Recurrent Instability After Arthroscopic Bankart Repair in Patients With Hyperlaxity and Near-Track Lesions

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Background: Recurrent anterior shoulder instability after arthroscopic Bankart repair presents a challenging clinical problem, with the primary stabilization procedure often portending the best chance for clinical success.

Purpose: To determine if capsuloligamentous laxity affects failure (recurrent dislocation, subluxation, and/or perceived instability symptoms) after arthroscopic Bankart repair in patients with near-track lesions (ie, those with smaller distance to dislocation [DTD]).

Study Design: Case-control study; Level of evidence, 3.

Methods: The authors retrospectively reviewed consecutive patients who underwent primary arthroscopic Bankart repair for recurrent anterior glenohumeral instability at a single institution between 2007 and 2019 and who had at least 2 years of follow-up data. Patients with glenoid bone loss >20%, off-track lesions, concomitant remplissage, or rotator cuff tear were excluded. Capsuloligamentous laxity, or hyperlaxity, was defined as external rotation >85° with the arm at the side and/or grade ≥2 in at least 2 planes with the shoulder at 90° of abduction. Near-track lesions were defined as those with a DTD <10 mm.

Results: Included were 173 patients (mean age, 20.5 years; mean DTD, 16.2 mm), of whom 16.8% sustained a recurrent dislocation and 6.4% had recurrent subluxations (defined as any subjective complaint of recurrent instability without frank dislocation), for an overall recurrent instability rate of 23.1%. The rate of revision stabilization was 15.6%. The mean time to follow-up was 7.4 years. Independent predictors of recurrent instability were younger age ($P = .001$), smaller DTD ($P = .021$), >1 preoperative instability episode ($P < .001$), and the presence of hyperlaxity during examination under anesthesia ($P = .013$). Among patients with near-track lesions, those with hyperlaxity had a recurrent instability rate almost double that of patients without hyperlaxity (odds ratio, 34.1; $P = .04$). The increased rate of failure and recurrent dislocation in the near-track hyperlaxity cohort remained elevated, even in patients with no bone loss.

Conclusion: Capsuloligamentous shoulder laxity was a significant independent risk factor for failure after primary arthroscopic Bankart repair without remplissage and was more predictive of failure in patients with versus without near-track lesions.

Keywords: anterior instability; glenoid track; laxity; distance to dislocation

Recurrent anterior shoulder instability after arthroscopic Bankart repair presents a challenging clinical problem, and the primary stabilization procedure is largely considered to portend the best chance for clinical success. Numerous studies have identified risk factors for failure (ie, recurrent instability) after primary arthroscopic anterior shoulder stabilization, including younger age, male sex, increased generalized and shoulder laxity, bone loss, participation in contact sports, and <3 anchors.[1,6,8] With a growing armamentarium of management options for patients with recurrent instability and subcritical bone loss, including arthroscopic Bankart repair with or without remplissage, open Bankart repair, open bone block procedures (eg, Bristow, Latarjet, distal tibial allograft, distal clavicle autograft, and iliac crest autograft), and arthroscopic bony augmentation, choosing the appropriate initial

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stabilization procedure while considering the individual patient and procedure complication profile can be a daunting task. The arthroscopic Bankart repair without remplissage remains a good option for select patients, but indications for alternate procedures and augmentation continue to expand as more research delineates patient populations with a higher risk of failure.

The glenoid track (GT), introduced by Yamamoto et al,[28] accounts for bipolar glenoid and humeral bone loss. Using the GT concept, off-track lesions have been shown to have a strong association with failure after arthroscopic Bankart repair alone without remplissage.[14,17,24] More recent studies have shown that the GT should be considered more dynamically, as a subset of on-track lesions may also be at increased risk of failure after arthroscopic Bankart repair. Specifically at risk are "near-track" lesions (those with a distance to dislocation [DTD] <10 mm) and "peripheral-track" lesions (those with Hill-Sachs occupancy ≥75% of the GT).[4,19,29]

While the GT concept continues to evolve, there are notable limitations. One limitation of the GT is that it is constrained to bony anatomy. Capsuloligamentous integrity or capsular laxity may play an important role in the glenohumeral contact points throughout shoulder range of motion (ROM). In the present study, we evaluated the effect of capsuloligamentous laxity and DTD on failure after arthroscopic Bankart repair. The purpose of this study was to determine whether shoulder capsuloligamentous laxity has a modifying effect on the GT, specifically for near-track lesions, which may help to explain why some on-track lesions are at an increased risk of recurrent instability. We hypothesized that ligamentous laxity and near-track lesions would be at increased risk of recurrent instability after arthroscopic Bankart repair.

METHODS

Study Population

Institutional review board approval was obtained for the study protocol, as it was a retrospective review of data participants did not need to provide informed consent. We reviewed consecutive patients who underwent primary arthroscopic Bankart repair for recurrent anterior glenohumeral instability between January 2007 and December 2019 at a single institution. The diagnosis of primary anterior glenohumeral instability was established based on history and examination findings consistent with anterior glenohumeral instability and evidence of anterior glenohumeral instability on preoperative magnetic resonance imaging (MRI), as described by Gartsman et al.[11]

Patients were included in the analysis if they had (1) clinical anterior instability findings confirmed with preoperative MRI, (2) intraoperative Bankart lesion or its variants, (3) documentation of shoulder examination under anesthesia (EUA) in the operative report, (4) undergone primary arthroscopic Bankart repair for anterior instability without remplissage, and (5) at least 2 years of clinical follow-up. Exclusion criteria were (1) lack of documentation of shoulder EUA, (2) presence of diagnosed connective tissue disorder, (3) concomitant rotator cuff tear, (4) <3 anchors used in repair, (5) off-track Hill-Sachs lesion, and (6) critical bone loss >20%.

Operative and Postoperative Protocols

All procedures were performed by 1 of 5 fellowship-trained orthopaedic sports medicine surgeons with >5 years of postfellowship experience (4 were authors: A.L., M.R., V.M., and B.P.L.). All surgeries were performed with the use of standard arthroscopic suture anchor techniques. Capsular plication was performed according to surgeon preference. Postoperatively, patients underwent a similar physical therapy protocol and returned to activities as tolerated after 5 months. Patients used a sling for 4 to 6 weeks with initiation of gradual passive ROM starting around 4 weeks postoperatively. Gradual increase of external rotation and progressive active ROM started at 6 weeks. Strengthening was initiated around 12 weeks. Return to sport was determined by surgeon preference, guided by criteria-based return-to-sport testing.

Outcome Evaluation

Patient and clinical data were prospectively collected and retrospectively reviewed at a single academic institution. This included patient sex, age at time of surgery, extremity involved, contact sport, number of anchors used, number of preoperative instability episodes, and associated pathology noted intraoperatively. Clinical notes were reviewed to obtain clinical outcomes including postoperative recurrent

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dislocation, recurrent subluxation, or subjective instability episodes and the need for revision stabilization surgery. Recurrent dislocation was defined as a postoperative dislocation event, and recurrent instability was defined as any postoperative recurrent subluxation event or subjective return of instability symptoms. Failure was defined as any recurrent instability event (recurrent dislocation, subluxation, and/or perceived instability symptoms) after the initial stabilization procedure. The duration of clinical follow-up was recorded.

Quantification of Capsuloligamentous Laxity. Classification of shoulder capsuloligamentous laxity (ie, hyperlaxity) was based on EUA, which was performed at the time of surgery before arthroscopy. Examination included anterior load and shift, posterior load and shift, and sulcus testing, which was graded according to Antoniou et al[2] on humeral head translation with respect to the glenoid rim: grade 0 indicated no translation, grade 1 reflected translation of the humeral head up to the glenoid rim, grade 2 reflected humeral head translation over the glenoid rim with spontaneous reduction, and grade 3 reflected humeral head translation over the glenoid rim without spontaneous reduction. Consistent with previous studies, capsuloligamentous laxity was defined as external rotation $>85°$ with the arm at the side and/or a grade ≥ 2 in at least 2 planes (anterior, posterior, and/or inferior) with the shoulder at 90° of abduction.[13,24,25] Total capsuloligamentous laxity was quantified by adding the values for anterior translation, posterior translation, and sulcus to get a total glenohumeral laxity score.

Measurement of Glenoid Bone Loss, GT, and DTD. Measurements of glenoid bone loss and length of Hill-Sachs lesions were performed as previously described using 3-T T1-weighted MRI sequences.[16] Glenoid bone loss was measured on sagittal plane MRI at the level of the coracoid base using the circle of best fit method.[16] The percentage of glenoid bone loss was calculated as the ratio between the diameter of the maximal anterior glenoid bone loss (d) and the diameter of the glenoid using the best-fit circle (D). The Hill-Sachs interval (HSI) was measured on axial plane images at the level of the widest medial extent of the Hill-Sachs lesion.[15] HSI was measured as the distance between the medial-most edge of the Hill-Sachs lesion and the insertion of the infraspinatus tendon. The GT was calculated based on the method described by Yamamoto et al[28]: $GT = 0.83(D - d)$. Measurements of D, d, and HSI were performed by an independent orthopaedic sports medicine surgery fellow and repeated 2 weeks later, and a subset of 30 measurements were made by a separate reviewer (J.D.H.). Intra- and interrater reliability were calculated using the intraclass correlation coefficient (ICC).

DTD was calculated according to Li et al[19] as $GT - HSI$. With this classification system, on-track Hill-Sachs lesions have a DTD >0 mm, off-track lesions have a DTD ≤ 0 mm, and near-track lesions are defined as 0 mm $<$ DTD $\leq$ 10 mm.[19]

Statistical Analysis

Quantitative variables were expressed as means ± standard deviations, and categorical variables were expressed as frequencies and percentages. The chi-square, Fisher exact, and Student t tests were used to validate the inclusion or exclusion of a priori major confounders of the relationship between shoulder laxity and recurrent instability. The relationship between shoulder laxity and recurrent instability was evaluated via logistic regression analyses with and without adjustment for several covariates. When building the multivariate regression model, a set of variables were considered important a priori major confounders of the relationship: baseline age, sex, DTD, number of preoperative instability episodes, and contact sport athlete. The statistical validity of including each of these variables in the final multivariate model was assessed using the likelihood ratio tests. Significance was set to $P < .05$. All analyses were conducted using Stata 17 software (StataCorp).

RESULTS

Of 251 patients initially identified, 213 met inclusion criteria; 17 were excluded due to inadequate documentation of EUA, 10 due to the use of <3 anchors in their instability repair, 5 for the presence of concomitant rotator cuff tear, 4 for the presence of an off-track Hills-Sachs lesion, and 4 for critical bone loss >20%. Thus, 173 patients were included in the analysis: 136 male (78.6%) and 37 female (21.4%) patients with a mean age of 20.48 ± 6.0 years at time of surgery and a mean follow-up time of 7.37 years (range, 2.2-14.4 years). Average DTD was 16.2 ± 5.7 mm. A total of 29 patients (16.8%) sustained a recurrent dislocation and 11 patients (6.4%) reported recurrent subluxations (any subluxation event or subjective instability). The combined overall failure rate (patients with any recurrent dislocation, subluxation, and/or perceived instability) was 23.1% (16.8% recurrent dislocation, 6.4% recurrent instability). The overall rate of revision stabilization was 15.6%. The inter- and intrarater reliability for DTD were good (ICC, 0.71) and excellent (ICC, 0.82), respectively.

When compared with patients without recurrent instability after arthroscopic Bankart repair (n = 133), patients with recurrent instability (n = 40) were younger ($P = .002$) and had larger-diameter glenoid bone loss ($P < .001$), a larger percentage of glenoid bone loss ($P < .001$), a longer Hill-Sachs lesion length ($P = .01$), a smaller DTD ($P = .004$), a higher incidence of >1 preoperative instability episode ($P < .001$), and an increased intraoperative shoulder laxity ($P = .007$) (Table 1). When comparing patients with hyperlaxity (n = 26) versus normal laxity (n = 147), the patients with hyperlaxity included significantly fewer contact sport athletes ($P = .008$), had a larger number of anchors used during instability repair ($P < .001$), and had a higher grade for total intraoperative laxity ($P < .001$) and sulcus testing ($P < .001$) (Table 2). When comparing patients with and without recurrent dislocation after arthroscopic Bankart repair (n = 29 vs n = 144, respectively), the patients with recurrent dislocation were significantly younger ($P = .02$) and had more glenoid bone loss, measured both directly and as a percentage ($P < .001$ for both); a larger Hill-Sachs

TABLE 1
Comparison of Patients With and Without Recurrent Instability (Subluxation and Dislocation) After Arthroscopic Bankart Repair[a]

	Recurrent Instability (n = 40)[b]	No Recurrent Instability (n = 133)[b]	*P*
Age, y	17.9 ± 2.4	21.3 ± 6.6	**.002**
Female, No. of shoulders	12 (30.0)	32 (24.1)	.45
Contact sport	33 (82.5)	96 (72.2)	.19
Glenoid bone loss, mm	2.1 ± 2.0	0.58 ± 1.2	**<.001**
Glenoid bone loss, %	7.0 ± 6.9	2.0 ± 4.0	**<.001**
Hill-Sachs lesion length, mm	7.7 ± 4.9	5.5 ± 4.8	**.01**
No. of anchors	4.4 ± 1.4	4.3 ± 1.7	.74
Degrees of tear (of 360°)	123.6 ± 47.5	140.7 ± 58.1	.35
Distance to dislocation, mm	13.9 ± 6.1	16.8 ± 5.4	**.004**
>1 preoperative instability event	35 (87.5)	65 (48.9)	**<.001**
Associated pathology			
SLAP tear	10 (25.0)	31 (23.3)	.83
Bony Bankart lesion	1 (2.5)	6 (4.5)	>.99
Loose body	4 (10.0)	18 (13.5)	.79
GLAD lesion	5 (12.5)	24 (18.0)	.41
ALPSA lesion	4 (10.0)	18 (13.5)	.56
HAGL lesion	1 (2.5)	1 (0.8)	.41
Intraoperative laxity grade			
Total laxity	4.1 ± 1.3	3.4 ± 1.4	**.007**
Anterior load and shift	2.3 ± 0.5	2.1 ± 0.6	**.03**
Posterior load and shift	0.9 ± 0.6	0.8 ± 0.7	.19
Sulcus	0.9 ± 0.7	0.6 ± 0.6	**.008**

[a]Data are reported as mean ± SD or n (%). Boldface *P* values indicate a statistically significant difference between groups ($P < .05$). ALPSA, anterior labroligamentous periosteal sleeve avulsion; GLAD, glenoid labrum articular disruption; HAGL, humeral avulsion glenohumeral ligament; SLAP, superior labral tear from anterior to posterior.
[b]Symptoms of recurrent dislocation, recurrent subluxation, or both.

TABLE 2
Baseline Characteristics of Patients With and Without Hyperlaxity at the Time of Arthroscopic Bankart Repair[a]

	Hyperlaxity (n = 26)	Normal Laxity (n = 147)	*P*
Age, y	22.7 ± 7.6	20.1 ± 5.7	.05
Female, No. of shoulders	9 (34.6)	35 (23.8)	.24
Contact sport athlete	14 (53.8)	115 (78.2)	**.008**
Glenoid bone loss, mm	0.7 ± 1.3	1.0 ± 1.6	.47
Glenoid bone loss, %	2.4 ± 4.5	3.3 ± 5.4	.43
Hill-Sachs length, mm	6.7 ± 4.9	5.8 ± 4.9	.41
No. of anchors	5.5 ± 2.3	4.1 ± 1.4	**<.001**
Distance to dislocation, mm	15.9 ± 5.8	16.2 ± 5.7	.76
>1 preoperative instability event	19 (73.1)	81 (55.1)	.09
Associated pathology			
SLAP tear	9 (34.6)	32 (21.8)	.16
Bony Bankart lesion	1 (3.8)	6 (4.1)	.72
Loose body	2 (7.7)	20 (13.6)	.54
GLAD lesion	3 (11.5)	26 (17.7)	.57
ALPSA lesion	2 (7.7)	20 (13.6)	.54
HAGL lesion	0 (0.0)	2 (1.4)	>.99
Intraoperative laxity grade			
Total laxity	5.5 ± 0.9	3.2 ± 1.2	**<.001**
Anterior load and shift	2.3 ± 0.5	2.1 ± 0.6	**.02**
Posterior load and shift	1.7 ± 0.7	0.6 ± 0.6	**<.001**
Sulcus	1.5 ± 0.8	0.6 ± 0.5	**<.001**

[a]Data are reported as mean ± SD or n (%). Boldface *P* values indicate a statistically significant difference between groups ($P < .05$). ALPSA, anterior labroligamentous periosteal sleeve avulsion; GLAD, glenoid labrum articular disruption; HAGL, humeral avulsion glenohumeral ligament; SLAP, superior labral tear from anterior to posterior.

TABLE 3
Comparison of Patients With and Without Recurrent Dislocation After Arthroscopic Bankart Repair[a]

	Recurrent Dislocation (n = 29)	No Recurrent Dislocation (n = 144)	*P*
Age, y	18.1 ± 2.7	21.0 ± 6.4	**.02**
Female, No. of shoulders	7 (24.1)	37 (25.7)	.86
Contact sport athlete	23 (79.3)	106 (73.6)	.52
Glenoid bone loss, mm	2.2 ± 2.2	0.5 ± 1.1	**<.001**
Glenoid bone loss, %	7.4 ± 7.6	1.8 ± 4.1	**<.001**
Hill-Sachs length, mm	7.4 ± 4.6	5.5 ± 5.1	**.04**
No. of anchors	4.2 ± 1.3	3.9 ± 1.3	.19
Distance to dislocation, mm	13.9 ± 5.7	16.2 ± 5.6	**.03**
>1 instability event preoperatively	26 (89.7)	74 (51.4)	**<.001**
Associated pathology			
SLAP tear	9 (31.0)	32 (22.2)	.31
Bony Bankart lesion	1 (3.4)	6 (4.2)	>.99
Loose body	2 (6.9)	20 (13.9)	.38
GLAD lesion	3 (10.3)	26 (18.1)	.42
ALPSA lesion	4 (13.8)	18 (12.5)	.77
HAGL lesion	1 (3.4)	1 (0.7)	.31
Intraoperative laxity grade			
Total laxity	4.0 ± 1.2	3.5 ± 1.4	.07
Anterior load and shift	2.2 ± 0.5	2.1 ± 0.6	.17
Posterior load and shift	0.9 ± 0.6	0.8 ± 0.7	.41
Sulcus	0.9 ± 0.7	0.7 ± 0.6	.06

[a]Data are reported as mean ± SD or n (%). Boldface *P* values indicate a statistically significant difference between groups ($P < .05$). ALPSA, anterior labroligamentous periosteal sleeve avulsion; GLAD, glenoid labrum articular disruption; HAGL, humeral avulsion glenohumeral ligament; SLAP, superior labral tear from anterior to posterior.

TABLE 4
Multivariate Predictors of Recurrent Instability, Dislocation, or Subluxation After Primary Arthroscopic Bankart Repair[a]

	Failure (n = 173)[b]		Recurrent Dislocation (n = 173)		Recurrent Subluxation (n = 144)	
	OR (95% CI)	*P*	OR (95% CI)	*P*	OR (95% CI)	*P*
Age	0.80 (0.70-0.91)	**.001**	0.84 (0.74-0.96)	**.009**	0.74 (0.55-0.98)	**.001**
Distance to dislocation	0.92 (0.85-0.99)	**.021**	0.89 (0.82-0.97)	**.007**	0.99 (0.88-1.12)	.91
>1 preoperative instability episode	8.10 (2.77-23.6)	**<.001**	7.50 (2.08-27.1)	**.002**	6.62 (1.25-35.1)	**.03**
Hyperlaxity	4.37 (1.37-13.9)	**.013**	2.78 (0.84-9.22)	.09	5.42 (1.04-28.3)	**.04**

[a]Boldface *P* values indicate statistical significance ($P < .05$). OR, odds ratio.
[b]Recurrent dislocation, subluxation, and/or perceived instability.

length ($P = .04$); a smaller DTD ($P = .03$); and a higher incidence of >1 instability event preoperatively ($P < .001$) (Table 3).

Multivariate analysis demonstrated that significant independent predictors of failure after arthroscopic Bankart repair were younger age (odds ratio [OR], 0.80; 95% CI, 0.70-0.91; $P = .001$), smaller DTD (OR, 0.92; 95% CI, 0.85-0.99; $P = .021$), >1 preoperative instability episode (OR, 8.10; 95% CI, 2.77-23.6; $P < .001$), and the presence of hyperlaxity during EUA (OR, 4.37; 95% CI, 1.37-13.9; $P = .013$) (Table 4).

When controlling for glenoid bone loss, patients with near-track lesions and hyperlaxity had significantly higher failure rates than those without hyperlaxity (60.0% vs 38.5%; $P = .04$) with an OR of 34.1 and an area under the receiver operating characteristic curve (AUC) of 0.91 (Table 5). Patients with hyperlaxity and on-track lesions also had higher failure rates than those without hyperlaxity (33.3% vs 16.5%; $P = .04$), with an OR of 2.94 and a less predictive AUC of 0.68 (Table 5). A similar significant trend was found with regard to recurrent dislocations, with the largest effect of hyperlaxity seen in patients with near-track lesions (60.0% vs 30.8%; $P = .04$; OR, 28.5; AUC, 0.86).

Recurrent dislocation was a strong predictor of secondary surgery (OR, 225.6; 95% CI, 50.6-1006.4; $P < .001$), whereas recurrent subluxation was not predictive of reoperation risk ($P = .28$). Approximately 83% (24/29) of patients who experienced a recurrent dislocation underwent reoperation, while only 27% (3/11) of patients who experienced recurrent subluxation underwent secondary surgery.

TABLE 5
Recurrent Instability and Dislocation Rates Stratified by DTD, Shoulder Laxity, and Glenoid Bone Loss[a]

	Hyperlaxity (n = 26)	Normal Laxity (n = 147)	AUC[b]	OR (95% CI)[c]	*P*[c]
Recurrent instability rates					
All patients	38.5 (10/26)	20.4 (30/147)	0.75	3.60 (1.35-9.62)	**.01**
Near-track lesion[d]	60.0 (3/5)	38.5 (10/26)	0.91	34.1 (1.25-926.2)	**.04**
Glenoid bone loss	100 (1/1)	62.5 (10/16)	—	—	—
No glenoid bone loss	50.0 (2/4)	0 (0/10)	—	—	—
On-track lesion[e]	33.3 (7/21)	16.5 (20/121)	0.68	2.94 (1.01-8.57)	**.04**
Glenoid bone loss	28.6 (2/7)	29.0 (9/31)	—	—	—
No glenoid bone loss	35.7 (5/14)	12.2 (11/90)	—	—	—
Recurrent dislocation rates					
All patients	26.9 (7/26)	15.0 (22/147)	0.73	2.84 (0.98-8.23)	*.05*
Near-track lesion[d]	60.0 (3/5)	30.8 (8/26)	0.86	28.5 (1.25-647.7)	**.04**
Glenoid bone loss	100 (1/1)	50.0 (8/16)	—	—	—
No glenoid bone loss	50.0 (2/4)	0 (0/10)	—	—	—
On-track lesion[e]	19.0 (4/21)	11.6 (14/121)	0.63	1.98 (0.57-6.90)	.28
Glenoid bone loss	14.3 (1/7)	19.4 (6/31)	—	—	—
No glenoid bone loss	21.4 (3/14)	8.9 (8/90)	—	—	—

[a]Data are reported as % (n/total for that group). Statistically significant differences between groups are indicated by italic ($P < .10$) and boldface ($P < .05$) *P* values. Dashes indicate areas not applicable. AUC, area under the receiver operating characteristic curve; DTD, distance to dislocation; OR, odds ratio.

[b]Based on the computed bivariate logistic regression models.

[c]Based on bivariate regression analysis on hyperlaxity as a predictor for recurrent instability or recurrent dislocation after adjusting for percentage of glenoid bone loss and stratifying by near-track versus on-track status.

[d]0 mm < DTD ≤ 10 mm

[e]DTD >10 mm.

DISCUSSION

The major finding of this study was that shoulder capsuloligamentous laxity at the time of EUA was independently predictive of recurrent instability after arthroscopic Bankart repair alone for primary anterior glenohumeral instability, with a 4.37-fold increased odds of failure. This effect was magnified in patients with near-track lesions, with a 34.1-fold increased odds of failure in this high-risk patient population. In patients with near-track lesions and hyperlaxity, a 60% failure rate was found, with high failure rates even in patients with no glenoid bone loss (Table 5).

The GT concept was introduced by Yamamoto et al[28] and was described in a dichotomous manor with lesions falling into 1 of 2 categories: on-track or off-track. Patients with on-track lesions are generally thought to be appropriate candidates for arthroscopic Bankart repair without remplissage. Despite this delineation, there appears to be a subset of patients with on-track lesions who remain at higher risk of failure after arthroscopic Bankart repair alone, and recent research has attempted to further delineate which patients may be particularly at risk. Li et al[19] introduced the concept of near-track lesions, defined as on-track lesions with a DTD <8 mm, which were shown to have a significantly higher rate of failure after arthroscopic Bankart repair alone using receiver operating characteristic curves.[19] They also found a lower threshold of 10 mm for patients <20 years of age.[19] This same threshold of <10 mm was also demonstrated by Barrow et al[4] with exponentially higher failure rates at lower values. Both studies demonstrated that failure rates increase as DTD decreases and suggested that clinicians should consider DTD as a continuous variable in surgical decision-making.[4,19] Our results corroborate the results of these studies and demonstrate that DTD is a significant predictor of failure and recurrent instability in patients undergoing primary arthroscopic Bankart repair without remplissage.

Shoulder hyperlaxity has been associated with an increased recurrence risk after primary arthroscopic Bankart repair alone and is included in the Instability Severity Index Score as a risk factor for failure.[3] However, the clinical validity and predictive value of the Instability Severity Index Score have been called into question by more recent studies that have failed to find a correlation between outcomes and Instability Severity Index Score using the previously established cutoff threshold.[7,22] Shoulder hyperlaxity has been shown to be an independent risk factor for failure after revision arthroscopic anterior shoulder stabilization.[25] Similarly, studies have shown an increased risk of recurrence after arthroscopic Bankart with a stretched inferior glenohumeral ligament or anterior hyperlaxity (external rotation, >90°).[6] An increase in capsular volume is correlated with higher risks of redislocation and a positive apprehension sign as well as with poorer Rowe scores.[21] While hyperlaxity is a well-established predictor of failure of arthroscopic anterior shoulder stabilization, to date no study has evaluated the interplay that hyperlaxity may have with the GT. We found the presence of a positive sulcus sign to be significantly

associated with shoulder laxity, and it may be a hallmark of shoulder capsuloligamentous laxity. Our findings are consistent with the existing literature that shows shoulder laxity increases failure rates after arthroscopic Bankart repair alone, but more importantly they suggest that laxity plays a much more significant role in patients with a smaller DTD with or without glenoid bone loss.

Preoperative instability has also been well established as a risk factor for failure after arthroscopic Bankart repair alone.[10,12,18,25] In concordance with the results of numerous previous studies that have shown a higher risk of failure in patients with an increasing number of preoperative instability events, our study also found that >1 preoperative instability episode was a significant predictor of failure after arthroscopic Bankart repair alone. In these cases, other surgical approaches including remplissage augmentation, open approaches, or bony augmentation can be considered.

Athletes of contact sports have been identified as patients at higher risk of failure after arthroscopic Bankart repair alone.[26] Our study had a large proportion of contact athletes (74.6%), which may help explain the higher recurrent instability rate found in our study. Contact athletes were less likely to have hyperlaxity, which may be attributable to athlete sport self-selection (ie, athletes with shoulder laxity may self-select out of contact sports) or may be a result of sport-specific shoulder adaptations. We did not find contact sport status to be independently predictive of recurrent instability in our cohort, which may be attributable to the older age of patients in our study. The mean age of patients in our study was 20.5 years, and contact athletes who have been identified as particularly at risk for recurrence are <20 years. The older age of patients in this study is likely in part due to the increasing trend to manage anterior instability in younger contact athletes with surgeries other than arthroscopic Bankart repair alone.

While our study is not the first to identify capsuloligamentous shoulder laxity as a risk factor for failure after arthroscopic Bankart repair, it may be the first to quantify the significant increased odds of failure in patients with shoulder hyperlaxity noted at the time of EUA and the first to suggest that this is even more critical in patients with a smaller DTD. While arthroscopic Bankart repair alone remains a good option for select patients with anterior shoulder instability, we caution against its use in patients with near-track lesions, patients with hyperlaxity, and patients with >1 preoperative instability episode. Alternate procedures are not without their own risks and complication profiles. Despite knowledge of increased risk of failure in young athletes with subcritical bone loss, many surgeons still attempt arthroscopic Bankart repair alone because of concerns for complications associated with alternate procedures. It is important to recognize the role that capsuloligamentous laxity plays in these patients, as a 60% failure rate in patients with a small DTD would be deemed unacceptable by most surgeons. Additionally, the failure rate seen in patients with hyperlaxity and near-track lesions remains high (50%) even in patients with no glenoid bone loss.

Several alternative approaches can be considered for this challenging patient population. The open Bankart repair has been shown to have lower reoperation rates and reliable long-term outcomes; however, some techniques require a takedown of the subscapularis tendon, which may result in loss of external rotation or long-term dysfunction with failure of healing.[20] Remplissage is becoming increasingly popular as an adjunct to both arthroscopic Bankart repair and bone augmentation procedures, but there remains concern in some studies regarding lower rates of return to sport at the same level in patients and external rotation stiffness risks after remplissage.[5,9] Open bone block procedures, including Bristow, Latarjet, distal clavicle autograft, and distal tibia allograft, among others, also carry well-known associated complications, including neurological injury, screw prominence, osteolysis, and nonunion, among others.[13,27] As arthroscopic bone block procedures continue to advance, they may become more desirable as they provide bony augmentation while minimizing the risk of neurovascular injuries and preserving the subscapularis. Further research is needed to evaluate these alternate stabilization procedures and the role that capsuloligamentous laxity may play in their outcomes.

While DTD is calculated preoperatively, shoulder hyperlaxity is best evaluated during EUA. Therefore, patients should be counseled preoperatively that the surgical plan may change intraoperatively based on EUA findings. Discussion of alternate surgical options should be thoroughly discussed in the clinic, so that patients are prepared for adjunct procedures when indicated and can participate in informed decision-making on the use of alternate procedures, especially in high-risk patients.

Limitations

The results of this study should be interpreted with consideration of certain limitations. Because of the retrospective nature of this study, it is prone to inherent biases. Additionally, patient-reported outcomes and return-to-sport information were not routinely collected and therefore not included in analysis. Shoulder capsuloligamentous laxity was defined using a validated and commonly used scoring system; however, the EUA was performed by the operating surgeon only, which may increase the risk of subjective scoring differences. Additionally, Beighton criteria were not routinely documented and therefore were not used in this study, but they should be a standard part of the documentation for shoulder instability patients, as the criteria provide information regarding generalized ligamentous laxity, which is a known risk factor for failure of arthroscopic stabilization.

While EUA data on the operative shoulder were collected, this study did not compare shoulder stability in the contralateral shoulder, so it is difficult to distinguish between inherent laxity in the shoulder and laxity secondary to capsular damage. Despite this limitation, our results suggest that regardless of the cause of shoulder laxity, the presence of laxity places the patient at significantly increased risk of failure after arthroscopic Bankart repair

alone, and patients with laxity and a small DTD are placed at an even higher risk. The failure rate of 23.1% may be considered high compared with reported rates in the literature. The reasons for this are likely twofold and include the relatively long follow-up of our cohort in addition to the inclusive definition of failure to include any subjective complaints of instability and not just recurrent dislocation. We believe this definition is a more realistic representation of postoperative patient concerns. Additionally, a recent study of long-term outcomes after Bankart repair demonstrated long-term recurrent instability rates of 27% in patients with on-track lesions, which helps to corroborate our results.[23]

The largest limitation of the study is the relatively small number of patients with near-track lesions and hyperlaxity, which limits the ability to interpret our results. While the sample size for this group is quite small, this is likely in part attributable to the unique patient population that falls into this category, and possible selection bias, as these patients may undergo arthroscopic Bankart repair with remplissage or some other alternative procedure at this institution. Additionally, even with the small sample size, the difference in recurrent instability was statistically significant. Future research with a more robust patient cohort may help delineate the effect of capsuloligamentous shoulder laxity on patients with near-track lesions.

CONCLUSION

This study demonstrated that capsuloligamentous shoulder laxity is a significant independent risk factor for failure after primary arthroscopic Bankart repair without remplissage and is much more predictive of failure in patients with near-track lesions. Our results suggest that hyperlaxity significantly increases the risk of failure in patients with a small DTD (near-track lesion) with or without bone loss. As our understanding of the GT continues to evolve, surgeons may need to consider the GT concept as a continuum with evolving surgical algorithms other than an arthroscopic Bankart alone, particularly in patients with near-track lesions and capsuloligamentous shoulder hyperlaxity.

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