Etiology of Shoulder Arthritis in Young Patients

Michael S. Laidlaw, MD, Harrison S. Mahon, MD, Brian C. Werner, MD*

KEYWORDS

• Young adult • Arthritis • Glenoid dysplasia • Glenohumeral instability
• Postarthroscopic glenohumeral chondrolysis • Osteonecrosis
• Radiofrequency ablation

KEY POINTS

- Both congenital and acquired glenoid defects may contribute to glenohumeral instability and lead to early arthritis.
- Postarthroscopic glenohumeral chondrolysis is multifactorial.
- Chondrolysis occurs in the young adult compared with primary osteoarthritis.
- Biologic etiologies such as osteonecrosis and inflammatory arthropathy also contribute to the development of arthritis in the young adult.

INTRODUCTION

Glenohumeral arthritis in the young adult is often a multifactorial problem that can be challenging to manage. Although shoulder replacement procedures remain an option for older individuals, treatment for younger patients is much more diverse and must take into account the underlying etiology. Many potential causes of glenohumeral arthritis have been implicated in the young adult. These include genetic factors, congenital and acquired glenoid abnormalities, focal chondral defects, infection, capsuolorraphy-related chondrosis, osteonecrosis, pain pump-mediated chondrolysis, thermal capsulorraphy, and inflammatory arthropathies. Although each of these etiologies have been studied in other contexts, their role in terms of the development of early glenohumeral arthritis has not been extensively studied. A better understanding of these factors is needed to treat and prevent this devastating problem.

GENETIC FACTORS

Many genetic and environmental factors contribute to the development of glenohumeral arthritis. Certain etiologies, like glenoid dysplasia (GD) and rheumatoid arthritis,

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Department of Orthopaedic Surgery, University of Virginia, 400 Ray C. Hunt Drive, Suite 300, Charlottesville, VA 22903, USA
* Corresponding author.
E-mail address: BCW4X@hscmail.mcc.virginia.edu

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have fairly strong genetic contributions that have been described in the literature. However, there is some limited information regarding which genes and biomarkers may play a significant role in the development of primary glenohumeral osteoarthritis (OA). One study sampled cartilage from arthritic and nonarthritic humeral heads and used real-time polymerase chain reaction to determine which genes were expressed in each group. Cx43, Cox-2, versican, collagen type I, ADAMTS5, MMP3, and tumor necrosis factor-α expression, were significantly increased in the arthritic group, with Cx43 having a 85-fold increase in the OA group.[1] This molecule is a gap junction protein that has been found to be increased in both the synovial lining cells and cartilage of patients with OA.[2,3]

CONGENITAL ABNORMALITIES

GD is a scapular developmental abnormality characterized by bony deficiency of the posteroinferior glenoid.[4] Before the recent exponential increase in axial imaging, it was thought to be exceedingly rare. However, recent data suggest that it is underdiagnosed and that it may contribute to premature shoulder OA.[5,6] The pathogenesis of GD seems to be related to abnormal ossification. Imaging and arthroscopy studies in patients with GD have demonstrated thickened inferior cartilage and a hypertrophic posterior labrum, suggesting normal development of the precartilage.[7,8] Two patterns of bony glenoid deficiency have been described based on axial computed tomography scan. The lazy J pattern is characterized by rounding of the posteroinferior rim, whereas the delta pattern has a sharp triangular area of bony deficiency in the same region.[9]

The link between GD and the development of early OA is poorly understood. GD has been associated with instability and posterior labral tears, which may be considered an entirely separate cause of early OA. It is important to note that, in contrast with the more common posterior subluxation and eccentric wear pattern seen in primary OA (Walch type B2), the thickened posterior soft tissues in GD maintain a more centered humeral head location.[10] Although the overall contribution of GD to early shoulder OA is low, several studies have demonstrated a correlation. Most patients with GD remain asymptomatic for years before the development of any symptoms, with most becoming symptomatic in the fifth or sixth decades.[11] However, Allen and colleagues[12] reported on 20 patients who underwent shoulder arthroplasty for GD and found that 9 of the patients were under the age 50. One patient was 39 years old at the time of surgery. Of course, GD patients still make up a small percentage of those requiring arthroplasty. Edwards and colleagues[13] reported that only 3.5% of patients undergoing total shoulder arthroplasty for OA had GD.

INSTABILITY

Glenohumeral instability can be loosely grouped into acute and chronic etiologies. Anterior and posterior instability is usually due to an acute dislocation event. Chronic instability is typically due to multidirectional instability. By far, the most common cause of shoulder instability is acute anterior dislocation, which accounts for 90% of all shoulder dislocations. Male sex, white race, age less than 30 years, contact sports, and enlistment in the military are all risk factors for anterior instability.[14,15]

Most of the literature on glenohumeral instability as a cause of arthritis focuses on patients treated with a stabilizing procedure (see section on capsulorraphy-related chondrosis). However, there is some research that focuses on patients treated nonoperatively. Neer and colleagues[16] first described anterior instability as a cause of arthritis in 1982. Samilson and Prieto[17] later coined the term “dislocation arthropathy”

after reporting on 74 patients with arthritis and prior instability. They found that posterior dislocation and older age at the time of initial dislocation was associated with the development of more severe arthritis, but that the number of prior dislocations was not.[17] Since then, data have shown that recurrent instability is associated with the development of arthritis. Marx and colleagues[18] found that patients with a history of dislocation were 10 times more likely to develop arthritis necessitating total shoulder arthroplasty compared with controls, and that patients with recurrent dislocation had an even higher risk. Hovelius and Saeboe[19] followed 223 shoulders after primary anterior dislocation for 25 years and found that age greater than 25 years at initial dislocation, high-energy sports, and alcohol abuse were associated with development of arthropathy. Of the shoulders that did not recur, 18% had moderate to severe arthropathy. For patients with recurrent dislocations, 39% of those treated nonoperatively developed arthropathy and 26% of surgically stabilized shoulders did. Buscayret[20] examined risk factors for both preoperative and postoperative arthritis in 570 patients treated with an instability procedure. In addition to age at onset, they found that time from onset to surgery, rotator cuff tear, bony glenoid lesions, and humeral head impaction fracture were associated with preoperative arthritis.

FOCAL CHONDRAL DEFECTS

As with other joints, full-thickness cartilage defects within the glenohumeral joint have a limited capability to heal. These lesions may alter the congruity of the joint and progress to OA. There are many etiologies of focal cartilage abnormalities within the shoulder, including posttraumatic defects, iatrogenic injuries, and osteochondritis dissecans (OCD). Other more diffuse etiologies such as osteonecrosis, chondrolysis, and instability-related defects are covered elsewhere in this article.

The most commonly reported and studied articular defects are those related to instability. However, other lesions related to both acute and chronic trauma have been described. Carroll and colleagues[21] reported on focal articular cartilage lesions located in the posterosuperior humeral head that were medial to the expected Hill-Sachs lesion location, likely owing to a compression injury against the acromion. The largest one in the series was 2×3 cm. The patients in this group who were imaged within 4 weeks of their traumatic event showed subchondral marrow edema, suggesting an acute cause. Traumatic shear-type injuries of the inferior humeral head have also been described in contact sport athletes.[22]

Chronic traumatic lesions of the glenohumeral joint are generally related to other causes of shoulder pathology. Jobe and Jobe[23] grouped these overuse athletic injuries of the shoulder into those related to rotator cuff injury, instability, and impingement syndrome. Full-thickness rotator cuff tears in the young patient are usually related to acute traumatic dislocation. However, the recent literature suggests that elite throwing athletes are at risk for developing partial thickness rotator cuff tears that occur owing to overuse.[24] Among all age groups, rotator cuff tears are associated with the development of chondral defects. These are usually found in the posterosuperior humeral head and inferior glenoid.[25,26] The incidence for humeral and glenoid defects in patients diagnosed with impingement has been reported at an average value of 16% and 10%, respectively.[27]

OCD is a poorly understood disease that most commonly affects the knee, ankle, and elbow. A recent review from the ROCK study group (Research on OsteoChondritis of the Knee) defined OCD as focal, idiopathic lesions that involve subchondral bone. These lesions increase the risk of articular cartilage instability and subsequent development of arthritis.[28] Both humeral head and glenoid fossa lesions have been

described in the literature but are quite rare. Humeral head OCD has 12 reported cases, with most being found in males between 12 and 19 years of age. The most common location was anterosuperior. Glenoid fossa OCD has 10 reported cases that suggested a predominance in overhead throwing athletes. For both humeral head and glenoid fossa OCD, the presumed etiology was repetitive microtrauma.[29]

For focal glenohumeral cartilage lesions owing to any cause, the clinical relevance in terms of development of arthritis has not been well-defined in the literature. In a recent series on 14 patients treated with humeral head and/or glenoid microfracture for isolated full-thickness defects, most patients had positive outcomes at an average of 27.8 months of follow-up. One patient underwent subsequent total shoulder replacement owing to progression of arthritis.[30]

GLENOID FRACTURE

Glenoid fractures are generally rare injuries and few require operative treatment. The most common types are anterior avulsion and rim fractures, which comprise 75% to 85% of all glenoid fractures and are usually associated with traumatic dislocation. The majority of these can be managed without surgery provided there is a concentrically reduced glenohumeral joint. Glenoid fossa fractures are generally seen in higher energy mechanisms of injury but can also be managed nonoperatively if there is minimal displacement and articular step-off. Kavanagh and associates[31] reported on 9 patients treated with open reduction and internal fixation for an intraarticular glenoid fracture displaced more than 2 mm. The average follow-up time was 4 years (range, 2–10 years) and no patient developed radiographic evidence of OA.[31] Mayo and colleagues[32] reported on 27 patients treated surgically for intraarticular displacement of more than 5 mm and found that only 2 had subtle joint space narrowing after a mean follow-up time of 43 months. There are few data regarding nonoperative management of glenoid fossa fractures but, in general, minimally displaced fractures can be treated conservatively with positive outcomes. Severely comminuted fossa fractures are associated with a high rate of OA and instability.[33]

SEPTIC SHOULDER

The modes of the introduction of bacteria into a joint are many; however, the most common two are direct inoculation by traumatic arthrotomy and by hematogenous seeding. Although weight-bearing joints are the most commonly afflicted joints associated with septic arthritis, upper extremity joints such as the shoulder joint are not immune, with roughly 10% to 15% of all septic arthritis cases involving the upper appendicular system, with potentially higher reported rates among intravenous drug users.[34,35] *Staphylococcus* species and β-hemolytic streptococcus species are the most commonly involved in nongonococcal septic arthritis, combining for more than 90% of reported cases.[36] The shoulder joint is seldom involved in septic arthritis cases, with a published incidence of 8%.[37] The concern for septic arthritis is given the chondrotoxic nature host immune response to the bacteria with associated enzymes and break down degradative products, cytokines and reactive oxygen species within the immunologically privileged joint.[38] It is well-known that the development of postseptic arthritis can occur in patients afflicted with a septic joint secondary to the chondrotoxic degradative process; however. there is no published literature to suggest that lower extremity versus upper extremity septic joint involvement portends a worse outcome based on the location. Previous studies have shown that either needle aspiration or open lavage are successful at treating the offending bacteria; however, more favorable and reproducible results of improved long-term functional range of

motion are seen with open irrigation/lavage and debridement.[34,39] The baseline importance of surgical treatment with complementary bacterial-sensitive antibiotics rests on the decrease of the bacterial load and resultant host immunologic response with degradative and chondrotoxic byproducts.

CAPSULORRAPHY-RELATED CHONDROSIS

Chondrolysis, by definition, is the ultimate death of chondrocytes, mainly from their inability to produce cartilage matrix or from apoptosis itself. The distinguishing features that sets this pathologic process apart from primary glenohumeral arthrosis is not only the time course of its development, the former being within months of the inciting event versus years, but also that it afflicts younger individuals. Several known factors have been involved in chondrolysis, including gentian violet, chlorhexidine, methyl methacrylate, menisectomy, thermal radiofrequency energy, prominent hardware, chronic synovitis, and fibronectin.[40] Recent literature suggests that postarthroscopic glenohumeral chondrolysis (PAGCL) might have an association with arthroscopic capsulorraphy stabilization procedures.[40] PAGCL is a term applied to the rapid development of destructive changes to the shoulder joint after an arthroscopic procedure. There are many trends that have been noted in the development of this clinically progressive arthrosis and the presence of PAGCL is likely multifactorial. Implant-related chondrosis can occur by either proud implants, loose body implants, or even chondral damage associated with implant placement, potentially too far onto the face of the glenoid articular surface.[41,42] In the absence of implant-related chondrosis, the actual procedure choice has been suggested as a potential risk when addressing glenohumeral instability. With the use of Putti-Platt, Magnuson-Stack, and Bankart repair procedures, the theory is that the overtightening of the anterior capsule causes eccentric load and wear on the posterior glenoid articular surface, ultimately resulting in an articulation imbalance and chondral degeneration.[43] Furthermore, the anterior capsular tightness with associated internal rotation contracture with posterior glenoid wear can further lead to posterior humeral head subluxation.[44] This posterior subluxation or even residual anterior instability can lead to postcapsulorraphy-related chondrosis. Thus, the balance of adequate postoperative joint mobility during capsular repair and retensioning the local soft tissue envelope for indicated instability procedures is of upmost importance and great care should be taken to limit the likelihood of overtensioning the repair to limit this potential complication and outcome.

OSTEONECROSIS

Osteonecrosis of the proximal humerus is the second most frequently involved site in the body, after the hip.[45] It can occur due to multiple etiologies, either atraumatic, which is the most common occurrence and is frequently secondary to systemic corticosteroids, or alcohol abuse, chemotherapy/radiation, hematopoietic diseases, and Caisson's disease.[46] The mechanism behind glucocorticosteroids-induced osteonecrosis is that there is an increase in marrow fat, which increases the intraosseous pressure and decreases the relative bone perfusion.[47] This process results in a change in local osteocyte apoptosis secondary to changes in local bone vascularity with diminishing hydrolytic support and the development of small fatigue fractures, which ultimately culminate in subchondral collapse at the most extreme cases.[47] This disease process can many times be symptomatically silent while there are minimal chondral and subchondral changes, given that the shoulder is not a weight-bearing joint, in contradistinction to the lower extremity. It is not uncommon for patients to become progressively symptomatic until there has been subchondral progression or even collapse with

resultant chondrosis/arthrosis. Corticosteroid-induced osteonecrosis of the humeral head was first detailed by Cruess in 1976 and is the most common atraumatic cause of osteonecrosis.[48–50] Although not as frequent as corticosteroid-induced osteonecrosis of the humeral head, osteonecrosis has also been reported in the setting of extraarticular proximal humerus fracture fixation. Despite there being no gross involvement of the glenohumeral articulation at the time of the trauma, the reported rates of osteonecrosis of the humeral head range between 0% and 34% after proximal humerus fractures, with 3- and 4-part fractures resulting in this greatest incidence, and has been reported at even higher rates with attempted fixation of 3- and 4-part fractures.[51,52] For many years, the anterolateral branch of the anterior humeral circumflex artery was thought to be the main blood supply to the humeral head; however, this finding did not correlate with the relatively low rates of osteonecrosis given the anterior humeral circumflex artery was disrupted in 80% of these fractures.[53] A more recent anatomic study has revealed that the posterior humeral circumflex artery is the predominant blood supply at 64% of the humeral head.[51] From a clinical and technical perspective, this anatomic finding is more in line with previous studies suggesting that the posteromedial hinge and the length of the posteromedial metadiaphyseal extension, where the posterior circumflex artery exists, are of strict importance in humeral head perfusion and that great care should be taken during posteromedial exposure and retractor placement or pin placement during operative fixation of these fractures.[54]

PAIN PUMP-MEDIATED CHONDROLYSIS

Local intraarticular pain pumps were developed as a perioperative adjunct given the increasing use of outpatient surgery and the need to limit the patient's exposure to narcotic use, owing to the potential for addiction and prescription of controlled substances. In the past, the patient would routinely manage the use of these devices postoperatively for 48 hours, normally when the most severe and acute pain is present immediately after surgery. Multiple studies show the chondrotoxic effects of bupivacaine on local chondrocytes when used intraarticularly both in vitro and in vivo.[55–57] Chondrocyte exposure to 0.5% bupivacaine in vitro resulted in 42% chondrocyte death and increased to 75% when the baseline articular surface was damaged.[55] For cases involving instability surgery, prior Hill-Sachs lesions could be the potential nidus of chondral damage that allowed for a more rapid progression of chondrolysis when pain pumps were used. The cytotoxic effects of decreasing chondrocyte density after even a single dose of bupivacaine has been reported; however, long-term outcomes have not been reported on an isolated dose in humans.[56] The use of pain pumps with a continuous infusion of bupivacaine has been suggested as a cause of PAGCL and has been reported in the literature.[40,58] With regard to PAGCL and pain pump use, the average age range reported in two of the largest reported cases series was between 19 and 30 years of age.[40,59,60] The radiographic changes and clinical presentation as detailed by Hansen and colleagues[40] routinely develop by 5 months postoperatively in the setting of prior intraarticular pain pump use. The rapid development of arthritis after this treatment modality leaves many patients with limited options for pain relief, further complicated by its devastating development in those that are young. Although complex biologic resurfacing salvage procedures are considered, arthroplasty options are many times the only remaining definitive treatment measures.[59]

RADIOFREQUENCY/THERMAL CAPSULORRAPHY

Radiofrequency ablation (RFA) device use in thermal capsulorraphy to "tighten up" or "shrink" the capsule in the treatment of shoulder instability has been fraught with

complications of capsule and nerve injuries as well as the possible contribution to PAGCL, where 33% of surgeries diagnosed with postoperative PAGCL involved RFA device use.[40,58,60,61] The hypothesis is that the increased temperatures created by the RFA device might in fact be a mechanism for chondrotoxicity, given that the purpose of the procedure is to denature the collagen in the capsule with untoward effects on the collagen present in articular cartilage. Initial studies suggesting the benefit of thermal capsulorraphy in the setting of shoulder instability were likely confounded by the fact that many of the treated patients also underwent Bankart repair at the same time.[62–65] Cadaveric as well as fluid chamber studies revealed the tip of the probe as the greatest focal point of temperature elevation and that, with continuous RFA use without arthroscopic fluid flow or lavage, temperatures can increase to as high as 80°C within the local arthroscopic surgical field.[66,67] The use of fluid pump flow rates from 50% to 100% of inflow capability has the ability to more readily cool the arthroscopic surgical field when using an RFA device and potentially protect the articular cartilage.[68] The occurrence of PAGCL has also occurred in patients where RFA was used in the setting of capsular releases for adhesive capsulitis and also extends to cases where RFA was used in cases without postoperative intraarticular pain pumps, further providing evidence that PAGCL is multifactorial, given the numerous factors reported in the development of this clinically relevant entity.[69,70]

INFLAMMATORY ARTHROPATHY

There is a paucity of reported literature detailing the influence of inflammatory arthropathies leading to truly early shoulder arthrosis in the young patient. In this patient demographic afflicted with this systemic disease, the term “young” is relative but important to discern from the age range of more traditional definitive treatment options, such as arthroplasty, for patients without an inflammatory arthropathy. In a prospective study to determine the potential predictors of shoulder joint destruction in patients with rheumatoid arthritis treated with biologics, the only variable that was important was the presence of synovitis on PET or MRI.[71] Given the periarticular synovitis, periarticular osteopenia, glenoid and humeral erosion, bone loss as well as rotator cuff degeneration, it is within reason to consider these intraarticular findings as part of a spectrum and process of joint inflammatory destruction.[72,73]

SUMMARY

The manifestation of early glenohumeral arthritis in the young adult is a devastating occurrence. This review article details the underlying etiologies and reviews their occurrence and contributions to the development of this clinical entity. The multifactorial nature of this disease process and the early age at presentation compounds the difficulty in the overall treatment of early shoulder arthritis in the young adult.

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