

# Postarthroscopic Glenohumeral Chondrolysis of the Shoulder

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**Abstract:** Postarthroscopic glenohumeral chondrolysis is a devastating, poorly understood, and relatively rare complication. True chondrolysis involves the dissolution of articular cartilage, including the matrix and cellular elements, leading to premature and irreversible articular cartilage loss. Several factors have been implicated in this phenomenon; however, to date, no study has conclusively ascertained the causation. Potential causative agents include subclinical infection, high volume intra-articular infusion of certain anesthetics, arthroscopic implants, suture material, and thermal energy. One must also consider the possibility that chondrolysis represents an ongoing immunogenic process interrupted or possibly potentiated by surgical intervention. The complex homeostasis of articular cartilage is undoubtedly sensitive to agents introduced into the joint including mechanical, chemical, and temperature-dependent interventions. To date, several papers have described the phenomenon and the potential associations; however, there is no definitive answer although the use of high-dose bupivacaine as an intra-articular anesthetic seems to be contraindicated. The purpose of this article is to review the basic science regarding chondrolysis and to assess the current literature which focuses on postarthroscopic glenohumeral chondrolysis, as well as innovative treatment alternatives. It is unlikely that postoperative chondrolysis will be clearly understood until controlled studies are available, of which there are currently none.

**Key Words:** chondrolysis, shoulder, arthroscopy, cartilage

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Several studies have reported on the catastrophic phenomenon known as postarthroscopic glenohumeral chondrolysis (PAGCL), primarily involving younger patients undergoing stabilization procedures.<sup>1-7</sup> True chondrolysis involves the dissolution of articular cartilage, including the matrix and cellular elements, leading to premature and irreversible articular cartilage loss. Several factors have been implicated in this phenomenon; however, to date, no study has conclusively ascertained the causation. Potential causative agents include subclinical infection, high volume intra-articular infusion of certain anesthetics, arthroscopic implants, suture material, and thermal energy. One must also consider the possibility that chondrolysis represents an ongoing immunogenic process interrupted or possibly potentiated by surgical intervention.

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## ARTICULAR CARTILAGE PHYSIOLOGY

A unique tissue, articular cartilage allows for nearly frictionless mechanical motion of the glenohumeral joint,<sup>8</sup> and is composed of hyaline cartilage which covers the articulating surfaces.<sup>9</sup> Five percent of articular cartilage is made up of chondrocytes, which develop from chondroblasts derived from mesenchymal cells. These chondrocytes are crucial in controlling the turnover of the other 95% of cartilage, the extracellular matrix, through the production of collagen, proteoglycans, and enzymes. The extracellular matrix of cartilage is composed of water (75%), proteoglycans (20%), and collagen (mainly type II), other enzymes, growth factors, lipids, and adhesives making up the remaining 5%.<sup>8</sup>

In terms of the actual structure of cartilage, there are 4 zones.<sup>9</sup> The surface or superficial tangential zone is composed primarily of type II collagen, oriented tangentially to the surface, and is able to resist shearing forces. The transitional/middle zone is obliquely arranged, and made up of almost entirely proteoglycans, and the radial/deep zone distributes the mechanical load and resists compression. The deepest layer, otherwise known as the calcified zone has mainly type X collagen, and contains a basophilic demarcation between the calcified and uncalcified cartilage.<sup>9</sup>

Mature articular cartilage has no blood supply, innervation, or lymphatic system. Movement of fluid, nutrients, and oxygen occurs via diffusion from the synovial fluid, and net flow is determined by the normal weight bearing function of the synovial joints.<sup>8</sup> As the cartilage ages, chondrocytes increase in size while proteoglycans are reduced in size and mass. Cartilage water content also decreases with age, but increases in cases of osteoarthritis. When the hyaline cartilage of the body calcifies, chondrocyte death occurs, and the matrix disintegrates.<sup>8</sup>

With respect to the molecular pathway behind chondrocyte destruction, Ding et al<sup>10</sup> have shown that in bovine chondrocytes, proteolytic cleavage of fibronectin, and the creation of fibronectin fragments which have chondrolytic activities, leads to the elevation of matrix metalloproteinase expression which has been implicated in cartilage damage. Decreased matrix metalloproteinase upregulation subsequently leads to a decrease in fibronectin fragment injury to the cartilage.

In 2008 Chu et al<sup>11</sup> demonstrated the cytotoxic effects of bupivacaine on chondrocytes and showed the toxicity is both dose and pH dependent. The authors proved that, in vitro, 0.5% and 0.25% bupivacaine solutions were toxic to both human and bovine articular chondrocytes. The chondrotoxicity of 0.25% bupivacaine increased with longer duration of exposure and time after exposure, and cellular death occurred faster with 0.5% than with 0.25% bupivacaine for both human and bovine chondrocytes.

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For human chondrocytes, exposure to 0.5% bupivacaine solutions resulted in cell viability of 41% after 15 minutes, 4% after 30 minutes, and no living chondrocytes after 60 minutes. However, at a lower dose, 0.125% bupivacaine, the authors found that there was no significant difference in the viability of the chondrocytes compared to the saline-exposed controls. Because of this Chu et al postulated that human chondrocytes may be able to tolerate exposure to 0.125% bupivacaine for up to 1 hour.

Instead of confirming the detrimental effect of bupivacaine on chondrocytes, in 2009 when Gomoll et al<sup>12</sup> studied the effect of intra-articular bupivacaine infusion on a rabbit shoulder model, they found there was no permanent impairment of cartilage function at 3 months. Thirty-six rabbits were randomized into 3 groups and infused for 48 hours with saline, bupivacaine, or bupivacaine plus epinephrine. After 3 months, the rabbits were sacrificed, and their tissue samples evaluated. Rather than observe chondrocyte death, the authors found an increase in chondrocyte anabolic activity, which they speculated to be an indication of a reparative reaction to the earlier toxic stimulus. The authors hypothesized that for their experimental model, the cartilage was only transiently impaired by the use of bupivacaine; however, they extrapolated from these findings the possible need for additional noxious stimuli in addition to the bupivacaine to cause permanent damage.

Similar investigative work by Good et al<sup>13</sup> shed light on the effects of thermal energy on cartilage injury. In 2009, they conducted a study to assess the effect of thermal energy on glenohumeral joint fluid temperature during an arthroscopic procedure. The authors measured the temperature at 4 locations within 10 cadaver shoulder joints with respect to method of heating, fluid pump flow rate, and the location of the radiofrequency probe. In all the trials, the joint fluid was increased to greater than 45°C at some point in time during the experiment. The authors concluded that the use of a thermal probe during arthroscopy may cause joint fluid temperatures to reach high enough levels that chondrolysis can occur, and that irrigation fluid flow is critical for maintaining lower joint fluid temperatures.

Like Good et al, Zoric et al<sup>14</sup> also explored the effects of radiofrequency devices on intra-articular fluid temperatures. Using 10 human cadaver shoulders, the intra-articular temperature was measured at 0, 5, 10, 20, 30, 40, 50, 60, 90, and 120 seconds of ablation time at distances of 1, 3, 5, and 10 mm away from the device. Temperatures above 50°C were seen after a longer duration of use, a decreased distance between the thermometer and probe and a decreased irrigation fluid flow rate. The authors noted that the flow rate was the most significant predictor of intra-articular temperatures. They concluded, like Good et al, that management of irrigation fluid flow rate across the joint during arthroscopic procedures is especially important, and that even short periods of low flow could result in cartilage damage.

In summary, given the fragile nature of articular cartilage and its lack of healing potential, injuries can result from repetitive trauma, a single major traumatic episode, metabolic or systemic illnesses, autoimmune disease, iatrogenic disruption, or from one or more elements which can be introduced during arthroscopic surgery, including infusion of anesthetic agents, unintended heat generation, and improper anchor or suture placement.

## CLINICAL STUDIES: A REVIEW OF THE LITERATURE

Current techniques and protocols employed during arthroscopic shoulder surgery often obscure the potential causes of chondrolysis in that unintended articular scuffing may occur in conjunction with the use of intra-articular anesthesia combined with implant usage and, in the past, associated thermal energy. Under these circumstances, with multiple uncontrolled variables, the ability to identify a single causative agent becomes extremely difficult, if not impossible. It may be that one factor potentiates the deleterious effect of another, yet this, too, is very difficult to ascertain and continues to underline the limitations present in the current published literature. With that said a review of the current literature follows.

Solomon et al<sup>15</sup> conducted a systematic literature review to identify factors associated with the development of PAGCL. The authors concluded that the most frequently cited factors correlated with this issue were: (1) direct surgical insults to cartilage owing to radiofrequency and/or thermal devices, (2) proud anchor implants and suture knots on the articular surface, and (3) exposure to high concentrations of anesthetics administered via intra-articular pain pumps. They speculated that the causal pathways leading to PAGCL involve primary and secondary cartilage injury that occurs owing to mechanical, chemical, or thermal events, with the consequence of such an event being an inflammatory response. The authors discussed how this inflammation leads to eventual chondrocyte apoptosis and disturbance of cellular metabolism, with the increased friction and accelerated wear resulting in PAGCL. From their literature search, the authors identified 6 studies in which the presence of pain pumps containing bupivacaine or lidocaine had been associated with the development of chondrolysis, and based on these published case reports concluded that 67% of patients with PAGCL used a postoperative pain pump.

With regard to intra-articular pain pumps, and in support of Solomon et al's findings, there are several clinical reports indicating the possible detrimental effects of postoperative infusion of anesthetics. In 2008 Greis et al<sup>16</sup> described 2 individuals who underwent bilateral arthroscopic shoulder procedures and subsequently developed severe chondrolysis in both shoulders. Thermal devices were not used during surgery; however, the 2 patients were treated postoperatively with a continuous intra-articular infusion of 0.5% bupivacaine without epinephrine at a rate of 4 mL/h for 48 hours. The authors speculated that placement of the catheter in the intra-articular position exposed the cartilage to a higher volume and concentration of bupivacaine compared to pain pump placement in the subacromial space. They also reasoned that the use of an intra-articular pain pump with continuous bupivacaine infusion in these 2 cases may have been the inciting event for chondrolysis, as other causes, such as infection or thermal devices, did not play a role. Because of this Greis et al concluded that the use of continuous infusion of intra-articular bupivacaine may be contraindicated after arthroscopy.

In 2009 Bailie and Ellenbecker<sup>4</sup> reported on the harmful effect of the continuous intra-articular infusion of anesthetics. The authors described a series of 23 cases of postarthroscopic chondrolysis discovered over a 2 years period. Fourteen of these cases involved a labral repair with bioabsorbable fixation devices, whereas 7 patients had undergone thermal treatment for capsular laxity. For all 23

cases, the degree of chondrolysis was determined by both radiographs and magnetic resonance imaging (MRI) scans. Seventeen of the 23 patients had an intra-articular pump inserted postoperatively, with administration of 250 to 300 mL of 0.25% bupivacaine over a period of 48 hours. Although their analysis revealed no single factor as causative, the authors expressed concern over the routine use of intra-articular bupivacaine. It is noteworthy that after the discovery of cartilage damage, 9 patients underwent humeral head resurfacing whereas 11 had an arthroscopic debridement and capsular release. Operative findings included both humeral head and glenoid articular cartilage loss whereas no case revealed mechanical abrasion from an anchor or the pain pump catheter itself.

Hansen et al<sup>3</sup> reviewed the cases of 12 shoulders that developed PAGCL. The authors compared these cases with 165 arthroscopic shoulder surgeries performed during the same period of time. Of the common factors identified in the shoulders demonstrating chondrolysis, a high flow intra-articular pain pump was the only new addition to the author's procedure. Of the 177 shoulders, 19 patients had intra-articular pain pump catheters consisting of 0.25% bupivacaine and epinephrine infused at a rate of 4.16 mL/h for 48 hours. Twelve of the 19 patients eventually developed chondrolysis. Thermal radiofrequency was used in 4 of the cases and loose metallic hardware occurred in 1 patient; however, the authors believed that neither of these issues was the initiating factor for the chondrolysis. It should be noted that most patients' initial symptoms of pain and progressive motion loss occurred 3 to 5 months postoperatively, yet the authors suspected that the damage to the articular cartilage occurred during the intraoperative and perioperative period. They speculated that the delay in onset was due to the immediate postoperative protected mobilization and lack of significant compression forces on the joint.

In contrast to the studies discussed above, Rapley et al<sup>16</sup> found chondrolysis to occur in only 16% of their patients receiving anesthetic infusion postarthroscopy. The authors divided 65 patients into 2 groups: group 1 received 100 mL of 0.5% bupivacaine at an infusion rate of 2.08 mL/h and group 2 received 270 mL of 0.5% bupivacaine infused at a rate of 4.16 mL/h. Twenty-nine of these patients had glenohumeral catheters (13 from group 1 and 16 from group 2), and the rest had subacromial pain pumps (19 from group 1 and 17 from group 2). Those with subacromial catheters scored better on the postoperative Constant, American Shoulder and Elbow Surgeons (ASES), Rowe, Single Assessment Numeric Evaluation, and Simple Shoulder Test (SST) scores than the patients who used a glenohumeral pump. Only 3 of the glenohumeral catheter patients were diagnosed with chondrolysis, and they had all received the higher infusion rate of the anesthetic. All of the clinical symptoms and radiographic evidence of chondrolysis occurred within a year of the surgery. Patients using a subacromial catheter and/or patients with bupivacaine infusion 2.08 mL/h for 48 hours did not develop cartilage toxicity. In accordance with Rapley et al, Busfield and Romero determined that in contrast to intra-articular pain pumps, subacromial pain pumps were associated with lower complication rates and have been shown to be effective without resulting in PAGCL.

Both suboptimal anchor placement and certain implant types have been shown to be factors in cartilage injury. In a study conducted by McNickle et al,<sup>7</sup> not only

were intra-articular pain pumps found to cause chondrolysis in some patients, but previous hardware placement was also a factor. The authors collected data on 20 patients over a 5-years period, all referred to their practice presenting with PAGCL. Each patient met the following inclusion criteria: (1) age below 35 years, (2) previous glenohumeral arthroscopy with capsular or labral procedure, (3) intact glenohumeral cartilage at the index procedure, and (4) ongoing postoperative symptoms of shoulder pain. It was discovered that after the initial procedure, a postoperative intra-articular glenohumeral pain pump was used in 16 of the patients. Furthermore, they reported that 2 of the patients with metal anchors placed along the anterior glenoid for a Bankart repair had to have the hardware removed owing to prominence. From their findings, the authors postulated that glenohumeral pain pumps and hardware complications, such as suboptimal anchor placement, could contribute to severe cartilage damage. It should be noted that in this study, the most common form of treatment for the chondrolysis was a humeral head allograft and lateral meniscus interposition.

The study conducted by Athwal et al<sup>17</sup> describing 4 cases of osteolysis after the use of bioabsorbable knotless suture anchors showed that this implant design could be a causative agent in PAGCL. In this study, bioknotless anchors were implanted in 25 shoulders for repair of a superior labrum anterior posterior lesion and/or Bankart lesion; in 4 of these patients the bioknotless device failed and led to glenoid osteolysis, anchor pull-out, and consequently, severe damage to the articular cartilage. The authors speculated that the osteolysis may have occurred secondarily to micromotion after the anchors lost their initial stability rather than from a chemical degradation process. They listed the factors leading to anchor instability as (1) the number of anchors used (5 in each patient), (2) the knotless concept, and (3) the anchor design. Based on their study, the authors recommended against using bioknotless-type suture anchors.

Thermal devices have also been implicated as a possible initiator of cartilage toxicity. In one of the earliest reviews of the literature published in 2004 Petty et al<sup>1</sup> described in their study glenohumeral chondrolysis in 3 cases occurring in the young throwing athlete. In 2 of the shoulders, a radiofrequency device was used along with a debridement. In the third shoulder, a 0.5% bupivacaine with epinephrine glenohumeral pain pump was used after a rotator cuff debridement and subacromial decompression (it was implied but not specified, that the placement of catheter was intra-articular). For all 3 cases, a repeat arthroscopy with limited debridement of the labrum and synovium was performed to confirm the development of glenohumeral chondrolysis. Physical therapy and nonsteroidal anti-inflammatory drugs were recommended, and all the 3 patients were allowed to return to their sports as tolerated. The authors concluded that there was very little published or known about the process underlying chondrolysis; however, they recommended avoiding the use of thermal energy in the glenohumeral joint of young athletes.

In 2007 Good et al<sup>2</sup> retrospectively reviewed 8 cases in which glenohumeral chondrolysis occurred postshoulder arthroscopy in which thermal energy was used. The authors speculated that the heating of joint fluid contributed to cartilage death. None of the patients used a postoperative pain pump, and each patient eventually demonstrated radiographic evidence of chondrolysis. Repeat arthroscopy was performed to confirm the diagnosis. More recently, in

2009, Busfield and Romero<sup>5</sup> discussed the role of radio-frequency thermal instruments and postoperative intra-articular pain pumps in cartilage damage. Based on their review of the literature, they recommended that thermal capsulorrhaphy should be used cautiously, and intra-articular pain pumps should be avoided.

As one can see from this extensive literature review, there are obviously multiple factors involved in the development of chondrolysis. Although thermal energy, infusion of local anesthetic, anchors (both permanent and absorbable), sutures of many kinds, subclinical infection and early reinjury have been associated with its development, there is no one clear factor that produces chondrolysis. The occurrence, while tragic, cannot be blamed on an isolated factor, but most likely represents a combination of patient disease and multiple sensitivities to these various entities. This disorder is the current focus of much research into etiology and prevention.

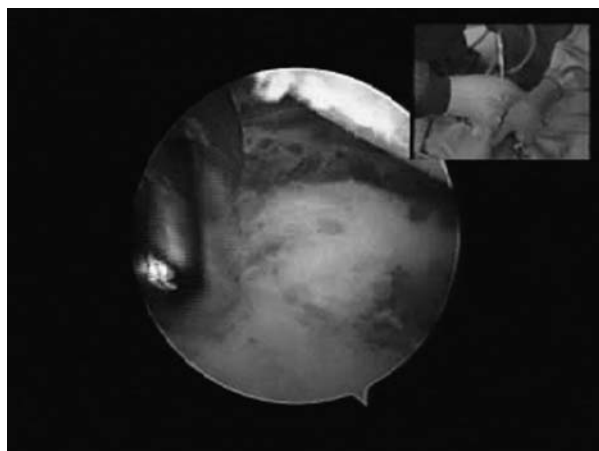
### Prevention (author's opinion, not supported by fact)

Although there is no definitive evidence linking one particular device or treatment to the etiology of chondrolysis, the severity of the disorder in the young patient warrants as much concern as possible to minimize the risk of it developing. We currently recommend avoidance of intra-articular pain pumps, thermal devices, and nonabsorbable anchors shaped in a way that would allow the most proximal part to erode out of the subchondral bone. Patients should be questioned as to sensitivity to metal and plastic, and if present, these materials should be avoided. Surgical site preparation should include topical agents effective against *P. Acnes* in addition to the usual antistaph and antistrep preparations. Anchors placed into the glenoid should be placed on the corner of the neck-face junction and buried more deeply than most guides allow, preventing them from becoming prominent. Suture techniques should be used that avoid having the suture or the knot placed between the soft tissue and the humerus. If absorbable anchors are used, the soft tissue should be mobilized to completely cover the hole in the subchondral bone created when placing the anchor.

### TREATMENT ALTERNATIVES

Once PAGCL occurs in patients, routine treatment options are often inadequate. Early arthroscopic debridement and capsular release may provide a temporizing strategy and modest pain relief while decreasing some of the acute inflammatory changes (Fig. 1). The off-label use of hyaluronic acid compounds may also provide some temporary relief. The choice and timing of the proper definitive surgery remains an issue in the young patient population. Currently available options include humeral or glenoid resurfacing, humeral hemiarthroplasty, total shoulder arthroplasty, and arthrodesis.

A diagnosis of glenohumeral chondrolysis should be considered if excessive pain and/or pain out of proportion to expectations are present. Radiographs should be obtained, and if a decrease in the preoperative joint space or cystic changes in the glenoid or humerus are noticed, then chondrolysis should be suspected (Figs. 2, 3). An MRI and a joint aspirate for culture should be obtained. The MRI should be reviewed for the presence and amount and degree of cyst formation in the glenoid (Fig. 4) and for



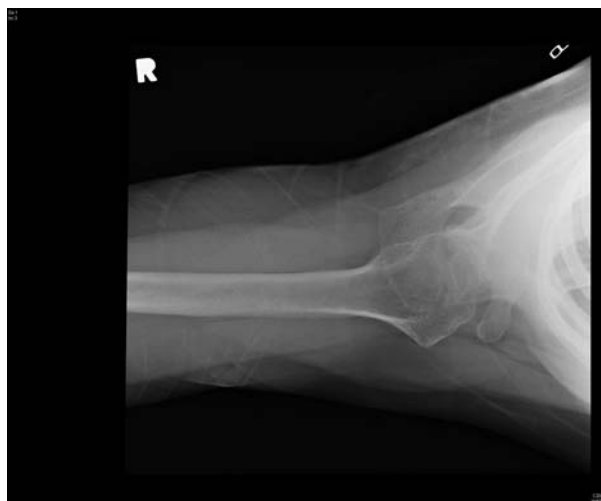
**FIGURE 1.** The arthroscopic view of early chondrolysis is one of massive destruction of the articular cartilage with diffuse inflammation and capsular damage.

deformity and evidence of avascular necrosis in the humerus (Fig. 5). A conservative treatment program should be started, including anti-inflammatory modalities and therapy, with a focus on maintenance of range of motion and an attempt to decrease the load crossing the affected glenohumeral joint.<sup>1</sup> However, there are no studies that clearly show the benefit of these interventions for PAGCL.<sup>15</sup> In most cases, the disease will progress and further intervention is required.

Resurfacing of the humeral head (Copeland-type procedure) (Fig. 6), biologic glenoid resurfacing (Fig. 7), humeral hemiarthroplasty, and total shoulder arthroplasty have all been described as treatment alternatives for severe chondrolysis. McNickle et al<sup>7</sup> described resurfacing using a humeral head allograft and lateral meniscus interposition. Thirteen of the 20 individuals who underwent biological resurfacing had little change in mean range of motion (from 132 to 133 degrees of forward flexion and from 42 to 41



**FIGURE 2.** A standard posteroanterior view of the shoulder may be supplemented by one in which the patient attempts to abduct the arm, revealing contact between the humeral head and the glenoid.



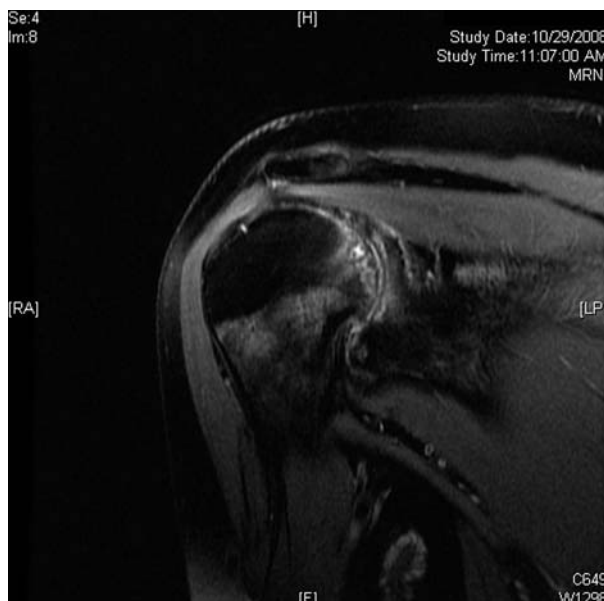
**FIGURE 3.** The axillary lateral view may be difficult to obtain but can provide valuable evidence of the shape of the humeral head and the need for humeral resurfacing or replacement. Note the anterior and posterior “squaring” seen on this view.

degrees of external rotation at the side). However, 8 patients who completed surveys postoperatively experienced mean outcome score improvement on the SST (from 7 to 10) and ASES scale (from 51 to 71), with a decrease on the visual analog scale for pain (from 5 to 3).

Savoie et al<sup>18</sup> described their experience using an arthroscopic interposition resurfacing of the glenoid with the Restore patch (DePuy Orthopaedics, Raynham, MA), a pluripotent cell construct with the purpose of providing normal native tissue in growth into damaged surfaces. Twenty-three consecutive patients, ranging from ages 15 to 58 years (mean age 32 y), underwent this treatment. The

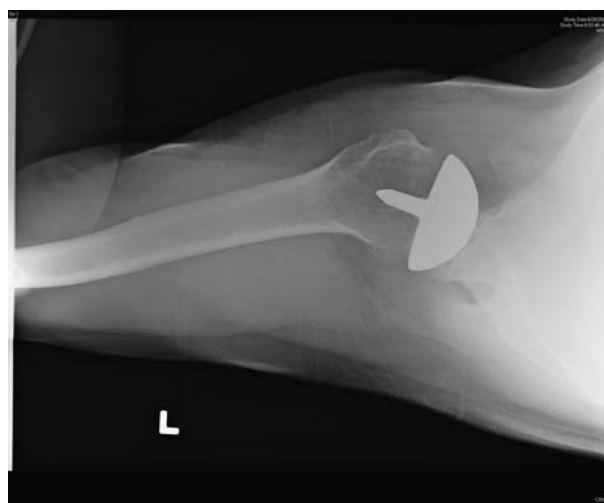


**FIGURE 4.** The magnetic resonance imaging scan will reveal the degree of cyst formation within the glenoid, detailing whether evacuation or resurfacing (or both) of the glenoid will be necessary.

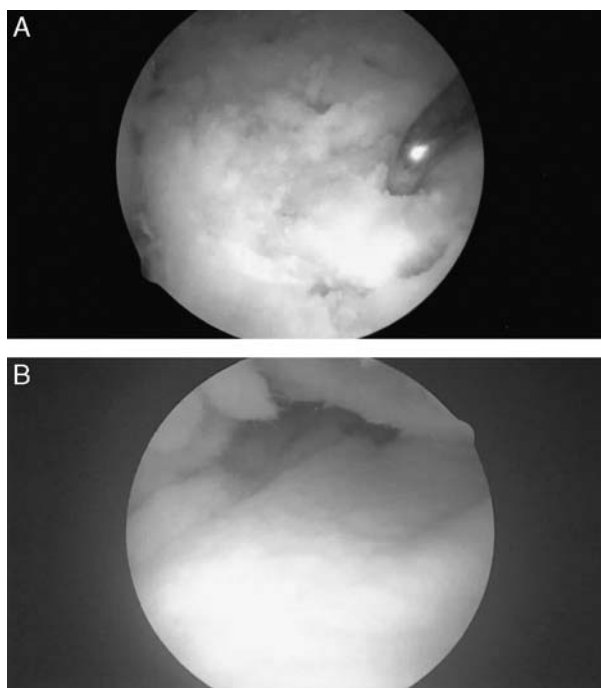


**FIGURE 5.** The magnetic resonance imaging may also show changes in the humeral head similar to those seen in avascular necrosis with or without collapse, predicting whether humeral replacement with biologic or metal devices may be required.

initial results demonstrated marked improvement both clinically and radiographically with bone sparing (Fig. 2). Seventy-five percent of patients reported a satisfactory result, and there was a statistically significant improvement between the pre- and postoperative shoulder measurements. The study included postchondrolysis patients along with post-traumatic and degenerative patients. There was no statistical difference in the results between the 3 groups, although the authors noted that the chondrolysis patients tended to have greater improvements for each of the study's measurements postoperatively. The success of the procedure was confirmed by similar studies by DeBeer et al,<sup>19</sup> using a human skin



**FIGURE 6.** Humeral resurfacing may provide satisfactory results when deformity occurs. However, this must be combined with biologic glenoid resurfacing or cyst decompression when there are cysts present in the glenoid.



**FIGURE 7.** A, This superior view of the shoulder demonstrates microfracture of the glenoid after reaming in preparation for resurfacing. B, Biologic glenoid resurfacing over the decompressed cysts often is a temporizing technique to “buy time” by decreasing pain and improving motion in these young patients.

patch. Burkhead et al<sup>20</sup> have reported satisfactory results with biologic glenoid resurfacing in a variety of patient groups, whereas Warner<sup>21</sup> has reported less than satisfactory results with biologic glenoid resurfacing for osteoarthritis. It is apparent in reviewing multiple studies that, similar to its etiology, the treatment of PAGHC involves multiple factors and no single treatment is perfect. In the author's opinion, the presence of cysts within the glenoid indicates the need for pressure relief resurfacing of the glenoid. In these young patients a biologic covering is the most logical first step in the management of these patients.

Levy et al<sup>22</sup> retrospectively reviewed 11 patients (average age 39 y) treated with total shoulder arthroplasty after chondrolysis associated with shoulder arthroscopy. Six of the patients had undergone arthroscopic Bankart repair, 2 had a superior labrum anterior posterior repair, 2 had an arthroscopic release, and 1 underwent an arthroscopic thermal capsulorrhaphy. Nine of the 11 patients had indwelling pain catheters in the joint postoperatively; however, the anesthetic agent was not specified in operative notes. Posttotal shoulder arthroplasty, 10 of the 11 patients rated their outcome as good or excellent, and there were statistically significant improvements in shoulder abduction (89 to 123 degrees), external rotation (26 to 48 degrees), and total ASES scores (30 to 77), and SST scores (3 to 8).

### Author's Preferred Surgical Treatment

In the chondrolysis patient, one must determine the side of the joint most affected. In cases in which there are large glenoid cysts, the glenoid must be resurfaced or reamed and the cysts evacuated. In patients in whom this is the primary area affected, the procedure is carried out arthroscopically. The lateral decubitus position is used

most commonly. Three portals are used: anterior superior, anterior inferior, and posterior. The glenoid is reamed to a smooth surface from the posterior portal and the cysts unroofed and evacuated. The glenoid is then microfractured (and sometimes drilled) to improve chances of healing and decrease inflammation. Anterior inferior and anterior superior anchors are placed. If the posterior labrum is hypertrophied, it can be used as an anchor, otherwise posterior superior and posterior inferior anchors are placed as well. The sutures are placed through the graft outside the shoulder and the graft is pulled into the joint and flattened. The sutures are then tied in sequence. Specific tips from the author are to place the sutures in the edge of the graft securely to prevent pull through, be attentive to suture management, and correctly size the graft using the measuring technique of DeBeer.

In cases in which there is humeral deformity, we recommend arthroscopically-assisted surface replacement. This is usually performed in the beach chair position, with an arthroscopic synovectomy, removal of foreign material (anchors and sutures), capsular release, and tenotomy of the biceps. An anterior deltopectoral approach is then performed, splitting the subscapularis in a raphe located in the lower part of the tendon (usually 1/2 to 2/3 down from the upper border), leaving the superior part intact. The bottom of the subscapularis is reflected medially to create a window through which the humeral head can be dislocated and replaced. The shoulder is reduced and the subscapularis repaired. The subscapularis sparing or window approach minimizes the risk of subscapularis muscle failure and allows early rehabilitation. Surgical tips are to follow the line of the humerus when taking down the inferior 1/3 of the tendon and to elevate it off the bone using a cautery to avoid injury to the 3 veins located near the inferior border.

## CONCLUSIONS

PAGCL is a devastating, poorly understood and relatively rare complication. The complex homeostasis of articular cartilage is undoubtedly sensitive to agents introduced into the joint including mechanical, chemical, and temperature-dependent interventions. To date, several articles have described the phenomenon and the potential associations, including high flow intra-articular pain pumps, thermal devices, nonabsorbable and absorbable anchors, and various sutures; however, level 1 evidence of the etiology does not exist. The prevention, etiology, and definitive treatment remain multifocal and the area of much research.

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