Shoulder Instability with Concomitant Bone Loss in the Athlete

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KEYWORDS

• Glenoid insufficiency • Bony Bankart • Shoulder instability • Dislocation

KEY POINTS

- The prevalence of clinically relevant bone loss is likely underestimated, with critical bone deficiency present in a high percentage of patients with multiple recurrent dislocations or prior failed stabilization surgery.
- Of the several options for the imaging quantification of bone loss, 3-dimensional computed tomography reconstruction is the current gold standard.
- As little as 6 mm of bone loss can translate into approximately 20% to 25% of the native glenoid surface; when glenoid bone loss exceeds 20%, glenoid bone augmentation is the currently accepted form of surgical management.
- Adopting an algorithmic approach to the athlete with glenohumeral instability and associated bone deficiency will help achieve the most predictable outcomes with regard to stability and return to activities.

INTRODUCTION

Shoulder instability is common in athletes, often resulting from an initial traumatic dislocation event. Glenohumeral dislocations and subluxations, especially those occurring at a young age, can lead to recurrent instability events, which can jeopardize performance in elite athletes. Following initial traumatic dislocation, repeated trauma to the glenoid can result in glenoid rim fractures, attritional bone loss, and soft-tissue injury. Over time this may lead to glenoid, humeral, or dual-sided bony deficiency and a change in the overall architecture of the shoulder. Previous failed fixation of bony Bankart and labral abnormality may also lead to insufficiency, prompting consideration of further options.

Thorough evaluation of the athlete with persistent shoulder instability and appropriate use of imaging modalities can help quantify the severity of bony deficiency. Based on obtained imaging and examination, surgical and nonsurgical methods can be considered. In many situations both the humeral-sided and glenoid-sided bone loss must be addressed. Depending on the extent of bone loss, athletic demands, and surgeon experience, arthroscopic or open surgical options can provide shoulder stability and return athletes to their prior level of activity.

NATURAL HISTORY OF BONE LOSS

The ability to counsel athletes presenting with varying degrees of bone loss depends on a proper understanding of the natural history of glenoid and humeral bone defects. The unconstrained nature of the glenohumeral joint in terms of osseous stability imparts a predisposition for recurrent joint

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instability. Young athletes and those who participate in contact sports are most prone to developing recurrent dislocation or subluxation events after an initial traumatic dislocation.[1,2] These events most commonly result in anterior inferior "Bankart" labral tears, occurring in approximately 97% of dislocations.[3,4]

Age has long been regarded as the most important factor for recurrence.[1,5] Sachs and colleagues[6] reported on predictive factors for recurrence and identified young age and contact sports as primary risk factors for recurrence. Glenoid fractures and Hill-Sachs lesions can be present from the initial dislocation event as well, and may go unrecognized; this can alter the necessary bony constraint within the glenohumeral joint.[7] Burkhart and De Beer[8] identified that failure to address the bony aspect of injury, including Hill-Sachs lesions, may lead to failed instability surgery.[7,8] This finding has led to recent discussion regarding proper treatment of first-time dislocators.[9] Even if there is not an initial bony lesion, repeated dislocation events over time will often lead to glenoid erosion and development of a characteristic pattern of bone loss, known as the "inverted pear" glenoid.[8]

Some athletes may be predisposed to glenohumeral instability based on their native glenoid architecture and glenoid version. Some amount of bony deficiency is present in approximately 22% of patients after initial dislocation. The incidence climbs to nearly 90% after failed prior stabilization surgery.[8] Most of these defects are located on the anterior inferior glenoid between 2 and 6 o'clock, consistent with the common direction of instability events.[10] Posterior glenoid fractures and insufficiency do occur, though much less commonly.

The type of sport may also influence the morphology of the glenoid lesion depending on the mixture of direction of axial and shear force applied to the joint at the time of injury, as demonstrated by Burkhart and De Beer.[8] Smooth-appearing lesions without a clear fracture fragment may be found in the setting of lower-energy chronic dislocations presenting over a longer term, whereas glenoid rim fractures are more common among football players presenting more acutely.[8] In the setting of recurrent dislocations persistent for longer than 6 months, the glenoid may remodel completely.[7,11]

The Hill-Sachs lesion is a compression defect of the humeral head that is associated with shoulder instability. This lesion occurs in approximately 40% to 90% of athletic shoulder instability and generally occurs in the abducted, externally rotated position.[12] Those humeral-sided lesions that do not engage are typically not parallel to the glenoid face when the shoulder is in abduction and external rotation. Reverse Hill-Sachs lesions (anterosuperomedial humerus lesion) occur in posterior shoulder instability.

The humeral head defect may also remodel, and the progression on the humeral side combined with attritional glenoid-sided loss can combine to lead to an "engaging" Hill-Sachs lesion (**Fig. 1**). These bipolar lesions need to be assessed by quantification of both the glenoid and humeral involvement, and treatment in this setting can be challenging. In the setting of isolated humeral-sided bone loss with engagement, which is less common than isolated glenoid-sided loss, a specific subset of surgical options can be considered.

BIOMECHANICAL EFFECTS OF BONE LOSS

The stability of the native glenohumeral joint depends on both static and dynamic restraints. Provencher and colleagues[13] identify 3 key contributors to joint stability: concavity-compression and the bony architecture, the glenohumeral capsuloligamentous structures (static stabilizers), and the coordinated contraction of the rotator cuff and periscapular musculature (dynamic stabilizers).

Bone loss on the glenoid and/or humeral sides can lead to a well-described cascade of events. Loss on the glenoid side can create a discrepancy in the articular area.[11,14] The concave surface of the glenoid provides a point on which the humerus can be compressed, assisted by the labrum, which deepens the concavity of the glenoid and serves as a site of ligament attachment. Lack of the labral buttress and disruption of bony architecture can lead to instability events, which can contribute to a cycle whereby both the glenoid-sided and humeral-sided lesions increase in size, compounding the problem. The cancellous architecture of the posterolateral humerus is prone to continued compression injury, with the cortical anterior glenoid bone as the assailant. In addition, the capsule has been demonstrated to stretch in the setting of recurrent instability.[15] Restoration of the proper glenoid articular arc is one method to help restore stability that is affected by these numerous disadvantageous factors.[16]

The concept of glenoid track is influenced by bone loss. As described by Yamamoto and colleagues,[17] the glenoid contact area shifts from inferomedial to superolateral portion of the posterior aspect of the humeral head, termed the glenoid track. These investigators reported that a Hill-Sachs lesion is at higher risk of engagement if it extends medially over the medial margin of the glenoid track.[17] For the athlete, increased glenoid