Idiopathic Glenohumeral Chondrolysis: A Case Report

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"GLENOHUMERAL CHONDROLYSIS HAS GAINED INTEREST IN THE PAST FEW YEARS AND HAS BEEN REPORTED IN MULTIPLE CASE REPORTS AND CASE SERIES AS A POTENTIAL POSTOPERATIVE COMPLICATION."

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Introduction
Chondrolysis is the disappearance of articular cartilage resulting from dissolution of the cartilage matrix and cells. It is accompanied by progressive loss of joint space and increased stiffness in the involved joint.[1,2] Chondrolysis has been documented in the hip, knee, ankle, and shoulder. The cause is often unknown. Recently, there have been a number of published reports of glenohumeral joint chondrolysis.[1,3-13] Although the etiology has been postulated to be multifactorial, associations with arthroscopy, pain pumps,[1,7,11] radiofrequency energy devices,[6,8] infection,[4,9] and suture anchors[3] have been documented. In the present article, we describe the first reported case of idiopathic glenohumeral chondrolysis not associated with any known risk factor.

Case Report
A 32-year-old man complained of shoulder pain and stiffness beginning in his early twenties. He reported no injury or traumatic event. His primary care physician treated him with 3 steroid injections over the course of 4 years. The exact location of the injections, the drug type and dose, and the timing between injections are unknown. With worsening pain, he was evaluated by an orthopedic surgeon approximately 7 years after the onset. He complained of anterior shoulder pain and also had feelings of shoulder instability. His range of motion was 80° of forward flexion, 70° of abduction, and 45° of external rotation. He did not have a history of severe acne or other known sources of potential infection. Radiographs demonstrated a concentrically located glenohumeral joint with a well-preserved joint space and a normal acromiohumeral index. Magnetic resonance imaging revealed osteochondritic changes of the humeral head with bony erosions and synovitis of the glenohumeral joint, a partial-thickness tear of the supraspinatus tendon, and fraying of the superior glenoid labrum. Laboratory evaluation included complete blood count (white blood cell [WBC] count 9.7), C-reactive protein (0.13), rheumatoid factor (<4), and antinucleotide antibody (<80), all within normal limits.

His shoulder pain required chronic pain management with narcotic analgesia. His local orthopedic surgeon examined him under anesthesia and found no instability but significant tightness. His passive range of motion was 90° of forward flexion, 90° of abduction, 30° of external rotation, and 25° of internal rotation. Diagnostic arthroscopy revealed a global chondrolysis with a 1.0 cm × 1.5 cm × 3.5 cm area of full-thickness cartilage defect, loose

bodies, synovitis, and degenerative fraying of the superior labrum and long head of the biceps (Figure 1). He underwent global capsular release, debridement of the superior labrum and long head of the biceps, chondroplasty, extensive synovectomy, loose body removal, subacromial decompression, and distal clavicle excision. A pain pump was inserted at the conclusion of the surgery.

One month after surgery, his forward flexion had increased to 150°, abduction to 155°, internal rotation to 60°, and external rotation to 55°. His pain dramatically decreased and only affected him at night and during physical therapy. Seventeen months after the initial surgery, he had another surgery performed by the same orthopedic surgeon because of continued activity-related pain. Arthroscopic evaluation demonstrated progressive glenohumeral joint chondrolysis, synovitis, and a thickened subacromial bursitis. The operative procedure was chondroplasty of the glenohumeral joint, extensive synovectomy, and subacromial bursectomy. After the second surgery, he experienced persistent pain and diminished range of motion.

Subsequently, he consulted the senior author (B.J.C.). He described "a sensation that there is always a knife in my shoulder." On exam, he had forward flexion to 60°, abduction to 40°, external rotation to 10°, and internal rotation to the buttock. Radiographs confirmed joint space narrowing without evidence of sclerosis or osteophytes (Figure 2). His activities of daily living were severely restricted, and he was taking 80 mg of OxyContin up to 10 times a day for pain relief. Repeat steroid injection did not improve symptoms.

The patient underwent shoulder hemiarthroplasty and biceps tenodesis. The glenoid was pristine and did not require a glenoid component. The thickened and flattened biceps tendon was released and tenodesed distally (Figure 3). Three months after the last surgery, he had an improved range of motion with 140° forward flexion, 140° abduction, and 60° external rotation and described minimal pain, occurring only at night. He was no longer taking pain medication. At the time of preparation of this report, 20 months after his last surgery, we have been unable, in spite of multiple attempts, to locate him in order to document his current status.

Discussion

The present report is the only case in the published literature of idiopathic glenohumeral chondrolysis. The patient presented with insidious onset of progressive shoulder pain and diminished global range of motion refractory to nonoperative treatment. At the initial presentation, the magnetic resonance imaging study demonstrated glenohumeral joint chondrolysis and synovitis. The patient also underwent laboratory evaluation for infectious or inflammatory etiology for his shoulder pathology, but these studies were unremarkable. Although the patient had 3 steroid injections early in his nonoperative management, single injections (as opposed to continuous infusion pain pumps) of Marcaine or lidocaine have not demonstrated chondrotoxicity.[14] The initial arthroscopic inspection demonstrated dramatic glenohumeral joint chondrolysis, and an indwelling pain pump was inserted following the procedure. The second arthroscopy also demonstrated severe glenohumeral joint chondrolysis.

Glenohumeral chondrolysis has gained interest in the past few years and has been reported in multiple case reports and case series as a potential postoperative complication. Although causes of postoperative chondrolysis have not been identified definitely, potential associated factors include thermal treatment,[6,8] continuous infusion of local anesthetics,[1,7,11] infection with *Propionibacterium acnes*,[4,9] high arthroscopic irrigation fluid temperatures,[5,10,15] injection of gentian violet,[12] anchor loosening and subsequent trauma,[3] and iatrogenic injury.

Postoperative shoulder chondrolysis is a rare but devastating complication. Patients are usually young, presenting with an un-

Figure 1. Arthroscopic images from the patient's initial surgery, displaying chondrolysis of the humeral head.

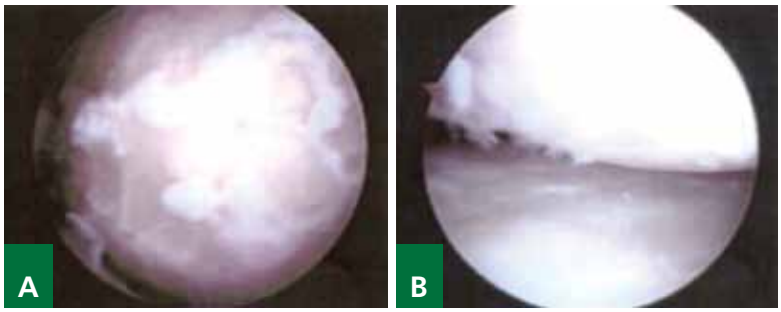

Figure 2. Preoperative anteroposterior and axillary radiographs prior to evaluation for hemiarthroplasty. The patient has joint space narrowing but does not display sclerosis or osteophytes.

Figure 3. Postoperative anteroposterior and axillary radiographs.

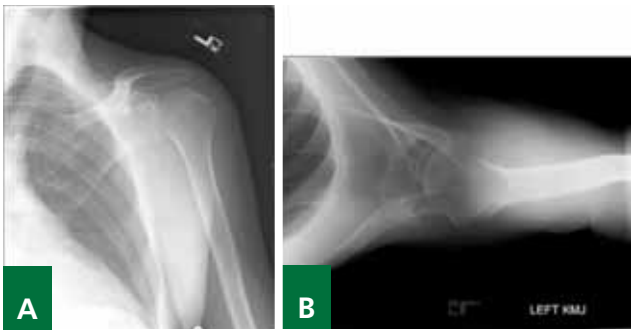

Figure 2

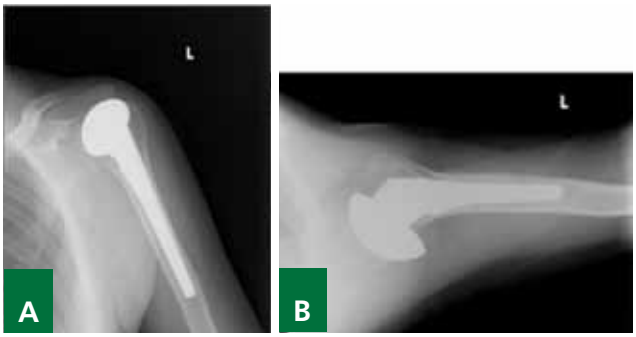

Figure 3

eventful postoperative course followed by rapid onset of shoulder pain at 6-12 months after the index surgery.[1,7] There have been no reliable treatments once glenohumeral chondrolysis is diagnosed. Bailie and Ellenbecker report on 23 cases of shoulder chondrolysis that were treated with oral and intra-articular steroids, nonsteroidal anti-inflammatory drugs, debridement, and hyaluronic acid injections.[1] Nine patients of 23 underwent shoulder arthroplasty. In a series of 20 patients with glenohumeral chondrolysis, patients were treated with a variety of biologic procedures, including microfracture, autologous chondrocyte implantation, allografts of the humeral head, concomitant humeral head allograft and lateral meniscal interposition, and capsular release.[11] In both case series, patients demonstrated improvement in the short term.

Chondrolysis has been described in multiple joints, including the knee, the ankle, and most commonly the hip (Table 1). Chondrolysis of the hip is well documented, with causes including sequelae of untreated slipped capital femoral epiphysis (SCFE),[13,22-24] penetration of the articular surface by pins during surgical treatment,[25] extended immobilization, exposure to methacrylate,[26] and septic arthritis. Idiopathic chondrolysis of the hip (ICH) is characterized by a rapid course of progressive chondrolysis that commonly occurs in adolescents.[27] ICH presents as pain and stiffness in the joint, with loss of articular space. Eisenstein and Rothschild suggest that chondrolysis is linked with an immune abnormality that makes the cartilage susceptible to articular cartilage damage.[28] Adib et al, in a case series of children presenting with painful stiff joints, discuss 14 patients with chronic hip arthritis in which juvenile idiopathic arthritis (JIA), septic hip, and reactive arthritis had been ruled out.[29] The authors suggest that the patients' arthritis is a result of chronic inflammatory arthritis and may even represent a separate subtype of JIA.

Regardless of the cause, chondrolysis of the hip in young patients is difficult to treat. Korula et al present a case series of patients (average age, 13 years) with idiopathic chondrolysis of the hip.[23] Patients were treated with capsulectomy, and the results report a less-than-satisfactory outcome for patients. Carney et al found chondrolysis in 16% of patients with SCFE, and most patients had poor outcomes.[22]

Chondrolysis of the knee, although uncommon, has been described following meniscectomy.[16,17] Charrois et al state that knee chondrolysis of the lateral compartment had been reported in young athletes following meniscectomy.[17] Alford et al present two cases of severe chondral damage within 1 year of meniscectomy.[16] The rapid presentation of chondrolysis in these cases suggests a cause other than mechanical wear. Furthermore, knee chondrolysis has been associated with radiofrequency procedures,[18] exposure to chlorhexadine,[19] and physical and surgical trauma.[20]

In a case report by Bojescul et al,[2] the authors report a case of idiopathic ankle chondrolysis. The patient presented with chronic (5 years) lateral ankle instability, and arthroscopic findings included moderate synovitis, grade II anterolateral chondrolysis, and an anterior talar osteophyte. Following reconstruction of the ligament, the patient reported stiffness and pain at 11 months postoperatively and had radiographic evidence of chondrolysis. Of note, this patient had a pain pump after the first scope.

Conclusions

We present the case of a young patient with long-standing shoulder pain and stiffness. Our patient had none of the factors reported as possible etiologies in cases of chondrolysis of the glenohumeral and other joints. He had had 3 intra-articular steroid injections prior to the diagnosis of chondrolysis, leading us to consider whether some

Table 1. Summary of Described Chondrolysis Etiologies

Affected Joint	Etiology
Knee	Following meniscectomy[16,17] Radiofrequency procedures[18] Exposure to chlorhexadine[19] Physical and surgical trauma[20] Idiopathic[21]
Shoulder (glenohumeral)	Thermal treatment[6,8] Intra-articular pain pumps[1,7,11] Infection[4,9] High-temperature irrigation fluid[5,10,15] Gentian violet[12] Anchor loosening and subsequent trauma[3]
Hip	Untreated slipped capital femoral epiphysis (SCFE)[13,22-24] Incorrect pin placement[25] Extended immobilization Exposure to methacrylate[26] Septic arthritis Idiopathic[23,27] Immune abnormality[28]

idiosyncratic reaction to the injected material or unrecognized infection from the injections could have occurred and caused the chondrolysis. However, he had symptoms prior to the injections, the materials injected were short-acting, shoulder joint injections are exceedingly common and not known to be associated with chondrolysis, laboratory testing showed no evidence of infection, and the pristine condition of the glenoid cartilage found at the last surgery suggested a pathologic process originating in the humeral head as opposed to the joint space. For all these reasons, we concluded that, though the possibility of a relationship between the injections and the chondrolysis could not be eliminated, it is probable that there was no causal relationship, and therefore the etiology, in this case, is best considered idiopathic. ■

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