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Clinical Outcome after Surgical Treatment of Recurrent Shoulder Dislocation with Small Bony Bankart

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Background: The consensus is that a bony Bankart lesion shorter than 25% of the length of glenoid does not affect the clinical result; hence, such lesions were often neglected. However, small bony Bankart lesions are associated with various types of capsulolabral lesions.
Methods: A total of 82 patients who had undergone arthroscopic capsulolabral lesion repair surgery for anterior shoulder dislocation were reviewed. The prevalence rates of early and late type of capsulolabral lesions were compared between a group of patients with and a group without small bony Bankart lesions. In addition, the types of accompanying capsulolabral lesion were analyzed according to the type of bony Bankart lesion. Finally, the clinical outcomes were evaluated (active range of motion, American Shoulder and Elbow Surgeons score and Rowe's score).
Results: Among the 13 patients who had small bony Bankart lesions, the prevalence rate of early and late type of capsulolabral lesions was 38.5% and 61.5%, respectively. Among the 69 patients without bony Bankart lesion, the prevalence rates of early and late type of capsulolabral lesions were 74% and 26%, respectively. Significantly worse clinical outcome was observed for the group of patients with both small bony Bankart lesions and late type of capsulolabral lesion.
Conclusions: More severe type of small bony Bankart lesion appears to be associated with late type of capsulolabral lesion. The significantly worse clinical outcome for patients with both small bony Bankart lesion and late type of capsulolabral lesion indicates that small bony Bankart lesions cannot always be neglected.
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Key Words: Recurrent shoulder instability; Small bony Bankart; Capsulolabral lesion

Introduction

Traumatic injury, the most common cause of recurrent shoulder dislocation,[1,2] can induce which can induce various types of capsulolabral detachment from the glenoid rim. Consequently, capsulolabral lesions are the most common pathological lesions in patients with recurrent shoulder subluxation and dislocations.[3-8] Bony glenoid defects also contribute to the instability of the shoulder. Such defects may occur when a traumatic force is applied to the glenoid fossa through the humeral head.[6,8] This compromises the static restraint of the shoulder and worsens its stability, thereby predisposing it to dislocation.[9-18] Significant work has been conducted on both capsulolabral lesions and bony Bankart lesions.[4-9] However, few studies have reported on the relationship between the two lesions, and smaller bony Bankart lesions have been largely neglected.[6,19] The general consensus is that a bony Bankart lesion shorter than 25% of the length of the glenoid does not require repair because it would not result in significant clinical improvements.[9,20]

However few studies on the relationship between the presence of small bony glenoid lesions shorter than 25% of the length of the glenoid and specific types of capsulolabral lesions have been reported. The hypotheses of our study were 1) the different types of capsulolabral lesions would be seen, depending on the presence or absence of a bony Bankart lesion less than 25% of the glenoid rim deficiency, 2) patients with such

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small bony lesions concomitant with a specific type of capsulolabral lesion would show inferior clinical outcomes after arthroscopic Bankart repair. The purpose of this study was to compare the frequencies of different capsulolabral lesions depending on the presence or absence of a bony Bankart lesion[21] and to compare the clinical outcomes among groups who were subdivided according to the presence or absence of a bony Bankart lesion and the type of accompanying capsulolabral lesion.[22]

Methods

After obtaining approval from the by Inje University Busan Paik Hospital Institutional Review Board (No. 11-069), we searched a senior author's database (Seung Suk Seo, M.D.) and found 140 patients who had undergone arthroscopic surgery for recurrent anterior shoulder dislocation between January 2003 and April 2009. Among the 140 patients, 82 patients who satisfied our inclusion criteria were selected. Included patients were those whose first shoulder dislocation was the result of a traumatic event and who had at least 3 recurrences of traumatic anterior shoulder dislocation. Those with multidirectional shoulder instability, spontaneous voluntary dislocation, a glenoid bony defect longer than 25% of the length of the glenoid, or a humeral-side bony defect that was large and engaged were excluded. Those who had undergone shoulder surgery previously, revision of a prior shoulder surgery, or re-dislocation after an operation were also excluded. Glenoid-side bony defects were evaluated by arthroscopy at the time of the operations. The capsulolabral lesions were evaluated by the arthroscopic findings recorded in the medical notes, X-ray (axillary view, west point view) and the results of preoperative magnetic resonance arthrography (MRA) or magnetic resonance imaging (MRI). The size of the Bankart lesion was estimated by X-ray (axillary view, west point view). The maximum width of the remnant glenoid was measured and expressed as a percentage of the width of intact glenoid. All patients had a minimum follow-up duration of 3 years. The patients were first classified according to the presence or absence of bony Bankart lesions. The group of patients with bony lesions was labeled as group A, and the group of patients without bony lesions was labeled as group B. The mean age of patients in group A (n=13) was 23.1 years (range 18 to 34 years), and that of patients in group B (n=69) was 23.15 years (range 17 to 35 years). The type of capsulolabral lesions in each patient in each group was evaluated using Habermeyer's classification system, which outlines the chronological evolution of capsulolabral lesions. Capsulolabral lesions were classified as two types:

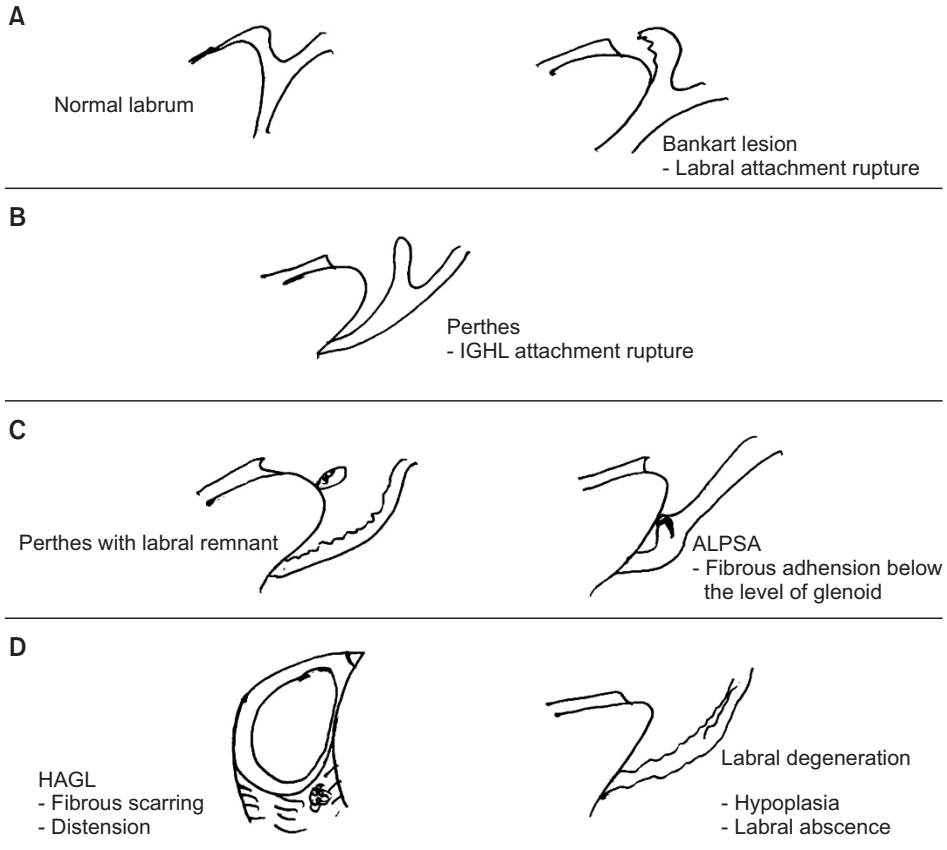

Fig. 1. Illustration of the capsulolabral lesions. (A) Isolated labral detachment (Bankart lesion). (B) Combined labral and inferior glenohumeral ligament (IGHL) attachment, continuous sublabral periosteal extension (Perthes). (C) Progressive degenerative changes of detached structures below the level of the glenoid, formation of fibrous scarring (anterior labroligamentous periosteal sleeve avulsion, ALPSA). (D) Degenerative process is extended above the level of glenoid, progressive disappearance of the labrum-ligament complex begins. HAGL: humeral avulsion of the glenohumeral ligament.

early and late (Fig. 1). The 'early type of capsulolabral lesions' included labral attachment ruptures, such as Bankart lesions, and inferior glenohumeral ligament attachment ruptures, such as Perthes lesions. The 'late type of capsulolabral lesions' included Perthes lesions with labral remnants, fibrous adhesion below the level of the glenoid, such as anterior labral periosteal sleeve avulsion lesions, subglenoid rupture with labral degeneration (labral hypoplasia, labral absence), and humeral avulsion of the glenohumeral ligament.[6] The prevalence rates of early and late types of capsulolabral lesions in each group were then compared (Fig. 2). In addition, in group A, the frequencies of the capsulolabral lesion types depending on Bigliani's bony Bankart types were compared. Bony lesions were classified according to Bigliani et al.[9] Type I lesions were defined as un-united fragments attached to a separated labrum; Type II lesions were defined as malunited glenoid fragments detached from the labrum; Type IIIa lesions were defined as anterior glenoid defects shorter than 25% of the total glenoid length; and, finally; Type IIIb was defined as defects longer than 25%.

Postoperative functional data were obtained on all patients during the follow-up evaluation, which was performed at least 36 months after the operation by an independent evaluator who did not participate in the original surgical procedures. At the follow-up, data on the patients' active forward flexion, and external rotation at 90 degrees abduction were obtained in addition to the assessment using American Shoulder and Elbow Surgeons (ASES) score and the modified Rowe's score. For comparison of the postoperative functional data, group A and group B were further subdivided into 4 groups according to the types of capsulolabral lesions. Patients in group A with the early-type capsulolabral lesions were assigned to a group labeled group A-I (n=5, mean age: 66.3 years, range 48 to 70 years), and those in group A with the late-type capsulolabral lesions were assigned to a group labeled group A-II (n=8, mean age: 64.8 years, range 50 to 73 years). Likewise, the group of patients in group B with the early-type capsulolabral lesions was named group B-I (n=51, mean age: 70.5 years, range 47 to 75 years), and the group of patients in group B with the late-type capsulolabral lesions was named group B-II (n=18, mean age: 67.2 years, range 49 to 78 years). Statistical tests were used for comparison of the long-term postoperative functional outcomes among the 4 groups.

Surgical Procedure

All operations were performed by the same surgeon using a standardized technique. After induction of general anesthesia, all patients were examined under anesthesia for assessment of translation of the shoulder. The patient was positioned in the lateral decubitus position, with the arm in a 3-direction traction device with 7 kg of traction (3-point shoulder distraction system; Arthrex, Naples, FL, USA).

Three arthroscopic portals were used. Arthroscopic evaluation of all associated intra-articular lesions was performed via a

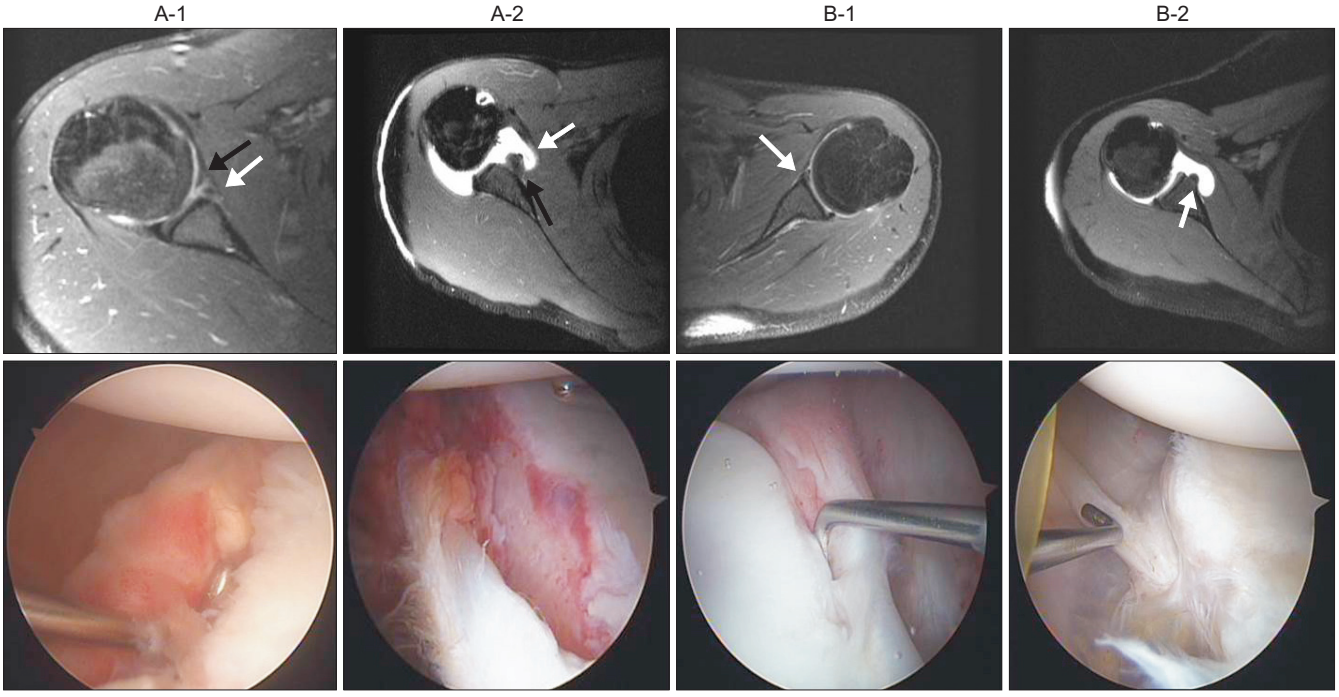

Fig. 2. The magnetic resonance imaging and magnetic resonance arthrography axial plane images and arthroscopic images of four groups are divided according to the presence of bony Bankart lesion and type of capsulolabral complex. A-1: early capsulolabral lesion with a bony Bankart lesion, A-2: late capsulolabral lesion with a bony Bankart lesion, B-1: early capsulolabral lesion without a bony Bankart lesion, B-2: late capsulolabral lesion without a bony Bankart lesion (white arrows: capsulolabral lesion, black arrows: bony Bankart lesion).

Table 1. Frequency of the Type of Capsulolabral Lesion according to the Presence or Absence of Small Bony Bankart Lesion

Group	Type		Total	*p*-value*
	Early (I)	Late (II)		
Group A	5	8	13	0.017
Group B	51	18	69	0.017

Values are presented as number of case.
Group A: with bony Bankart, Group B: without bony Bankart, I: with early capsulolabral lesion, II: with late capsulolabral lesion.
*Fisher test.

standard posterior portal. The anterior-superior portal was established using an inside-out technique, and the anterior-inferior portal was created just above the superior margin of the subscapularis tendon. An arthroscopic elevator was used to dissect medially approximately 1.5 to 2.0 cm along the anterior glenoid neck. A motorized shaver was used for removal of soft tissue from the glenoid rim and neck and to debride the neck down to bleeding bone.

The number of anchors used varied, depending on the severity of the lesions and the capsulolabral separation. Typically, 3 or 4 holes were drilled into the glenoid rim at the 2, 3, 4, and 5 o'clock position. After establishing the anchor, the surgeon retrieved one suture strand from the anchor through the anterior-superior portal. Next, a suture hook with a no. 1 nylon thread was used to penetrate the inferior glenohumeral ligament and labrum through the anterior-inferior portal. The pulling suture was retrieved through the anterior-superior portal and tied to the outer strand of a single anchor suture outside the anterior-superior portal. The pulling suture loaded with the outer strand of the anchor suture was then pulled back through the capsulolabral structures from the anterior-inferior portal to create a simple stitch. The capsulolabral complex was sutured to its anatomic position using a sliding knot. Additional anchors were placed in a similar manner. The surgeon performed only soft tissue Bankart repair. Bony Bankart lesions, regardless of amount or length, were left in situ without repair or excision.

In all cases, the patient's arm remained in a sling for 6 weeks, and they were allowed only a passive range of motion during this period. At 6 weeks, gradual full active motion was instituted, progressing to resistive strengthening, which was continued for a total of 3 to 4 months.

Statistical Evaluation

SAS (SAS Institute, Cary, NC, USA) was used for statistical evaluations, and all statistical analyses were performed by a specialized biostatistician. The level of significance was set at 0.05. Fisher's test was used to compare the prevalence rate of the early and late forms of capsulolabral lesions, depending on the presence or absence of small bony Bankart lesions. The Kruskal-Wallis test was used to compare the follow-up evaluation scores in the 4 different groups (group A-I, group A-II, group B-I. group B-II), and the Bonferroni test was used for post-hoc analysis.

Table 2. Frequency of the Type of Capsulolabral Lesion according to the Type of Bony Bankart Lesion

Type of bony Bankart	Early (I)	Late (II)
Biglinani type I	4	0
Biglinani type II	1	3
Biglinani type IIIa	0	5
Total	5	8

Values are presented as number of case.
I: with early capsulolabral lesion, II: with late capsulolabral lesion.

Results

Among the 82 patients who met our inclusion and exclusion criteria, 13 patients had bony Bankart lesions (group A), and 69 patients did not (group B). Among patients in group A, 5 patients (38.5%) had the early type of capsulolabral lesion, and 8 patients (61.5%) had the late type of capsulolabral lesion. In contrast, in group B, 18 patients (26%) had the late type of capsulolabral lesion, and 51 patients (74%) had the early type of capsulolabral lesion. The percentage of patients with the late type of capsulolabral lesion was higher for the bony Bankart group when compared to its counterpart and vice versa. These differences were statistically significant (Table 1). Among the 5 patients with the early type of capsulolabral lesion with bony Bankart lesion, 4 patients had a type I bony Bankart lesion, and one patient had a type II bony Bankart lesion. Among the 8 patients with the late type of capsulolabral lesion, 3 patients had type II bony Bankart lesions, and 5 patients had type IIIa bony Bankart lesions. Patients with type II and IIIa bony Bankart lesions had higher incidences of concomitant late-type capsulolabral lesions (Table 2).

The ASES score and the score on Rowe's scale for the patients in group A-I (bony Bankart, early type of capsulolabral lesion) was 96.00 and 95.00, respectively. The ASES score and the modified Rowe's score for those in group A-II (bony Bankart, late type of capsulolabral lesion) was 78.33 and 75.63, respectively. Patients in group B-I (non-bony Bankart, early type of capsulolabral lesion) had an ASES score of 95.66 and a modified Rowe's score of 95.80. Finally, those in group B-II (non-bony Bankart,

Table 3. Postoperative Clinical Outcomes of the Patients

Clinical outcome	Group A		Group B		*p*-value*
	Early (I)	Late (II)	Early (I)	Late (II)	
ASES	96.00 ± 3.46	78.33 ± 13.63	95.66 ± 3.12	91.85 ± 5.56	0.002
Rowe's score	95.00 ± 5.12	75.63 ± 12.37	95.80 ± 5.14	93.33 ± 8.29	0.003
Stability	50.00 ± 0.00	30.00 ± 10.69	49.20 ± 4.11	45.56 ± 8.82	<0.001

Values are presented as mean ± standard deviation.
Group A: with bony Bankart, Group B: without bony Bankart, I: with early capsulolabral lesion, II: with late capsulolabral lesion, ASES: American Shoulder and Elbow Surgeons score.
*Krusakal-Wallis test.

Table 5. Postoperative Range of Motion for the Patients

Outcome	Group A		Group B		*p*-value*
	Early (I)	Late (II)	Early (I)	Late (II)	
FF ROM (°)	174.8 ± 8.1	175.4 ± 11.1	173.4 ± 12.1	169.1 ± 8.9	0.31
Abd ROM (°)	175.3 ± 11.5	173.1 ± 12.8	172.8 ± 15.4	168.0 ± 18.1	0.341
ER ROM (°)	73.0 ± 12.5	74.1 ± 19.8	74.5 ± 18.4	68.5 ± 9.5	0.135

Values are presented as mean ± standard deviation.
Group A: with bony Bankart, Group B: without bony Bankart, I: with early capsulolabral lesion, II: with late capsulolabral lesion, FF: forward flexion, ROM: range of motion, Abd: abduction, ER: external rotation.
*Wilcoxon signed rank test.

late type of capsulolabral lesion) had an ASES score of 91.85 and a modified Rowe's score of 93.33. The stability component of the modified Rowe's score for group A-I, group A-II, group B-I, and group B-II was 50.00 ± 0.00, 30.00 ± 10.69, 49.20 ± 4.11, and 45.56 ± 8.82, respectively. The ASES score, the modified Rowe's score, and the stability component of the modified Rowe's score were significantly lower in group A-II than in the other groups (Table 3, 4). Except for group A-II, results of the between-group comparison showed no significant differences in the scores. There were also no statistically significant differences in the range of motion among the 4 groups (Table 5).

Discussion

In the current literature, comparison of the types of capsulolabral lesions according to the presence or absence of small bony glenoid lesions revealed a definite pattern, with the frequency of certain types of capsulolabral lesions associated with a specific type of small bony Bankart lesion. Habermeyer et al.[6] evaluated numerous capsulolabral lesions in 91 post-traumatic, unidirectional, antero-inferior shoulder instability patients. Based on the number of shoulder dislocations, they suggested a chronological evolution and spectrum of capsulolabral lesions. However, they did not evaluate the capsulolabral lesions with regard to bony Bankart lesions. Bigliani et al.,[9] who studied anterior glenoid rim lesions in detail in their study of 25 shoulders with recurrent shoulder dislocation and instability, classified the bony lesions. Type I lesions were defined as un-united fragments attached to

Table 4. Two-way Bonferoni Test Results for Clinical Outcomes of the Patients

Clinical outcome	Group (mean ± standard deviation)		*p*-value*
ASES	A-I (96.00 ± 3.46)	B-I (95.66 ± 3.12)	1
	A-I (96.00 ± 3.46)	A-II (78.33 ± 13.63)	0.11
	A-I (96.00 ± 3.46)	B-II (91.85 ± 5.56)	0.801
	B-I (95.66 ± 3.12)	A-II (78.33 ± 13.63)	<0.01
	B-I (95.66 ± 3.12)	B-II (91.85 ± 5.56)	0.191
	A-II (78.33 ± 13.63)	B-II (91.85 ± 5.56)	0.117
Rowe's score	A-I (95.00 ± 5.00)	B-I (95.80 ± 5.14)	1
	A-I (95.00 ± 5.00)	A-II (75.63 ± 12.37)	0.022
	A-I (95.00 ± 5.00)	B-II (93.33 ± 8.29)	1
	B-I (95.80 ± 5.14)	A-II (75.63 ± 12.37)	<0.01
	B-I (95.80 ± 5.14)	B-II (93.33 ± 8.29)	1
	A-II (75.63 ± 12.37)	B-II (93.33 ± 8.29)	0.027
Stability	A-I (50.00 ± 0.00)	B-I (49.20 ± 4.00)	1
	A-I (50.00 ± 0.00)	A-II (30.00 ± 10.69)	0.022
	A-I (50.00 ± 0.00)	B-II (45.56 ± 8.82)	1
	B-I (49.20 ± 4.00)	A-II (30.00 ± 10.69)	<0.001
	B-I (49.20 ± 4.00)	B-II (45.56 ± 8.82)	0.449
	A-II (30.00 ± 10.69)	B-II (45.56 ± 8.82)	0.038

ASES: American Shoulder and Elbow Surgeons score, A-I: with Bony Bankart and early type of capsulolabral lesions, A-II: with Bony Bankart and late type of capsulolabral lesions, B-I: without Bony Bankart and early type of capsulolabral lesions, B-II: without Bony Bankart and late type of capsulolabral lesions.
*Bonfenoni test.

a separated labrum; Type II lesions were defined as malunited glenoid fragments detached from the labrum; Type IIIa lesions were defined as anterior glenoid defects shorter than 25% of the total glenoid length; and, finally; Type IIIb was defined as defects longer than 25%. Bigliani et al.[9] studied only bony Bankart lesions, not capsulolabral lesions. Neither Habermeyer et al.[6] nor Bigliani et al.[9] examined the relationship between bony Bankart lesions and capsulolabral lesions. In our study, more severe type of small bony Bankart lesions appear to be associated with late type of capsulolabral lesion and patients with both small bony Bankart lesion and late type of capsulolabral lesion showed significantly worse clinical outcome.

We found a relationship between bony Bankart lesions and the type of capsulolabral lesion. A high prevalence of early-type capsulolabral lesions (74%) was observed in the group without bony Bankart lesions, whereas the group with these lesions had a high frequency of late-type capsulolabral lesions (61.5%). Based on the results, there is a high probability that bony Bankart lesions will be accompanied by the late type of capsulolabral lesions. In addition, all patients with type I bony Bankart lesions and one patient with a type II bony Bankart lesion had early capsulolabral lesions, such as Perthes and Bankart. On the other hand, all patients with type II bony Bankart lesions, except for one, and all patients with type III bony Bankart lesions had concomitant late-type capsulolabral lesions.

Elucidating the specific pathomechanism responsible for this phenomenon is beyond the scope of this study. This study also does not explain which lesion–bony Bankart or capsulolabral–occurs first in the patients. The chronological evolution and the interplay of the two lesions are difficult to assess due to the limitations in patient history taking. However, in our opinion, after a patient first suffers a small bony Bankart lesion resulting from blunt traumatic injury, repeated dislocation and subluxation in the presence of a bony Bankart lesion (eroded or not) imparts unusual and continuous tension and pressure on the capsulolabral ligament complex above, below, or even at the bony Bankart area where the capsulolabral ligament is inserted. On the other hand, we suspect that after a patient first suffers a capsulolabral lesion resulting from a traumatic injury, repeated dislocation and subluxation of the shoulder in the presence of the capsulolabral lesion could lead to erosion, or even fracture, of the glenoid rim. The compromise in the static restraint of the shoulder could, in turn, burden the capsulolabral ligament complex, worsening the existing capsulolabral lesion and leading to the late type of capsulolabral lesion. Again, although knowledge regarding the evolution and the interplay of the two lesions is limited, it is clear that small bony Bankart lesions are frequently accompanied by the late type of capsulolabral lesion. In addition, the fact that the frequencies of the types of capsulolabral lesions differ with respect to the different bony Bankart lesions is clinically relevant. Bony Bankart type II, which are retracted malunited lesions, and type IIIa, which are erosive lesions, were more frequently accompanied by the late type of capsulolabral lesions. Another notable point is that all patients with bony Bankart lesions also had capsulolabral lesions. This finding is different from the results of Bigliani et al.[9] In their study,[9] the Bankart type I lesions had normal labrum attached to the bony lesion. They did not describe a concomitant capsulolabral lesion with the type III Bankart lesions. Only the type II Bankart lesion was accompanied by detachment of the labrum from the malunited bony fragment. The different findings between our study and those of Bigliani et al.[9] could be due to the different diagnostic methods. Bigliani et al.[9] used computed tomography (CT), X-ray, and gross findings during the open Bankart repair operation to define bony defects and labral lesions. These methods might be best for evaluation of bony parts. However, they are not optimal for evaluation of capsulolabral lesions. Thus, soft tissue lesions might not have been properly evaluated. In contrast, we used MRI, MRA, and arthroscopic findings for diagnosis of bony and soft tissue lesions. The different evaluation methods might explain the different prevalence of concomitant capsulolabral lesions with bony Bankart lesions.

In addition to elucidating the relationship between bony Bankart and capsulolabral lesions, we examined the clinical outcomes among the 4 groups classified according to the presence or absence of bony Bankart lesions and the type of capsulolabral lesions. Numerous biomechanical studies[21,23-26] have elucidated the inverse relationship between the stability of the shoulder and the size of glenoid bony defects. In a biomechanical study using cadavers, Itoi et al.[14] found that glenoid bony defects longer than 21% of the total glenoid length cause shoulder instability. Other clinical studies[8,9,20] have demonstrated that bony defects longer than 20% to 25% of the glenoid length are an important cause of re-dislocation after arthroscopic Bankart repair. Burkhart and De Beer[11] who also suggested that glenoid defects longer than 25% of the glenoid length are a key factor in recurrence after surgery, argued that more rigorous examination and treatment are required for such large bony Bankarts. Although the clinical significance of bony Bankarts longer than 25% of the glenoid length has been widely recognized, smaller bony Bankarts have not received sufficient attention. Some studies[9,11,20,22,27] reported that repair of smaller bony lesions did not provide significant improvement in the stability of the shoulder.

However, in our study, when compared to the other patient groups, the group of patients with small bony Bankart lesions accompanied by the late type of capsulolabral lesions had significantly lower ASES scores and lower modified Rowe scores, in addition to lower scores for the stability component of the modified Rowe score. This finding deserves attention. None of the subjects in the study had recurrent dislocations after surgery (i.e., the patients had no postoperative complications or dislocations). Based on our results, even after successful surgery, they

could continue to experience shoulder discomfort. Specifically, the stability component of Rowe's score indicated the presence of subluxation and a feeling of discomfort in some positions in the absence of definite dislocations.

This retrospective study cannot shed light on the reason for the inferior clinical outcomes of the patients with bony Bankart and late-type capsulolabral lesions. Our speculation is that the loss of bony buttress, when accompanied by the late type of capsulolabral lesions, compromises the stability of the shoulder to a noticeable degree while not resulting in re-dislocation. The results are clinically relevant because they suggest that the current clinical recommendation of neglecting small bony Bankart lesions might not be the best course for some patients. If such lesions are accompanied by the late type of capsulolabral lesions, the reduction in the arch of glenoid, even if the reduction is small, affects the stability of the shoulder. In such cases, performing both soft tissue Bankart repair and bony Bankart repair may improve postoperative shoulder stability.

There are a number of limitations of this study in addition to its retrospective design. For evaluation of correlation between bony Bankart and capsulolabral lesions, we used X-ray (axillary view, west point view), MRI, MRA, and intraoperative arthroscopic findings; however CT was not used, even though it has benefit for detection of bony Bankart lesions. Not all patients underwent CT and detection of capsulolabral lesions is difficult. Therefore, some osseous lesions might not have been detected. The study included only patients whose operation was successful and who experienced re-dislocation after surgery. In addition, to eliminate potential bias and to focus on the correlation between small bony Bankart lesions and capsulolabral lesions, patients with larger bony Bankart lesions and other bony lesions, such as large and engaging Hill-Sachs lesions, were excluded. Hence, our study population is not representative of all patients with recurrent shoulder dislocation. The study also did not compare clinical outcomes between a patient group who underwent bony Bankart surgery and a group who underwent soft tissue surgery. Thus, we can only conclude that the patients with small bony Bankart and the late type of capsulolabral lesions showed the worst clinical outcomes. The case for the role of small bony Bankart lesions in the stability of the shoulder would have been stronger if we had been able to perform both bony Bankart repair and soft tissue repair surgeries in patients with both lesions and compare the outcomes with those of a similar group of patients who underwent only soft tissue repair. However, the senior author performed soft tissue repair only for cases with concomitant small bony Bankarts because this course of action was clinically recommended at the time. The results of this study suggest otherwise, and further study on small bony Bankarts will refine clinical recommendations. This study also did not assess the amount of glenoid defect, although we suggest that loss of bony buttress compromises the stability of the shoulder to a noticeable degree. Finally, because the number of patients with small bony Bankart lesions was too small, we could not perform statistical analyses of the different frequencies of capsulolabral lesion types according to the bony Bankart type. However, comparison of the frequencies in this study suggests that the late type of capsulolabral lesions are more frequently associated with Type II and IIIa small bony Bankart lesions than with the other types of small bony Bankart lesions. The relationship between the type of bony Bankart and concomitant capsulolabral lesion will be elucidated once a larger sample is obtained.

Conclusion

More severe type of small bony Bankart lesion appears to be associated with late type of capsulolabral lesion. The significantly worse clinical outcomes for patients with both small bony Bankart lesion and late type of capsulolabral lesion indicate that small bony Bankart lesions cannot always be neglected.

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