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SCIENTIFIC ARTICLE

Bennett lesions in overhead athletes and associated shoulder abnormalities on MRI

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Abstract

Objective To determine if a Bennett lesion and its size are associated with additional MRI shoulder abnormalities in an overhead athlete.

Materials and methods An IRB-approved retrospective review of our database from 1 January 2012 to 1 April 2018 identified 35 overhead athletes with a Bennett lesion on MRI. A control group consisting of 35 overhead athletes without a Bennett lesion were matched for age, level of play (professional vs non-professional), and type of study (arthrogram vs non-arthrogram). Each study was assessed independently by two MSK fellowship trained radiologists. The sizes of the Bennett lesions were measured. Each MRI was assessed for the presence of a labral tear, posterior glenoid cartilage abnormality, humeral head notching or cysts, and fraying or tear of the supraspinatus or infraspinatus tendons. Statistical analyses were performed using Student's *t* test, Fisher's exact test, and Chi-squared test.

Results There was an increased incidence of posterior glenoid cartilage abnormalities in athletes with Bennett lesions vs those without (23% vs 3%, *p* value = 0.01). There was no difference in any other MRI abnormalities, including labral tears and findings of internal impingement between these two groups (*p* value range = 0.09–0.46). There was no association between the size of a Bennett lesion and the presence of glenoid cartilage lesions, labral tears, internal impingement, age, professional status, or need for surgery (*p* value range = 0.08–0.96).

Conclusion Symptomatic overhead athletes with Bennett lesions have an increased frequency of posterior glenoid cartilage abnormalities, but not labral tears or findings of internal impingement compared to those without Bennett lesions.

Keywords Bennett lesion · Overhead athlete · MRI

Introduction

The Bennett lesion has been identified as a common finding in baseball players, with an incidence ranging between 22% and 33% [1, 2]. George Bennett first described it in 1941 as an exostosis or deposit of bone at the posteroinferior border of the glenoid fossa found in professional pitchers [3]. He originally hypothesized the cause to be traction of the long head of the triceps tendon from repetitive overhead throwing [3, 4]. However, in 1977, Lombardo et al. localized the lesion to within the posteroinferior joint capsule and found it to be characterized by chronic inflammation and new bone formation [5]. The authors suggested traction of the posterior band of the inferior glenohumeral ligament during the deceleration phase of pitching or humeral head impingement of the posterior capsule and glenoid rim during the cocking phase of pitching as alternative mechanisms [5]. Debate as to the exact mechanistic cause of the lesion persists today, although its extra-articular location has since been confirmed at both arthroscopy and with arthrographic studies [6].

Both operative and non-operative management of a symptomatic Bennett lesion have been shown to be successful [2, 6, 7]. Treatment decisions, however, are more complicated if there are associated shoulder injuries, as these may need to be addressed either separately or concurrently. Previous studies, including a recent retrospective study by Park et al. have had conflicting results regarding the incidence of labral tears and internal impingement associated with a Bennett lesion [2,

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5, 8]. To the best of our knowledge, however, no prior study has assessed the incidence of glenoid cartilage abnormalities or correlated Bennett lesion volume with associated shoulder injuries.

A Bennett lesion diagnosis can be made radiographically on axillary, Bennett, or Stryker notch views [3]. Computed tomography (CT) is more sensitive for the detection of the capsular mineralization. However, magnetic resonance imaging (MRI) can detect an early Bennett lesion when there is non-mineralized posterior glenoid or periosteal enthesopathic change and is the gold standard for evaluating associated soft-tissue abnormalities in an overhead athlete [9].

The purpose of this study is to determine if a Bennett lesion and its size are associated with additional MRI shoulder abnormalities in an overhead athlete.

Materials and methods

This is a retrospective case–control study approved by the institutional review board, who waived the requirement for informed consent, and was compliant with the Health Insurance Portability and Accountability Act.

A consecutive case group of 35 overhead athletes with a Bennett lesion on shoulder MRI were identified from a review of MRI reports issued from 1 January 2014 to 1 April 2018 in our picture archiving and communication system (PACS). A consecutive control group consisting of 35 overhead athletes without a Bennett lesion on shoulder MRI were matched for age, level of play (professional vs non-professional athlete), and type of study (MR arthrogram vs non-arthrogram) during the same time period (Table 1). All patients from both groups were symptomatic and experienced pain with throwing.

Magnetic resonance imaging was acquired using a 1.5-T or 3-T MR scanner (GE Healthcare, Waukesha, WI, USA; Siemens Healthcare, Erlangen, Germany) with sequences that included (slice thickness in millimeters, repetition time/echo time in milliseconds): axial proton density-weighted, fat-saturated (3.5 mm, 2,000–3,500/30), coronal proton density-weighted, fat-saturated (3.0 mm, 2,000–3,500/30), coronal T2-weighted fat-saturated (3.0 mm, 3,800–5,000/68), sagittal proton density (3.0 mm, 2,000–3,500/30), and sagittal proton density-weighted fat-saturated (3.0 mm, 2,000–3,500/30). An interslice gap of 0.3 mm, matrix of 320 × 224, and field of view of 14 cm were used.

Magnetic resonance arthrograms were acquired using a 1.5-T or 3-T MR scanner (GE Healthcare; Siemens Healthcare) with sequences that included (slice thickness in millimeters, repetition time/echo time in milliseconds): axial T1-weighted fat-saturated (3.0 mm, 600–900/4–15), coronal T1-weighted fat-saturated (3.0 mm, 600–900/4–15), sagittal T1-weighted fat-saturated (3.0 mm, 600–900/4–15), coronal T2-weighted fat-saturated (3.0 mm, 3,800–5,000/68), and sagittal T1-weighted (3.0 mm, 600–900/4–15). An interslice gap of 0 or 0.5 mm, matrix of 320 × 224, and field of view of 14 cm were used.

Clinical data were obtained from the electronic health record. In the Bennett group, 34 of the 35 patients identified baseball as their primary sport (pitcher, $n = 29$; fielder, $n = 4$; and recreational/no designated position, $n = 1$). One patient in the Bennett group (1 out of 35) identified football (wide receiver) as his primary sport, although he had previously played baseball extensively as both a pitcher and a fielder. In the non-Bennett group, all 35 patients identified baseball as their primary sport (pitcher, $n = 33$; fielder, $n = 2$).

All studies were assessed on a PACS workstation independently by two musculoskeletal fellowship-trained radiologists (JKK and MJR with 12 and 14 years of radiology experience respectively) who were blinded to the clinical history. Each MRI was assessed for labral tear, posterior glenoid cartilage injury, humeral head notching or cysts, and posterior supraspinatus or infraspinatus tendon fraying or tear. Of these, superior labrum anterior to posterior (SLAP) tears, humeral head notching or cysts, and posterior supraspinatus or infraspinatus fraying or tear were considered findings of internal impingement. Bennett lesions were measured in three dimensions with the final volume for each lesion, (length × width × height) / 2, assuming an ellipsoid shape, determined by the average between the two readers. Labral tears were localized using a clock-face descriptor. Cartilage injuries included

Table 1 Patient demographics

	Bennett lesions ($n = 35$)	Non-Bennett lesions ($n = 35$)	p value
Number of professional players	17	17	1
Mean age (years)[a]	26.7 (± 3.5)	25.3 (± 3.8)	0.25
Number of MR arthrograms	2	0	0.48
Number of non-professional players	18	18	1
Mean age (years)[a]	20.3 (± 3.9)	19.4 (± 2.5)	0.42
Number of MR arthrograms	8	8	1

[a] Mean age reported in years ± standard deviation

Table 2 Associated MRI abnormalities in Bennett versus non-Bennett athletes

	Bennett (%)	Non-Bennett (%)	*p* value
Superior labral tear	18/35 (51)	11/35 (31)	0.09
Posterior labral tear	18/35 (51)	11/35 (31)	0.09
Posterior glenoid cartilage abnormality	8/35 (23)	1/35 (3)	0.01*
Humeral head notching or cysts	27/35 (77)	22/35 (63)	0.19
Supraspinatus or infraspinatus tendon articular surface abnormality	15/35 (43)	12/35 (34)	0.46
Antero-inferior labral tear	9/35 (26)	6/35 (17)	0.38

*Denotes statistical significance with $p \leq 0.05$

fissuring, delamination, and defects. All discrepancies were adjudicated by a third musculoskeletal fellowship-trained radiologist (TTW with 11 years of radiology experience). The discrepant findings requiring adjudication were as follows: SLAP tear ($n = 7$), posterior labral tear ($n = 15$), antero-inferior labral tear ($n = 16$), posterior glenoid cartilage abnormality ($n = 5$), humeral head notching/cysts ($n = 5$), and articular surface rotator cuff fraying/tear ($n = 20$).

Statistical analyses were performed using Student's *t* test, Fisher's exact test, and Chi-squared test for continuous and categorical variables. Cohen's kappa was used for inter-observer variability. All statistics were performed using Microsoft Excel version 1710 and PSPP version 0.10.4.

Results

Associated MRI abnormalities in the Bennett compared with the non-Bennett overhead throwing athlete groups are reported in Table 2. Posterior glenoid cartilage injuries were identified more frequently in those with Bennett lesions ($p = 0.01$; Figs. 1, 2). There was no statistical difference in the incidence of labral tears or findings of internal impingement (Figs. 3, 4). Cohen's kappa (k) values for inter-observer agreement between the two primary readers are as follows: superior labral tear (0.80), posterior labral tear (0.54), posterior glenoid cartilage abnormality (0.70), humeral head notching or cysts (0.82), supraspinatus/infraspinatus tendon articular surface abnormality (0.42), and antero-inferior labral tear (0.40). The reference ranges for kappa interpretation are: < 0: none, 0–0.20: slight, 0.21–0.40: fair, 0.41–0.60: moderate, 0.61–0.80: substantial, and 0.81–0.99: almost perfect.

Among the 35 Bennett lesions, the average Bennett lesion volume was 624 $mm^3 \pm 598$. There was no statistically significant association between Bennett lesion volume and the MRI abnormalities assessed (Tables 3, 4).

A total of 27 out of 35 Bennett patients (77%) and 17 out of 35 non-Bennett patients (49%) had a clinical follow-up at our institution after their initial MRI, with a mean follow-up time of 18.6 months ±24.0 (range 1–111 months). Excluding the patients who were lost to follow-up, a total of 10 out of 27 Bennett patients (37%) vs 6 out of 17 non-Bennett patients (35%) underwent arthroscopic shoulder surgery ($p = 0.91$). Of the Bennett

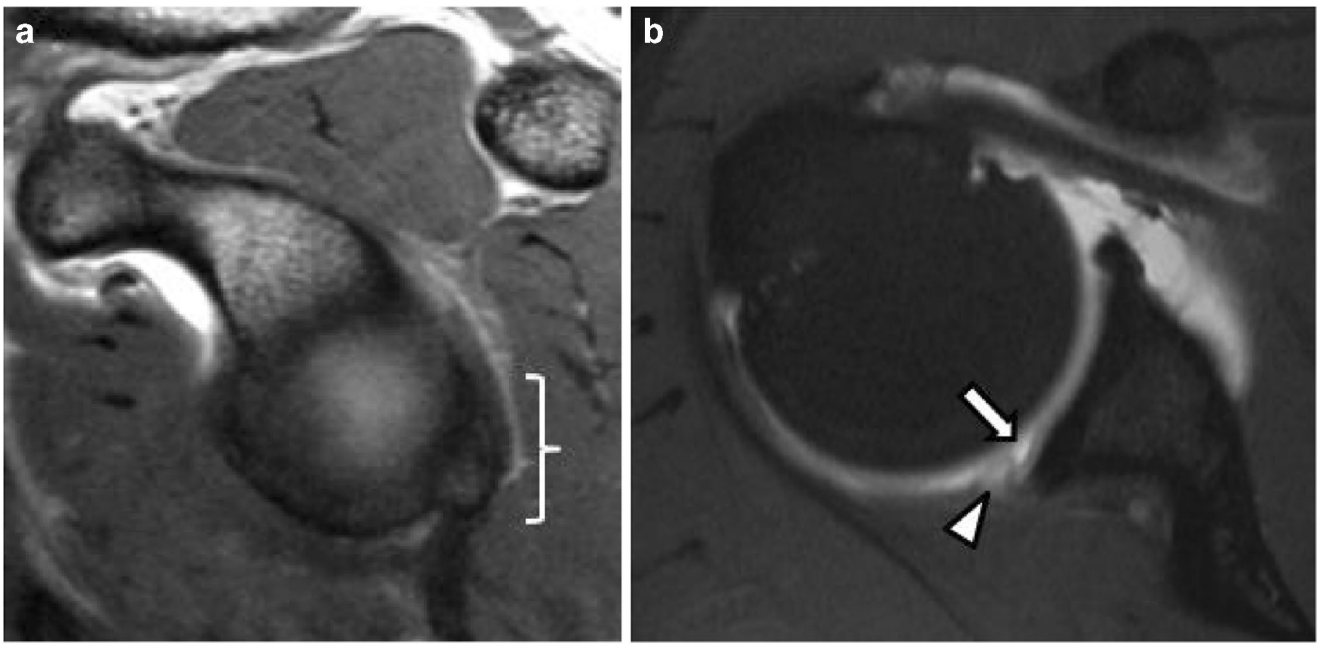

Fig. 1 A 20-year-old male non-professional athlete with a Bennett lesion. **a** Magnetic resonance arthrogram with sagittal T1-weighted and **b** axial T1-weighted fat-saturated. Bennett lesion (*bracket* in **a**), posterior labral tear (*arrowhead* in **b**), and partial thickness posterior glenoid cartilage defect (*arrow* in **b**)

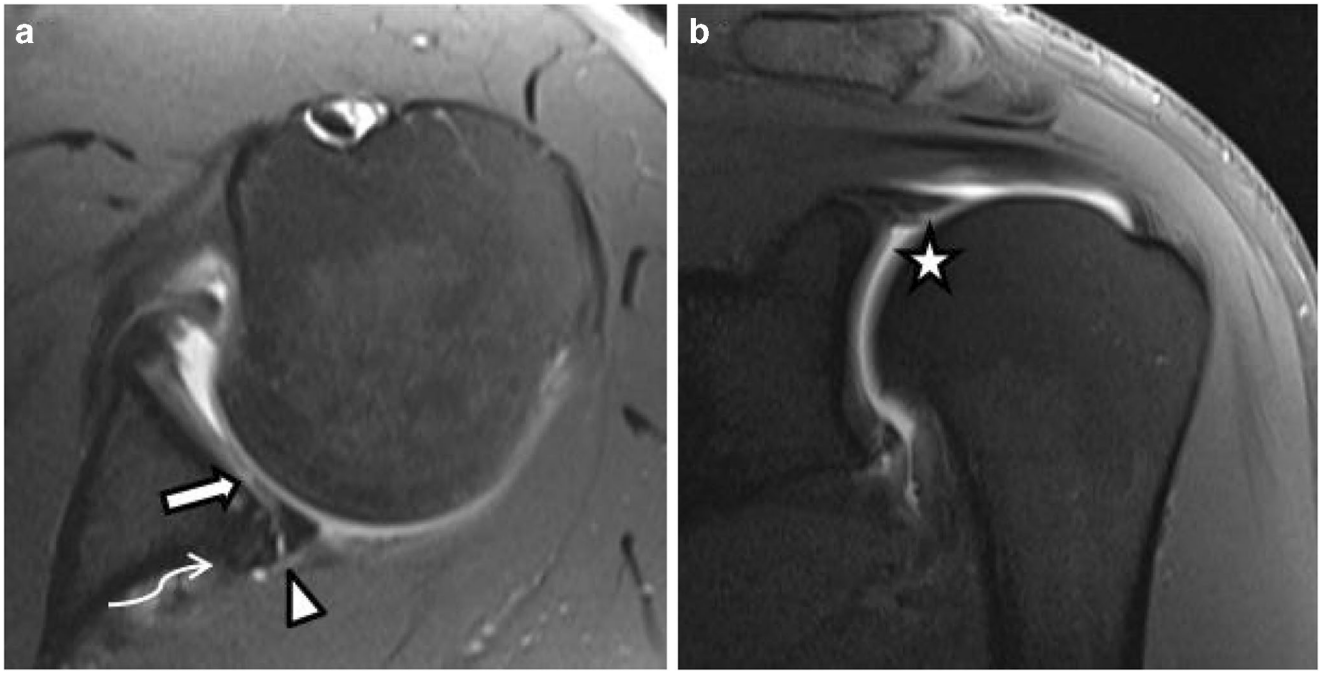

Fig. 2 A 29-year-old male non-professional athlete with a Bennett lesion. **a** Magnetic resonance arthrogram with axial T1-weighted fat saturation and **b** coronal T1-weighted fat saturation. Bennett lesion (*curved arrow* in **a**), posterior labral tear with paralabral cyst (*arrowhead* in **a**), fissure in posterior glenoid cartilage (*arrow* in **a**), and superior labral tear (*above the star* in **b**)

patients who had surgery, 2 out of 27 (7.4%) had Bennett lesion resections, with MRI lesion volumes of 144 mm^3 and 423 mm^3 respectively.

Of the total number of patients who had surgery, 13 out of 16 (81%) had more than one type of procedure performed. For the Bennett patients, the various procedures included labral repair ($n = 5$), labral debridement ($n = 5$), coracoclavicular ligament reconstruction ($n = 1$), semitendinosus allograft ($n = 1$), posterior capsular release or debridement ($n = 4$), Bennett lesion resection ($n = 2$), synovitis removal ($n = 1$), rotator cuff debridement ($n = 4$), and bursectomy with subacromial decompression ($n = 1$). The locations of the five labral repairs were superior and posterior ($n = 3$), superior only ($n = 1$), and antero-inferior only ($n = 1$). The locations of the five labral debridements were superior and posterior ($n = 3$), superior only ($n = 1$), and posterior only ($n = 1$).

For the non-Bennett patients, the various procedures included labral repair ($n = 1$), labral debridement ($n = 5$), posterior capsular release or debridement ($n = 1$), rotator cuff debridement ($n = 3$), bursectomy with or without subacromial decompression ($n = 2$), subscapularis repair ($n = 1$), spinoglenoid cyst decompression ($n = 1$), and glenoid chondroplasty ($n = 1$). The locations of the one labral repair were superior and posterior. The locations of the five labral debridements were superior and posterior ($n = 1$), superior only ($n = 2$), posterior only ($n = 1$), and antero-inferior only ($n = 1$).

Discussion

Since the Bennett lesion was first described in 1941, controversy has persisted regarding its etiology, associated injuries, and treatment. Our results suggest that symptomatic overhead athletes with Bennett lesions have an increased frequency of posterior glenoid cartilage abnormalities compared with those without a Bennett lesion, but not labral tears or findings of internal impingement. We found that the size of a Bennett lesion is not associated with level of play, age, other typical overhead throwing injuries in the shoulder, or need for surgery.

Internal impingement is seen in overhead athletes as the arm is repetitively put in extreme abduction and external rotation, causing impingement of the supraspinatus and infraspinatus tendons between the humeral head and posterior glenoid. MRI findings of internal impingement include undersurface fraying or tear of the posterior supraspinatus and anterior infraspinatus tendons, notching and cystic change of the humeral head, and posterosuperior labral injury [10]. An association between internal impingement and a Bennett lesion has been described in the past [5, 11]. However, these prior studies had smaller sample sizes than our own and lacked control groups. We found no increase in the findings of internal impingement in patients with Bennett lesions. This result is supported by a recent larger retrospective study by Park et al. and may provide some insight into the mechanism of Bennett lesion development [8]. Considering that patients

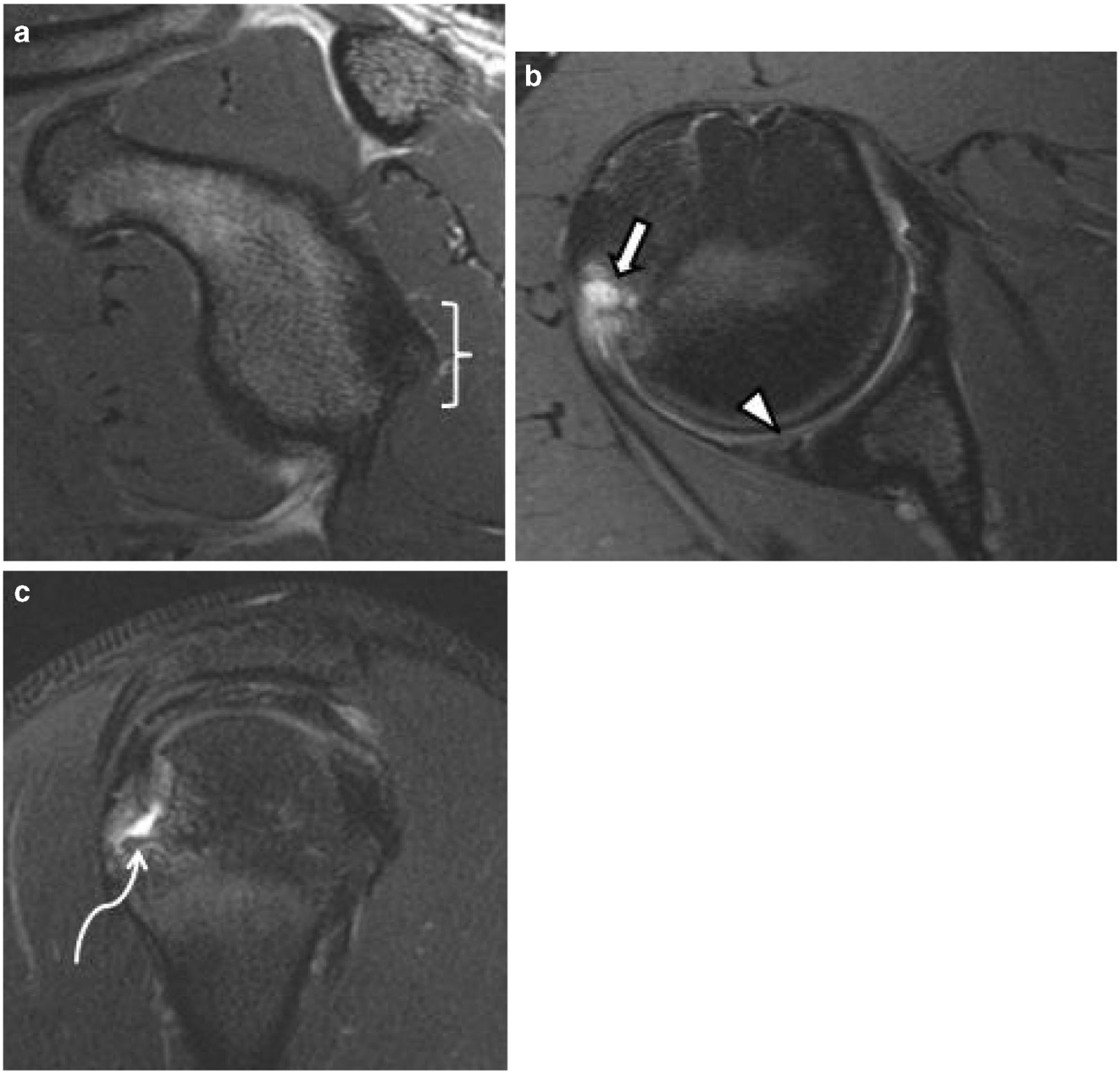

Fig. 3 A 20-year-old male professional athlete with a Bennett lesion. **a** Magnetic resonance imaging with sagittal proton density weighting, **b** axial proton density-weighted fat saturation, and **c** sagittal proton density-weighted fat saturation. Bennett lesion (*bracket* in **a**), posterior labral tear (*arrowhead* in **b**), humeral head cysts (*arrow* in **b**), and partial thickness infraspinatus tendon tear (*curved arrow* in **c**)

with Bennett lesions experience shoulder pain during the release to the follow-through phases of pitching, our study and prior work favor a Bennett lesion developing during the deceleration phase rather than during the cocking phase [7, 8].

We found that there was no increase in the incidence of labral tears in patients with Bennett lesions. This is similar to outcomes of some previous studies, although other literature has suggested contradicting results [6–9]. Ferrari et al. evaluated 7 baseball players with symptomatic Bennett lesions treated arthroscopically, 6 of whom had posterior labral tears [6]. A study by Miniaci et al. evaluated asymptomatic professional baseball pitchers and found that 29% had Bennett lesions and 79% had labral abnormalities [9]. A potential reason for discrepant results between these studies and ours may be the way in which a labral tear is determined. One study used arthroscopy as the gold standard, whereas others, including our own, used imaging with a mixture of arthrograms and non-arthrographic studies. This examination heterogeneity produces different accuracies and biases in determining a labral tear.

We found that patients with Bennett lesions have an increased incidence of posterior glenoid cartilage abnormalities,

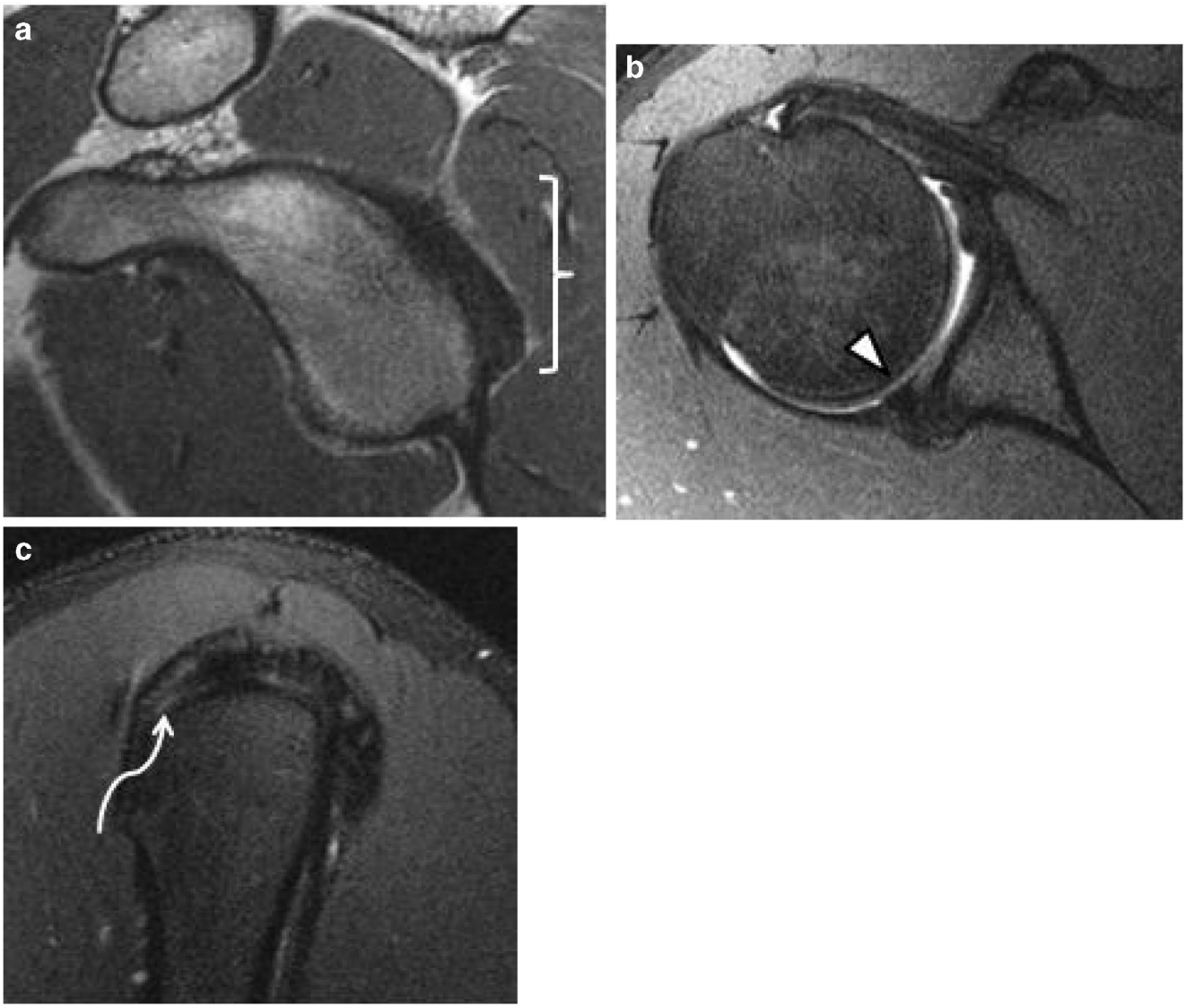

Fig. 4 A 20-year-old male non-professional athlete with a Bennett lesion. **a** Magnetic resonance imaging with sagittal proton density-weighting, **b** axial proton density-weighted fat saturation, **c** and sagittal proton density-weighted fat saturation. Bennett lesion (*bracket* in **a**), normal posterior labrum (*arrowhead* in **b**), and infraspinatus tendinosis without a tear (*curved arrow* in **c**)

including fissuring, delamination, and partial- or full-thickness defects ($p = 0.01$). One possible mechanistic explanation is that posterior glenoid cartilage abnormalities are caused by a tight posterior capsule, which causes posterior translation of the humeral head during the late cocking phase of pitching, resulting in compression and shear forces being applied to the posterior glenoid fossa [12, 13]. None of the athletes with Bennett and glenoid cartilage lesions on MRI underwent surgical management of their cartilage injury, although this does not mean that their cartilage lesion was asymptomatic. The one case of glenoid chondroplasty performed in the non-Bennett group was evaluated as having normal cartilage on MRI by both readers. Therefore, the clinical significance of a cartilage lesion in the setting of a Bennett lesion is unclear.

Table 3 Mean Bennett lesion volume and demographic associations

	Mean Bennett volume (mm^3)	*p* value
Professional athlete	708 ± 721	0.43
Non-professional athlete	545 ± 461	
<21 years old	619 ± 592	0.96
≥21 years old	628 ± 618	
Surgical patient	749 ± 731	0.48
Non-surgical patient	581 ± 555	

Although Bennett lesions were originally thought to require surgical excision, recommendations were later

Table 4 Mean Bennett lesion volume and MRI injury associations

	Mean Bennett volume (mm^3)	*p* value
Superior labral tear	710 ± 742	0.43
Normal superior labrum	533 ± 398	
Antero-inferior labral tear	386 ± 168	0.17
Normal antero-inferior labrum	707 ± 671	
Posterior labral tear	614 ± 604	0.85
Normal posterior labrum	660 ± 622	
Posterior glenoid cartilage abnormality	807 ± 460	0.33
Normal glenoid cartilage	570 ± 950	
Humeral head cysts or notching	677 ± 668	0.35
Normal humeral head	448 ± 193	
Supraspinatus or infraspinatus tendon articular surface abnormality	826 ± 740	0.08
Normal supraspinatus and infraspinatus	473 ± 424	

revised because of poor surgical outcomes [3, 4]. Limited subsequent studies reached contradicting conclusions [6, 11, 14]. One study evaluating Bennett lesion size concluded that lesions greater than 100 mm^2 have poorer outcomes and should therefore be debrided [11]. Our average Bennett lesion volume was greater in professional compared with non-professional athletes, although this was not statistically significant. Most of our Bennett patients, regardless of lesion size, did not require excision. We found no significant association between the size of a Bennett lesion and age, level of play, need for surgery, or other shoulder injuries. This suggests that either the lesion itself is asymptomatic and perhaps adaptive in most athletes or that referable symptoms can be managed non-operatively.

There are a few limitations to our study. First, selection bias is a concern given the intrinsic limitations of this small, single-institution retrospective study. However, bias was minimized by using matched case and control groups. Second, we lack long-term clinical follow-up regarding return to play and level of play upon return. There were also a large number of patients who did not return to our institution after their initial MRI. Given a motivated patient population of professional and aspiring professional athletes, some patients lost to follow-up likely received evaluation or treatment elsewhere. Third, we had a low inter-observer agreement for detection of posterior and antero-inferior labral tears and rotator cuff tears. Although we controlled for the type of MRI examination, it would have been ideal to only use MR arthrograms in our study to increase the sensitivity for the diagnosis of labral tears and pathological conditions of the undersurface of the rotator cuff [15, 16]. However, the overall examination heterogeneity is representative of the referral pattern we see at our institution.

In conclusion, overhead athletes with Bennett lesions have an increased frequency of posterior glenoid cartilage abnormalities, but not labral tears or findings of internal impingement, compared to those without Bennett lesions. The clinical relevance of a cartilage lesion in this patient population is unclear, although it may contribute to posterior shoulder pain. The size of a Bennett lesion was not associated with player age, level of play, need for surgery, or other shoulder injuries.

Compliance with ethical standards

This is an IRB-approved study.

Conflicts of interest Tony T. Wong is a Consultant for SoundStim Therapeutics (not relevant to this work).

The authors declare that they have no other conflicts of interest.

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