Purpose: To quantify the magnetic resonance arthrography (MRA) findings in rugby players during preoperative workup for anterior surgical stabilization for glenohumeral instability. Methods: All patients who underwent glenohumeral instability surgery in our institution between 2008 and 2018 were considered for inclusion. Rugby players were identified using the patient’s medical notes, with subsequent identification of all professional players. All rugby player’s preoperative MRA findings were recorded and analyzed. Results: Overall, 267 rugby players were included, 261 of whom were male (97.8%), with a mean age of 22.7 years (range 13-55 years). There were 58 professional rugby players (21.7%). The mean number of pathologies in nonprofessional rugby players was 5.0 pathologies versus 6.2 pathologies in the professional rugby players, with a significant difference in nonprofessional rugby players with up to 3 pathologies versus professional rugby players (26.3% vs 10.3%, \( P = .01 \)). Professional rugby players had a statistically significant increased incidence of bicipital tendon lesions (25.9% vs 13.9%, \( P = .009 \)), acromioclavicular joint degeneration (60.3% vs 42.1%, \( P = .016 \)), glenohumeral bone loss (87.9% vs 69.9%, \( P = .006 \)), and degenerative changes (67.2% vs 44.0%, \( P = .002 \)) on their MRAs. Conclusions: Rugby players undergoing surgical stabilization for glenohumeral instability often have a significant number of pathologies identified on MRA at the time of surgery. Professional rugby players showed concerningly greater frequencies of early degenerative changes when compared with nonprofessional rugby players. Level of Evidence: III; Retrospective Cohort Study

Glenohumeral instability is a common clinical problem among collision athletes, with an incidence being reported as 14,800 per 100,000 adolescent rugby players, compared with 8 per 100,000 in the general population.\(^1\)\(^-\)\(^5\) This injury has become increasingly more common in rugby players over the past 2 decades, with the literature reporting high incidences of glenohumeral instability in collision-sport athletes, with rates of 0.40 injuries per 1000 athlete exposures.\(^6\)

In modern practice, magnetic resonance (MR) has been used as a gold-standard preoperative diagnostic investigation in evaluating labral injuries.\(^7\) Magnetic resonance arthrography (MRA) with contrast has been found to be the most useful single modality for evaluating labral lesions.\(^7\) Although not as immediately useful as computed tomography for evaluating bone loss, MR has been found to be adequate for surgical decision-making and has advantages of being without high radiation while allowing excellent soft-tissue visualization.\(^9\)

The frequency of pathologic MR findings in rugby players undergoing stabilization for glenohumeral instability is unknown. Therefore, the purpose of this study was to quantify the MRA findings in rugby players during preoperative workup for anterior surgical stabilization for glenohumeral instability. Our hypothesis was that rugby players would have a substantial number of pathologies identified on their
MRAs, with professional rugby players having a significantly greater frequency of pathologic findings on their MRAs.

Methods
Using an electronic database, we retrospectively identified all patients who underwent arthroscopic Bankart repair, open Bankart repair, or a Latarjet procedure by a single surgeon between 2008 and 2018. Inclusion criteria for this study were the following: rugby union athlete, presented with anterior shoulder instability, and underwent a preoperative MRA in our institution. Included patients were subsequently assigned a randomized study number to maintain anonymity throughout the study.

A 3-Tesla magnet was used for all MRAs (TwinSpeed 8; GE Medical Systems, Milwaukee, WI). A dedicated shoulder surface coil was used, and patients were positioned with the shoulder in a neutral position. T1-weighted fat-saturated coronal, sagittal, and axial images and T2-weighted fat-saturated coronal images were obtained. The MRAs were assessed at the time by the shoulder surface coil was used, and patients were positioned with the shoulder in a neutral position. T1-weighted fat-saturated coronal, sagittal, and axial images and T2-weighted fat-saturated coronal images were obtained. The MRAs were assessed at the time by a fellowship-trained musculoskeletal radiologist.

Pathologies were defined to include the following: (1) labral tear, (2) SLAP tear, (3) glenolabral articular disruption lesion, (4) humeral avulsion of the glenohumeral ligament, (5) anterior labral periosteal sleeve avulsion, (6) acromioclavicular joint degenerative, (7) bicipital lesion, (8) rotator cuff lesion, (9) Hill–Sachs (HS) lesion, (10) reverse HS lesion, (11) degenerative changes, (12) Bankart lesion, and (13) bony cysts. Glenoid bone loss was defined to include (1) Bankart lesions and (2) acromioclavicular joint degeneration, whereas glenohumeral bone loss was defined to include (1) glenoid bone loss, (2) HS lesions, and (3) reverse HS lesions.

Statistical Analysis
Qualitative statistical analysis was performed using the SPSS (version 22.0. IBM Corp., Armonk, NY). \(\chi^2\) analysis was performed comparing pathologies found between professional rugby players and nonprofessional rugby players.

Results
Patient Demographics
There were 267 rugby players who underwent surgical stabilization for glenohumeral instability enrolled in the study, 261 of whom were male (97.8%). The mean age of the rugby players was 22.0 years (range 17-32). These findings are further illustrated in Table 1.

MR Findings
Overall, 249 rugby players had labral tears (93.3%), including 87 SLAP lesions (32.6%) identified on MRA. There were 109 rugby players with rotator cuff lesions (40.8%), 44 with bicipital tendon lesions (16.5%), and 163 HS lesions (61.0%). In addition, 132 rugby players had anterior glenoid bone loss (49.4%), 197 had glenohumeral bone loss (73.8%), and 104 had bone loss in both anatomical areas (39.0%) identified on MR. There were 131 with degenerative changes (49.1%), 123 with acromioclavicular joint degeneration (46.1%), and 61 with bony cysts of the glenoid or humeral head (22.8%) identified on MR. These findings are further illustrated in Table 2.

Comparison Between Professional and Nonprofessional Rugby Players
Overall, 58 professional rugby players were compared with the 209 nonprofessional rugby players. There was a nonstatistically significant difference between the mean number of pathologies described on their MRAs in the nonprofessional rugby players and the professional rugby players: 5.0 pathologies versus 6.2 pathologies, respectively \((P > .05)\). Nonprofessional rugby players were significantly more likely to have up to 3 pathologies described on their MRA (when compared with professional rugby players; 26.3% vs 10.3%, \(P = .01\)). There were no other significant differences in the combined numbers of pathologies between the professional and nonprofessional rugby players identifiable on MRA (\(P > .05\) for all). Professional rugby players had a statistically significant increased prevalence of bicipital tendon lesions (25.9% vs 13.9%, \(P = .009\)), acromioclavicular joint degeneration (60.3% vs 42.1%, \(P = .01\)), degenerative changes (67.2% vs 14.8%, \(P = .002\)), and glenohumeral bone loss (87.9% vs 69.9%, \(P = .006\)) identified on MRA. There were no other significant differences in the prevalence of other pathologies between the professional and nonprofessional rugby players \((P > .05\) for all). These findings are further illustrated in Fig 1.

Table 1. Patient Demographics

| Characteristic         | Number (%) | Nonprofessionals | Professionals |
|------------------------|------------|------------------|---------------|
| Number                 | 267        | 209 (78.3%)      | 58 (21.7%)    |
| Male                   | 261 (97.8%)| 205 (98.1%)      | 56 (96.5%)    |
| Mean age, y            | 22.66      | 22.83            | 22.04         |
| Left shoulders         | 140 (52.4%)| 106 (50.7%)      | 34 (58.6%)    |
| Right shoulders        | 127 (47.6%)| 103 (49.3%)      | 24 (41.4%)    |
| Arthroscopic Bankart   | 175 (65.5%)| 131 (62.7%)      | 44 (75.9%)    |
| Open Bankart           | 15 (5.6%)  | 13 (6.2%)        | 2 (3.5%)      |
| Latarjet               | 77 (28.8%) | 60 (28.7%)       | 17 (29.3%)    |
Discussion

The most important finding in this study was that rugby players undergoing surgical stabilization for glenohumeral instability had a high number of pathologies identified on their MRAs. Both the professional and nonprofessional rugby players had multiple pathologies identified on their MRAs, with labral tears seen in more than 90% of rugby players included. This study demonstrated that significantly greater frequencies of degenerative changes, glenohumeral bone loss, acromioclavicular degeneration, as well as bicipital tendon lesions were identified on the MRAs of the professional rugby players when compared with those of the nonprofessional rugby players.

The incidence of glenohumeral instability is significantly greater in rugby players, with studies describing rates of glenohumeral instability as more than 1500 times more likely to occur in rugby players when compared with the general population.\(^1,10-12\) The reason for this is thought to be primarily due to the increased number of collisions, as well as a number of other factors. Second, the establishment of the professional playing era in the mid-1990s has seen a significant increase in mean elite rugby players’ mass, from 88 kilograms in the amateur era to 104 kilograms today.\(^13\) The authors of this study believe that a combination of the aforementioned factors have resulted in the greater frequencies of shoulder pathologies being reported on MRA in not only the professional rugby player, but the nonprofessional or previously deemed “social” player,

Table 2. Comparison of Pathologies Found Between Professional and Nonprofessional Rugby Players

| Associated Lesions               | Total | Professionals | Nonprofessionals | \(P\) value |
|----------------------------------|-------|---------------|-----------------|------------|
| Total                            | 267   | 58 (21.7%)    | 209 (78.3%)     | −          |
| Age                              | 22.66 | 22.03         | 22.89           | −          |
| Left                             | 140   | 34 (58.6%)    | 106 (50.7%)     | −          |
| Right                            | 127   | 24 (14.1%)    | 103 (49.3%)     | −          |
| Labral tear                      | 249   | 57 (23.0%)    | 191 (78.3%)     | .07        |
| SLAP tear                        | 87    | 20 (23.5%)    | 67 (31.2%)      | .73        |
| GLAD lesion                      | 30    | 6 (10.3%)     | 24 (11.5%)      | .81        |
| HAGL lesion                      | 8     | 3 (5.2%)      | 5 (2.4%)        | .27        |
| ALPSA lesion                     | 5     | 1 (1.7%)      | 4 (1.9%)        | .92        |
| AC joint degeneration            | 123   | 35 (28.4%)    | 88 (68.2%)      | .014       |
| Bicipital lesion                 | 44    | 15 (25.9%)    | 29 (13.9%)      | .009       |
| Rotator cuff lesion              | 109   | 29 (50.0%)    | 80 (38.3%)      | .11        |
| HS lesion                        | 163   | 35 (20.9%)    | 128 (62.7%)     | .42        |
| RHS lesion                       | 17    | 5 (8.6%)      | 12 (5.7%)       | .43        |
| Degenerative changes             | 131   | 39 (29.7%)    | 92 (63.3%)      | .002       |
| Bankart lesion                   | 106   | 23 (22.4%)    | 83 (76.7%)      | .99        |
| Bony cysts                       | 61    | 13 (22.4%)    | 48 (23.0%)      | .93        |
| Glenoid bone loss                | 132   | 32 (25.7%)    | 100 (69.5%)     | .32        |
| Glenohumeral bone loss           | 197   | 51 (26.1%)    | 146 (71.7%)     | .006       |

AC, acromioclavicular; ALPSA, anterior labral periosteal sleeve avulsion; GLAD, glenolabral articular disruption; HAGL, humeral avulsion of the glenohumeral ligament; HS, Hill–Sachs; RHS, reverse Hill–Sachs.

Fig 1. Comparison of the percentage of combined pathology frequencies identified on magnetic resonance arthrography for the professional and nonprofessional rugby players.
raising concern for these young athletes across the board. Rugby has seen a continuous evolution of the tackle and “poacher” mechanisms of defensive play, which has subsequently led to an increase in frequency of defending players using their shoulders for head-on collisions at greater forces. These mechanisms are also the most likely etiology in developing a single episode of glenohumeral dislocation, as well as recurrent dislocations and instability. The mechanism of this pathology in cases of glenohumeral instability involve the humeral head being displaced anteriorly from the glenoid labrum; this theoretically reinforces the rationale for the high frequencies of bony and chondral pathologies in collision athletes such as rugby players. Although the literature widely reports that American football athletes have a similarly increased risk of glenoid labral tears (among other pathologies) secondary to collisions, reported that not only unilateral tears are commonplace in this cohort but so too are recurrent and bilateral tears on MR imaging. Despite this, they found that this did not directly affect future short-term participation in the National Football League.

Following shoulder dislocations, rugby players have an average time of 12 weeks before they are able to return to play. Such injuries affect professional rugby players in a similar manner, with a mean of 18% of a professional rugby club’s registered rugby players having a shoulder injury at any one time. As professional rugby players often endure planned collision-protected training, the incidence of such shoulder injuries leading to absence from play in rugby players is 90 times more likely to occur during competitive matches compared with club training sessions. Due to the high tackle rates in their game play, back-row players have been reported to have the greatest incidence of shoulder injury of all rugby players, with one event reported per 37 hours of play. In addition, rugby players with glenohumeral dislocations proceed to experience further dislocations and subsequent instability in nearly two thirds of cases and often require surgical stabilization in up to 50% of cases, with professional rugby players subsequently continue to pursue playing rugby post-stabilization. Similarly, our study demonstrated significantly greater frequencies of glenohumeral bone loss, degenerative changes, bicipital tendon lesions, and acromioclavicular joint degeneration on MRA at the time of surgery. Professional rugby players tend to have concerningly greater frequencies of findings when compared with nonprofessional rugby players.

**Conclusions**

Rugby players undergoing surgical stabilization for glenohumeral instability often have a significant number of pathologies identified on MRA at the time of surgery. Professional rugby players tend to have concerningly greater frequencies of findings when compared with nonprofessional rugby players.

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