Severe chondrolysis after shoulder arthroscopy: A case series

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Summary Chondrolysis of the glenohumeral joint can be a devastating complication of shoulder arthroscopy. It has rarely been reported, and its etiology is unknown. We report 23 cases of glenohumeral chondrolysis after various types of arthroscopic shoulder procedures.

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Chondrolysis is defined as the disappearance of articular cartilage as the result of lysis or dissolution of the cartilage matrix and cells. For our discussion, this definition should be further refined to include the term “rapid” (onset of radiographic loss of joint space within 12 months of documented normal joint during arthroscopy) to describe the dissolution of cartilage matrix and chondrocytes. Because “acute” typically indicates onset within several weeks to a few months and “chronic” implies long-term presence, we chose the term “rapid” to describe the onset of degeneration in cases of chondrolysis to emphasize the difference from other more common reasons for degenerative disease of the shoulder. Historically, chondrolysis was mostly associated with the hip as an idiopathic disorder in the developing hip5 or associated with slipped capital epiphysis.16 The etiology remains elusive but may be multifactorial, given the variety of reported associations and presumed causes. Chondrolysis has been reported to occur after both physical and surgical trauma,11 after meniscectomy,1 and with the use of irrigation fluid,25 thermal devices,8,10,15,24 or bioabsorbable implants.23 Chondrolysis of the glenohumeral joint after arthroscopic surgery appears to be rare, being reported in only a few published studies.2,8,14,19,22 The reported association of the occurrence of chondrolysis with high-volume intraarticular pain pumps, bioabsorbable labral fixation devices, and thermal probes is most concerning, given the frequency of use of these technologies in modern shoulder arthroscopy. However, the actual cause of even the reported cases has not been confirmed, and the associations are speculative at this juncture. No randomized or prospective studies have been performed. Given the severity of this devastating complication and the young patients affected, published anecdotal observations can be useful.

We report a series of 23 cases of chondrolysis of the glenohumeral joint occurring after shoulder arthroscopy in young patients. The suspected causative associations, presenting characteristics, and our treatment experience are discussed.

Materials and methods

We reviewed the records of suspected cases of glenohumeral chondrolysis that presented to our office after shoulder
arthroscopy from 2005 to 2006. This research study was submitted
to and approved by the Institutional Review Board of Physio-
therapy Associates (Exton, PA) before the review of patient data.

This time period was used because we had not been aware of
a single case of glenohumeral chondrolysis in our clinic before
2005. A total of 23 cases were identified. Chondrolysis was sus-
ppected in those patients who presented with a delayed increase in
shoulder pain after arthroscopy (typically 8-12 months after the
index procedure) and whose radiographs showed a clear loss of
joint space. In all of these cases, radiographs taken before the
index surgery were interpreted as normal, that is, no radiographic
evidence of loss of joint space, osteophytes, cystic changes, or
other findings that could represent early arthritis.

Patients with any known history or diagnosis of arthritis or
infection before the index surgery were excluded. All operative
reports were reviewed, in addition to any available preoperative
imaging studies and intraoperative photographs from the index
surgery. A thorough history was obtained and physical examinations
performed on all suspected chondrolysis patients. Additional radi-
ographs and magnetic resonance imaging (MRI) scans were obtained
in some cases. In addition, because of the concern initially over
infection, the first 10 cases encountered had blood work performed,
including complete blood count, sedimentation rate, and C-reactive
protein level. None had a bone scan or nuclear white cell scan.

Eleven of the cases occurred in patients in whom the index
operation was performed by the senior author. The remaining
patients had been referred to us for further care and their surgery
performed elsewhere. None of the latter had been given the
diagnosis of chondrolysis before being evaluated in our clinic. The
11 cases represented less than 1% of the total shoulder surgeries
performed during the time when an indwelling pain catheter was
used. Although symptoms may have been present, they were not
sufficient to warrant a return to our office before 8 months after the
index surgery. In addition, these numbers do not account for
patients in whom chondrolysis may have developed to a lesser
degree and who did not feel the need to return to the clinic for
evaluation or those who may have sought opinions elsewhere. We
did not attempt to contact all shoulder surgery patients during
a specific time period for this study.

We evaluated demographic data, including gender and age,
time of onset (as indicated by the patient’s history of a rapid
change in pain) from the index surgery, and initial pathology. In
addition, surgical variables of concern including the use of bio-
absorbable fixation devices, intra-articular pain pumps, and
thermal probes were documented from the patient history and
medical records. Treatment methods are presented but outcomes
are not yet available and are not the purpose of this report.

Results

There were 23 documented cases of chondrolysis of the
glenohumeral joint. The details are summarized in Table I.

The mean age was 30.5 years (range, 15-47 years). The
onset of symptoms occurred at a mean of 9.1 months after
the index operation (range, 8-12 months). No patient indi-
cated an intervening injury during the time from index
surgery to presentation to our office. Review of all available
preoperative and intraoperative data failed to show any
evidence of degenerative arthritis, infection, or inflamma-
tory condition before the index surgery in any patient.

There were 14 cases involving labral repairs with bio-
absorbable fixation devices (suture anchors and/or tacks),
including 5 Bankart-type tears and 9 superior labrum ante-
rior-posterior tears. There were 7 cases with documented use
of a thermal probe for capsular laxity (3 monopolar and 4
bipolar), but the wattage used was not documented in the
medical records. Seventeen cases involved the use of an intra-
articular high-volume pain pump catheter, with 250 to 300
mL of 0.25% bupivacaine administered over a 48-hour
period. One of these catheters was reportedly placed in the
subacromial space after a mini-open rotator cuff repair. The
remainder had been placed within the glenohumeral joint
itself. Epinephrine appeared to have been used in 6 known
cases, but not all operative reports reflected the actual
concentration or amount. Other records (ie, nursing operative
records and anesthesia records) were not available, except in the
patients who had their index surgery at our clinic. In 8 addi-
tion, there were 4 cases in which there was no recorded
use of fixation anchors, thermal probes, or pain pumps. Two
of these had only an arthroscopic debridement of a frayed
labrum and/or partial rotator cuff tear. However, all 4 did
have a documented intra-articular bolus of 20 to 30 mL of
0.25% bupivacaine with 1:200,000 epinephrine at the end of
the procedure.

Initially, the first 10 cases were also evaluated for occult
infection, despite a lack of constitutional symptoms,
including a complete blood count, sedimentation rate, and
C-reactive protein level, which were normal in all cases. In
addition, 4 of these had a negative joint aspirate with no
bacteria seen on Gram stain and no growth after 21 days’
incubation (aerobic, anaerobic, and fungal). After these 10
negative infection workups, the remaining patients did not
have these tests performed because of the lack of other clinical
findings that would lead us to suspect an infectious etiology.

The clinical presentation was similar in all patients. All
had initially done well after surgery, with nearly complete
resolution of their original symptoms. Most had already
completed a rehabilitation program and returned to func-
tion. At a mean of 9.1 months, they noticed an increase in Q6
pain that rapidly escalated over the next 4 to 6 weeks. Nine
patients also had a rapid loss of function and loss of range of
motion. The remainder described significant pain but had
preserved motion and function.

Radiographic findings were similar as well. Compared
with preoperative radiographs, there was a diffuse loss of joint
space ranging from 1 mm to complete loss (Figure 1). None
had any evidence of substantial bone loss develop on plain
radiographs, and osteophytes were not apparent. In some
cases, MRI scans were obtained. These showed profound loss
of articular cartilage and symmetric subchondral cysts on
both sides of the joint (Figure 2).

In all 23 cases, treatment initially consisted of the use of
injected and/or oral corticosteroids, nonsteroidal anti-
inflammatory medication, and/or physical therapy. This was
not effective in any case to the point that patients could return to desired activity. Of the cases, 9 (39.1%) ultimately went on to undergo cementless humeral head resurfacing arthroplasty with the Copeland Macrobond Implant (Biomet, Warsaw, IN). These cases had initially presented with severe loss of motion and function and, at the time of surgery, had more diffuse loss of articular cartilage on the humeral head (Figure 3) and a contracted, thickened capsule. In 6 of these cases (26.1%), arthroscopic debridement and capsular release were performed before arthroplasty had not improved their range of motion, and the patients had continued pain. The remainder (14 cases [60.9%]) underwent successful arthroscopic debridement and/or capsular release, with 11 also receiving subsequent hyaluronic acid injections beginning 6 weeks later.

Surgical findings at arthroscopy indicated a paucity of loose bodies, nearly complete dissolution of the articular cartilage on the glenoid, and more central cartilage erosion of the humeral head (Figure 4). None of the cases had evidence of mechanical abrasion by a pain catheter or broken labral fixation device, leading us to conclude that the etiology was biochemical. Furthermore, the surrounding intra-articular soft tissues appeared to be unaffected except in a few cases in which the labral tear may not have healed.

### Discussion

Given the total number of shoulder arthroscopies done each year, the occurrence of chondrolysis still appears to be rare. Of the 3 commonly used modalities implicated by various authors as potential contributors to chondrolysis (pain pumps, thermal devices, and bioabsorbable tissue anchors), devices used for tissue shrinkage initially had the greatest research support as a potential cause for chondrolysis. The association for both pain pumps and bioabsorbable anchors was initially more tenuous. Gobezie et al. reported on 687 consecutive patients who had received pain

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**Table 1** Summary of chondrolysis cases including demographics, pathology, time of onset, and ultimate treatment

<table>
<thead>
<tr>
<th>Age (y)</th>
<th>Sex</th>
<th>Original complaint/surgery</th>
<th>Onset</th>
<th>Anchor</th>
<th>Thermal</th>
<th>Pump</th>
<th>Treatment</th>
</tr>
</thead>
<tbody>
<tr>
<td>40 M</td>
<td>Traumatic instability Bankart</td>
<td>9</td>
<td>BIOABS</td>
<td>Monopolar</td>
<td>IA</td>
<td>Arthroplasty</td>
<td></td>
</tr>
<tr>
<td>45 F</td>
<td>SLAP (thrower)</td>
<td>10</td>
<td>BIOABS</td>
<td>IA</td>
<td>Arthroplasty</td>
<td></td>
<td></td>
</tr>
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<td>IA</td>
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<tr>
<td>47 M</td>
<td>P/O RTC repair</td>
<td>10</td>
<td>Metal</td>
<td>SUB AC</td>
<td>Arthroplasty</td>
<td></td>
<td></td>
</tr>
<tr>
<td>22 F</td>
<td>MDI with chondral blisters</td>
<td>12</td>
<td>BIOABS</td>
<td>Bipolar</td>
<td>IA</td>
<td>Debridement</td>
<td></td>
</tr>
<tr>
<td>27 M</td>
<td>SLAP (thrower)</td>
<td>9</td>
<td>BIOABS</td>
<td>IA</td>
<td>Arthroplasty</td>
<td></td>
<td></td>
</tr>
<tr>
<td>15 F</td>
<td>SLAP (direct blow impact)</td>
<td>8</td>
<td>BIOABS</td>
<td>IA</td>
<td>Arthroplasty</td>
<td></td>
<td></td>
</tr>
<tr>
<td>33 M</td>
<td>SLAP (fall)</td>
<td>10</td>
<td>BIOABS</td>
<td>IA</td>
<td>Arthroplasty</td>
<td></td>
<td></td>
</tr>
<tr>
<td>26 M</td>
<td>Posterior labrum</td>
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<td>BIOABS</td>
<td>IA</td>
<td>Debridement/HA</td>
<td></td>
<td></td>
</tr>
<tr>
<td>47 F</td>
<td>PASTA/type I SAD</td>
<td>8</td>
<td>BIOABS</td>
<td>IA</td>
<td>Arthroplasty</td>
<td></td>
<td></td>
</tr>
<tr>
<td>28 M</td>
<td>SLAP (fall)</td>
<td>10</td>
<td>BIOABS</td>
<td>IA</td>
<td>Debridement</td>
<td></td>
<td></td>
</tr>
<tr>
<td>29 M</td>
<td>Traumatic instability Bankart</td>
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<td>BIOABS</td>
<td>IA</td>
<td>Debridement/HA</td>
<td></td>
<td></td>
</tr>
<tr>
<td>27 F</td>
<td>MVA partial bursal cuff</td>
<td>12</td>
<td>BIOABS</td>
<td>IA</td>
<td>Debridement/HA</td>
<td></td>
<td></td>
</tr>
<tr>
<td>29 M</td>
<td>SLAP (fall)</td>
<td>7</td>
<td>BIOABS</td>
<td>IA</td>
<td>Arthroplasty</td>
<td></td>
<td></td>
</tr>
<tr>
<td>32 M</td>
<td>SLAP (thrower)</td>
<td>8</td>
<td>BIOABS</td>
<td>IA</td>
<td>Arthroplasty</td>
<td></td>
<td></td>
</tr>
<tr>
<td>46 F</td>
<td>Idiopathic frozen shoulder</td>
<td>9</td>
<td>IA</td>
<td></td>
<td>Arthroplasty</td>
<td></td>
<td></td>
</tr>
<tr>
<td>16 F</td>
<td>MDI (thrower)</td>
<td>9</td>
<td>Monopolar</td>
<td>IA</td>
<td>Arthroplasty</td>
<td></td>
<td></td>
</tr>
<tr>
<td>18 M</td>
<td>SLAP (thrower)</td>
<td>8</td>
<td>BIOABS</td>
<td>IA</td>
<td>Debridement/HA</td>
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<td></td>
</tr>
<tr>
<td>22 M</td>
<td>Bankart</td>
<td>10</td>
<td>BIOABS</td>
<td>Bipolar</td>
<td>IA</td>
<td>Debridement/HA</td>
<td></td>
</tr>
<tr>
<td>26 M</td>
<td>Bankart</td>
<td>10</td>
<td>BIOABS</td>
<td>Monopolar</td>
<td>IA</td>
<td>Debridement/HA</td>
<td></td>
</tr>
<tr>
<td>24 M</td>
<td>SLAP I/PASTA</td>
<td>9</td>
<td>BIOABS</td>
<td>IA</td>
<td>Debridement/HA</td>
<td></td>
<td></td>
</tr>
<tr>
<td>31 F</td>
<td>MDI</td>
<td>8</td>
<td>Bipolar</td>
<td>IA</td>
<td>Debridement/HA</td>
<td></td>
<td></td>
</tr>
<tr>
<td>30 M</td>
<td>Interval repair</td>
<td>8</td>
<td>IA</td>
<td></td>
<td>Debridement/HA</td>
<td></td>
<td></td>
</tr>
</tbody>
</table>

*BIOABS, Bioabsorbable anchor; IA, intra-articular pain pump; SLAP, superior labrum anterior-posterior; HA, injection series of hyaluronic acid.*

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Figure 1 Glenoid with nearly complete loss of articular cartilage 9 months after suture capsulorrhaphy and use of an intra-articular pain pump in a 17-year-old female athlete. This shoulder had normal articular cartilage at the index surgery.
may have deleterious effects on cartilage metabolism.\textsuperscript{4,7} More recently, 0.5\% bupivacaine was shown to be significantly more toxic to human chondrocytes than ropivacaine.\textsuperscript{20} Whereas the degradation products of bioabsorbable devices have been shown to be chondrotoxic, chondrolysis associated with the use of these devices may more likely be the result of mechanical factors (dislodgement and fragmentation) than a biochemical sequela.\textsuperscript{21}

As noted earlier, the available literature on the pathophysiology of chondrolysis after arthroscopic shoulder surgery is sparse. Whereas other authors have correlated the occurrence of chondrolysis with modalities such as pain pumps, bioabsorbable anchors, and thermal devices, the actual etiology would appear to be much more complex and multifactorial. The demographic data of the patients in this series do little to clarify potential etiologies, because they are a fairly representative patient sampling of a typical orthopaedic practice. We were unable to identify a single specific modality that we could implicate in the initiation of postoperative chondrolysis. More likely, we believe that chondrolysis is an unfortunate convergence of multiple factors that may initiate rapid articular cartilage dissolution and degenerative changes. Realistically, these may include not only the modalities mentioned previously but such factors as genetic predisposition of the patient, irrigation fluids used during the procedure (type, temperature, and pressure), the surgical procedure itself, and any medications the patient has or is taking. These factors remain to be defined. Certainly, with the addition of recent laboratory evidence of chondrocyte toxicity of local anesthetics,\textsuperscript{4,7,20} caution should be used in considering the use of indwelling pain catheters near hyaline cartilage.

In our series, we identified some common presenting factors that may aid in making the diagnosis of chondrolysis. Most of our patients had surgery elsewhere and presented with advanced disease, that is, the process of chondrolysis was already mature, and extensive damage to the joint surfaces had already occurred. Upon interviewing...
arthroscopy whereas those without fragmentation might not require arthroscopy. We did not find nuclear imaging studies helpful, and blood work findings were not abnormal. However, if infection is suspected, these 2 tests should be performed, in conjunction with arthroscopic lavage.

Unfortunately, because of the rarity of this condition, the treatment regimen for postarthroscopy chondrolysis of the shoulder is not defined. Our initial approach to treatment has been conservative/palliative, using intra-articular and oral corticosteroids and nonsteroidal anti-inflammatories. Of the 23 patients in this report, 9 subsequently underwent arthroplasty because of loss of function after failure of conservative management. The remainder responded well to debridement with and without intra-articular injections of hyaluronic acid. Long-term follow-up will be necessary to determine the success of the treatment in this series. A trend seemed to exist in short-term follow-up that suggests that those with severe loss of motion and capsular thickening do not rapidly respond to any form of treatment. Therefore, we would recommend arthroscopic debridement/capsular release and aggressive rehabilitation before performing arthroplasty, even when the joint space appears to be completely lost.

In conclusion, chondrolysis is fortunately a rare sequela of arthroscopic shoulder surgery. However, its long-term consequences can be particularly disabling, especially in the young patient. It is difficult to implicate a single factor as the etiology of this condition. Rather, we believe that this condition results from an unfortunate convergence of multiple factors. These factors are not adequately defined but probably include genetic, traumatic, biomechanical, and biochemical elements. Likewise, treatment options are poorly defined. Whereas some patients have responded favorably to conservative management, arthroplasty has been required to restore function in others. Clearly, more research is indicated to identify the initiating factors in this pathology.

A high index of suspicion is needed to make an early diagnosis, and patients with any change in symptoms after arthroscopy of the shoulder should be encouraged to return for prompt evaluation and followed up closely over time.

References


