both PTV70 and PTV56. Plans generated with MSW values of 1.5 cm and 2.0 cm failed to meet the clinical D95 requirement for PTV70, while the 2.0 cm MSW also did not satisfy requirements for PTV56. HI increased with larger MSW, whereas Dmean and Dmax remained statistically unchanged across the four plan groups ($p > 0.05$). The MSW 0.5 cm plans produced significantly higher D95 values ($p < 0.02$). All OAR doses remained within recommended tolerance limits. Monitor units and treatment time decreased as MSW increased; however, the MSW 0.5 cm plans required the highest MU (1016) and demonstrated reduced delivery efficiency.

Conclusion: Although larger MSWs improved delivery efficiency, smaller MSWs yielded superior dosimetric quality. An MSW of 1.0 cm provided the optimal balance between target coverage, plan conformity, and delivery efficiency, making it the most suitable setting for BOT cancer IMRT planning.

JK-Practitioner 2026-31(2):63-69

Introduction

Base-of-tongue (BOT) cancer is a subtype of oropharyngeal squamous cell carcinoma and is increasingly associated with human papillomavirus (HPV) infection.[1,2] Owing to the complex anatomy of the tongue base, its deep position , and its close adjacency to several normal structures, the management of BOT cancer demands highly precise and advanced radiotherapy approaches. Intensity-Modulated Radiation Therapy (IMRT) has become a preferred technique in this setting, as it allows the delivery of radiation with modulated beam intensities, enabling improved dose conformity to the tumor while minimizing radiation exposure to surrounding OARs. [3] In the Monaco Treatment Planning System (TPS), the MSW specifies the smallest permissible width of segments formed by the multi-leaf collimator (MLC). Within this system, MSW values typically range from 0.5 cm to 2.0 cm, and this parameter plays a key role in influencing both the quality of the treatment plan and its deliverability. [4-10] Despite its significance, there is a lack of published research specifically examining how MSW variations affect IMRT plans for patients with base-of-tongue (BOT) cancer. Therefore, the objective of this study is to evaluate the influence of different MSW settings on the dosimetric quality and deliverability of BOT cancer IMRT plans.

Author Affiliations

Priya Saini, Department of Radiological Physics, S.M.S Medical College & Hospitals, Jaipur-302004 (Rajasthan) India and Department of Physics, Institute of Applied Science and Humanities, GLA University, Mathura-281406(U.P), India. **Mary Joan,** Department of Radiation Oncology, Christian Medical College, Ludhiana, Punjab, India. **Anirudh Pradhan,** Centre for Cosmology, Astrophysics and Space Science, GLA University, University, Mathura-281406(U.P), India.

Correspondence

Priya Saini,
Department of Radiological Physics, S.M.S Medical College & Hospitals, Jaipur-302004 (Rajasthan) India and Department of Physics, Institute of Applied Science and Humanities, GLA University, Mathura-281406(U.P), India.
Email: priyasaininkt@gmail.com
Phone no. +9109869807392

Indexed

EMBASE, SCOPUS, IndMED , ESBCO, Google Scholar besides other national and international databases.

Cite this article as

Saini P, Joan M, Pradhan A.An institutional study on determining the optimal minimum segment width for Intensity Modulated Radiation Therapy plans in base-of-tongue cancer. JK-Pract2026;31(2):63-69

Full length article available at **jkpractitioner.com** one month after publication

Keywords

Intensity modulated radiation therapy, dose volume histogram, BOT cancer, minimum segment width

Material and Methods

Simulation and Preparation of Patients

This study included thirty patients with BOT cancer who received IMRT at our institution from March 2022 to March 2023. For each patient, CT simulation was performed in a stable and reproducible supine position using a four-clamp, customized thermoplastic immobilization mask mounted on an acrylic base plate with an appropriate headrest. A slice thickness of 2.5 mm was used to acquire images on a GE Discovery 16-slice spiral CT scanner. The resulting CT datasets were then transferred to the Monaco Treatment Planning System in DICOM format via the local area network for further treatment planning.

Structures Delineation and Radiation Dose Prescription

Gross tumor volume (GTV), clinical target volume (CTV), and the surrounding normal structures were delineated by a skilled oncologist in accordance with RTOG guidelines. A uniform margin of 10 mm was expanded around the GTV, including involved lymph nodes, and a 3–5 mm margin was added around the CTV to generate the planning target volumes (PTVs). These were prescribed doses of 70 Gy (PTV70) and 56 Gy (PTV56), respectively. Critical organs such as the spinal cord, parotid glands, lips, brainstem, and thyroid were contoured as OARs.

Treatment Plan Development Using the Monaco TPS

For each patient, four IMRT plans were developed in the Monaco TPS (version 6.1.2.0) using MSW settings of 0.5 cm, 1.0 cm, 1.5 cm, and 2.0 cm. They were named MSW 0.5, MSW 1.0, MSW 1.5 , and MSW 2.0 respectively. Only MSW value was varied in all plans. Cost functions and remaining parameters were kept constant. Dose calculation grid size was used 3mm and maximum dose rate was used 600 MU/min. Treatment plans were delivered with 6MV X-ray photon beam using elekta versa HD linear accelerator. Nine fields with gantry angles 200º, 250º, 300º, 0º, 50, 100º and 150º were used to designed IMRT plan.

Figure 1: Elekta versa HD linear accelerator

Analysis of Treatment Plan Quality

The dosimetric quality of IMRT plans designed with different MSW was evaluated using a range of quantitative parameters. These included the median dose (D50%), maximum dose (D2%), minimum dose (D98%), dose homogeneity index (HI), conformity index (CI), the percentage of the PTV covered by 95% of the prescription dose, and dose–volume histogram (DVH) metrics for all relevant organs at risk (OARs). Plan complexity was assessed through the total number of monitor units (MUs), while plan delivery efficiency was determined using the plan delivery time (PDT) and the gamma passing rate (GPR) from patient-specific quality assurance.

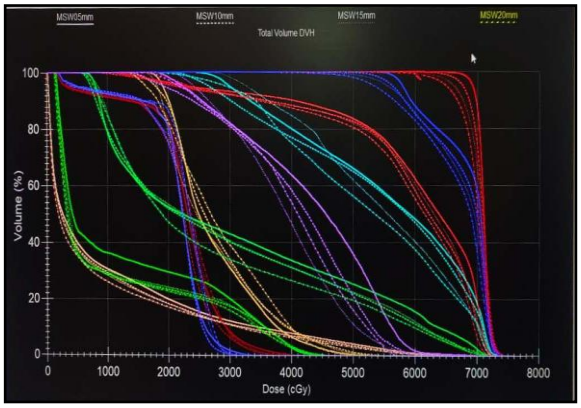
The CI and HI values for the target volumes were calculated according to the recommendations of the ICRU Report 83 using equations (1) and (2): [6]

$$HI=(D_{2\%}-D_{98\%})/D_{50\%} \quad (1)$$

$$CI = (TV_{RI})^2 / (TV * V_{RI}) \quad (2)$$

In these equations, $D_{x\%}$ denotes the minimum dose received by (x %) of the PTV; (TV) represents the total target volume; TV_{RI} indicates the target volume encompassed by the prescription isodose; and V_{RI} refers to the overall volume covered by the prescription dose.

Figure 2: The Dose volume histogram of four different IMRT plans with different MSW for a typical BOT cancer patient



Verification of Treatment Plan

Octavius II system was used for dosimetric verification of the plan. The criterion for GPR was dose to distance agreement 3%, 3mm. MU, GPR, and PDT were noted.

Statistical Analysis

Statistical evaluations were performed using Primer software. The Wilcoxon signed-rank test was applied to compare the dosimetric and delivery metrics among

the different MSW groups. The MSW 0.5 cm group served as the reference for all comparisons. p-values were calculated for each parameter, with a value of $p < 0.05$ considered indicative of a statistically significant difference.

Result:

Target Dose Analysis

Target Dosimetric parameters of four different MSW IMRT plan group were listed in Table 1and Table 2. CI values [Figure 3and 4] and PTV coverage (D_{95}) of both PTVs are decreased as the MSW value increased. For PTV 70, MSW1.5 and MSW2.0 IMRT plans group failed to satisfy clinical criteria, i.e., for MSW 2.0 group $D_{95\%}$=65.31 Gy (93.3 % of 70 Gy) and for MSW 1.5 group $D_{95\%}$ = 65.06 (92.94% of 70Gy). For PTV 56 IMRT plan group MSW 2.0 failed to satisfy clinical criteria, i.e, D95% = 50.31 (71.87% of 70 Gy). HI values for both PTVs are increased as MSW value increased [Figure 5 and 6]. The mean and maximum dose of PTV70 and PTV56 were not markedly different among the all four plans ($p>0.05$). In term of dose coverage $D_{95\%}$ MSW 0.5 plan group had significant difference from another three group for both PTVs ($p<0.02$).

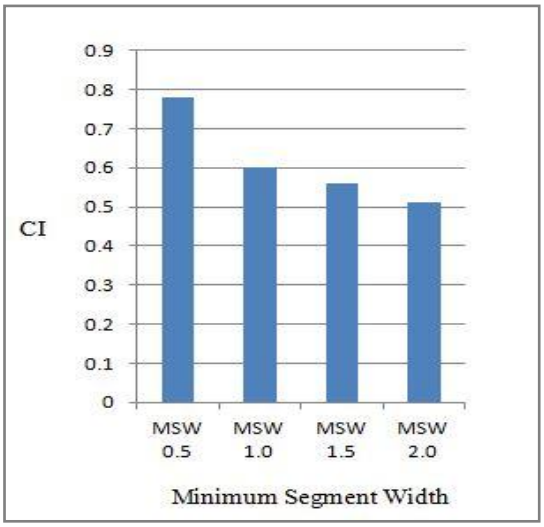


Figure 3: Graphical representation of CI for PTV70 of four different MSW settings
CI: Conformity Index

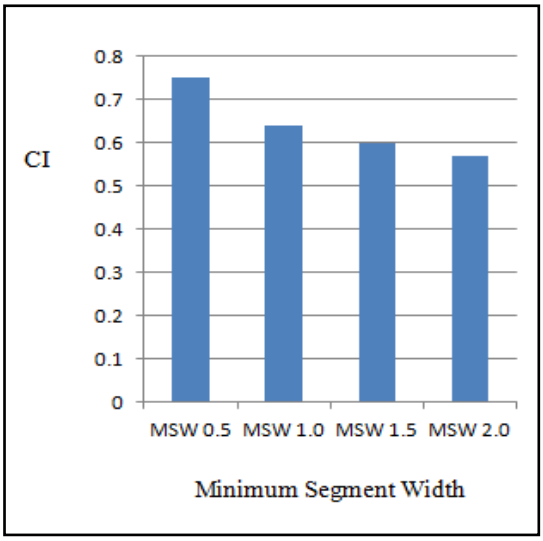


Figure 4: Graphical representation of CI for PTV56 of four different MSW setting
CI: Conformity Index

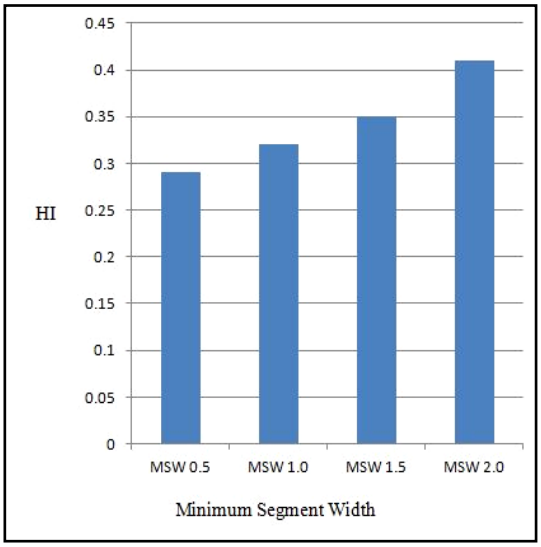


Figure 5: Graphical representation of HI for PTV56 of four different MSW settings
HI: Homogenity Index

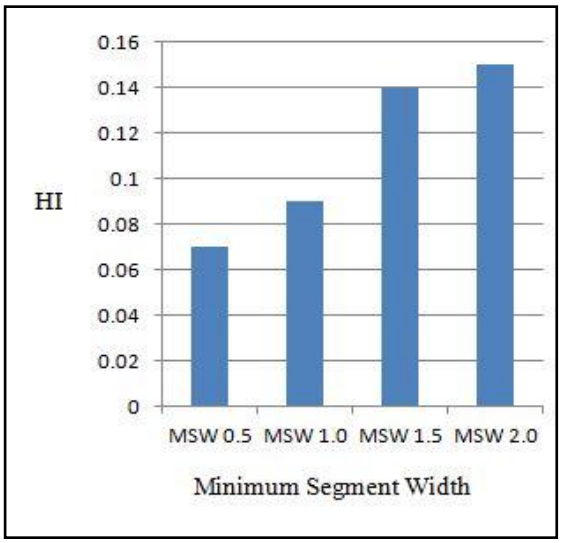


Figure 6: Graphical representation of HI for PTV70 of four different MSW settings
HI: Homogenity Index

Table 1. Dosimetric comparison of PTV56 for four MSW settings of BOT Cancer plans

Structure	Parameter	MSW 0.5	MSW 1.0	MSW 1.5	MSW 2.0	P1	P2	P3	P4
PTV56	PTV Coverage 95% (Gy)	55.2 ± 0.47	53.4 ± 0.52	53.38 ± 0.42	50.31 ± 0.42	<0.02	<0.02	<0.02	>0.05
	Mean Dose (Gy)	57.22 ± 0.25	57.19 ± 0.43	56.31 ± 0.30	56.10 ± 0.30	>0.05	>0.05	>0.05	>0.05
	Maximum Dose (Gy)	61.8 ± 0.30	60.91 ± 0.54	61.47 ± 0.54	61.31 ± 0.54	>0.05	>0.05	>0.05	>0.05
	HI	0.29 ± 0.03	0.32 ± 0.02	0.35 ± 0.01	0.41 ± 0.01	>0.05	>0.05	>0.05	>0.05
	CI	0.75 ± 0.10	0.64 ± 0.18	0.60 ± 0.17	0.57 ± 0.16	>0.05	>0.05	>0.05	>0.05

P1: p-value of comparison between MSW 0.5 and MSW 1.0, **P2:** p-value of comparison between MSW 0.5and MSW 1.5, **P3:** p-value of comparison between MSW 0.5and MSW 2.0, **P4:** p-value of comparison between MSW1.0 and MSW 1.5, HI: Homogeneity index, CI: Conformity Index

Table2. Dosimetric comparison of PTV70 for four MSW settings of BOT Cancer plans

Structure	Parameter	MSW 0.5	MSW 1.0	MSW 1.5	MSW 2.0	P1	P2	P3	P4
PTV70	PTV Coverage 95% (Gy)	68.72 ± 0.17	67.09 ± 0.27	65.00 ± 0.35	65.31 ± 0.30	< 0.02	< 0.02	< 0.02	> 0.05
	Mean Dose (Gy)	70.98 ± 0.45	69.20 ± 0.35	69.38 ± 0.41	69.59 ± 0.32	> 0.05	> 0.05	> 0.05	> 0.05
	Maximum Dose (Gy)	75.86 ± 0.30	74.91 ± 0.54	75.35 ± 0.72	75.47 ± 0.72	> 0.05	> 0.05	> 0.05	> 0.05
	HI	0.07 ± 0.01	0.09 ± 0.01	0.14 ± 0.01	0.15 ± 0.01	< 0.02	< 0.02	< 0.02	> 0.05
	CI	0.78 ± 0.08	0.60 ± 0.06	0.56 ± 0.12	0.51 ± 0.12	< 0.02	< 0.02	< 0.02	> 0.05

P1: p-value of comparison between MSW 0.5 and MSW 1.0, **P2:** p-value of comparison between MSW 0.5and MSW 1.5, **P3:** p-value of comparison between MSW 0.5and MSW 2.0, **P4:** p-value of comparison between MSW1.0 and MSW 1.5, HI: Homogeneity index, CI: Conformity Index

OARs Dose Analysis

OARs doses of all four groups were shown in Table 3. All OARs doses were within tolerance, although they were not directly comparable for all four group plans

Dosimetric verification, MU and plan delivery time

GPR, PDT and MUs for all four groups were shown in Table 4. Dosimetric plan evaluation was done using a comparison between measured planer dose and calculated dose by TPS studying the gamma passing criteria of a 3% dose difference (DD) and a 3mm distance to agreement (DTA). MSW2.0 group plan had highest GPR value and MSW0.5 group plan had lowest GPR value. MUs of the IMRT plans decreased as the MSW increased (Table 4).The MSW0.5 group

had the highest MUs (1016MUs).The PDT decreased with increasing MSW ($p < 0.05$). However plans with 0.5 cm MSW had worse delivery accuracy and efficiency than other groups. There were no significant difference between MSW 1.5 and MSW1.0 group in term of MUs (p4>0.05).

.

Table3. Dosimetric comparison of OAR doses across four MSW settings of BOT Cancer plans

OARs	**Parameter**	**MSW 0.5**	**MSW 1.0**	**MSW 1.5**	**MSW 2.0**	**P1**	**P2**	**P3**	**P4**
Brainstem	Dmax (Gy)	32 ± 2.33	34.9 ± 2.25	36.5 ± 2.50	37.9 ± 2.56	P>0.05	P>0.05	P>0.05	P>0.05
Spine	Dmax (Gy)	36 ± 2.00	34.9 ± 3.80	35.9 ± 2.96	36.6 ± 3.15	P>0.05	P>0.05	P>0.05	P>0.05
Thyroid	Dmean (Gy)	41.2 ± 4.13	42.7 ± 5.46	42.5 ± 4.40	42.5 ± 6.00	P>0.05	P>0.05	P>0.05	P>0.05
Lip	Dmean (Gy)	26.4 ± 13.3	27.1 ± 8.36	26.6 ± 12.8	27.2 ± 4.56	P>0.05	P>0.05	P>0.05	P>0.05
Right Parotid	Dmean (Gy)	22.7 ± 3.20	21.7 ± 3.25	21.3 ± 2.15	21.3 ± 2.50	P>0.05	P>0.05	P>0.05	P>0.05
Left Parotid	Dmean (Gy)	23.6 ± 2.0	22.9 ± 3.27	23.8 ± 1.50	22.4 ± 1.23	P>0.05	P>0.05	P>0.05	P>0.05

Dmax: maximum dose, Dmean ; Mean Dose

Table4. Monitor units and plan delivery time comparison of four MSW settings of BOT Cancer plans

Parameter	**MSW 0.5**	**MSW 1.0**	**MSW1.5**	**MSW 2.0**
MUs	1016.76±54.414	716.32± 46.23	667.55±53.11	693.12±109.64
PDT(mint)	5.00 ± 0.50	4.43 ± 0.32	4.20±0.30	4.02±0.40
GPR	95.5± 1.32	96.37±1.30	96.40±1.45	97.2±1.5

MSW: Minimum segment width, MUs: Monitor units, PDT: Plan delivery time
IMRT: Intensity modulated radiation therapy, MSW: Minimum Segment Width

Discussion

The sequencing parameters in a treatment planning system can influence both plan quality and clinical deliverability to a degree comparable to that of dosimetric constraints.[11-19] In the Monaco TPS, MSW is a key sequencing parameter that specifies the smallest allowable width of multileaf collimator (MLC) segments.[20] Designing IMRT plans for base-of-tongue (BOT) cancer often results in numerous small, narrow, and irregular apertures. Because MSW directly influences the shape and size of these apertures, it plays a critical role in generating optimized segments. When MSW values are not selected appropriately, plans may contain excessively small or elongated segments, which can compromise verification results and even cause interruptions during clinical delivery. [20-26] In this study, four IMRT optimization schemes were compared; each created using a different MSW value. The findings indicate that MSW not only affects target dose distribution and sparing of organs at risk (OARs) but also has a notable impact on PDT and MU. The plan generated with MSW 0.5 cm served as the reference group. This group achieved the highest PTV coverage at the 95% dose level, whereas MSW 2.0 cm produced the lowest coverage. For PTV70, the MSW 1.5 cm and MSW 2.0 cm groups failed to meet clinical criteria, and for PTV56, the MSW 2.0 cm group also did not satisfy requirements. Doses to

OARs across all four MSW groups remained within acceptable tolerance levels.

As MSW increased, both MUs and PDT decreased, while gamma passing rate (GPR) increased. The MSW 1.0 cm plans exhibited the highest MU and PDT values but showed the lowest GPR. Conversely, although the MSW 0.5 cm group demonstrated superior dose conformity, homogeneity, and overall dose distribution, its treatment efficiency and delivery accuracy were inferior to those of the other groups. No statistically significant differences were observed between the MSW 1.0 cm and MSW 1.5 cm groups regarding PTV95% coverage, mean dose, maximum dose, or OAR doses ($p > 0.05$) for both target volumes.

Plans created with MSW 2.0 cm and MSW 1.5 cm showed poorer dose distribution and failed to meet clinical target coverage criteria; however, they achieved better treatment efficiency and higher delivery accuracy. Overall, increasing MSW values resulted in fewer MUs, shorter PDT, and higher GPR. The MSW 0.5 cm group required the greatest number of MUs and longest PDT but produced the lowest GPR (>95%) among all groups. Reductions in MU and PDT achieved with larger MSW settings may help minimize patient movement during treatment, enhance treatment efficiency, and potentially improve biological treatment effectiveness**.**

Conclusion

Based on a comprehensive assessment of all dosimetric and delivery parameters—including PTV95% coverage, OAR sparing, homogeneity index (HI), conformity index (CI), maximum and mean doses, monitor units (MUs), planning delivery time (PDT), and gamma passing rate (GPR)—we observed that the MSW 1.5 cm and MSW 2.0 cm groups did not fulfill the clinical requirements for acceptable IMRT plan quality. Although the MSW 0.5 cm group achieved strong dose conformity and homogeneity, it demonstrated poorer delivery accuracy and lower efficiency compared with the other groups. Overall, our findings indicate that an MSW value of 1.0 cm provides the most favorable balance between dosimetric quality and delivery efficiency for IMRT planning in base-of-tongue cancer. Therefore, an MSW of 1.0 cm may be considered the optimal setting for BOT patients undergoing IMRT technique.

Financial support and sponsorship

Nil.

Conflicts of interest

There are no conflicts of interest.

References:

1. Ang KK, Harris J, Wheeler R, Weber R, Rosenthal DI, Nguyen-Tân PF, et al. Human papillomavirus and survival of patients with oropharyngeal cancer. N Engl J Med 2010; 363(1):24–35.
2. Yom SS, Torres-Saavedra P, Caudell JJ, Waldron JN, Gillison ML, Xia P, et al. Reduced-dose radiation therapy for HPV-associated or pharyngeal cancer. J clin oncol 2021; 39(9):956-965.
3. Nutting CM , Morden JP, Harrington Kj, Harrington TG, Bhide SA, Clark C, et al. (2011). Parotid-sparing IMRT versus conventional RT in head and neck cancer (PARSPORT): a phase 3 multicentre randomised controlled trial. Lancet Oncol 2011; 12(2):127–136.
4. Sun H, Wang N, Wang X, Huang G, Chang Y, Liu Y. A study of different minimum segment area parameters on automatic IMRT plans for cervical cancer using Pinnacle3 910 TPS. Medicine (Baltimore) 2022; 101:29290.
5. Clements M, Schupp N, Tattersall M, Brown A, Larson R. Monaco treatment planning system tools and optimization processes. Med Dosim 2018;43:106–17.
6. International Commission on Radiation Units and Measurements. ICRU Report 83: Prescribing, Recording, and Reporting IMRT.Bethesda (MD): ICRU 2010.
7. Sun X, Xia P, Yu N. Effects of the intensity levels and beam map resolutions on static IMRT plans. Med Phy 2004; 31:2402–11.
8. Sun X, Xia P. A new smoothing procedure to reduce delivery segments for static MLC-based IMRT planning. Med Phys 2004; 31:1158–65.
9. Wang N, Chen L, Huang G, Sun H. Influence of minimum segment width on intensity-modulated radiotherapy plan for left sided breast after breast conversing surgery. Medicine (Baltimore) 2023; 102(46):e36064.
10. Jiménez-Puertas S, González Rodríguez A, Lozares Cordero S, González González T, Díez Chamarro J, Hernández Hernández M, et al. Evaluation of the Minimum Segment Width and Fluence Smoothing Tools for Intensity-modulated

techniques in Monaco Treatment Planning System. J Med Phys 2024; 49(2):250-260.
11. Sung H, Ferlay J, Siegel RL, Laversanne M, Soerjomataram I, Jemal A, et al. Global cancer statistics 2020: GLOBOCAN estimates of incidence and mortality worldwide for 36 cancers in 185 countries. CA cancer J clin 2021; 71:209-49.
12. Coselmon MM, Moran JM, Radawski JD, Fraass BA. Improving IMRT delivery efficiency using intensity limits during inverse planning. Med phys 2005; 32: 1234-45.
13. Ur Rehman J, Ahmad N, Khalid M, Gilani ZA, Ullah I, Nasar G, et al. Intensity modulated radiation therapy: A review of current practice and future outlooks. J Radiat Res Appl Sci 2018; 11: 361-7.
14. Baba MH, Singh BK, Wani SUQ: Skin sparing in intensity modulated radiation therapy of nasopharyngeal carcinoma. J Med Phys 47(3): 243-249, 2022. DOI: 10.4103/jmp.jmp_27_22
15. Guowei Z, Ziping J, Shepard D, Earl M, Yu C. Effect of beamlet step-size on IMRT plan quality. Med phys 2005; 32: 3448-54.
16. Sutterley C, Gautier Q, Graves Y, Li N, Jia X, Jiang S. SU-E-T-570: Optimizing Dose Calculation Parameters for GPU-Based Treatment Planning. Med Phys 2013; 40:336.
17. Hong J, Han JH, Luo HL, Song YQ. Optimization of Minimum Segment Width Parameter in the Intensity-Modulated Radiotherapy Plan for Esophageal Cancer. Int J Gen Med 2021; 16: 9913-21.
18. Wang Y, Chen L, Zhu F, Guo W, Zhang D, Sun W. A study of minimum segment width parameter on VMAT plan quality, delivery accuracy, and efficiency for cervical cancer using Monaco TPS. J Appl Clin Med Phys 2018; 19: 609-15.
19. Nguyen D, O'Connor D, Yu VY, Ruan D, Cao M, Low DA, et al. Dose domain regularization of MLC leaf patterns for highly complex IMRT plans. Med Phys 2015; 42: 1858-70.
20. Wu M, Jin J, Li Z, Kong F, He Y, Liu L, et al. Influence of beamlet width on dynamic IMRT plan quality in nasopharyngeal carcinoma. Peer J 2022; 10: 13748.
21. Coselmon MM, Moran JM, Radawski JD, Fraass BA. Improving IMRT delivery efficiency using intensity limits during inverse planning. Med phys 2005; 2: 1234-45.
22. Yoosuf AM, Ahmad MB, AlShehri S, Alhadab A, Alqathami M. Investigation of optimum minimum segment width on VMAT plan quality and deliverability: A comprehensive dosimetric and clinical evaluation using DVH analysis. J Appl Clin Med Phys 2021; 22: 29-40.
23. Jain N, Alok K, Ashok K. Investigating the influence of minimum segment width in volumetric-modulated arc planning of prostate cancer. Turk J Oncol 2024;39:84–91.
24. Nithiyanantham K, Kadirampatti Mani G, Subramani V, Karukkupalayam Palaniappan K, Uthiran M, Vellengiri S, et al. Influence of segment width on plan quality for volumetric modulated arc based stereotactic body radiotherapy. Rep Pract Oncol Radiother 2014; 19:287–95.
25. Moon YM, Bae SI, Choi CW, Jeon WW, Kim JY, Lee MW, et al. Effect of minimum segment width on gamma passing rate considering MLC position error for volumetric modulated arc therapy. J Korean Phys Soc 2019; 74:724–30.
26. Baba MH, Singh BK. In-vivo skin dose measurement using gafchromic EBT3 film dosimetry in the radiation therapy of Head and Neck cancers: 2DRT versus IMRT. J Radiat Res Appl Sci. 2022;15(2):170–174, ISSN 1687–8507. https:// doi. org/ 10. 1016/j. jrras. 2022. 05. 019. https:// www. scien cedir ect. com/ scien ce/ artic le/ pii/ S1687 85072 20718 01.

SUBMISSION CHECKLIST

Please make sure you have satisfied the points on the following checklist before submitting your manuscript.

1. Email addresses

Do you have a list of the email addresses for all the co-authors of the manuscript? You will need to provide these so that we can inform the co-authors when the manuscript has been received and when it is published.

2. Instructions for Authors , peer reviewers and editorial board selection

Have you read the detailed Instructions for Authors and are aware of the peer review and selection process.

3. Covering letter

Have you prepared a covering letter for your submission

4. Manuscript files

Do you have all the files for the manuscript in an acceptable format

5. Article-processing charges

An article processing charges are payable for articles accepted for publication in JK-Practitioner

6. Are you one of the authors of this article?

If not, you cannot submit the article on behalf of the authors. As part of the submission process you will assign a corresponding author for the article, and this can be yourself or a co-author Once the manuscript is submitted the contact author takes responsibility and receive correspondence sent throughout the peer review and production process.

7. Conditions of submission and JK-Practitioner Copyright and License Agreement

Do you confirm that all the authors of the manuscript have read and agreed to its content and are accountable for all aspects of the accuracy and integrity of the manuscript in accordance with ICMJE criteria? Do you confirm that you have ethical approval for any human or animal experimentation ? Do you confirm that the manuscript is original, has not already been published in a journal and is not currently under consideration by another journal? By submitting the manuscript , you are confirming these points and agreeing to these and all other terms of the JK-Practitioner Copyright and License Agreement

Yes, I am the submitting author of this article and I accept the conditions of submission and the JK-Practitioner Copyright and License Agreement.

Authorship Form

Each author should meet all criteria below (A, B, C, and D) and should indicate general and specific contributions by reading criteria A, B, C, and D and checking the appropriate boxes.

A. I certify t hat

• The manuscript represents original and valid work and that neither this manuscript nor one with substantially similar content under my authorship has been published or is being considered for publication elsewhere, except as described in the journal's submission form and cover letter submitted with the manuscript, and copies of closely related manuscripts have been provided; and I agree to be accountable for all aspects of the work in ensuring that questions related to the accuracy or integrity of any part of the work are appropriately investigated and resolved; and If requested, I will provide the data or will cooperate fully in obtaining and providing the data on which the manuscript is based for examination by the editors or their assignees; and For papers with more than 1 author, I agree to allow the corresponding author to serve as the primary correspondent with the editorial office, to review the edited manuscript and proof, and to make decisions regarding release of information in the manuscript to the media, federal agencies, or both; or, if I am the only author, I will be the corresponding author and agree to serve in the roles described above.

B. I have given final approval of the submitted manuscript.

C. I have participated sufficiently in the work to take public responsibility for (check 1 of 2 below)

- part of the content
- the whole content.

D. To qualify for authorship, you must check at least 1 box for each of the 3 categories of contributions listed below.

I have made substantial contributions to the intellectual content of the paper as described below.

1. Check at least 1 of 2 below
- conception or design
- acquisition, analysis, or interpretation of data

2. Check at least 1 of 2 below
- drafting of the manuscript
- critical revision of the manuscript for important intellectual content

3. Check at least 1 below
- statistical analysis
- obtaining funding
- administrative, technical, or material support
- supervision
- no additional contributions
- other

Acknowledgement Statement.

Authors should obtain written permission from all individuals named in an Acknowledgment, since readers may infer their endorsement of data and conclusions. The corresponding author must check the following box:

A. I certify that all persons who have made substantial contributions to the work reported in this manuscript (eg, data collection, analysis, writing or editing assistance) but who do not fulfill the authorship criteria are named, along with their specific contributions in an Acknowledgment in the manuscript and all such persons have provided me with written permission to be named.

Confirmation of Reporting of Funding and Conflicts of Interest. Please check A and B. Also check C or D.

A. I certify that all funding, or other financial support, and material support for this work are clearly identified in the manuscript.

B. I have completed the ICMJE for Disclosure of Potential Conflicts of Interest (available at http://www.icmje.org) and will submit it to JK-Practitioner

C. I certify that all my potential conflicts of interest (as reported in the ICMJE), are disclosed in the Acknowledgment section of the manuscript.

OR

D. I have no potential conflicts of interest.