

Posterior Shoulder Instability: A Clinical Review



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KEYWORDS

• Posterior shoulder instability • Reverse hill-sachs • Glenoid bone loss • Posterior labral tear

KEY POINTS

- In the absence of glenoid bone loss, arthroscopic labral repair has a high success rate.
- Bone block augmentation procedures should be considered for glenoid bone loss exceeding 13.5%.
- Soft tissue procedures are effective for treating small to moderate-sized reverse Hill-Sachs defects, while larger defects may require bone grafting or arthroplasty procedures.

INTRODUCTION

Posterior shoulder instability has emerged as an increasingly prominent concern for surgeons treating shoulder pathology. Previously estimated to account for only 5% to 10% of cases in the general population, it is now recognized that the true incidence is likely underestimated. Recent studies report rates of posterior shoulder instability as high as 24%, particularly among high-risk populations such as weightlifters, football linemen, and military recruits.^{1–3} This growing recognition highlights the need for tailored treatment strategies. Research addressing posterior shoulder instability has been driven by the challenges of recurrence and the critical role of bone loss.^{4,5}

Successful treatment of posterior shoulder instability relies on a comprehensive understanding of the anatomic structures contributing to its pathology. Glenohumeral joint stability relies on the integrity of both static and dynamic stabilizers. Given the limited osseous constraint provided by the glenoid, even relatively small bony deficiencies can have a significant impact. For instance, research suggests that glenoid bone loss as low as 13.5% can increase the risk of instability recurrence.⁶ This is particularly

relevant in recurrent posterior shoulder instability, where the incidence of glenoid bone loss is dramatically higher—6% in first-time dislocations compared with 69% in recurrent cases. Among these, 22% exhibit subcritical bone loss between 13.5% and 20%.^{5,6} The effect of glenoid bone loss is further amplified by the presence of glenoid retroversion, which redirects forces across the glenoid posteriorly.⁵ Cadaveric studies have shown that retroversion greater than 10° significantly increases the risk of posterior instability.⁷ A case-control study by Owens and colleagues found that each degree of retroversion increased the risk of posterior instability by 17%.⁸ Consequently, evaluation of posterior glenohumeral instability must include an assessment of osseous contributions during presurgical planning and intraoperative decision making to restore native shoulder kinematics.

Dynamic and static restraints of the glenohumeral joint are essential for maintaining stability and must be addressed to effectively manage posterior instability and restore optimal shoulder function. While osseous anatomy plays a role in glenohumeral stability, particularly in cases of recurrence, soft tissue structures are critical for establishing and maintaining constraint. The rotator cuff, labrum, glenohumeral ligaments, and

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Abbreviations	
AP	anterior-posterior
ASES	American Shoulder and Elbow Surgeons
rHSL	reverse Hill-Sachs lesion

capsule are key to providing stability throughout shoulder range of motion. The periscapular and rotator cuff musculature function as dynamic stabilizers and serve as the cornerstone of nonoperative management aimed at preventing recurrent instability. Acting in synergy with these dynamic forces, the labrum deepens the glenoid concavity and preserves a negative pressure environment, making labral disruption a primary focus of surgical intervention. In addition to the posterior labrum, the posterior capsule and posterior inferior glenohumeral ligament serve as essential static stabilizers. These structures are thought to become attenuated due to repetitive microtrauma in at-risk populations, increasing the likelihood of dislocation, particularly during shoulder flexion and internal rotation.⁹

PATIENT EVALUATION

The evaluation of posterior shoulder instability requires a comprehensive history and physical examination. Unlike with anterior instability, patients with posterior instability seldom present with a distinct dislocation or subluxation event.² Instead, patients often report vague, deep-seated shoulder pain accompanied by mechanical symptoms like popping or clicking.^{4,10} Patients may also describe difficulty participating in physical activities or a noticeable decline in athletic performance.¹⁰ Pertinent elements of the clinical history include key factors such as a personal or family history of instability, underlying medical conditions like seizure disorders, involvement in high-risk activities or contact sports, military service, and the precise mechanism of injury. These elements are crucial for identifying potential causes and contributing factors to the patient's pain and instability. It is also essential to consider other potential sources of pain that may mimic posterior instability symptoms including referred neck pain, radiculopathy, or impaired scapulothoracic motion.

The physical examination should be thorough, evaluating strength, sensation, and range of motion of the entire upper extremity to identify concomitant pathology. Screening for hyperlaxity, using validated tools such as the Beighton score, can provide valuable insight into the

integrity of the patient's soft tissues, which may influence treatment decisions. Additionally, performing an examination of the contralateral upper extremity is necessary to establish a patient-specific baseline for comparison.

Several clinical examination maneuvers are used to evaluate posterior instability, including the Kim test, jerk test, load-and-shift test, and posterior stress test.¹⁰ While the posterior load-and-shift test is 100% specific for posterior instability, its sensitivity is limited, with one study reporting a sensitivity of only 14%.¹¹ In comparison, the Kim and jerk tests demonstrate greater clinical utility, offering higher sensitivity (80% and 73%, respectively) and specificity (94% and 98%, respectively). Combining these maneuvers is recommended to optimize diagnostic accuracy. Findings from Kim and colleagues indicate that using both the Kim and jerk tests achieves a sensitivity of 97%.¹² Additionally, the dynamic posterior instability test has demonstrated high sensitivity and specificity, though it has yet to gain widespread clinical adoption.¹³ To ensure a reliable assessment of posterior shoulder instability and guide appropriate management strategies, a comprehensive physical examination incorporating multiple maneuvers is recommended for each patient evaluation.

IMAGING

Imaging standards for posterior shoulder instability align closely with those used for anterior shoulder instability. Plain radiographs remain the primary modality for initial evaluation. A standard radiographic series should include anterior-posterior (AP), Grashey, scapular Y, and axillary views of the affected shoulder, with optional contralateral imaging for comparison. Initial radiographs are essential for identifying fractures, osseous defects of the glenohumeral joint and suspensory complex, radiopaque hardware from prior surgeries, loose bodies, and joint subluxation or dislocation. Although the utility of the axillary view has been debated—Cruz and colleagues reported a 97.5% nonutility rate compared with AP, Grashey, and scapular Y views—it remains highly valuable in cases of suspected or confirmed shoulder instability.¹⁴ The axillary view is essential for confirming concentric joint reduction and detecting humeral or glenoid pathologies that may not be visualized on other views.¹⁵

Advanced imaging provides a detailed assessment of both soft tissue and bony anatomy. Magnetic resonance imaging (MRI) is typically the preferred modality, offering effective assessment

of soft tissue structures while also providing valuable insights into osseous anatomy. A recent study demonstrated that both conventional 3T MRI and 3T MR arthrography were 100% specific for posterior labral tears; however, MR arthrography demonstrated superior sensitivity at 95% compared with 84% for conventional 3T MRI.¹⁶ It is also important to recognize that posterior instability differs from anterior instability, as posterior bone defects most commonly occur in the posterior-inferior quadrant of the glenoid, approximately 30° off the long axis of the glenoid.¹⁷

Computed tomography (CT) remains the gold standard for evaluating bony morphology and pathology. While Yanke and colleagues found MRI and CT equivalent for estimating glenoid bone loss in anterior instability, this finding has not been replicated for posterior glenoid bone loss.¹⁸ Glenoid bone loss is typically defined as the percentage of the defect width relative to the native glenoid width. A best-fit circle along the inferior two-thirds of the glenoid is used to measure its diameter and quantify the defect. Cadaveric studies have demonstrated that glenoid bone loss exceeding 20% significantly compromises glenohumeral stability, indicating the need for bony stabilization procedures.¹⁹ To evaluate glenoid retroversion, the Friedman method remains one of the most widely used and reproducible techniques. This method measures the angle between a line connecting the anterior and posterior mid-glenoid rims and a line perpendicular to the scapular body axis (Fig. 1).²⁰ Additional morphologic factors, such as acromial slope, bone loss slope, and glenoid concavity, have also been linked to posterior instability and recurrence, though their role in clinical decision-making remains limited.²¹

TREATMENT

Nonoperative Management

Nonoperative management remains a viable treatment strategy for select patients with posterior shoulder instability. A recent international expert consensus outlined several relative indications for nonoperative care, including first-time dislocations, ligamentous laxity or connective tissue disorders, seizure disorders, and in-season athletes aiming to return to play.²² Patients with functional instability in the absence of structural lesions should also be managed without surgery.²³ Evidence further suggests that patients experiencing isolated pain without significant instability tend to achieve more favorable outcomes with nonoperative management compared with those with pronounced instability.²⁴

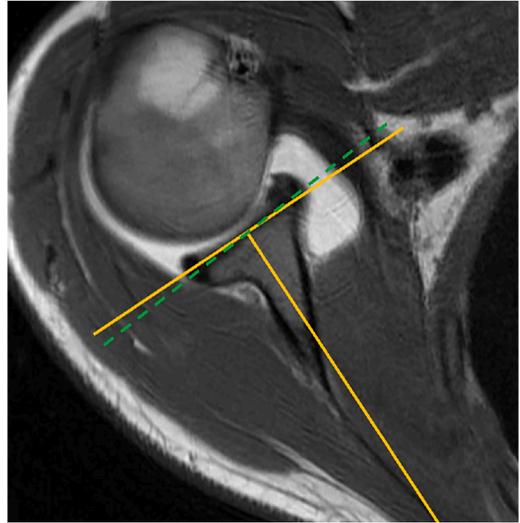


Fig. 1. Axial MRI showing measurement of glenoid retroversion using the Friedman method. The dashed line represents the axis of anterior and posterior glenoid rims. The angle between the dashed line and the line perpendicular to the scapular body axis represents the degree of retroversion.

The foundation of nonoperative treatment is a targeted physical therapy program focused on scapulothoracic control, rotator cuff strengthening, and proprioception.¹ This approach leverages muscular control to improve dynamic stability and mitigate symptoms, particularly in patients whose primary complaint is pain rather than recurrent instability. Neuromuscular electrical stimulation, an emerging adjunct to traditional physical therapy, has shown promise in improving outcomes in functionally unstable shoulders when integrated with standard rehabilitation protocols.²⁵

Patient selection is crucial for predicting treatment success. Studies indicate that nonoperative management is less effective in cases of acute traumatic injuries, where muscular compensation may be insufficient to address traumatic pathology, and surgical intervention often yields superior outcomes.²⁶ Identifying glenohumeral pathomorphological features is essential to recognizing patients at risk of treatment failure.²⁷ The overall long-term success of nonoperative protocols is variable; one study reported a 46% conversion rate to surgery within 10 years, with an overall surgical rate of 70%.²⁸ Despite these challenges, nonoperative treatment remains a valuable option. Consideration should be based on the specific pathology and patient goals, particularly for those prioritizing surgery avoidance or for whom surgical intervention carries elevated risks.

Surgical Management

Patients who fail to respond to nonoperative management and a structured rehabilitation program, or those with chronic pain, recurrent instability, and functional deficits associated with capsulolabral or osseous injuries, are candidates for surgical management. When treating chronic posterior shoulder instability, it is essential to recognize repetitive microtraumatic stress on the capsulolabral complex or the presence of inherent capsular laxity, as these factors differentiate chronic instability from acute traumatic injuries. Techniques that imbricate the capsule, such as the "Hospital Corner Repair," can effectively address these sources of instability.²⁹ For patients with reverse Hill-Sachs lesions (rHSLs), reverse remplissage is a viable option to fill the defect, making the lesion extra-articular and restoring the coupling forces of the shoulder's dynamic stabilizers.

Arthroscopic capsulolabral repair

Patients with isolated posterior labral tears or subcritical osseous pathology are ideal candidates for arthroscopic labral repair. Specifically, cases with glenoid bone loss less than 11% and nonengaging rHSL are well-suited for this approach, as these findings are associated with favorable outcomes.^{30,31} Studies have shown significant improvements in functional outcomes and high return-to-sport rates.^{32–34} Bradley and colleagues reported an improvement in American Shoulder and Elbow Surgeons (ASES) scores from 45.9 to 85.1, with 90% of patients returning to sport and 64% resuming activity at their previous level.³⁵ Similarly, adolescent athletes have demonstrated comparable results, with ASES scores improving from 48.6 to 85.7, an 89% return to sport rate, and 71% achieving their preinjury performance level.³⁶

Despite the high success rates of arthroscopic labral repair, recognizing risk factors for revision is essential to improving outcomes. Surgical techniques, including knotted versus knotless fixation or positioning in the lateral decubitus versus beach chair position, have not demonstrated significant differences in clinical results.^{37,38} Although the lateral decubitus position may offer improved visualization and facilitate anchor placement, studies indicate no significant advantage over the beach chair position, allowing surgeons to choose the approach that is most consistent and reproducible in their practice. Furthermore, a recent study identified underestimation of labral pathology as a significant risk factor for repair failure. The use of 3 or fewer anchors is associated with higher failure

rates, emphasizing the importance of adequate fixation to restore the bumper effect of the labrum.³⁹

Additionally, glenoid morphology remains one of the most critical risk factors for isolated labral repair. Patients with increased glenoid bone width demonstrate lower failure rates and better functional outcomes.⁴⁰ Posterior glenoid bone loss exceeding 13.5% has been shown to significantly reduce return-to-duty rates in military populations, although other clinical outcomes and revision rates appear unaffected.⁶ As previously discussed, glenoid retroversion may amplify instability associated with bone loss, collectively contributing to higher reoperation rates.^{5,7,8} Thorough evaluation of patient-specific anatomic factors is essential to optimize surgical strategies and outcomes. Addressing these factors during surgical planning can improve the likelihood of success and reduce the risk of complications.

Reverse Hill-Sachs lesions

Although labral pathology is the most common finding in posterior shoulder instability, humeral head lesions should also be thoroughly assessed and managed appropriately during surgical intervention. A rHSL, often associated with traumatic posterior shoulder dislocations, significantly increases the risk for recurrent instability, with a reported incidence ranging from 21.5% to 86%.⁴¹ These lesions result from an impaction fracture on the anterior humeral head due to engagement with the posterior glenoid rim, which compromises joint stability and increases the likelihood of failure if left untreated.²⁹ Lesion size is a key determinant in selecting the appropriate treatment for rHSLs. Small lesions, involving less than 25% of the articular surface, are typically managed nonoperatively. Medium-sized lesions (25%–50%) often require surgical intervention, incorporating soft tissue or bony procedures. The reciprocal relationship between the humeral head articular surface and the glenoid determines joint stability. Consequently, either glenoid bone loss or humeral head articular loss can alter joint biomechanics, leading a noncritical rHSL to engage in the presence of concomitant glenoid deficiency.^{8,42}

Several surgical strategies effectively address humeral-sided pathology. Soft tissue techniques include the McLaughlin procedure, where the subscapularis is released and transferred into the defect, or the modified McLaughlin technique, which incorporates the lesser tuberosity into the repair. Alternatively, reverse remplissage can be performed, utilizing the subscapularis

tendon to fill the defect while maintaining its attachment to the lesser tuberosity. While soft tissue repairs are effective for moderate defects, larger or chronic lesions may require bony reconstruction or joint-preserving procedures to optimize outcomes and maintain function. Techniques such as disimpaction and fixation, bone block/grafting, or, in rare cases, rotational osteotomies can effectively disengage the rHSL from the posterior glenoid rim. For lesions exceeding the intermediate size threshold, arthroplasty may be necessary, particularly in chronic cases or when advanced degenerative changes are present.^{43–46}

Evidence guiding treatment decisions is limited due to the low incidence of rHSLs. A systematic review of 26 studies, including 291 patients, reported favorable outcomes across various treatment approaches. Arthroscopic procedures were typically performed for defects averaging 29% of the humeral head, while graft-filling techniques were used for larger defects, averaging 38%.⁴⁶ Graft-filling procedures have shown promising results in preserving glenohumeral joint function, although long-term reports on degenerative joint disease remain variable.^{8,22,47} These findings highlight the importance of tailoring treatment strategies based on lesion size, characteristics, and concomitant pathology to optimize outcomes and preserve joint integrity.

Posterior bone block procedures

Indications for posterior bone block procedures include critical posterior glenoid bone loss, posterior glenoid bone loss with glenoid retroversion, failed primary repair, and predisposing risk factors for recurrent instability and failure.¹⁵ Both decreased native glenoid bone width and glenoid bone loss of 13.5% are associated with an increased risk for failure.^{6,40} Using data extrapolated from anterior instability studies, recommendations for bony augmentation based on glenoid bone loss percentages have been proposed. Arner and colleagues defined critical posterior bone loss thresholds, reporting a 10-fold increase in failure risk with 11% loss and a 25-fold increase with 15% loss. Their study utilized reformatted MRI scans to improve measurement precision along the glenoid anatomic axis.³⁰ While glenoid retroversion is recognized as a contributing factor to instability, especially when combined with bone loss, studies have not established a direct correlation between isolated retroversion and arthroscopic treatment failure.^{5,8,21,30} Consequently, current recommendations primarily rely on the percentage of bone

loss to determine the need for posterior bone block procedures.

Posterior bone block procedures, performed open or arthroscopically, aim to restore glenoid bone width using either autograft (clavicle, acromion, or iliac crest) or allograft (distal tibia allograft [DTA]). Outcomes reported in the literature demonstrate significant variability likely reflecting the heterogeneity of the patient populations and surgical indications. A systematic review by Cognetti and colleagues identified recurrence rates as high as 73%.⁴⁸ Conversely, Mojica and colleagues reported a much lower recurrence rate of 9.8% with a complication rate of 13.8%.⁴⁹ Saeed and colleagues, in a systematic review, noted a recurrence rate of up to 12.5% for arthroscopic iliac crest autograft procedures, while open procedures demonstrated higher recurrence rates, reaching 36.4%.⁵⁰ In a small case series, Gilat and colleagues reported a 20% revision rate for patients undergoing a DTA bone block procedure for glenoid lesions averaging 26% in size.⁵¹ Clavert and colleagues found a 12% recurrence rate with 18% of patients experiencing persistent pain after iliac crest or acromial-based bone block procedures. However, due to significant heterogeneity in their cohort—including 52% with posterior glenoid damage, 59% with reverse Bankart lesions, and 20% with rHSLs—these findings have limited generalizability.⁵²

Although definitive indications for posterior bone block procedures are still evolving, identifying patients at high risk for failure of isolated capsulolabral repair is essential. Addressing critical osseous defects during the index procedure allows surgeons to optimize outcomes and reduce the likelihood of recurrence.

CASE EXAMPLE

We present the case of a 38-year-old male who sustained a traumatic injury after a vehicle rolled over his upper body and left shoulder. Initial evaluation at an outside emergency department included radiographs, which showed no evidence of acute fracture or dislocation. Two weeks later, he presented to clinic with vague, posterior shoulder pain and discomfort during overhead movements.

On physical examination, findings included anterior capsule tenderness, a positive biceps tension test, a positive jerk test, and rotator cuff weakness associated with pain. Notably, anterior and posterior load-and-shift tests demonstrated no evidence of instability. Given the traumatic mechanism of injury and the

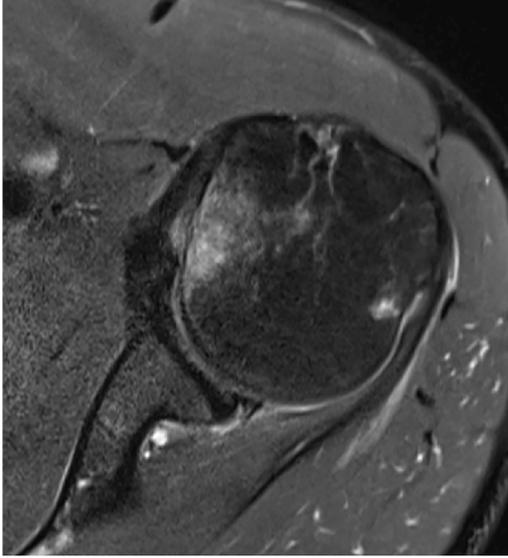


Fig. 2. Axial T2 MRI showing posterior labral tear and anterior humeral head impaction consistent with a dislocation event.

abovementioned clinical findings, an MRI was performed, revealing a tear involving the posterior-superior, posterior, and posterior-inferior labrum. Additional findings included an impaction contusion with edema of the posterior humeral head consistent with a small rHSL (**Fig. 2**). The patient underwent a trial of physical therapy with minimal symptomatic relief. He continued to experience pain and mechanical symptoms with provocative findings consistent with posterior labral pathology. As a result, he was indicated for a left shoulder arthroscopy, posterior labral repair, and possible subscapularis advancement.

A standard diagnostic arthroscopy was performed with the patient in the lateral decubitus position. Extensive labral tearing was identified

from the 2 o'clock to 6 o'clock position, while the remainder of the inferior labrum was intact (**Fig. 3**). There was no evidence of anterior labral pathology or a SLAP lesion. Subtle softening of the anterior humeral head adjacent to the subscapularis was noted, corresponding to a shallow rHSL, which was nonarticulating.

The posterior labrum was elevated using a liberator elevator, and a motorized shaver was used to gently debride the labral edge and lightly abrade the exposed glenoid neck and bony surface to facilitate healing. To optimize anchor placement, an accessory posteroinferior portal at the 7 o'clock position was created. A double-loaded 2.9-mm anchor was inserted at the 5:30 position. The 2 sutures from this anchor were passed across the posterior-inferior labral tear. The first pass started at the 6 o'clock position, advancing the labrum superiorly and laterally with a full-thickness labral pass to address the defect. The second suture was passed as a plication stitch, incorporating a portion of the posterior-inferior capsule (notably patulous in this case) along with a full-thickness labral bite to create a "hospital corner" repair.⁵³ This construct effectively centralized the humeral head within the glenoid articular surface.

Two additional double-loaded anchors were placed sequentially from inferior to superior. Sutures were advanced with full-thickness labral passes and capsular plication stitches, which reduced capsular volume and restored the labrum to its anatomic position along the bony edge. This configuration recreated the posterior buttress and achieved humeral head centralization with appropriate tissue tension. The capsular defects posteriorly were then closed using #1 PDS (**Fig. 4**). The number of anchors used was determined by the extent of the labral pathology.



Fig. 3. Intraoperative arthroscopic images showing extent of posterior labral tearing.

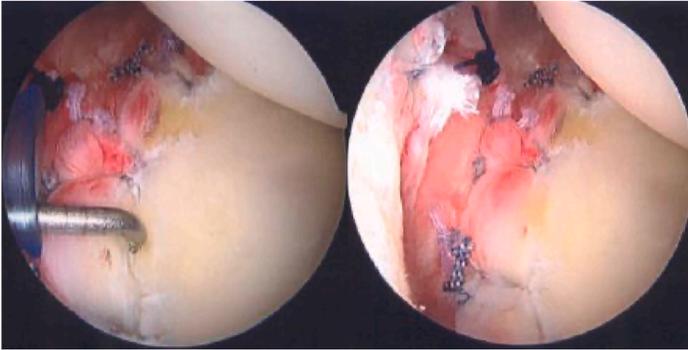


Fig. 4. Intraoperative arthroscopic images showing posterior labral repair and capsular closure.

SUMMARY

Treatment of posterior shoulder instability should be differentiated from anterior instability, as these conditions present distinct biomechanical challenges and clinical presentations. Unlike anterior instability, posterior instability rarely involves a dislocation event and often results from repetitive microtrauma. Patients frequently present with pain as the primary complaint. Identifying anatomic risk factors, such as glenoid bone loss, is critical to guiding treatment strategies and achieving successful outcomes. Comprehensive evaluation and appropriate surgical techniques are essential to restore joint stability and function.

CLINICAL CARE POINTS

- Maintaining a high degree of clinical suspicion is essential for diagnosing posterior shoulder instability, as most cases lack a history of overt dislocation.
- Certain patient populations may benefit from earlier or more aggressive intervention to restore stability, minimize recurrence, and prevent further progression of pathology.
- Both soft tissue and bony abnormalities should be carefully evaluated and addressed, as adequate restoration of shoulder stability often requires a comprehensive approach.
- Surgical management options include soft tissue stabilization and bony procedures, with the treatment plan customized to the patient's specific pathologic findings.

DISCLOSURE

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