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Critical Glenoid Bone Loss in Posterior Shoulder Instability

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Abstract

Background: There is currently no consensus regarding the amount of posterior glenoid bone loss that is considered critical. Critical bone loss is defined as the amount of bone loss that occurs in which an isolated labral repair will not sufficiently restore stability

Purpose: The purpose of this study was to identify the critical size of the posterior defect.

Methods: 11 cadaver shoulders were tested. With the use of a custom robot device, a 50-N compressive force was applied to the glenohumeral joint and the peak force that required to translate the humeral head posteriorly and the lateral displacement that occurred with translation were measured. The defect size was measured as a percentage of the glenoid width. Testing was performed in 11 conditions: (1) intact glenoid and labrum, (2) simulated reverse Bankart lesion, (3) the reverse Bankart lesion repaired, (4) a 10% defect, (5) the reverse Bankart lesion repaired, (6) a 20% defect, (7) the reverse Bankart lesion repaired (8) a 30% defect, (9) the reverse Bankart lesion repaired, (10) a 40% defect, and (11) the reverse Bankart repaired.

Results: Force and displacement decreased as the size of the osseous defect increased. The mean peak force that occurred with posterior displacement in specimens with a glenoid defect 20% and a reverse Bankart repair (13 ± 9 N) was significantly lower than the peak force that occurred in specimens with an isolated reverse Bankart repair (22 ± 10 N) ($p=0.0451$). Additionally, the mean lateral displacement was significantly lower in the specimens with a 20% glenoid defect and a reverse Bankart repair (0.61 ± 0.57 mm) compared with the lateral displacement that occurred in specimens with an isolated reverse Bankart repair ($1.6 \text{ mm} \pm 0.78 \text{ mm}$) ($p=0.0058$).

Conclusions: An osseous defect that is 20% of the posterior glenoid width remains unstable after isolated reverse Bankart repair.

Introduction

Posterior glenoid bone loss is a relatively uncommon scenario when compared to its' anterior counterpart. Despite this, it is reported as a contributing factor associated with recurrent posterior shoulder instability^{9,11,14}. It is important to understand that posterior bone deficiency often occurs concomitantly with other pathologies such as posterior labral tears, capsular laxity, posterior inferior glenohumeral ligament avulsions, and in the setting

of glenoid dysplasia¹⁴. Patients with a large defect are likely to be at an increased risk for recurrent instability after isolated capsulolabral repair. The size of this defect that is considered critical remains controversial⁹. Etiologies for posterior bone loss include: congenital hypoplasia, a traumatic event such as dislocation, or repetitive microtrauma often seen in young male athletes^{12,14,19}.

Previous investigations have examined the critical amount of bone loss in anterior shoulder instability^{6,17,20}. These results have demonstrated that with as little as a 15% anterior defect, a Bankart repair may fail to properly restore resistance to glenohumeral translation and stability, therefore necessitating a bony restoration procedure¹⁷. To our knowledge, there is no analogous biomechanical study that has quantified the critical bone defect involving the posterior glenoid.

The majority of patients with atraumatic posterior shoulder instability can improve with conservative treatment; however, a small subset will go on to require surgical intervention. In contrast, it has been demonstrated that up to 84% of patients with a traumatic etiology will ultimately fail conservative treatment^{3,12}. Arthroscopic posterior stabilization has proven to be a reliable option in most cases, albeit the risk of recurrence has demonstrated to be as high as 10%¹⁴. One of the primary factors that increase risk for recurrent instability is a failure to treat concomitant pathology such as anterior labral lesions, excessive glenoid retroversion, and posterior glenoid bone loss¹. There are multiple described posterior glenoid reconstruction procedures in cases of large amounts of bone loss; however, the indications for when to utilize these procedures remains unclear^{10,15,16}. The purpose of this present study was to identify the critical size of the posterior glenoid defect.

Materials and Methods

Preparation of Specimens

Eleven fresh-frozen cadaver shoulders (11 male donors) were used in this study. The average age of the subjects at the time of death was 58 years (range, 55 to 63 years). We excluded specimens with moderate to severe osteoarthritis present on pre-testing computerized tomography (CT) or during specimen preparation. We also excluded any specimens over the age of 65. The shoulders were thawed overnight at room temperature prior to testing. The skin, subcutaneous tissue, and posterior rotator cuff tendons were removed leaving the anterior structures intact, including the biceps tendon. A posterior capsulotomy was performed to gain access to the posterior glenoid and labrum. A posterior approach and capsulotomy was utilized in order for improved access and to mirror a posterior instability state in which the capsule and ligaments are typically redundant, loose and providing minimal restraint to posterior translation. The medial margin of the scapula was osteotomized along a line parallel to the glenoid surface. The humeral shaft was osteotomized 10 cm distal to the most superior portion of the humeral head. Both the scapula and humerus were potted in a two-part urethane compound (300Q, Smooth-On, Macungie, PA) utilizing custom fixtures. Prior to potting, the exposed bony ends were transfixed with two bicortical screws to increase rotational stability at the bone-potting interface. The glenoid surface was potted parallel to the horizontal plane while the humerus was potted in 30 degrees of abduction and 30 degrees of flexion in respect to the glenoid.

This positioning was chosen in order to simulate a load and shift test in which majority of the stability is being provided by the glenohumeral articulation including the humeral head and labrum rather than ligamentous and tendinous structures. After potting, all specimens underwent CT scanning prior to mounting.

A caliper (Mitutoyo, Takatsu-ku, Kawasaki, Kanagawa, Japan) was used to measure the posterior labral thickness for each specimen. The pre-testing CT volume images were used to measure the depth of glenoid, which was defined as the distance from the most posterior reference point on the rim to the deepest portion of the glenoid.

Testing Apparatus

All testing was performed with a six-axis industrial robot (KUKA KR 6 R700, Augsburg, Germany) that is integrated with simVITRO labview-based control software (Cleveland Clinic, Ohio, US). The robot is equipped with a multi-axis load cell (SI-580N, ATI, Apex, NC) at the end effector for the measurement of forces and torques in all three Cartesian directions. This interface provides a flexible musculoskeletal simulator to control and report joint kinetics and kinematics. The specimen was then mounted to the robot with the glenoid parallel to the base and humerus attached to the robot end effector (Fig 1). After mounting, the spatial relationships between the robot, load cell, and the specimen were established using a 6-DOF digitizing probe (Optotrak, NDI, CA). Glenoid coordinate system was generated via the digitization of bone-based anatomic landmarks determined within a specimen-specific CT scan.

A 50-N compressive force was constantly applied to the humerus to keep the humeral head compressed in the glenoid fossa. The initial reference position was determined to be the center of the glenoid based on the CT scan data. This reference position was confirmed when both the x and y forces recorded were minimized (~0 N) prior to translation. Starting from the initial reference position, the humeral head was translated in the posterior direction for 10 mm at a rate of 1.0 mm/sec. This 10 mm displacement protocol was utilized for the entire cohort as the glenoid widths were all measured as 30 ± 1 mm. These parameters were utilized in prior studies that examined the effect of anterior glenoid bone defects on anterior shoulder instability^{6,20}. The specimens were hydrated with normal saline prior to mounting and before each test to minimize cartilage desiccation. The peak force that occurred during humeral head translation and the lateral displacement (z axis) of the center of the humeral head were recorded. Three trials were performed for each condition, and the mean value was used for data analysis.

Human Cadavers:

Our institution purchased human cadaveric specimens from MedCure (Cumberland, RI) which was blinded. Permission was not required for basic science research.

Test Conditions

The humerus was positioned in 30° of abduction, flexion, and neutral rotation relative to the scapula. Testing was performed under 11 conditions: (1) intact glenoid, (2) simulated reverse Bankart lesion, (3) the reverse Bankart lesion repaired, (4) a 10% defect, (5) The reverse

Bankart lesion repaired, (6) a 20% defect, (7) the reverse Bankart lesion repaired (8) a 30% defect, (9) the reverse Bankart lesion repaired, (10) a 40% defect, and (11) the reverse Bankart repaired. A reverse Bankart lesion was created by elevating the capsulolabral attachment periosteally from the glenoid from six o'clock to 11 o'clock in the right shoulder.

Glenoid Defect and Labral Repair

Glenoid defects were created in 10% increments of the overall glenoid width, which was measured from the pre-testing CT volume images. Osteotomy lines were drawn in the superior-inferior direction of the y axis in respect to the glenoid center, which was used as the reference point (Figure 2). Osteotomies were initially made with a sagittal saw then completed with an osteotome (Figure 3). The posterior labrum was sharply detached from the glenoid rim from approximately the 6 to 11 o'clock positions creating a reverse Bankart lesion. Reverse Bankart repairs were performed by first creating 4 drill holes in the exposed glenoid rim utilizing a 1.0 mm drill bill and subsequently passing #2 Fiberwire sutures (Arthrex, Naples, FL) (Figure 3). With increasing glenoid defects, we placed our sutures through the most posterior portion of the glenoid face that remained.

Data Analysis

One-way repeated-measures analysis of variance was used to compare the change in peak force across the various glenoid defects with and without reverse Bankart repair. When a significant effect was observed, it was further analyzed separately with the use of the Dunnett Test. The conditions with an isolated reverse Bankart lesion was used as the baseline condition compared to bone defects without and with a reverse Bankart repair, respectively. The effect of labral thickness (<3 vs. 3 mm) and glenoid depth on peak force and lateral displacement was also evaluated. 3 mm was chosen as the labral thickness cutoff as this allowed for an even distribution of specimens. The effect of labral thickness was evaluated using a two-way repeated-measures analysis of variance and a linear regression analysis for evaluation of glenoid depth. The level of significance was set at $p < 0.05$. All statistical tests were performed with GraphPad Prism (7.0c, San Diego, CA)

Results

Stability

The mean peak force that occurred with posterior translation decreased incrementally after the creation of the reverse Bankart lesion and each subsequent osseous defect (Figure 4). The force associated with a 20% defect ($11 \pm 8\text{N}$) was significantly smaller than the baseline force (reverse Bankart lesion without a bony defect) [$19 \pm 9\text{N}$] ($p=0.0448$). Similarly, the peak force in shoulders with a reverse Bankart repair and a 20% defect ($13 \pm 9\text{N}$) was significantly decreased compared with the baseline force (reverse Bankart repair with no bony defect) [$22 \pm 10\text{N}$] ($p=.0451$). The mean glenoid width was 30 mm; therefore, a 20% defect is equivalent to a 6-mm defect. This critical defect corresponds to a bone defect in which an isolated reverse Bankart repair can no longer restore the original stability to posterior translation.

Lateral Humeral Displacement

The lateral humeral displacement decreased as the size of the osseous defect increased (Figure 5). Displacement after creating a 20% defect without (0.63 ± 0.54 mm) and with labral repair (0.61 ± 0.57 mm) was significantly smaller than the baseline displacements (intact glenoid with reverse Bankart lesion (1.62 ± 0.73 mm) ($p=0.0057$) and with reverse Bankart repair (1.57 ± 0.78 mm) ($p=0.0058$)). The lateral displacement was not restored to baseline with an isolated reverse Bankart repair in any condition with a bone defect.

Other Stability Factors

There was no significant difference in peak force between specimens with a labral thickness < 3mm and those with a labral thickness ≥ 3 mm for any of the tested conditions. Lateral humeral displacement was significantly greater in specimens with a labral thickness ≥ 3 mm when compared to those with a labral thickness < 3mm for the intact ($p=0.0350$), reverse Bankart lesion ($p=0.0056$), and the reverse Bankart lesion repair ($p=0.0121$) conditions. For the remaining conditions, there was no significant difference (Figure 6). There was no linear relationship between glenoid depth and stability ($p=0.6604$).

Discussion

The purpose of this study was to identify the critical size of the posterior glenoid defect that is associated with posterior shoulder instability that cannot be addressed with an isolated reverse Bankart repair. Our results indicate that an osseous defect 20% of the glenoid width (average 30 mm), which corresponds to a 6-mm defect, was the critical size of a glenoid defect in posterior shoulder instability. To our knowledge, this is the first study to apply this previously studied concept in anterior shoulder instability to a posterior shoulder model.

Prior cadaveric studies that have focused on determining the critical size of an anterior glenoid defect have demonstrated similar results ranging from 15% to 25% of the glenoid width^{6,17,20}. Though we were able to restore some stability with reverse Bankart repairs of 20% defects, this stability was found to be significantly decreased when compared to baseline. The peak force obtained for the intact specimens without any defects in this study was overall smaller than the peak translation force reported in prior anterior studies (23 N vs. ~ 68 N). However, the percentage decrease in force for subsequent simulated labral lesions and bone defects was comparable²⁰. We hypothesize that these discrepancies occurred secondary to the positioning of the arm during testing. Itoi et al. reported that that anteroinferior instability was highly dependent on shoulder position. In the mid-range of motion (abduction and neutral rotation), there was little effect on stability with increasing glenoid defects because of ligamentous restraints in their cadaver model⁶. During pilot testing, as expected, we found that at extremes of internal rotation and flexion, anterior capsuloligamentous structures became taut and resisted posterior glenohumeral translation. This inhibited our ability to detect a change in force even with larger bony defects as the humerus was translated posteriorly. Therefore, to isolate the effect of the glenoid and labrum on stability, we tested the shoulders in a rather relatively neutral position.

Previous studies have demonstrated that the magnitude of lateral humeral displacement is a representation of the total depth of the glenoid socket ^{7,8}. Our results demonstrated a significant difference in lateral displacement with a 20% defect for shoulders without and with reverse Bankart repairs when compared to the baseline lateral displacements. This is additional data that supports that a defect that measures over 20% of the glenoid width is not adequately addressed with isolate soft tissue capsulolabral repair. Though labral repairs were able to restore stability with bone defects, the lateral displacement did not increase. To explain this finding, we hypothesize that the capsulolabral tissue provides initial resistance to translation; however, as the humeral head continues to translate it essentially compresses the tissue secondary to an axial load and therefore minimal additional lateral displacement is detected.

There are no specific studies that focus on quantifying the posterior glenoid defect; however, several techniques both pre-operatively and intra-operatively have been extrapolated from the anterior instability literature ^{2,4,5,18}. The best-fit circle method is the most common technique used in which the percentage of the defect is calculated based on an assumed circle of the inferior portion of the glenoid. The use of 3-D CT has improved the overall accuracy in measuring the defect and has even been able to predict the arthroscopic decision to perform an anterior bone stabilization procedure ⁴. In a cadaver simulated bone loss model, Rerko et al. compared several imaging techniques. He demonstrated that 3D-CT had a lowest mean prediction error of -3.3 ± 6.6 in comparison to the other imaging modalities ¹³. We attempted to limit error in estimating our created glenoid defects by utilizing en-face 3D-CT cuts. We also had the advantage of measuring our planned osteotomies from an intact glenoid rather than measuring the defects after they were made.

The present study had several limitations. First, osseous defects were created by osteotomizing the glenoid in a superior-inferior direction in 10 mm increments. It is unclear if this type of bone loss is the most common type encountered clinically in patients with posterior shoulder instability. Because we tested bone loss in 10 mm increments, we cannot be sure if the critical amount of bone loss is exactly 20 mm or lies somewhere between 10 mm and 20mm. Second, we sacrificed the posterior rotator cuff tendons and capsule in order to adequately expose the posterior glenoid and perform cuts and repairs. The posterior soft tissue structures have demonstrated to play a role in overall posterior shoulder stability ^{1,12}. We attempted to limit the effect of the soft tissue restraint to translation by testing our specimens in an overall neutral position; therefore, stability primarily relied on the restraint provided by the glenoid and labrum. Third, the labral tissue underwent multiple detachment and repair cycles which may have contributed to its inability to resist translation therefore demonstrating a lower average peak force with increasing glenoid defects. Finally, the bone and soft tissue quality of cadaveric shoulders may not represent the tissues found in younger patients who typically present with posterior shoulder instability. We chose all male specimens under the age of 65 in attempt to match this cohort more accurately.

In conclusion, an osseous defect 20% of the glenoid width may be the critical posterior defect in which an isolated Bankart lesion repair fails to restore stability. In these instances, reconstruction of the posterior glenoid with a bone block may be warranted.

References

1. Bigliani LU , Kelkar R , Flatow EL , Pollock RG , Mow VC . Glenohumeral stability. Biomechanical properties of passive and active stabilizers. Clin Orthop Relat Res. 1996;(330):13–30. PMID: . [PubMed: 8804270]
2. Burkhart SS , Debeer JF , Tehrany AM , Parten PM . Quantifying glenoid bone loss arthroscopically in shoulder instability. Arthroscopy. 2002;18(5):488–491.PMID: [PubMed: 11987058]
3. Burkhead WZ , Rockwood CA . Treatment of instability of the shoulder with an exercise program. J Bone Joint Surg Am. 1992;74(6):890–896.PMID: . [PubMed: 1634579]
4. Chuang T-Y , Adams CR , Burkhart SS . Use of preoperative three-dimensional computed tomography to quantify glenoid bone loss in shoulder instability. Arthroscopy. 2008;24(4):376–382.PMID:. [PubMed: 18375267]
5. Griffith JF , Antonio GE , Tong CWC , Ming CK . Anterior shoulder dislocation: quantification of glenoid bone loss with CT. AJR Am J Roentgenol. 2003;180(5):1423–1430.PMID: [PubMed: 12704062]
6. Itoi E , Lee SB , Berglund LJ , Berge LL , An KN . The effect of a glenoid defect on anteroinferior stability of the shoulder after Bankart repair: a cadaveric study. J Bone Joint Surg Am. 2000;82(1):35–46. PMID: [PubMed: 10653082]
7. Lazarus MD , Sidles JA , Harryman DT , Matsen FA . Effect of a chondral-labral defect on glenoid concavity and glenohumeral stability. A cadaveric model. J Bone Joint Surg Am. 1996;78(1):94–102.PMID: . [PubMed: 8550685]
8. Lippitt SB , Vanderhooft JE , Harris SL , Sidles JA , Harryman DT , Matsen FA . Glenohumeral stability from concavity-compression: A quantitative analysis. J shoulder Elb Surg. 1993;2(1):27–35.PMID:. [PubMed: 2295924]
9. Longo UG , Rizzello G , Locher J , et al. Bone loss in patients with posterior gleno-humeral instability: a systematic review. Knee Surg Sports Traumatol Arthrosc. 2016;24(2):612–617.PMID: . [PubMed: 25051908]
10. Millett PJ , Schoenahl J-Y , Register B , Gaskill TR , van Deurzen DFP , Martetschläger F . Reconstruction of posterior glenoid deficiency using distal tibial osteoarticular allograft. Knee Surgery, Sport Traumatol Arthrosc. 2013;21(2):445–449. PMID: [PubMed: 23114865]
11. Moroder P , Plachel F , Tauber M , et al. Risk of Engagement of Bipolar Bone Defects in Posterior Shoulder Instability. Am J Sports Med. 2017;45(12):2835–2839.PMID: [PubMed: 28658583]
12. Provencher MT , LeClere LE , King S , et al. Posterior instability of the shoulder: diagnosis and management. Am J Sports Med. 2011;39(4):874–886.PMID: [PubMed: 21131678]
13. Rerko MA , Pan X , Donaldson C , Jones GL , Bishop JY . Comparison of various imaging techniques to quantify glenoid bone loss in shoulder instability. J shoulder Elb Surg. 2013;22(4):528–534.PMID: [PubMed: 22748926]
14. Robinson CM , Aderinto J . Recurrent Posterior Shoulder Instability. J Bone Jt Surg. 2005;87(4):883PMID: [PubMed: 15805222]
15. Schwartz DG , Goebel S , Piper K , Kordasiewicz B , Boyle S , Lafosse L . Arthroscopic posterior bone block augmentation in posterior shoulder instability. J Shoulder Elb Surg. 2013;22(8):1092–1101.PMID: [PubMed: 23337111]
16. Servien E , Walch G , Cortes ZE , Edwards TB , O'Connor DP . Posterior bone block procedure for posterior shoulder instability. Knee Surg Sports Traumatol Arthrosc. 2007;15(9):1130–1136. PMID: [PubMed: 17370061]
17. Shin S-J , Koh YW , Bui C , et al. What Is the Critical Value of Glenoid Bone Loss at Which Soft Tissue Bankart Repair Does Not Restore Glenohumeral Translation, Restricts Range of Motion, and Leads to Abnormal Humeral Head Position? Am J Sports Med. 2016;44(11):2784–2791. PMID: [PubMed: 27480979]
18. Sugaya H Techniques to evaluate glenoid bone loss. Curr Rev Musculoskelet Med. 2014;7(1):1–5. PMID: [PubMed: 24488759]
19. Wirth MA , Lyons FR , Rockwood CA . Hypoplasia of the glenoid. A review of sixteen patients. J Bone Joint Surg Am. 1993;75(8):1175–1184. PMID: . [PubMed: 8354676]

20. Yamamoto N , Muraki T , Sperling JW , et al. Stabilizing Mechanism in Bone-Grafting of a Large Glenoid Defect. *J Bone Jt Surgery-American* Vol. 2010;92(11):2059–2066. PMID: [PubMed: 20810855]

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Clinical Relevance:

A bony restoration procedure of the glenoid may be necessary in shoulders with a posterior glenoid defect that is 20 % of the glenoid width.

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What is known about the subject?

It is known that large posterior glenoid defects can be responsible for persistent shoulder instability. This can be effectively treated with a bone block type procedure. There are multiple studies regarding the critical size of an anterior glenoid bone defect in which a labral repair is not sufficient for stability.

What this study adds to existing knowledge:

This study quantifies the critical size of the posterior glenoid defect in which a labral repair is no longer sufficient to confer stability. To our knowledge, there is no analogous biomechanical study that has quantified the critical bone defect involving the posterior glenoid.

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Figure 1:
Kuka Industrial Robot (GmbH, Ausburg, Germany) with mounted specimen.

Figure 2.

En face view of the glenoid showing simulated osteotomy lines (blue) in 10% increments of the total glenoid width, which were drawn parallel to the y axis. The x and y axes pass through the center (C) which was determined from pre-testing CT scans.

Figure 3. Creation of a 20% bone defect (left) and typical transosseous reverse Bankart repair in an intact specimen (right).

Figure 4.

Histogram demonstrating stability. The peak force decreased with the creation of each subsequent defect size (blue bars) and those with reverse Bankart lesion repairs (grey bars).

* Compared with shoulders with a reverse Bankert lesion without a defect [$19 \pm 9\text{N}$], force decreased significantly with an osseous defect 20% of the glenoid width [$11 \pm 8\text{N}$] ($p < 0.05$). † Compared with shoulders with a reverse Bankert lesion repair without a defect [$22 \pm 10\text{N}$], the force decreased significantly in repairs with a defect 20% [$13 \pm 9\text{N}$] ($p < 0.05$).

Figure 5.

Histogram demonstrating the lateral humeral displacement. The lateral humeral displacement decreased with the creation of each subsequent defect size (blue bars) and those with reverse Bankart lesion repairs (grey bars). * Compared with shoulder with a reverse Bankart lesion without a defect [$1.6 \pm 0.73\text{N}$], the lateral displacement decreased significantly with an osseous defect 20% of the glenoid width [$0.63 \pm 0.54\text{N}$] ($p < 0.05$). † Compared with shoulders with a reverse Bankart lesion repair without a defect [$1.6 \pm 0.78\text{N}$], the lateral displacement decreased significantly in repairs with a defect 20% [$0.61 \pm 0.57\text{N}$] ($p < 0.05$).

Figure 6.

Graph demonstrating the effect of labral thickness on lateral humeral displacement. * Lateral humeral displacement was significantly greater in specimens with a labral thickness ≥ 3 mm when compared to those with a labral thickness < 3 mm for the intact ($p=0.035$), reverse Bankart lesion ($p=.0056$), and the reverse Bankart lesion repair ($p=0.0121$) conditions. There was no significant difference in the remaining conditions.