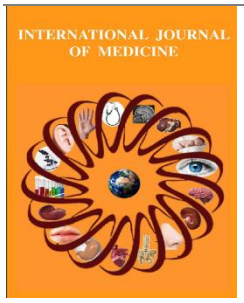
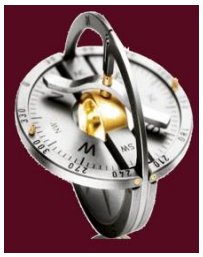
International Journal of Medicine 2026; 8(2): 95-97



International Journal of Medicine

https://www.theinternationalmedicine.com



Research Article

Periarthritis Shoulder: Basic Pathology and Multifaceted Management—A Narrative Review

Amol Khade[1], Chaitanya Kumar IS[2], Rishabh Sharma[1], Aditya Narayan Sahoo[1], Muni Srikanth I[3]

[1]*Department of Physical Medicine and Rehabilitation, AIIMS, Raipur*

[2]*Additional Professor, Department of Transfusion Medicine and Hemotherapy, All India Institute of Medical Sciences, Mangalagiri*

[3]*Department of Orthopaedics, AIIMS, Mangalagiri.*

Abstract

Background: Periarthritis shoulder, commonly known as adhesive capsulitis or frozen shoulder, is a debilitating condition characterized by painful restriction of shoulder motion. Despite its high prevalence, particularly among individuals with diabetes and thyroid disorders, the pathophysiology remains incompletely understood, and management strategies vary widely. ***Objectives***: To review the current understanding of periarthritis shoulder pathophysiology and evaluate the efficacy of various management strategies, including conservative, injection-based, and surgical interventions. ***Methods***: A comprehensive literature search was conducted across PubMed, PMC, and academic databases for studies published between 2010-2025. Data were extracted on pathological mechanisms, clinical stages, risk factors, and treatment outcomes. Results: Periarthritis shoulder involves a complex interplay of synovial inflammation and capsular fibrosis mediated by cytokines (IL-6, TNF-α, TGF-β) and fibroblast activation. The condition progresses through three stages: freezing (painful), frozen (stiff), and thawing (recovery). Diabetes increases risk 2-5-fold, with prevalence reaching 13.4% in diabetic populations. Conservative management with physical therapy achieves success in 67-90% of cases, with supervised neglect showing superior outcomes. Corticosteroid injections provide short-term pain relief (up to 12 weeks) but limited long-term benefit. Hydrodilatation demonstrates significant improvements in range of motion and functional scores. Surgical intervention via arthroscopic capsular release is reserved for recalcitrant cases. ***Conclusions***: Periarthritis shoulder represents an inflammatory-fibrotic cascade requiring stage-specific management. Early intervention with corticosteroid injection combined with supervised physical therapy offers optimal outcomes. Future research should focus on targeted anti-fibrotic therapies and standardized protocols.

Keywords: Periarthritis shoulder; adhesive capsulitis; frozen shoulder; pathology; management; physical therapy; corticosteroid injection

Introduction:

Periarthritis shoulder, clinically synonymous with adhesive capsulitis or frozen shoulder, represents a common yet poorly understood musculoskeletal disorder characterized by spontaneous onset of shoulder pain accompanied by progressive loss of active and passive range of motion[1]. The condition predominantly affects individuals aged 40-70 years, with a higher prevalence in females[2]. Epidemiological studies indicate that the incidence varies between 2-5% in the general population, escalating dramatically to 11-30% among patients with diabetes mellitus[3–5].

The pathological hallmark involves synovial inflammation followed by capsular fibrosis, leading to thickening and contracture of the glenohumeral joint capsule[6–8]. This results in the characteristic clinical triad of pain, stiffness, and functional disability. The natural history traditionally follows three overlapping phases: freezing (painful), frozen (stiff), and thawing (recovery), with total duration ranging from 12 months to 4 years[9–11].

Despite being considered self-limiting, periarthritis shoulder imposes substantial burden through prolonged pain, sleep disturbance, and impaired quality of life[12]. The multifactorial etiology includes both local and systemic factors, with diabetes mellitus, thyroid dysfunction, cervical spondylosis, and hyperlipidemia identified as significant risk factors4,5,13. Understanding the basic pathology is crucial for implementing appropriate stage-specific management strategies.

Address for Correspondence: Dr Muni Srikanth I , Department of Orthopaedics, AIIMS, Mangalagiri.
munisrikanth@aiimsmangalagiri.edu.in .

DOI: 10.61336/im/26-2-17

This is an Open Access article under the terms of the Creative Commons Attribution-Noncommercial 4.0 International License (https://creativecommons.org/licenses/by-nc/4.0/)

Review

Basic Pathology

Inflammatory-Fibrotic Cascade

The pathophysiology of periarthritis shoulder involves a complex interplay between inflammation and fibrosis, mediated through cytokine-driven cellular mechanisms[6–8]. The disease initiates with synovial hyperplasia and angiogenesis, characterized by inflammatory cell infiltration predominantly comprising T cells, B cells, macrophages, and mast cells5,14,15. This inflammatory milieu triggers the release of pro-inflammatory cytokines including interleukin-1α (IL-1α), IL-1β, IL-6, tumor necrosis factor-α (TNF-α), and cyclooxygenase-2 (COX-2)14,15.

Transforming growth factor-β (TGF-β) plays a pivotal role in driving fibroblast proliferation and differentiation into myofibroblasts, which are responsible for capsular contracture6-8. Recent molecular studies have identified IL-17A-producing T cells that induce profibrotic responses through enhanced IL-17RA expression on fibroblasts, activating TRAF6/NF-κB dependent pathways2,5. This cytokine cascade stimulates excessive deposition of type I and type III collagen, leading to capsular thickening and loss of axillary pouch volume6-8.

Histopathological Features

Histological examination reveals stage-specific changes. Stage I (freezing) demonstrates synovial hyperplasia with inflammatory cell infiltration6,7. Stage II (frozen) shows progressive synovial

proliferation with early adhesion formation and capsular contracture6,8. Stage III (thawing) exhibits dense collagenous tissue within the capsule, with resolution of synovitis but persistent fibrotic adhesions7,8. The rotator interval, particularly the coracohumeral ligament, becomes markedly thickened and fibrotic, contributing significantly to restricted external rotation6,7.

Advanced glycation end products (AGEs) and their receptors (RAGE) are overexpressed in diabetic patients, promoting oxidative stress and perpetuating synovial inflammation and fibroblast proliferation4,13. This mechanism partly explains the increased severity and refractory nature of periarthritis shoulder in diabetic populations3,5.

Stages of Disease Progression

Table 1: Clinical and Pathological Stages of Periarthritis Shoulder

Stage	Duration	Clinical Features	Pathological Features	Key Cytokines
Freezing	2-9 months	Severe pain, progressive stiffness	Synovial hyperplasia, inflammatory infiltration	IL-1β, IL-6, TNF-α, TGF-β14,15
Frozen	4-12 months	Pain at extremes, marked stiffness	Capsular fibrosis, adhesion formation	TGF-β, TIMP-1, TIMP-26-8
Thawing	5-24 months	Minimal pain, gradual improvement	Fibrosis resolution, tissue remodeling	MMP-1, MMP-26-7

Multifaceted Management

Conservative Management

Physical Therapy and Exercise

Conservative treatment remains the cornerstone of periarthritis shoulder management, with success rates of 67-90%11,12. The primary goals include pain reduction, restoration of range of motion (ROM), and functional recovery. Exercise protocols are tailored to disease stage and patient irritability level16,17.

Pendulum (Codman's) exercises constitute the foundation of early rehabilitation, utilizing gravity-assisted passive motion to prevent capsular adhesions without provoking pain. These exercises involve forward flexion at 75-90°, allowing the affected arm to swing freely in circular patterns for 3-5 minutes, repeated 5 times daily16,17.

Progressive stretching techniques target specific motion limitations. External rotation stretching in adduction, forward elevation, and cross-body adduction exercises are introduced as pain subsides. Strengthening exercises for rotator cuff and scapular stabilizers are implemented during the thawing phase to restore dynamic stability16,17.

Table 2: Evidence-Based Physical Therapy Protocols

Intervention	Stage	Frequency	Evidence Level	Key Outcomes
Pendulum exercises	Freezing	5× daily	I	Pain reduction, maintained ROM16,17
Joint mobilization	All stages	3× weekly	I-II	Improved flexion, abduction, external rotation16,17
Supervised neglect	Frozen	As tolerated	I	Superior ROM improvement vs. active therapy16,18
Core stability training	All stages	5× weekly	II	Enhanced SPADI scores, reduced pain16,17

Supervised neglect, defined as supportive therapy with patient-controlled activity within pain limits, has demonstrated superior outcomes compared to intensive physical therapy in idiopathic cases16,18. A systematic review reported that supervised neglect achieved the highest percentage improvement in flexion (57.9°), abduction (62.4°), external rotation (37°), and internal rotation (22.1°)16,18.

Injection Therapies

Corticosteroid Injections

Intra-articular corticosteroid injections provide significant short-term pain relief and accelerate recovery when administered during the freezing stage19,20. Triamcinolone acetonide (20-40 mg) is the most commonly used agent, with evidence suggesting 20 mg is as effective as 40 mg19,20. Ultrasound-guided injections achieve 92-95% accuracy compared to 72-79% with blind techniques21,22.

Systematic reviews demonstrate that corticosteroid injections are superior to placebo and physiotherapy for 4-12 weeks, with 77% of injected patients achieving successful resolution by 7 weeks versus 46% with physiotherapy alone19,20. However, long-term benefits beyond 24 weeks are limited, and repeated injections may accelerate cartilage degradation.

Hyaluronic Acid Injections

Hyaluronic acid (HA) injections aim to restore synovial fluid viscoelasticity and modulate inflammation. Studies report significant improvements in pain and function at 1, 3, and 6 months post-injection23,24. A randomized trial comparing HA to corticosteroid showed superior long-term outcomes with HA, though short-term relief was more pronounced with steroids. The combination of HA with physiotherapy demonstrates additive benefits.

Hydrodilatation (Capsular Distension)

Hydrodilatation involves injecting saline with corticosteroid to mechanically rupture capsular adhesions and reduce synovial inflammation19,20. Ultrasound-guided procedures achieve significant improvements in active abduction (84.7° to 121.3°), external rotation, and functional scores (ASES, CMS) within 1-3 months21,22. The technique is particularly effective for patients failing conservative therapy, with 70-80% avoiding surgical intervention.

Table 3: Injection Therapy Outcomes in Periarthritis Shoulder

Injection Type	Short-term Efficacy	Long-term Efficacy	Optimal Stage	Complications
Corticosteroid	Excellent (4-12 wks)	Limited (>24 wks)	Freezing	Skin atrophy (2-5%)19,20
Hyaluronic acid	Moderate (6-12 wks)	Good (6-12 months)	All stages	Minimal23,24
Hydrodilatation	Excellent (1-3 months)	Good (12 months)	Frozen	Capsular rupture (<1%)21,22

.

Surgical Management

Arthroscopic Capsular Release

Surgical intervention is reserved for recalcitrant cases failing 4-6 months of conservative management1,8. Arthroscopic capsular release (ACR) involves selective division of the coracohumeral ligament, rotator interval, and anterior capsule using radiofrequency probes. This minimally invasive approach allows 360° capsular release while preserving rotator cuff integrity.

Studies report high patient satisfaction with significant improvements in ROM and Constant-Murley scores1,8. Idiopathic cases demonstrate better outcomes compared to secondary adhesive capsulitis1,25. The procedure effectively shortens disease duration from years to weeks, with most patients achieving functional recovery within 3-6 months.

Manipulation Under Anesthesia

Manipulation under anesthesia (MUA) remains an alternative when arthroscopic expertise is unavailable. The technique

involves forced passive ROM to rupture adhesions, though it carries higher risk of humeral fracture, rotator cuff tear, and labral injury compared to ACR1,8. Current guidelines favor ACR over MUA due to superior precision and lower complication rates.

Limitations

Current evidence suffers from several methodological constraints. Most studies are retrospective or small randomized trials with heterogeneous populations16,19. The optimal timing, dosage, and combination of interventions remain undefined, with significant variation in physical therapy protocols and injection techniques16,20. Long-term follow-up data beyond 2 years are limited, and cost-effectiveness analyses are lacking19,20.

The lack of standardized diagnostic criteria and staging systems complicates comparison across studies1,8. Most trials exclude diabetic patients or fail to stratify results by glycemic control, despite clear evidence of differential treatment response3. Furthermore, the role of emerging biological therapies like Platelet rich plasma etc., targeting specific cytokines remains experimenta.

CONCLUSION

Periarthritis shoulder represents a complex inflammatory-fibrotic disorder requiring stage-specific, multifaceted management. The pathological cascade involves cytokine-driven synovial inflammation progressing to capsular fibrosis, with diabetes and thyroid dysfunction serving as major risk factors. Conservative management with supervised physical therapy remains first-line treatment, achieving success in most patients when appropriately staged. Corticosteroid injections provide valuable short-term relief during the freezing phase, while hydrodilatation offers effective minimally invasive intervention for refractory cases. Surgical capsular release is reserved for persistent disability, demonstrating excellent outcomes in carefully selected patients.

Future research should prioritize large-scale randomized trials comparing stage-matched interventions, development of validated biomarkers for disease progression, and investigation of targeted anti-fibrotic therapies. Standardized treatment protocols and predictive models for treatment response would optimize resource utilization and patient outcomes.

REFERENCES

1. Le HV, Lee SJ, Nazarian A, Rodriguez EK. Adhesive capsulitis of the shoulder: review of pathophysiology. Curr Rev Musculoskelet Med. 2016;10(1):1-8. doi:10.1007/s12178-016-9391-0
2. Sun G, Li H, Zeng X, et al. Risk factors and predictive models for frozen shoulder. Sci Rep. 2024;14(1):66360. doi:10.1038/s41598-024-66360-y
3. Dyer B, Razmjou H, Henry P, Gosselin-Fournier S, Holtby R. Diabetes as a risk factor for the onset of frozen shoulder: a systematic review and meta-analysis. BMJ Open. 2023;13(9):e069191. doi:10.1136/bmjopen-2022-069191
4. Akkoc Y, Karatay S, Tok F, et al. Shoulder adhesive capsulitis: is there an association with the metabolic syndrome? Arch Rheumatol. 2018;33(3):320-327. doi:10.5606/ArchRheumatol.2018.6625
5. Wang K, Ho V, Hunter-Smith DJ, et al. Risk factors for idiopathic frozen shoulder: a systematic review and meta-analysis. J Orthop Surg Res. 2023;18(1):1-12. doi:10.1186/s13018-023-04314-5
6. Bunker TD, Anthony PP. The pathology of frozen shoulder. A Dupuytren-like disease. J Bone Joint Surg Br. 1995;77(5):677-683. doi:10.1302/0301-620X.77B5.7559690
7. Hannafin JA, Chiaia TA. Adhesive capsulitis. A treatment approach. Clin Orthop Relat Res. 2000;(372):95-109. doi:10.1097/00003086-200003000-00012
8. Neviaser AS, Hannafin JA. Adhesive capsulitis: a review of current treatment. Am J Sports Med. 2010;38(11):2346-2356. doi:10.1177/0363546509348048
9. Reeves B. The natural history of the frozen shoulder syndrome. Scand J Rheumatol. 1975;4(4):193-196. doi:10.3109/03009747509097136
10. Shaffer B, Tibone JE, Kerlan RK. Frozen shoulder. A long-term follow-up. J Bone Joint Surg Am. 1992;74(5):738-746. doi:10.2106/00004623-199274050-00013
11. Hand C, Clipsham K, Rees JL, Carr AJ. Long-term outcome of frozen shoulder. J Shoulder Elbow Surg. 2008;17(2):231-236. doi:10.1016/j.jse.2007.05.009
12. Dias R, Cutts S, Massoud S. Frozen shoulder. BMJ. 2005;331(7530):1453-1456. doi:10.1136/bmj.331.7530.1453
13. Zreik NH, Malik RA, Charalambous CP. Adhesive capsulitis of the shoulder and diabetes: a meta-analysis of prevalence. Muscle Nerve. 2016;54(5):763-767. doi:10.1002/mus.25166
14. Rodeo SA, Hannafin JA, Tom J, Warren RF, Wickiewicz TL. Immunolocalization of cytokines and their receptors in adhesive capsulitis of the shoulder. J Orthop Res. 1997;15(3):427-436. doi:10.1002/jor.1100150315
15. Lho YM, Ha E, Cho CH, et al. Inflammatory cytokines are overexpressed in the subacromial bursa of frozen shoulder. J Shoulder Elbow Surg. 2013;22(5):666-672. doi:10.1016/j.jse.2012.07.001
16. Kelley MJ, McClure PW, Leggin BG. Frozen shoulder: evidence and a proposed model guiding rehabilitation. J Orthop Sports Phys Ther. 2009;39(2):135-148. doi:10.2519/jospt.2009.2916
17. Russell S, Jariwala A, Conlon R, et al. A blinded, randomized, controlled trial on conservative treatment with physiotherapy vs. intra-articular steroids in the treatment of frozen shoulder. Shoulder Elbow. 2014;6(3):173-178. doi:10.1111/sae.12047
18. Diercks RL, Stevens M. Gentle thawing of the frozen shoulder: a prospective study of supervised neglect versus intensive physical therapy in seventy-seven patients with frozen shoulder syndrome followed up for two years. J Shoulder Elbow Surg. 2004;13(5):499-502. doi:10.1016/j.jse.2004.01.004
19. Buchbinder R, Green S, Youd JM. Corticosteroid injections for shoulder pain. Cochrane Database Syst Rev. 2003;(1):CD004016. doi:10.1002/14651858.CD004016
20. Shah N, Lewis M. Shoulder adhesive capsulitis: systematic review of randomised trials using multiple corticosteroid injections. Br J Gen Pract. 2007;57(541):662-667. doi:10.3399/096016407782317955
21. Hashimoto T, Ozaki J. Intra-articular corticosteroid injection for idiopathic adhesive capsulitis of the shoulder: a comparative study of ultrasound-guided versus blind injection. J Orthop Sci. 2018;23(2):331-335. doi:10.1016/j.jos.2017.11.010
22. Park KD, Lee JH, Kim TK, et al. Ultrasound-guided versus blind corticosteroid injection for adhesive capsulitis of the shoulder: a randomized controlled trial. J Shoulder Elbow Surg. 2020;29(1):73-81. doi:10.1016/j.jse.2019.07.011
23. Blaine T, Moskowitz R, Udell J, et al. Treatment of persistent shoulder pain with sodium hyaluronate: a randomized, controlled trial. A multicenter study. J Bone Joint Surg Am. 2008;90(5):970-979. doi:10.2106/JBJS.G.00485
24. Hsieh LF, Hsu WC, Lin YJ, et al. Is ultrasound-guided injection more effective in chronic subacromial bursitis? Medicine (Baltimore). 2018;97(40):e12649. doi:10.1097/MD.0000000000012649
25. Kim WS, Kim JH, Park KD, et al. Arthroscopic capsular release for frozen shoulder: when etiology matters. J Orthop Surg Res. 2023;18(1):763. doi:10.1186/s13018-023-04045-7..