The Buford Complex Redefined

A Pathologic Morphology in Sheep's Clothing

Jasmine A. Scott,* MD, JD, Jerry R. Dwek,† MD, Karen Y. Cheng,‡ MD, Tracey P. Bryan,§ MA, and Eric W. Edmonds,§|| MD
Investigation performed at Rady Children's Hospital, San Diego, Department of Orthopedic Surgery, San Diego, California, USA

Background: Considered a normal anatomic variant, the Buford complex has not been studied in children.

Hypothesis: A Buford complex is not a normal anatomic variant and would, therefore, be present at a lower rate than that seen in the adult population.

Study Design: Cross-sectional study; Level of evidence, 3.

Methods: Measurements were recorded from magnetic resonance imaging performed over 13 years in children aged ≤11 years for various pathologies unrelated to glenohumeral instability. Interrater reliability was determined to identify Buford complexes, sublabral foramens and tears, and normal shoulders via 16 preadolescent and adolescent patients with confirmed arthroscopic correlation. The Buford complex and labral foramen rates were then compared with a published rate in adults using a binomial probability test.

Results: A total of 122 children (62 girls; mean age, 6.4 years [age range, 2 months-10.9 years]) were evaluated. Interrater reliability was 0.846 (95% CI, 0.56-1) to identify anterosuperior labral variants. The expected sublabral foramen count was 23 children, but only 1 was identified ($P < .001$). The expected Buford complex count was 8 children, but none could be identified ($P < .001$).

Conclusion: The absence of Buford complexes and the significant reduction in sublabral foramen abundance in younger children suggest that these anatomic variants are more likely to be developmental than congenital. The distinct possibility that these previously considered normal variants are truly pathologic findings cannot be ignored. Evidence of a Buford complex could potentially signify an underlying, long-term shoulder instability issue to the treating provider that warrants further investigation or management.

Keywords: Buford complex; children; general; glenoid labrum; imaging; magnetic resonance; pediatric sports medicine; shoulder; sublabral foramen

The glenohumeral joint is the most mobile joint in the human skeleton, supported by dynamic and static stabilizers.[1] The glenoid labrum lines the glenoid rim and increases the depth of the glenoid fossa to provide increased stability. The labrum comprises fibrous connective tissue rather than cartilage,[4] although it may be fibrocartilaginous at the chondrolabral junction. The labrum attaches to the glenoid rim and is the site of attachment of the glenohumeral ligaments.

The labrum is most securely affixed to the posterior superior glenoid.[4,17] The gross morphology of the glenoid labrum varies around its circumference.[14] Several investigations have established wide variability in the morphology of the anterosuperior labrum anteriorly between the origin of the long head of the biceps and the equator of the glenoid.[3-5,17,21] It is challenging to utilize magnetic resonance evaluation of the anterosuperior portion of the glenoid labrum to differentiate between normal anatomic variations and true labral pathology.[19] A sublabral foramen and sublabral sulcus are 2 such supposed variants. A sublabral sulcus is a groove that indents the labrum but does not perforate completely through its substance, while a sublabral foramen is an actual hole that perforates through the labral substance (Figure 1).

A sublabral foramen has an anatomic void between the anterior labrum and the glenoid fossa at approximately the 2-o'clock position (in reference to a right shoulder) of the

The Orthopaedic Journal of Sports Medicine, 12(6), 23259671241252834
DOI: 10.1177/23259671241252834

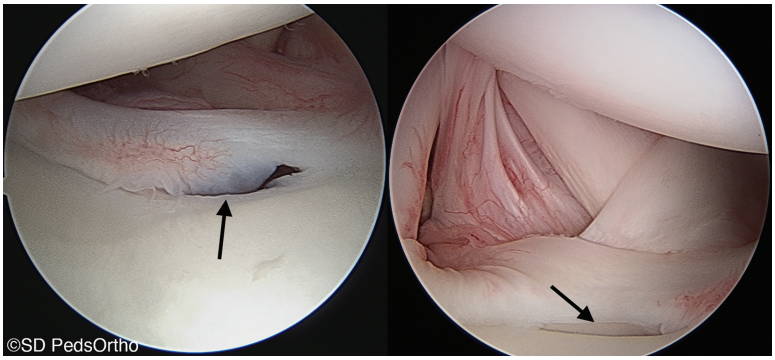

Figure 1. Two adolescent shoulders viewed from the posterior portal looking at the anterosuperior labrum with evidence of sublabral foramen (arrows) and no other traumatic pathology.

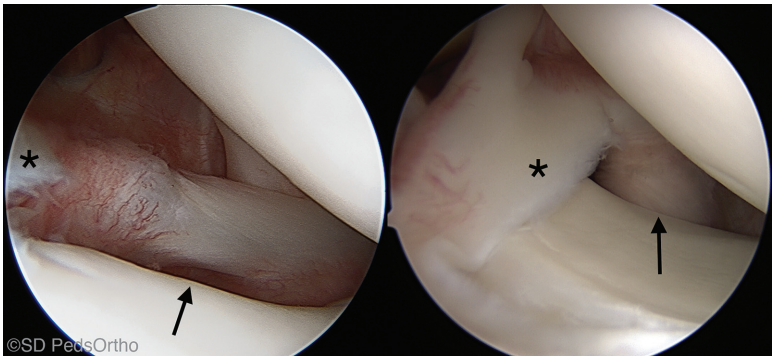

Figure 2. Two adolescent shoulders viewed from the posterior portal looking at the anterosuperior labrum (both of which have evidence of superior labral anteroposterior tears [asterisk]) with evidence of a Buford complex (arrows), as the labrum becomes a cord-like structure with the middle glenohumeral ligament and does not reattach to the anteroinferior labrum.

labrum.[6,11] The sublabral foramen reportedly varies in location, contour, and orientation from the pathologic superior labral anteroposterior (SLAP) tear despite having distinct similarities in location on the glenoid, especially with type 2 SLAP tears.[11,15,20] The presence of the sublabral foramen was first described in 1987 by Detrisac and Johnson[7] using arthroscopy. It was later confirmed in a cadaveric investigation by Cooper et al,[4] who published images showing the synovial lining of the sublabral foramen and no evidence of previous trauma. Another finding that has been previously considered a normal variant is the complete absence of the anterosuperior labrum at the anterosuperior glenoid rim called a Buford complex, especially as the superior labrum joins with the middle glenohumeral ligament (MGHL) in a cord-like fashion (Figure 2).

Although the MGHL has a classic "sheet-like" appearance, it may vary in size and thickness.[17,18] The MGHL arises most frequently from the anterosuperior aspect of the labrum, although it can also arise more inferiorly.[18] The MGHL inserts at the humerus at the base of the lesser tuberosity, although it also can blend with the capsule before reaching the tuberosity. This is in contrast to the Buford complex, defined as a cord-like MGHL that

‖Address correspondence to Eric W. Edmonds, MD, Rady Children's Hospital, San Diego, Department of Orthopedic Surgery, 3020 Children's Way, MC 5062, San Diego, CA 92123, USA (email: ewedmonds@rchsd.org) (Twitter/X: @radyortho).
*Naval Medical Center San Diego, Department of Orthopedic Surgery, San Diego, California, USA.
†Rady Children's Hospital, San Diego, Department of Radiology, San Diego, California, USA.
‡University of California, San Diego, Department of Radiology, San Diego, California, USA.
§Rady Children's Hospital, San Diego, Department of Orthopedic Surgery, San Diego, California, USA.
Final revision submitted November 6, 2023; accepted November 16, 2023.
One or more of the authors has declared the following potential conflict of interest or source of funding: E.W.E. has received education payments from Elevate Surgical Co; has received nonconsulting fees from Arthrex; has the following disclosures unrelated to this project: Elevate Surgical Co– Education; and is a board or committee member of the Pediatric Orthopaedic Society of North America. AOSSM checks author disclosures against the Open Payments Database (OPD). AOSSM has not conducted an independent investigation on the OPD and disclaims any liability or responsibility relating thereto.
Ethical approval for this study was obtained from the University of California, San Diego (ref No. 170519X).

attaches to the base of the biceps anchor with the absence of labral tissue on the anterosuperior glenoid.[15,17,21] In this complex, the MGHL originates from the lesser tuberosity of the humerus, extends toward the superior and anteromedial aspects of the glenoid, and attaches to the base of the origin of the long head of the biceps tendon at the superior labrum instead of attaching to the anterosuperior labrum.[17,21] Williams et al[21] determined this complex to be a normal anatomic variation and reported the prevalence of the Buford complex as 1.5% (3 of 200 patients), and subsequent studies have reported a range between 1.2% and 7.5%.[4,10,12,15,17] Ilahi et al[10] conducted an investigation that included 108 consecutive shoulder arthroscopies in adults and found a total of 20 (18.5%) to have a sublabral foramen and 7 (6.5%) to have the Buford complex.

The Buford complex is traditionally considered a normal anatomic variant, given the smooth transition zones seen arthroscopically from the superior labrum to the MGHL. This study aimed to utilize magnetic resonance imaging (MRI) of pediatric patients to identify the presence of a Buford complex and labral foramen in this younger population. We hypothesized that a Buford complex is not a normal anatomic variant and would, therefore, be present at a lower rate than that seen in the adult population.

METHODS

Patient Selection

Institutional review board approval was obtained before the inclusion of the participants in this study that complies with the Health Insurance Portability and Accountability Act. A waiver of informed consent was obtained. The institutional radiology database was searched to identify all patients aged $\leq$11 years who underwent shoulder MRI for an indication other than glenohumeral instability between November 1, 2008, and February 28, 2022.

A separate surgical database was queried to identify a subset of children with an arthroscopic correlation of findings consistent with Buford complex, sublabral foramens, labral tear, and normal anterosuperior labrum with available MRI scans that utilized the same protocols detailed below.

MRI Technique

The shoulder MRI examinations were performed on either a GE MR450 or HDxT 1.5T MRI scanner (General Electric Company) following the standard institutional protocol. Imaging protocols for noncontrast examinations consisted of a combination of axial proton density-weighted fat-suppressed (FS) sequence (repetition time/echo time [TR/TE], 1750-1850/40-55 ms) or axial T2-weighted FS sequence (TR/TE, 2050-2975/70-85 ms), coronal T2-weighted FS sequence (TR/TE, 2050-2975/70-85 ms), coronal T1-weighted sequence (TR/TE 400-500/14-20 ms), sagittal T2-weighted FS sequence (TR/TE, 2050-2975/70-85 ms), and sagittal T1-weighted FS sequence (TR/TE, 300-450/11-20 ms). Additional imaging parameters included a 12 $\times$ 12–cm field of view (FOV) up to 16 $\times$ 16–cm FOV, 192 $\times$ 256 matrix size, and 3 to 4 mm slice thickness.

Some children had MR arthrograms, and the same magnets were used. These examinations consisted of axial T1-weighted sequence (TR/TE, 480-550/13-16 ms), axial T1-weighted FS sequence (TR/TE 480-550/13-16 ms), coronal T1-weighted sequence (TR/TE, 480-550/13-16 ms), axial T1-weighted FS sequence(TR/TE, 480-550/13-16 ms), coronal T2-weighted FS sequence (TR/TE, 2050-2975/70-85 ms), sagittal T1-weighted sequence (TR/TE, 480-550/13-16 ms), sagittal T2-weighted FS sequence (TR/TE, 2050-2975/70-85 ms), and an abduction external rotation proton density FS sequence (TR/TE, 1750-1850/40-55 ms).

Image Analysis

The MRI examinations from each patient were reviewed independently by 2 musculoskeletal-trained radiologists with 25 years (J.R.D.) and 1 year of experience (K.Y.C.), respectively. Examinations were evaluated for a labral tear, Buford complex, or sublabral foramen. A sublabral foramen was defined as the presence of smooth contrast imbibition (on arthrogram) or fluid signal (on noncontrast examinations), separating the base of the anterosuperior labrum from the adjacent glenoid rim between the 1- and 3-o'clock positions on the glenoid clock face (right shoulder reference). Cases in which the margins of the contrast or fluid signal were irregular were considered to be tears and not sublabral foramens. A Buford complex was defined as a thickened and cord-like MGHL with an absent anterosuperior glenoid labrum. Cases of cord-like MGHLs with visibly torn and displaced or detached anterosuperior glenoid labrums were not considered Buford complexes.

Before the formal evaluation of the primary study MRI scans, each radiologist independently ranked the MRI findings of the small cohort of 16 preadolescent and adolescent children identified, demonstrating the findings of interest detailed above. In the cases not representing an anterior labral tear, the diagnoses included anterior instability unrelated to the labral tear, posterior labral tear, and multidirectional instability. Each radiologist was blinded to the findings and determinations of the other. They ranked each MRI scan as either normal or abnormal (representing any tear, foramen, or Buford complex) to determine the interrater kappa value, utilizing the arthroscopic findings as the "true" findings to confirm their ability to reliably detect and distinguish Buford complex, sublabral foramen, and labral tear.

Statistical Analysis

Interrater reliability was assessed using the kappa (κ) statistic with the interpretation of Landis and Koch[13]: <0, no agreement; 0 to 0.20, slight; 0.21 to 0.40, fair; 0.41 to 0.60, moderate; 0.61 to 0.80, substantial; and 0.81 to 1, almost perfect or perfect. The Buford complex and labral foramen rate in this study was compared with the published rate of

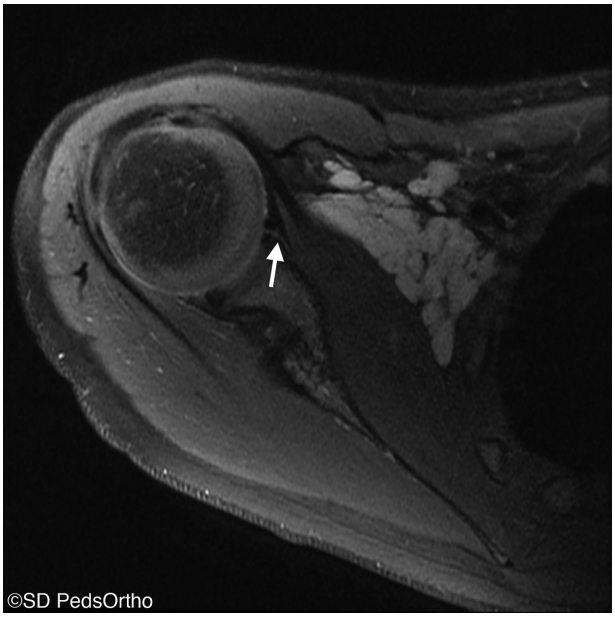

Figure 3. A 9-year-old girl with an identified sublabral foramen—underlying diagnosis was a venous malformation. The white arrow indicates the foramen at the glenoid surface.

6.5% in adults using a binomial probability test. $P < .05$ was set to declare significance. SPSS Version 27 (IBM Corp) was utilized for analyses.

RESULTS

A total of 122 children (62 girls, 60 boys) were evaluated (mean age, 6.4 years old [age range, 2 months-10.9 years). Indications for MRI in these patients included septic arthritis; brachial plexus injuries; pain or decreased shoulder range of motion; soft tissue or bone masses of the clavicle, humerus, and shoulder; Sprengel deformity; multiple hereditary exostosis; Noonan syndrome; juvenile rheumatoid arthritis; and shoulder pain with unknown etiology. A total of 25 examinations were performed with an arthrogram (20% of the total, 13 girls and 12 boys), and none were excluded from the analysis. The interrater reliability between the 2 musculoskeletal radiologists was 0.846 (95% CI, 0.56-1), indicating an almost perfect agreement in identifying anterosuperior labral pathology (inclusive of foramen and Buford complex) in the 16 preadolescent and adolescent patients with arthroscopic confirmation of pathology or normal findings.

None of the children examined in this cohort were identified as having a Buford complex. Binomial analysis between the adult control and our childhood study population indicates this finding is statistically significant ($P < .001$). Only 1 patient was identified with a sublabral foramen at the anterosuperior glenoid (Figure 3). The low prevalence of sublabral foramens in this childhood cohort was also significantly different from that of the historic adult shoulder control ($P < .001$).

DISCUSSION

Past studies of an adult population have demonstrated that the Buford complex is present in between 1.2% and 21.2% of shoulder arthroscopies, MRI evaluations, and anatomic dissections,[4,9,12,15,17,21] suggesting that this entity is a normal variant. Similarly, Ilahi et al[10] found that a Buford complex was present in 6.5% of the adult population undergoing surgical intervention, predominately for pathology other than shoulder instability. If, as previously stipulated, the Buford complex is a congenital anatomic variant, it should be present in the same proportion in a pediatric population as that seen in the adult population. We should have been able to identify approximately 8 Buford complexes in our series of 122 children; however, there were none.

With that said, our understanding of the Buford complex as a normal anatomic variant may be more complex. Detrisac and Johnson[7] described 5 normal variations in labral anatomy that are based on labral appearance being wedge-shaped or meniscoid. Subsequent investigations, including that by Cooper et al,[4] identified additional variants and morphologic differences between the anterior and inferior aspects of the labrum. They found that the inferior labrum was more rounded and firmly continuous with the articular cartilage, whereas the superior labrum generally is more mobile with loose attachments to the glenoid.[4,17] These morphologic differences between the superior and inferior labral attachments raise the possibility that the anterosuperior labrum is more prone to labral tears, resulting in the formation of these labral variants during development rather than being congenitally present. The Buford complex is clinically significant, perhaps more so than other labral variations, because it changes shoulder mechanics and has been associated with other labral pathologies.[16]

As described by Huber and Putz,[8] the labrum, glenohumeral ligaments, and inserting tendons create a periarticular fiber system that acts as a tension brace that provides hoop stresses at its periphery. It has been postulated that patients with a Buford complex (an absent anterosuperior labrum but a cord-like MGHL attachment to the superior labral tissue at the biceps tendon) create forces that are abnormally concentrated to the biceps origin and superior labrum that would potentiate pathology at this location.[2] This described force shifting in the absence of the anterosuperior labrum prevents load dispersion over a wider surface area; therefore, SLAP lesions, which have a higher reported occurrence in the presence of a Buford complex, have a mechanism to form.[16] In an investigation conducted by Bents and Skeete[2] on 250 consecutive shoulder arthroscopies performed at the US Air Force Academy, 6 patients (2.4%) had Buford complexes. Of those 6 patients with Buford complexes, 5 patients (83.3%) had SLAP lesions that required treatment. Compared with their patients without Buford lesions (n = 229), 40 patients (17.5%) had SLAP lesions documented, which was statistically significantly different from their cohort with the Buford complex.[2] In the investigation conducted by Ilahi et al,[9] the incidence of significant SLAP

lesions was significantly higher in shoulders with a sublabral foramen or a Buford complex than in the rest of the study population (56% vs 12%; $P < .05$). In an investigation conducted by Kanatli et al,[12] the reported presence of the Buford complex was found in 28 of 691 patients (4.1%) who underwent shoulder arthroscopy, and a SLAP lesion was present in 23 of the 28 patients with a Buford complex (82.1%) versus only 139 (21.0%) of the 663 patients without a Buford complex. In a recent study from Özer et al[16] that included 3129 consecutive shoulder arthroscopies in 1461 patients without labral tears or multidirectional instability, the prevalence of the Buford complex was 2.7%. The prevalence of the Buford complex in patients with and without labral pathologies was 4.6% and 0.3%, respectively ($P < .001$). The authors concluded that the presence of the Buford complex should prompt a thorough evaluation for concomitant SLAP lesions, as the Buford complex was statistically associated with pathology. The present study (performed in a nonlabral pathology cohort) had a similar, 0.8%, incidence of labral findings on the MRI scans to that of the Özer et al[16] cohort without pathology, further suggesting a direct correlation of Buford complex and labral pathology.

Our study demonstrates that despite the conventional wisdom, the Buford complex may not be a congenital anatomic variant but instead is more likely a developmental variant. With the known association with anterior shoulder instability in adults, this may then suggest that the Buford complex is a pathologic structure. It is beyond the scope of this study to examine what may lead to the development of the Buford complex, and this is an area where further research is needed. However, a reasonable conjecture would be that early shoulder injury during childhood and adolescence might result in the separation of the anterosuperior labrum and glenoid. The robust healing potential of children then lends to the torn labrum conjoining with the juxtaposed MGHL and forming the cord-like structure known as a Buford complex, or if not completely detached from the anteroinferior labrum at the glenoid equator, it merely forms a sublabral foramen. The mechanism of injury for this tear pattern to occur would require further study; perhaps future comparative anatomic animal studies could prove the potential for this developmental sequence to occur.

There are several limitations to this study. First is the inherent selection bias; only patients with shoulder problems necessitating advanced imaging were included. There was no control group of children without shoulder complaints who underwent MRI or arthroscopy. Furthermore, we do not know whether the issues that supported the acquisition of an MRI for each child could have affected their development overall. In addition, some children underwent an MR arthrogram. However, given the lack of Buford complexes and the paucity of sublabral foramens in this cohort, it is unlikely that the children had pathologies that caused changes to the normal labral structure. Second, not all the patients in this study who underwent MRI subsequently underwent shoulder arthroscopy to confirm the findings of the radiologists; however, this is balanced by the interrater reliability ($\kappa = 0.846$), identified via the known arthroscopic findings cohort. Last, the MRIs in this study only included MR arthrograms 20% of the time, potentially underreporting labral pathology. Different MRI machines were used across the cohort, which might have created a variation in the findings noted because of subtle differences in the protocols utilized.

Based on the reported incidences by Ilahi et al,[10] we would have expected to find 23 sublabral foramen and instead found 1. In addition, we would have expected to find 8 Buford complexes, but none were discovered. The lack of evidence for Buford complexes, or even the lack of sublabral foramen abundance, in the presented childhood cohort lends to a further need for investigation. Given the known associations of a Buford complex with labral pathology (both anterior instability and SLAP tears) coupled with our hypothesis being upheld—that Buford complexes occurred at a significantly lower rate in children than published rates in adults—the distinct possibility that these previously considered normal variants of the anterosuperior labrum are pathologic findings cannot be ignored. Further study in this younger cohort or a comparable anatomic study in animals may be beneficial to better understand how a Buford complex can be developmentally obtained. In the meantime, evidence of a Buford complex by preoperative imaging or intraoperative diagnostic arthroscopy should potentially signify an underlying, long-term instability issue to the treating provider that started in the patient's youth.

CONCLUSION

The absence of Buford complexes and the significant reduction in sublabral foramen abundance in children suggests that these anatomic variants are more likely to be developmental than congenital. The distinct possibility that these previously considered normal variants are truly pathologic findings cannot be ignored. Evidence of a Buford complex could potentially signify an underlying, long-term shoulder instability issue to the treating provider that warrants further investigation or management.

ORCID iD

Eric W. Edmonds https://orcid.org/0000-0002-5724-5645

REFERENCES

1. Almajed Y, Hall AC, Gillingwater TH, Alashkham A. Anatomical, functional and biomechanical review of the glenoid labrum. *J Anat*. 2022;240(4):761-771.
2. Bents RT, Skeete KD. The correlation of the Buford complex and SLAP lesions. *J Shoulder Elbow Surg*. 2005;14(6):565-569.
3. Clavert P. Glenoid labrum pathology. *Orthop Traumatol Surg Res*. 2015;101(suppl 1):S19-S24.
4. Cooper DE, Arnoczky SP, O'Brien SJ, Warren RF, DiCarlo E, Allen AA. Anatomy, histology, and vascularity of the glenoid labrum. An anatomical study. *J Bone Joint Surg Am*. 1992;74(1):46-52.

5. Davidson PA, Rivenburgh DW. Mobile superior glenoid labrum: a normal variant or pathologic condition? *Am J Sports Med*. 2004; 32(4):962-966.
6. De Maeseneer M, Pouliart N, Boulet C, et al. A bare area of the glenoid misdiagnosed as a cartilage ulceration. *JBR-BTR*. 2012;95(1): 22-24.
7. Detrisac DA, Johnson LL. *Arthroscopic Shoulder Anatomy: Pathologic and Surgical Implications*. Slack; 1986.
8. Huber WP, Putz RV. Periarticular fiber system of the shoulder joint. *Arthroscopy*. 1997;13(6):680-691.
9. Ilahi OA, Cosculluela PE, Ho DM. Classification of anterosuperior glenoid labrum variants and their association with shoulder pathology. *Orthopedics*. 2008;31(3):226.
10. Ilahi OA, Labbe MR, Cosculluela P. Variants of the anterosuperior glenoid labrum and associated pathology. *Arthroscopy*. 2002; 18(8):882-886.
11. Jin W, Ryu KN, Kwon SH, Rhee YG, Yang DM. MR arthrography in the differential diagnosis of type II superior labral anteroposterior lesion and sublabral recess. *AJR Am J Roentgenol*. 2006;187(4): 887-893.
12. Kanatli U, Ozturk BY, Bolukbasi S. Anatomical variations of the anterosuperior labrum: prevalence and association with type II superior labrum anterior-posterior (SLAP) lesions. *J Shoulder Elbow Surg*. 2010;19(8):1199-1203.
13. Landis JR, Koch GG. An application of hierarchical kappa-type statistics in the assessment of majority agreement among multiple observers. *Biometrics*. 1977;33(2):363-374.
14. Mileski RA, Snyder SJ. Superior labral lesions in the shoulder: pathoanatomy and surgical management. *J Am Acad Orthop Surg*. 1998;6(2):121-131.
15. Motamedi D, Everist BM, Mahanty SR, Steinbach LS. Pitfalls in shoulder MRI: part 1—normal anatomy and anatomic variants. *AJR Am J Roentgenol*. 2014;203(3):501-507.
16. Özer M, Kaptan AY, Ataoglu MB, et al. The Buford complex: prevalence and relationship with labral pathologies. *J Shoulder Elbow Surg*. 2021;30(6):1356-1361.
17. Rao AG, Kim TK, Chronopoulos E, McFarland EG. Anatomical variants in the anterosuperior aspect of the glenoid labrum: a statistical analysis of seventy-three cases. *J Bone Joint Surg Am*. 2003;85(4):653-659.
18. Shankman S, Bencardino J, Beltran J. Glenohumeral instability: evaluation using MR arthrography of the shoulder. *Skeletal Radiol*. 1999; 28(7):365-382.
19. Shortt CP, Morrison WB, Shah SH, Zoga AC, Carrino JA. Association of glenoid morphology and anterosuperior labral variation. *J Comput Assist Tomogr*. 2009;33(4):584-586.
20. Smith DK, Chopp TM, Aufdemorte TB, Witkowski EG, Jones RC. Sublabral recess of the superior glenoid labrum: study of cadavers with conventional nonenhanced MR imaging, MR arthrography, anatomic dissection, and limited histologic examination. *Radiology*. 1996;201(1):251-256.
21. Williams MM, Snyder SJ, Buford D. The Buford complex—the ''cord-like'' middle glenohumeral ligament and absent anterosuperior labrum complex: a normal anatomic capsulolabral variant. *Arthroscopy*. 1994;10(3):241-247.