Glenoid Dysplasia: Pathophysiology, Diagnosis, and Management

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Current Concepts Review

Glenoid Dysplasia

Pathophysiology, Diagnosis, and Management

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- ➤ Subtle forms of glenoid dysplasia may be more common than previously thought and likely predispose some patients to symptomatic posterior shoulder instability. Severe glenoid dysplasia is a rare condition with characteristic radiographic findings involving the posteroinferior aspect of the glenoid that often remains asymptomatic.
- ➤ Instability symptoms related to glenoid dysplasia may develop over time with increased activities or trauma. Physical therapy focusing on rotator cuff strengthening and proprioceptive control should be the initial management.
- ➤ Magnetic resonance imaging and computed tomographic arthrograms are useful for detecting subtle glenoid dysplasia by revealing the presence of an abnormally thickened or hypertrophic posterior part of the labrum, increased capsular volume, glenoid retroversion, and posteroinferior glenoid deficiency.
- ➤ Open and arthroscopic labral repair and capsulorrhaphy procedures have been described for symptomatic posterior shoulder instability. Glenoid retroversion of >10° may be a risk factor for failure following soft-tissue-only procedures for symptomatic glenoid dysplasia.
- ➤ Osseous procedures are categorized as either glenoid reorientation (osteotomy) or glenoid augmentation (bone graft), and no predictable results have been demonstrated for any surgical strategy. Glenoid osteotomies have been described for increased retroversion, with successful results, although others have noted substantial complications and poor outcomes.
- ➤ In severe glenoid dysplasia, the combination of bone deficiency and retroversion makes glenoid osteotomy extremely challenging. Bone grafts placed in a lateralized position to create a blocking effect may increase the risk of the development of arthritis, while newer techniques that place the graft in a congruent position may decrease this risk.

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Historically, glenoid dysplasia has been considered a rare condition attributed to brachial plexus birth palsy or malformation of glenoid ossification centers[1-4]. The classic constellation of radiographic findings includes glenoid and humeral head hypoplasia, varus angulation of the humeral head, and coracoid and acromial hyperplasia. Recently, with the use of advanced imaging studies, localized posteroinferior glenoid dysplasia has been found to be a relatively common clinical entity with potential

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clinical importance[5-9]. A variety of terms and definitions are used to describe abnormal glenoid morphology, including *dysplasia, hypoplasia, glenoid cleft,* and *retroversion,* which contribute to confusion regarding diagnosis and management[5-9]. The recognition of localized posteroinferior glenoid bone deficiency or hypoplasia may be important as these often subtle findings have been correlated with posterior labral tears and recurrent atraumatic posterior shoulder instability affecting shoulder function[6,8]. The degree to which glenoid dysplasia is clinically relevant, in terms of both diagnosis and outcomes after surgery for symptomatic shoulder instability, is not known. The spectrum of developmental anatomy, classification, radiographic findings, and treatment strategies for glenoid dysplasia involving symptomatic posterior instability are reviewed. Glenoid dysplasia associated with skeletal dysplasia is outside the focus of this review.

Glenoid Embryology and Anatomy

The osseous development of the scapula is characterized by intramembranous ossification through 8 different ossification centers. The glenoid consists of 2 ossification centers: a superior ossification center at the base of the coracoid, and a horseshoe-shaped growth center inferiorly. It is hypothesized that variable development of the superior growth center modulates glenoid development, and lack of stimulation of the inferior growth center results in glenoid hypoplasia[10]. The genes that govern the development of these various ossification centers have been well described and include PAX1 (acromion and scapular spine[10,11]), Emx2 (scapular body[10,12]), and Hoxc6 (coracoid and glenoid[10,13]).

Classification and Definitions: Glenoid Dysplasia and Retroversion

Glenoid dysplasia can be characterized as a condition stemming from deficiency of the osseous socket or rim with associated soft-tissue abnormalities of the labrum and capsule as well as malformation in the mechanical alignment or orientation of the socket (version). Glenoid rim deficiency has interchangeably been called dysplasia, hypoplasia, or a dentated glenoid[4]. An alteration in the articular alignment resulting in a posteriorly oriented glenoid surface relative to the scapular body is termed *retroversion.*

Glenoid Dysplasia

While severe glenoid dysplasia is obvious on plain radiographs, there is a spectrum of disease that is variable in presentation and severity, both clinically and radiographically. Various methods have been used to describe and define *dysplasia.* Edelson identified and attempted to define *hypoplasia of the glenoid* by studying scapular bone specimens from several museum collections[5]. *Posteroinferior hypoplasia* was defined as a "dropping away" of the normally flat plateau of the posterior part of the glenoid beginning 1.2 cm caudad to the scapular spine. This specific craniocaudal level on the scapular spine was chosen to eliminate false positives. Hypoplasia occurred in 19% to 35% of specimens. A prospective component of the study by Edelson found an 18% rate of hypoplasia on 300 magnetic resonance imaging (MRI) and computed tomographic (CT) scans made for rotator cuff disease or trauma[5].

Weishaupt et al.[8] further characterized glenoid abnormalities in a series of patients with atraumatic, recurrent posterior instability and found both increased retroversion and posteroinferior osseous deficiency. Utilizing CT arthrograms, they defined *glenoid dysplasia* as either a rounded "lazy J form" or a triangular osseous deficiency called the "delta form" (Fig. 1). They also evaluated glenoid retroversion, which was significantly increased in posterior shoulder instability in comparison with a cohort of patients with anterior instability. Additionally, Harper et al.[6] developed a classification system using MR arthrography (MRA), which recognized the coexistence of hypertrophied posterior glenoid cartilage with glenoid dysplasia (Fig. 2).

Glenoid Retroversion

Glenoid version is defined as the orientation of the glenoid articular surface relative to the axis of the scapular body. *Neutral version* is defined as a glenoid that is perpendicular to the scapular body[14]. A number of authors have sought to determine normal glenoid version using anatomical measurements with advanced imaging, and it is generally regarded to be from 4° to 7° of retroversion[6,14-18]. In addition, Kim et al.[19] identified not only osseous retroversion in posterior instability but also a soft-tissue component described as chondrolabral retroversion. The "loss of containment," as described by Kim et al., in atraumatic posterior shoulder instability is similar to and on the same spectrum as that described by Edelson, Harper et al., and Weishaupt et al.[5,6,8].

Several authors have described glenoid retroversion as a risk factor for the development of posterior instability. Bradley et al.[20] compared MRI scans of 100 patients undergoing arthroscopic posterior labral repair with a control group and found that patients with posterior instability demonstrated greater chondrolabral and osseous retroversion. Owens et al.[21] performed a prospective analysis of 714 military cadets using

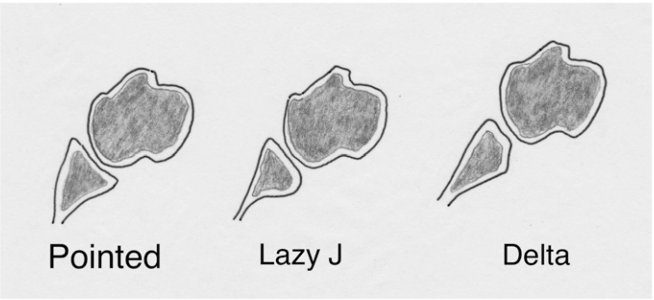

Fig. 1
The qualitative descriptions of glenoid dysplasia as developed by Edelson[5] and Weishaupt et al.[8]. (Redrawn from: Weishaupt D, Zanetti M, Nyffeler RW, Gerber C, Hodler J. Posterior glenoid rim deficiency in recurrent [atraumatic] posterior shoulder instability. Skeletal Radiol. 2000 Apr;29[4]:204-10.)

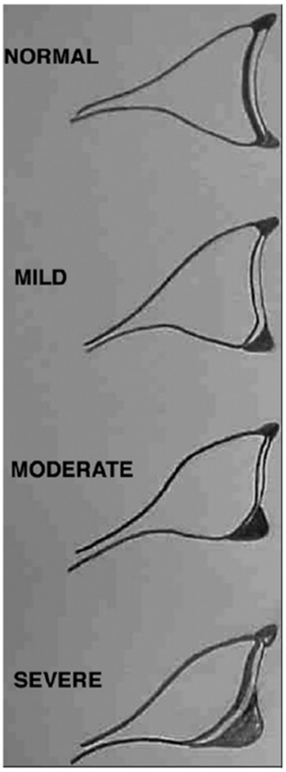

Fig. 2
The classification system developed by Harper et al. showing normal through severe dysplastic morphologies of the glenoid[6]. (Reproduced, with permission of the *American Journal of Roentgenology,* from: Harper KW, Helms CA, Haystead CM, Higgins LD. Glenoid dysplasia: incidence and association with posterior labral tears as evaluated on MRI. AJR Am J Roentgenol. 2005; 184[3]: 984-8.)

MRI and found that the median retroversion in uninjured cadets was 7.7° versus 17.6° in cadets who developed posterior instability. Hurley et al.[15] also found glenoid retroversion to be a risk factor for failure of both surgical and nonsurgical treatment of posterior shoulder instability.

History and Physical Examination

History

Patients with posterior shoulder instability and glenoid dysplasia without arthritis have a variable presentation, as younger, adolescent patients tend to have few or no symptoms[3,4]. Symptoms can develop over time through an increase in activity or physical demands from an occupation or from sporting events[22]. Pain or instability may be attributed to a trivial event or injury[23]. Patients with glenoid dysplasia may also have symptomatic osteoarthritis develop later in life[24]. Typical presenting complaints include pain, weakness, or a sensation of instability.

Physical Examination

Patients with symptomatic posterior glenoid dysplasia usually present with signs of posterior shoulder instability; however, variable presentation with a painful, diminished range of motion, resulting in a suggestion of weakness, can also occur. As such, clinical examination for posterior shoulder instability does not allow for clear and simple distinction among traumatic causes, collagen disorder-related laxity, and causes associated with glenoid dysplasia. Both active and passive ranges of motion are typically normal, although patients may demonstrate a loss of external rotation if pain and subluxation occur. Assessment for signs of generalized ligamentous laxity should be performed as these patients also can present with signs of posterior instability[25-27].

The hallmark feature of posterior instability is pain or apprehension with previously well-described posterior drawer, load, Kim, and jerk tests[28,29]. In the drawer test, which is performed with the patient supine, the examiner uses one hand to stabilize the scapula while the other hand translates the humeral head posteriorly, resulting in subluxation or dislocation with pain, a feeling of apprehension, or an uncomfortable sensation that reproduces the patient's symptoms[22,30,31]. Examiners must distinguish between a normal posterior drawer with painless posterior glenohumeral translation and a pathological examination that reproduces symptoms of instability and pain. The jerk test is performed with the patient in a sitting position. The examiner holds the scapula with one hand and positions the arm at 90° of abduction and internal rotation. An axial force is loaded with the examiner's other hand holding the patient's elbow, with a simultaneous horizontal adduction force applied. A sharp pain with or without a posterior clunk or click suggests a positive test result[32]. The Kim test is similar to the jerk test but is performed with the

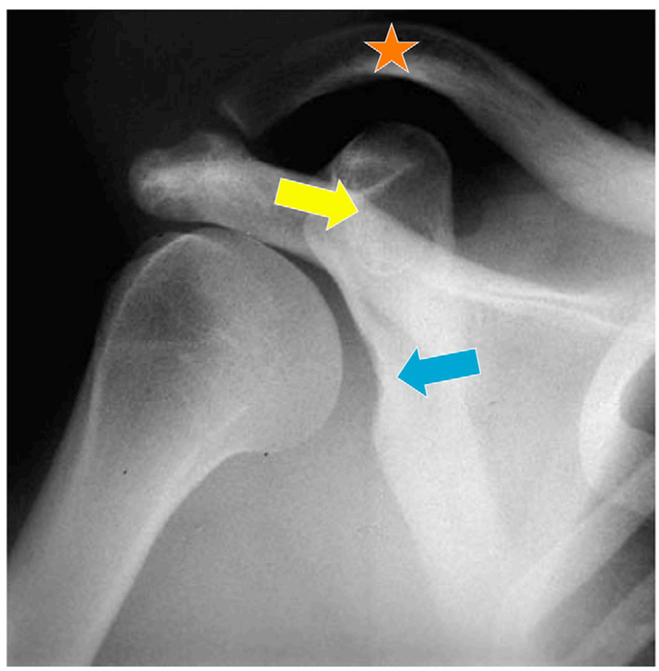

Fig. 3
Anteroposterior radiograph of severe glenoid dysplasia showing hooking of the distal end of the clavicle (orange star), absence of the glenoid neck (blue arrow), and coracoid enlargement (yellow arrow).

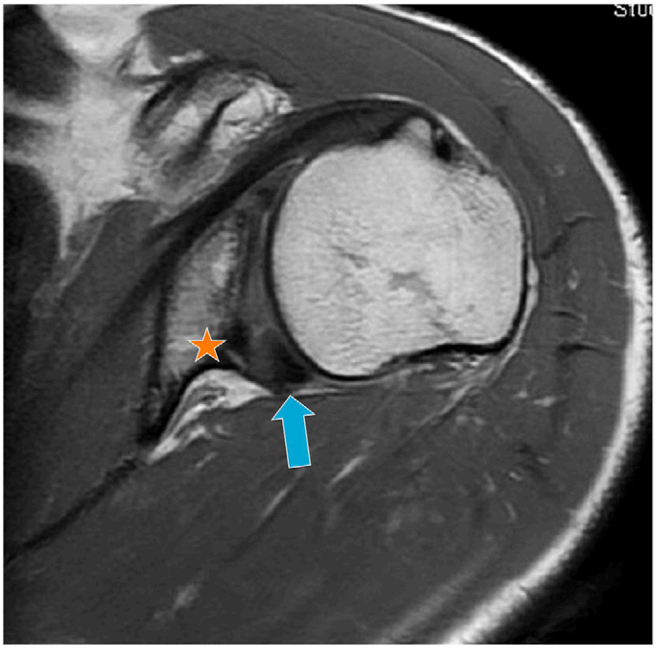

Fig. 4
Axillary T1-weighted MRI scan showing glenoid dysplasia with combined lesions of an enlarged posterior aspect of the labrum (blue arrow), retroversion, posterior humeral head subluxation, and posterior-inferior glenoid hypoplasia (orange star).

arm in 90° of abduction with the examiner holding the patient's elbow and lateral aspect of the proximal part of the arm. A simultaneous axial loading force and 45° of upward diagonal elevation are applied to the distal end of the arm, while an inferior and posterior force is applied to the proximal part of the arm. Pain with or without a clunk represents a positive test[29].

Imaging

Radiographs

Orthogonal radiographs including anteroposterior (Grashey) and axillary radiographs allow for diagnosis of severe glenoid dysplasia. Radiographic findings specific for glenoid dysplasia include hypoplasia of the scapular neck, shallowness of the glenoid cavity, and overgrowth or enlargement of the coracoid or acromion[1,3,4,9,21] (Fig. 3). Axillary images are also useful in identifying other features of posterior instability, including posterior humeral head subluxation and evidence of post-traumatic dislocation events such as humeral head impaction fractures, reverse Hill-Sachs lesions, and posterior glenoid rim fractures (Fig. 4).

Interpretation and diagnosis with radiographs must be performed with caution, given the potential for variation in technique[33]. For this reason, axillary radiographs are no longer considered adequate for the measurement of glenoid version[10,34]. Therefore, while radiographs offer useful qualitative information with regard to osseous anatomy, quantitative measurements of version and osseous and soft-tissue dysplasia can be made accurately and reproducibly only with advanced imaging.

CT

CT is particularly useful in characterizing the anatomy of the shoulder, including the identification and characterization of osseous anatomy such as osseous deficiency of the glenoid that is either traumatic in origin or due to developmental dysplasia[17,18]. Other specific characteristics relevant to humeral stability that are readily measured via CT include version and posterior humeral head subluxation. While various methods exist for determining version, the method described by Friedman et al. has been validated and is considered the accepted method of determining glenoid version[18] (Fig. 5). An arthrogram provides information about the soft tissues of the shoulder including labral pathology and capsular abnormalities such as increased capsular area and capsular tears or avulsions.

CT enables analysis of posterior humeral head subluxation, which is a measure of the static position of the humeral head relative to the glenoid. Similar to version, posterior humeral head subluxation is measured on the axial CT cuts. Increased humeral head posterior subluxation, while not specifically identified as a component of glenoid dysplasia, is often found coexistent with increased glenoid retroversion and is thought to be a risk factor for the development of arthritis[18,35]. The effect of posterior humeral head subluxation on patient outcomes in the treatment of unstable shoulders without arthritis is unknown, and no study, to our knowledge, has correlated posterior humeral head subluxation with outcomes in the setting of glenoid dysplasia and surgical reconstruction for instability in the young patient[36-38].

MRI

Similar to CT, MRI is also capable of providing detail regarding glenoid version, posterior humeral head subluxation,

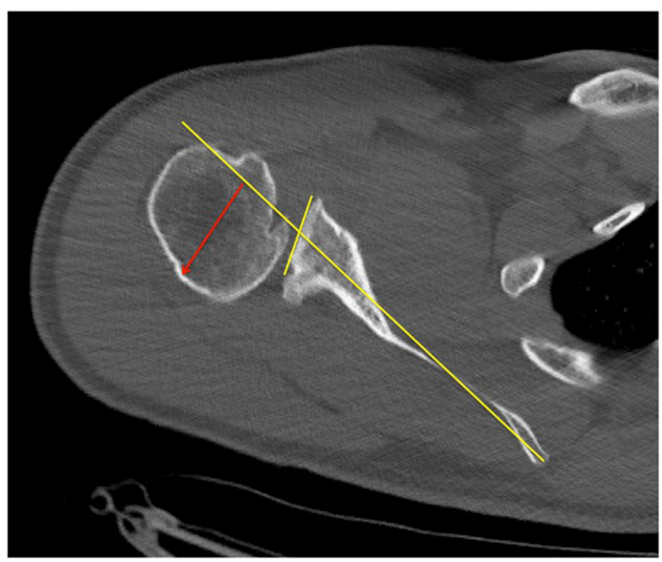

Fig. 5
Preoperative axial CT scan showing glenoid dysplasia and posterior humeral head subluxation. Yellow lines indicate glenoid retroversion of −23° using the Friedman technique. The red arrow indicates 81% posterior humeral head subluxation. Note that posterior instability is defined in relation to the glenoid face and not to the plane of the scapula.

TABLE I Outcomes of Posterior Bone-Block Procedures for the Management of Posterior Glenoid Dysplasia

Study*	Technique†	Graft Type‡	No. of Patients	Patient Types§	Length of Follow-up§ (yr)
Posterior bone block					
Servien et al.[56] (2007)	Open PBB, lateralized	ICBG	21	Trauma (21)	6
Meuffels et al.[54] (2010)	Open PBB, lateralized	ICBG	11	NS	18 (median)
Mowery et al.[65] (1985)	Open PBB, lateralized	ICBG	5	NS	4.2
Fronek et al.[66] (1989)	Open PBB, congruent with capsulorraphy	Acromial	5	Trauma (5)	NS
Scapinelli[67] (2006)	Posterior addition acromioplasty	Acromial	8	Mixed voluntary and involuntary instability	9.6
Essadki et al.[68] (2000)	Open PBB, lateralized	ICBG	6	Trauma (5), MDI (1)	3
Sirveaux et al.[69] (2004)	Open PBB (Group 1) and acromial-deltoid pedicle (Group 2)	ICBG (Group 1) and acromial (Group 2)	18 (9 in Group 1 and 9 in Group 2)	Involuntary posterior instability, including. 50% with signs of hyperlaxity	13 (Group 1) and 3.5 (Group 2)
Kouvalchouk et al.[70] (1993)	Open PBB, congruent	Acromial	5	Mixed trauma, voluntary instability and dysplasia	1.5
Barbier et al.[71] (2009)	Open PBB, congruent	ICBG	8	Glenoid fracture (6), labral tear (6), and dysplasia (1)	2.8
Schwartz et al.[60] (2013)	Arthroscopic PBB, congruent	ICBG	18 (19 shoulders)	Trauma (12), dysplasia (3), retroversion (1), and other (3)	1.7
Ahlgren et al.[46] (1978)	Open PBB, lateralized	ICBG	5	Trauma (5) and MDI (1)	NS
Glenoid osteotomy					
Hernandez and Drez[47] (1986)	Glenoid osteotomy	Acromial	8	Trauma (7) and MDI (1)	3
Bessems and Vegter[72] (1995)	Glenoid osteotomy	ICBG	10 (13 shoulders)	Mixed trauma and generalized laxity	9 (median)
Hawkins[53] (1996)	Glenoid osteotomy	ICBG and acromial	12	Mixed voluntary and involuntary instability	5
Hawkins et al.[33] (1984)	Glenoid osteotomy	NS	17	NS	NS
Graichen et al.[50] (1999)	Glenoid osteotomy	NS	16	Trauma (16); no patient had labral pathology or capsular laxity, they only had retroversion and glenoid "flattening"	5

*All studies had Level-IV evidence. †PBB = posterior bone-block procedure, NS = not specified, and NR = not reported. ‡ICBG = iliac crest bone graft. §The values are given as the mean unless otherwise noted. NR = not reported, and MDI = multidirectional instability. #WOSI = Western Ontario Shoulder Index. **EMG = electromyography, OA = osteoarthritis, and PBB = posterior bone-block procedure.

and other soft-tissue anatomy. MRI evaluation of posterior instability is useful to characterize associated capsular and labral lesions. An arthrogram with intra-articular contrast medium should generally be included as part of this radiographic study as it allows for a more accurate interpretation of the glenoid labrum and capsule[19]. Previous analyses performed have indicated that MRA is also specifically useful in identifying glenoid dysplasia by the presence of

TABLE I (*continued*)

Mean Outcome Scores#	Advanced Imaging	Complications§ *(no. of patients)*	Findings**	Shortcomings
Constant score of 93.3 points	CT scans	Meralgia paresthetica (1)	3 failures; 75% with bone loss	Assessed version (but not glenoid bone loss)
WOSI 60%	NR	Recurrent dislocations (4, including 2 that required fusion)	Poor long-term results and increased rate of glenohumeral OA	Mixed patient population and unclear graft positioning technique
NR	NR	Anterior dislocation (1) and scar revision (2)	Good or excellent outcome in all patients	No mention of bone characteristics, and no outcome scores reported
Pain and instability rating score	NR	NR	Satisfactory results	No advanced imaging, and no mention of bone characteristics
NR	NR	NR	Subjectively all had good outcomes; EMG studies revealed no postop. deficits; no OA	No advanced imaging, no mention of bone characteristics, and no outcome scores
NR	NR	NR	Nearly normal recovery; 100% returned to sports	No advanced imaging, no mention of bone characteristics, and no outcome scores
Walch-Duplay score of 70 (Group 1) and 86 (Group 2)	NR	Screw removal required (4)	Grade-IV OA in 2 patients in Group 1	No advanced imaging and no mention of bone characteristics
NR	NR	NR	Excellent	No outcome scores reported and no mention of bone characteristics
Constant score of 96.25 and Duplay score of 90	CT scan but no measurements	No short-term complications	Satisfactory results in 80%	Did not quantify bone characteristics
Rowe score improved from 18.4 to 82.1 and Duplay score, from 37.4 to 82.9	CT scan but no measurements	Screw removals (6) and graft resorption requiring revision (1)	36% complication rate but substantial improvement	No mention of bone characteristics
NR	NR	None reported	2 excellent, 1 very good, and 2 improved	No mention of bone characteristics, and no outcome scores reported
NR	NR	NR	Posterior labral tears and lax capsules	No mention of bone characteristics, and no outcome scores reported
Rowe score excellent for 12 and good for 1	NR	NR	All with good to excellent results	No mention of bone characteristics
NR	CT scan; mean version correction of 10.8°	Various complications (7)	High complication rate and variable version correction; technique should be used with caution	No outcome scores reported
NR	NR	Shoulder OA (3), and persistent pain of unknown etiology (1)	Recurrence of instability in 7 patients	No advanced imaging, no mention of bone characteristics, and no outcome scores reported
81% good to excellent (Constant and Rowe)	CT scan; mean version correction of 4.73°	NR	25% of patients with degenerative changes and 12.5% with recurrent instability	No MRIs performed

an abnormally thickened or hypertrophied posterior labrum[2,6,39,40] (Fig. 4).

Treatment Options

Nonoperative Treatment

Some patients with glenoid dysplasia receive the diagnosis incidentally and remain asymptomatic[3,9]. Physical therapy involving rotator cuff strengthening and proprioceptive control of the shoulder through symptomatic ranges of motion can be successful[3,9,23]. Despite these treatments, however, a return to a physically demanding occupation and sports may be limited for some patients[9,15]. A rotator cuff strengthening program was successful in avoiding surgery in 50% of patients with posterior shoulder instability[15]; however, 96% continued to experience symptoms. Exercises focusing on external rotation stretching should be considered in patients with symptomatic glenoid dysplasia and internal rotation contracture.

Operative Treatment

Operative treatment of symptomatic posterior shoulder instability in the setting of glenoid dysplasia has been performed in a variety of ways. Treatment may involve soft-tissue or osseous procedures, or a combination of these procedures. Soft-tissue procedures may be focused on the capsule, labrum, or both structures, and may be performed through either an open or an arthroscopic technique. Osseous procedures are generally categorized as glenoid reorientation (osteotomy) or glenoid augmentation (bone graft) procedures.

Soft-Tissue Procedures

Few studies have directly evaluated the outcomes of soft-tissue procedures in patients with glenoid dysplasia or have established a threshold of dysplasia or retroversion that is amenable to soft-tissue-only techniques. Previously, authors recognized the association between increased glenoid retroversion and inferior surgical outcomes after open capsular shift surgery. Hurley et al.[15] found that patients with symptomatic posterior instability and glenoid retroversion of >9° experienced higher recurrence rates after soft-tissue procedures alone. Similarly, Fuchs et al.[41] determined that excessive retroversion of the glenoid fossa remained unresolved and recommended a glenoid osteotomy for retroversion of >10° when performing an open posterior-inferior capsular shift for symptomatic posterior instability. Conversely, Bigliani et al.[42] performed CT scans for 16 of 35 shoulders prior to an open posterior capsular shift and found the average retroversion was –6°. Overall, their surgical cohort had an 80% success rate but they did not attribute their failures to osseous anatomy. Bradley et al.[20] described increased retroversion in his cohort of patients who were operatively treated for posterior instability in comparison with a control cohort, but they did not attribute retroversion as a factor in the outcome or as a risk of failure.

In general, open and arthroscopic labral repair and capsulorrhaphy procedures for symptomatic posterior shoulder instability have been described with generally good outcomes free from revision and with a return to activity. However, in most of those studies, the authors had not commented on the osseous architecture of the shoulder in terms of version or dysplasia, but rather focused on the soft-tissue abnormalities[19,20,42-45]. In conclusion, no specific recommendations can be made regarding the choice of a soft-tissue procedure versus an osseous procedure. More severe forms of dysplasia and retroversion of >10° may represent a risk for failure with a soft-tissue procedure, but further research on this topic needs to be conducted.

Osseous Procedures

The lack of precise documentation of the surgical technique is problematic when analyzing historical data from various augmentation procedures. This lack of uniformity in description and technique makes evaluation of outcomes difficult and comparison among techniques challenging. Additionally, most published historical techniques utilizing various glenoid bone-grafting and osteotomy procedures were performed before the advent of CT and MRI and therefore do not provide an assessment of glenoid morphology[46,47]. Furthermore, the lack of patient-reported outcome measures and inadequate follow-up do not allow for a true interpretation of outcomes. The combination of these factors makes most historical case series difficult to interpret in the current treatment of glenoid dysplasia. Table I lists the published case series with ≥5 patients, preoperative imaging analysis, a clear description of surgical technique, and a minimum follow-up of 1 year.

Glenoplasty procedures are opening-wedge osteotomies resulting in version reorientation[48]. Kretzler and Blue are recognized as the first to employ open glenoplasty for posterior shoulder instability[49]. Metcalf et al. noted that 79% of 236 patients in 21 different case series utilizing glenoplasty had a stable shoulder at the time of follow-up[48]. Graichen et al., in a study of 32 patients, reported good-to-excellent results in 81% of the patients undergoing glenoplasty with no reported complications and an absence of osteoarthritis after 5 years of follow-up[50]. Hawkins et al., however, described a complication rate of 29% (5 of 17 shoulders) and a recurrence rate of 41% (7 of 17 shoulders)[33]. Glenoplasty is technically demanding, and consequently, complications can be substantial, including loss of correction, intra-articular fracture, graft extrusion, and overcorrection with subsequent development of coracohumeral impingement[51,52]. Others have questioned the safety and effectiveness of glenoplasty procedures, given the potential complications and variability of correction[53].

Glenoid augmentation procedures are bone grafts from a variety of autograft and allograft sources, including the acromion, iliac crest, tibia, and ribs. These grafts can be placed in an extracapsular or intracapsular position. Furthermore, the graft may be placed congruent with the existing articular surface in an effort to extend the functional articulating surface or in a lateralized position in which they act as a true "bone block" to posterior humeral translation (Fig. 6). Lateralized bone-block procedures represent a subset of glenoid augmentation procedures that must be distinguished from congruent procedures. The posterior bone-block procedure using a tibial autograft fixed to the posterior part of the glenoid was described in 1938 by Fèvre and Mialaret[54]. Additional authors have since described similar methods of lateralized posterior glenoid augmentation[55].

Not all case series clearly differentiate graft positioning although determining this difference is essential as a lateralized graft may predispose the development of symptomatic osteoarthritis. Servien et al.[56] reported good results in all 21 shoulders in their study at 6 years after the use of a lateralized bone block; however, those patients had traumatic instability and bone loss and not dysplasia. Similarly, Meuffels et al.[54]

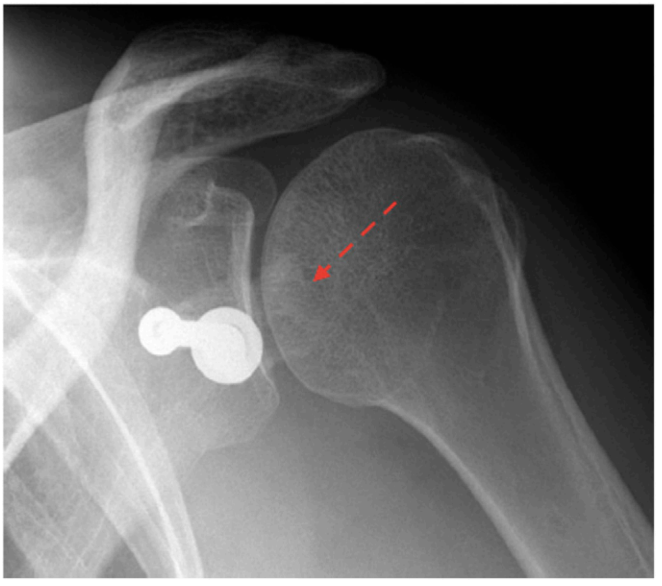

Fig. 6
Anteroposterior radiograph of the shoulder showing the lateralized posterior bone-grafting with the blocking (red dashed arrow) technique. (Reproduced, with modification, from: Meuffels DE, Schuit H, van Biezen FC, Reijman M, Verhaar JA. The posterior bone block procedure in posterior shoulder instability: a long-term follow-up study. J Bone Joint Surg Br. 2010 May;92(5):651-5. Reproduced with permission and copyright © of the British Editorial Society of Bone and Joint Surgery.)

described good results in 11 patients at 6 years after treatment with a lateralized bone block, but these results were not found to be durable over time. At an average of 18 years of follow-up, the outcomes had substantially declined because of recurrent instability and the development of osteoarthritis. The authors concluded that a bone-block procedure should not be performed in patients with hyperlaxity or multidirectional instability.

Because the majority of historical case series utilizing a posterior bone block were performed before the availability of CT or MRI, it is probable that most of these series included patients who did not have glenoid dysplasia but rather had either a torn posterior part of the labrum or symptomatic multidirectional instability. Therefore, the results of these case series cannot be reliably used to determine the success of posterior bone block procedures for posterior glenoid dysplasia (Table I).

Recent developments in arthroscopic instrumentation and techniques allow for arthroscopic posterior bone-graft procedures that provide a congruent extension of the existing articular surface[57-60]. This technique has the theoretical advantage of requiring less dissection, ensuring accurate graft placement and the ability to treat concomitant intra-articular pathology. The goal behind this treatment method is to increase the available surface area for the head to articulate and in doing so accept the native version. Biomechanically, this technique has the advantage of restoring the joint kinematics in situations involving posterior humeral head subluxation and asymmetric joint loading (Figs. 7-A through 7-D). The capsule is repaired over the graft using suture anchors, effectively making the graft extra-articular as described by Smith et al. and Schwartz et al.[57,60]. Techniques utilizing fresh tibial osteoarticular allograft have also been described for glenoid bone loss and fractures[61]. There may be concerns regarding the difficulty of learning and performing arthroscopic bone-grafting techniques; therefore, these techniques may not be applicable to all surgeons.

Patients who present with end-stage osteoarthritis in the setting of glenoid dysplasia (a Walch type-C glenoid with >25° of retroversion) are uncommon, and reports regarding treatment are sparse. In one case series describing shoulder arthroplasty outcomes, the authors suggested that acceptable short to intermediate-term outcomes are possible after the placement of a glenoid component in shoulders with a glenoid deformity if the humeral head is not subluxated and there is adequate glenoid vault depth[62]. Bonnevialle et al. reported good outcomes with hemiarthroplasty alone[63]; however, this has not been the experience of other investigators who have acknowledged difficulty in placing glenoid components in this clinical scenario, with worse outcomes for hemiarthroplasty, and recommended the use of bone-grafting or prosthetic augmentation to address the glenoid dysplasia[24,64].

Overview

The recognition of glenoid dysplasia is increasing because of the widespread availability of advanced imaging studies. These studies show that subtle and less demonstrative forms of dysplasia exist and are more prevalent than previously recognized. Recognition of glenoid dysplasia is important for patient counseling and treatment method selection. Variables to consider when choosing treatment options for symptomatic posterior shoulder instability in the setting of glenoid dysplasia include patient activity, occupation, previous procedures, severity of osseous or tissue deformity, and the presence of increased joint laxity or collagen disorders.

Analysis of published treatment types and outcomes is plagued by a lack of high-quality cohort studies with clear functional and clinical outcome assessment, small case series with mixed pathology, and the lack of advanced imaging to accurately characterize the type and severity of dysplasia. This lack of clarity makes technique comparison and outcome analysis problematic and precludes the establishment of absolute treatment recommendations. However, recognition of the limitations of various techniques allows for an informed approach in choosing patient-specific solutions for the pathological conditions encountered.

Soft-tissue repairs, glenoplasty, and bone augmentation techniques are described as achieving short-term success in the treatment of symptomatic posterior shoulder instability with glenoid dysplasia. Isolated soft-tissue repairs likely have higher failure rates at some threshold because of inherent biomechanical disadvantages of increased retroversion and dysplastic

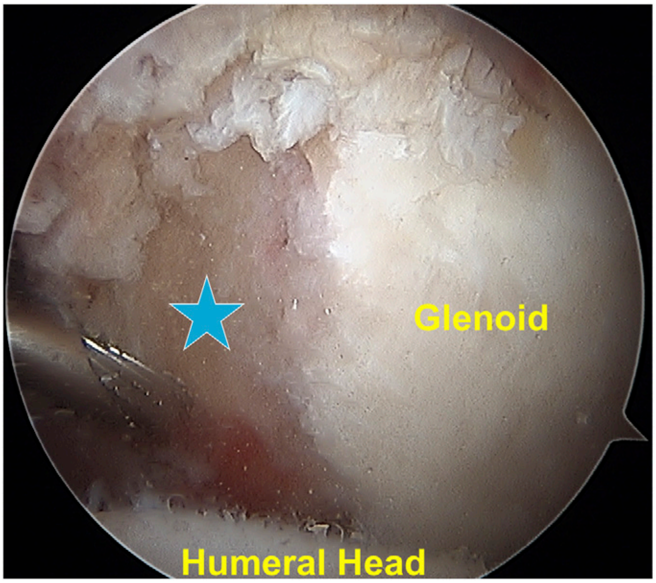

Fig. 7-A

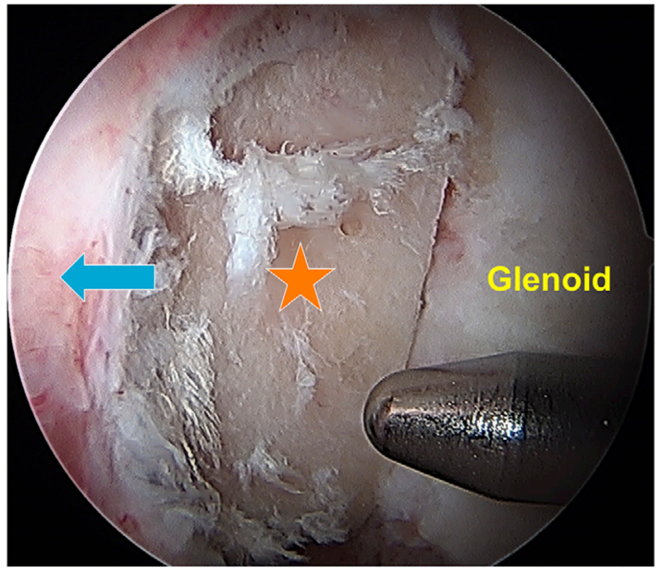

Fig. 7-B

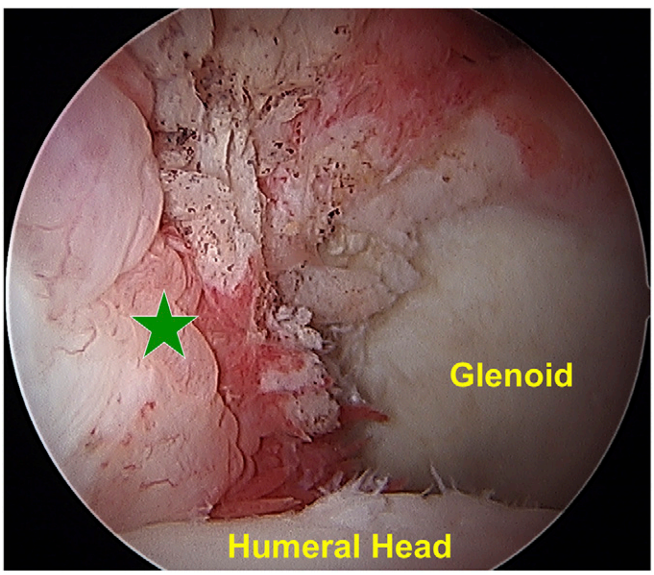

Fig. 7-C

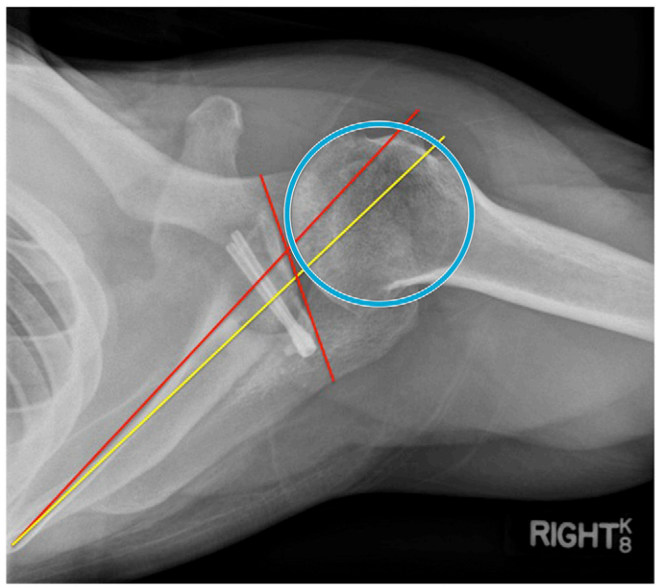

Fig. 7-D

Figs. 7-A through 7-D Arthroscopic posterior glenoid reconstruction with iliac crest bone graft (ICBG) for symptomatic glenoid dysplasia. **Fig. 7-A** Arthroscopic view of the posterior glenoid dysplasia and cartilage loss (blue star) before the reconstruction procedure. **Fig. 7-B** Arthroscopic posterior glenoid bone-grafting (orange star) with autogenous ICBG. The blue arrow is pointing to the posterior capsule. **Fig. 7-C** Arthroscopic repair of the posterior capsule (green star) over the ICBG to the native glenoid. **Fig. 7-D** Postoperative axillary radiograph made after arthroscopic bone-grafting of the glenoid with ICBG, showing recentering of the humeral head (blue circle) on the glenoid axis (yellow line). The original glenoid retroversion and posterior humeral head subluxation is illustrated with the red lines. (Note that the radiograph represents a static image without the arm in forward flexion.)

bone insufficiency. Glenoplasty or glenoid osteotomies are challenging procedures with a substantial risk of complications. Glenoid augmentation techniques must be differentiated between congruent and incongruent techniques. Incongruent or lateralized bone grafts can be effective at achieving stability but may predispose toward secondary osteoarthritis. Congruent bone-graft placement may be a more successful alternative, particularly with newer techniques and instrumentation. Given the dearth of robust outcome studies, technical difficulty of osseous procedures, and associated potential surgical risks, caution must be exercised when considering surgical options for symptomatic glenoid dysplasia. Future research efforts should focus on determining the threshold of clinically important glenoid dysplasia as well as thorough analysis of surgical intervention strategies. ■

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