Current Concepts Review

Joint Chondrolysis

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- Although the disease was first described in the hip, reports of chondrolysis in nearly all diarthrodial joints have since emerged with considerable variations in the literature.
- Despite speculation among clinicians and researchers about the implicit causal pathways and etiologic contributors associated with chondrolysis, definitive answers remain elusive.
- The term *chondrolysis* has been applied to varied levels of joint cartilage destruction from focal chondral defects to diffuse cartilage loss, revealing a lack of consistency in the application of diagnostic criteria to guide differential disease classification.
- Differentiating between the various potential etiologies associated with chondrolysis provides opportunities for the prevention of the disease.

Rapid loss of articular cartilage was first described as "acute cartilage necrosis," with, to our knowledge, the first reported case involving the hip published in 1930[1]. In that article, Waldenstrom identified the disease in a series of pediatric patients with slipped capital femoral epiphysis. He characterized this finding as marked loss of joint space attributed to capsular rupture with subsequent disruption of the nutrition to the articular surfaces, leading to acute cartilage necrosis occurring between five and eight months after manual reduction of the slipped capital epiphysis. By 1970, there were several additional reports of slipped capital femoral epiphysis preceding acute cartilage necrosis, with a mean prevalence ranging from 1% (two of 185 hips) to 28% (thirty-six of 127 hips)[2]. In 1971, the term *chondrolysis* was proposed in a case series of predominantly black adolescents with deterioration of the hip that followed a similar clinical and radiographic pattern described in previous reports of acute cartilage necrosis as seen with slipped capital femoral epiphysis[3]. Over the next several decades, the definitional criteria for "chondrolysis" of the hip evolved to include a rapid, progressive loss of joint space on radiographs and associated loss of clinical hip motion[4]. Although partial restoration of joint space and mobility has been noted, the disease progresses in the majority of patients to debilitating osteoarthritis or ankylosis, requiring subsequent hip arthrodesis or arthroplasty.

Reports of chondrolysis in other joints emerged in the 1980s. Chondrolysis of the shoulder was first reported, to our knowledge, in 1983[5], followed by reports of the disorder in the knee in 1984[6], the ankle in 1997[7], and the elbow in 2009[8]. A comparative review of reported cases of chondrolysis among patients in the United States and the broader international community revealed a rather uniform emphasis in the hip until about 2005, at which time a preponderance of publications began to focus on shoulder chondrolysis.

As a clinical syndrome, chondrolysis is a devastating condition that results in substantial pain and morbidity, leading to

Disclosure: None of the authors received payments or services, either directly or indirectly (i.e., via his or her institution), from a third party in support of any aspect of this work. One or more of the authors, or his or her institution, has had a financial relationship, in the thirty-six months prior to submission of this work, with an entity in the biomedical arena that could be perceived to influence or have the potential to influence what is written in this work. No author has had any other relationships, or has engaged in any other activities, that could be perceived to influence or have the potential to influence what is written in this work. The complete **Disclosures of Potential Conflicts of Interest** submitted by authors are always provided with the online version of the article.

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impairment of the affected joint in often young and otherwise active patients. Despite considerable speculation among clinicians and researchers[6-12] about the causal pathways and etiologic contributors associated with chondrolysis, definitive answers remain elusive. Moreover, there is no consensus about a clear set of diagnostic criteria to facilitate the differentiation of chondrolysis from other pathologic conditions of articular cartilage. Without specific criteria to guide diagnosis, a physician may inadvertently classify other joint injuries as chondrolysis, leading to suboptimal treatment and the inability to determine prognosis with accuracy.

Although the literature is replete with case reports and series of chondrolysis, no comprehensive review of the entire spectrum of joint chondrolysis exists. Moreover, no systematic assessment of the diagnostic attributes of joint chondrolysis is available to guide orthopaedic surgeons toward adopting a standardized approach to disease identification and appropriate treatment recommendations. Furthermore, there is a dearth of critical analysis pertaining to associated etiologic contributors of chondrolysis, which is imperative to advance disease prevention. Thus, the purpose of this article was to address these deficiencies in the literature by comparatively examining reports of chondrolysis across all diarthrodial joints to evaluate (1) variations in the clinical application of definitional criteria used to diagnose chondrolysis, (2) commonalities and differences in associated etiologies, and (3) the implications of proper disease recognition on treatment, practice, and research directions.

Approach to Case Identification and Clinical Review

Article Selection Process

Using an iterative keyword search strategy in MEDLINE, with no date or language delimiters, two independent reviewers (J.C.S. and E.A. Chavez) identified 384 published case reports and series involving joint chondrolysis. The following exclusion criteria were then applied: (1) duplicative reports of the same patient population, (2) unrecoverable publications (e.g., too dated to locate), (3) animal or in vitro studies, (4) chondrolysis not involving a joint (e.g., tracheal cartilage), (5) letters or editorial publications, and (6) transient chondrolysis (i.e., idiopathic joint-space narrowing that partially or completely recovered).

After applying the exclusion criteria, 128 published articles (fifty-three from the U.S. and seventy-five from the international community)[1-8,13-132] were analyzed in depth. Non-English-language articles were translated into English prior to data extraction and clinical review. Of the eighty-nine reports involving the hip[1-4,14-98], twelve involving the knee[6,99-109], and one on the elbow[8], most (64%, 75%, and 100%, respectively) were on groups of patients outside the U.S. In contrast, of the twenty-three cases involving the shoulder[5,13,110-130] and three involving the ankle[7,131,132], the majority (70% and 67%, respectively) originated from the U.S.

Cumulatively, there were 830 reports of joint chondrolysis. The hip was involved in 626 patients (339 patients [54%] were international and 287 [46%] were from the U.S.), although ascertaining the exact number of patients was not always possible because of a lack of sufficient detail in the articles. Reports on the shoulder included 167 patients (ninety-four [56%] were from the U.S., and seventy-three [44%] were international) and 171 affected joints (ninety-eight [57%] were in patients from the U.S. and seventy-three [43%] were in patients from the international community). Articles involving the knee included twenty-eight patients (twenty-three [82%] were international, and five [18%] were from the U.S.), accounting for twenty-nine joints (twenty-four [83%] and five [17%], respectively). The ankle was involved in three patients, two of whom were from the U.S. The single case involving the elbow was in a Turkish patient.

Clinical Metrics

The articles were thoroughly reviewed by two trained data abstractors in consultation with five orthopaedic surgeons, including four of us (M.T.P., D.J.S., B.J.C., and A.A.R.) and C.B. Dewing, to systematically extract the following information: (1) number of reported patients with chondrolysis per publication, (2) number of reported chondrolytic joints per publication, (3) primary presenting symptoms of the patients, (4) examination findings, (5) time to diagnosis secondary to related mechanical, chemical, or thermal exposure or insult, (6) time to reported symptom onset, (7) extent of joint destruction, (8) number and types of surgical interventions performed, (9) reported etiologic factors described in relation to loss of articular cartilage, (10) clinical criteria used by the treating physician to diagnose chondrolysis, (11) treatment modality, (12) patient age, (13) patient sex, and (14) the country of origin where the diagnosis and treatment occurred.

Assessment of Variations in the Definitional Criteria Used to Diagnose Joint Chondrolysis

Five orthopaedic surgeons independently and in paired groups systematically evaluated the definitional criteria used to diagnose chondrolysis in the original publications. The criteria were stratified into four categories including (1) temporal factors (e.g., time to symptom onset and time to diagnosis), (2) patient factors (e.g., age, medical history, and evidence of infection), (3) radiographic findings (e.g., imaging modality, extent of joint-space narrowing, and severity of articular cartilage damage), and (4) surgical findings (e.g., diffuse or nearly complete cartilage loss, unipolar changes, and focal cartilage damage). If publications provided no clinical criteria but declaratively stated that the patient had chondrolysis, such cases were grouped into a fifth category labeled as "no definitional criteria provided."

After variations in diagnostic criteria were determined, definitional trends were evaluated to generate a narrowed set of more definitive criteria to guide the differential diagnosis of chondrolysis. Subsequently, each case was independently classified as either chondrolysis or nonchondrolysis (i.e., an alternative diagnosis indicative of cartilage pathology) with use of a standardized working definition based on the following most commonly reported diagnostic criteria: (1) surgical, radiographic, or imaging findings demonstrating diffuse cartilage loss or joint-space narrowing due to involvement of apposing

articular surfaces, and (2) rapid cartilage destruction (i.e., within eighteen months after an insult). Cases in which a portion of the criteria lacked typical findings of chondrolysis were reviewed independently by two orthopaedic surgeons. This process was repeated until the definitional attributes of each case were identified and concordance was achieved. Cases with initial discordance between the primary surgeon reviewers were further evaluated by the senior author (B.J.C.) to render a final diagnosis.

Identification of Potential Etiologic Contributors to Joint Chondrolysis

Etiologic determinants reported as potential contributors to the loss of articular cartilage were stratified into four categories including (1) thermal (e.g., radiofrequency device, electrocautery, and holmium:YAG [yttrium-aluminum-garnet] laser), (2) chemical (e.g., intra-articular infusion of local anesthetics, chlorhexidine, and gentian violet), (3) mechanical (e.g., surgical insult or prominent hardware such as anchors, pins, or screws), or (4) other (e.g., infection or a history of traumatic joint injury). Commonalities and differences in etiologies were compared between U.S. and international publications.

Selection of Surgical Images for Case Illustration

To demonstrate the implications of proper disease recognition on treatment, illustrative examples of chondrolysis and non-chondrolysis in the hip (two cases), knee (four), and shoulder (three) were purposively selected from the surgical image collections of two authors (D.J.S. and B.J.C.).

Data Analysis

Descriptive statistics, bivariate analysis, and comparative proportional differences were calculated with use of Z-tests (version 9.1; SAS Institute, Cary, North Carolina). The level of significance was set at $p < 0.05$. Interrater reliability among surgeon evaluators was assessed with use of the Cohen kappa statistic[133] (range, 0.74 to 1.0). Two sets of sensitivity analyses were performed with use of range variations in the following diagnostic criteria for chondrolysis: (1) the severity of cartilage damage (i.e., bipolar and/or diffuse cartilage loss or focal cartilage damage), (2) time to symptom presentation or disease identification, or (3) presence of osteoarthritis.

Overview of Chondrolysis in the Literature

Variations in the Definitional Criteria Used to Diagnose Joint Chondrolysis

Standard medical dictionaries define chondrolysis as "the disappearance of articular cartilage as the result of lysis or dissolution of the cartilage matrix and cells."[134] As is often the case for an emerging disease, this broad definition fails to provide meaningful attributes to facilitate proper recognition and diagnosis. In the present review, over one-third (34%; forty-three) of the 128 articles on chondrolysis lacked an explicit and clear definition. Those lacking a clear definition included 48% (eleven) of the twenty-three involving the shoulder (eight of sixteen U.S. studies and three of seven international studies), five of the twelve involving the knee (one of three U.S. studies and four of nine international studies), and 30% (twenty-seven) of the eighty-nine involving the hip (19% [six] of thirty-two U.S. studies and 37% [twenty-one] of fifty-seven international studies).

The four most widely reported definitional attributes used to diagnose chondrolysis were (1) patient age (a surrogate risk factor for confounding cartilage-related maladies), (2) time of symptom onset or clinical presentation, (3) magnitude of cartilage loss (i.e., diffuse or focal), and (4) severity or depth of cartilage injury. Among the articles that provided descriptive clinical criteria, considerable heterogeneity was evident in the following indicators: (1) patient age, (2) time to symptom onset or clinical presentation, and (3) extent of cartilage damage. The age for patients with reported chondrolysis ranged from three to ninety-five years for the hip, fourteen to sixty-four years for the shoulder, fifteen to sixty-two years for the knee, and fifteen to twenty-one years for the ankle. No age was given for the single elbow case. Wide temporal variations were evident. In many cases, the time to the onset of symptoms was unknown as the patients presented to tertiary care centers or subspecialists, and the time to presentation or diagnosis was used instead. The time from index surgery to diagnosis was also highly varied for the hip (one month to fifteen years), shoulder (three months to seven years), and knee (five weeks to three years). For the ankle, the range was much tighter (four to eleven months). No time parameter was provided for the elbow case.

Variability in the extent of cartilage damage required before physicians diagnosed chondrolysis differed by joint. In the hip, some diagnosed chondrolysis if joint-space narrowing of ≥3 mm existed, while others specified extensive loss of articular cartilage of the femoral head and acetabulum. In the shoulder, the diagnosis of chondrolysis included a range of cartilage loss from focal to complete. In the knee, cartilage damage limited to one compartment was considered extensive enough to be labeled chondrolysis by some, while others defined chondrolysis when cartilage loss involved the three compartments of the knee. In the ankle, the extent of cartilage damage necessary to diagnose chondrolysis was not specified beyond joint-space narrowing. No information on the extent of cartilage damage was provided for the elbow case.

Misclassification of Diseases Associated with Cartilage Loss in Joints: An Empirical Pooling Effect

The lack of a standardized set of definitional attributes to facilitate diagnostic differentiation of diseases associated with cartilage loss in joints revealed an empirical pooling effect, whereby various types of pathologic conditions of articular cartilage were inappropriately grouped together as chondrolysis. Pooling was most prevalent in the knee (48% [fourteen] of twenty-nine knees overall, including one of five knees in patients from the U.S. versus 54% [thirteen] of twenty-four knees in international patients)[100,103-107], followed by the shoulder (9% [twelve] of 128 joints overall, including 6% [six] of ninety-eight from the U.S. versus 20% [six] of thirty joints among international patients)[112,115,121,125], the hip (7% [forty-six] of 626

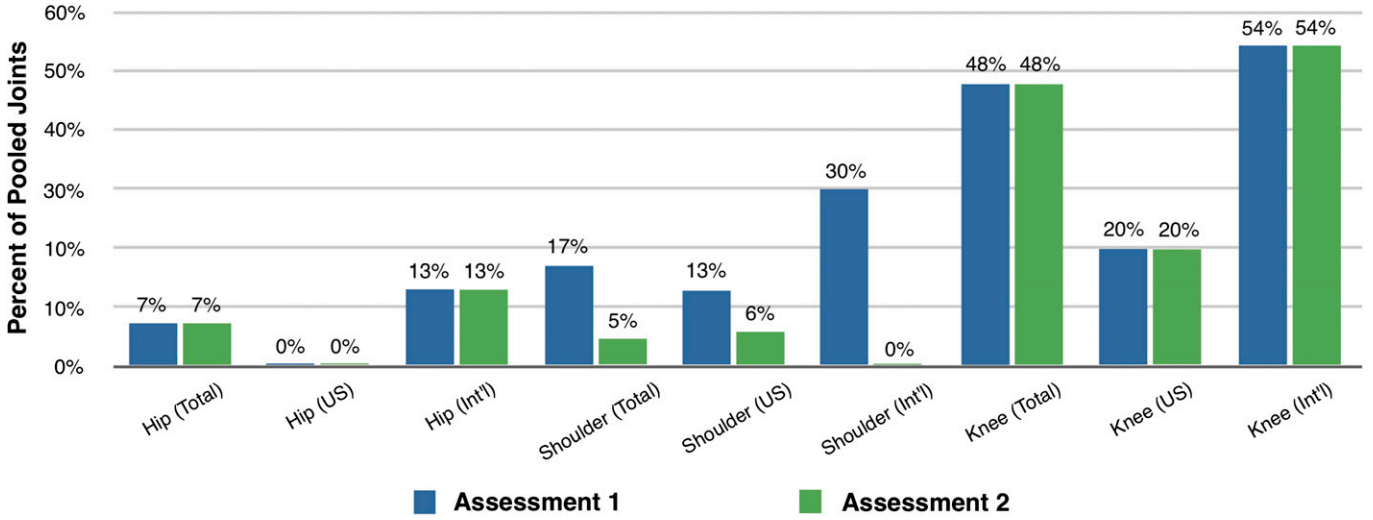

Fig. 1

Misclassification of diseases associated with cartilage loss in joints: an empirical pooling effect in the global literature. Two assessments of pooling (an effect whereby various types of pathologic conditions of articular cartilage are inappropriately grouped together as chondrolysis) were conducted to determine the fraction of pooled joints in the literature. No variations between assessments were evident for the hip and knee. However, for the shoulder, differences were seen between assessments in relation to case reports in both the U.S. and international community.

joints overall, including <1% [one] of 287 from the U.S. versus 13% [forty-five] of 339 joints among international patients)[25,50,53,57,58,67,73,92,94], and the elbow (one of one international patient)[8]. There was no pooling evident for the ankle.

To examine the effects of variations in the definitional criteria on the fractional portion of pooled cases, clinical sensitivity analyses were performed (Fig. 1). Results from sensitivity analyses revealed no change in the fraction of pooled cases for the hip and knee. For the shoulder, variations in the definitional criteria between the first and second assessments revealed a larger pooling effect to be present under the first assessment.

Examination of the clinical factors contributing to the pooling effect revealed commonalities across joints. In the knee, pooling was primarily associated with unicompartmental chondral damage, typically limited to the lateral compartment (all thirteen international cases versus one case from the U.S.). In addition, in the international knee literature, twelve of the thirteen of pooled joints were inappropriately diagnosed as chondrolysis instead of secondary arthritis following lateral meniscectomy.

In the shoulder, pooling was associated with an extended time to diagnosis (i.e., twelve months or more after an identifiable insult), which was indicative of more chronic conditions (e.g., osteoarthritis), for all six international cases. For U.S. cases, pooling was evident when focal chondral defects were combined with diffuse cartilage loss (83%; five of six cases). Nearly 100% (forty-five) of the forty-six pooled hip cases were among international patients, with the majority (47%; twenty-one) of the forty-five reports grouping patients with rapid onset of chondrolysis (i.e., within twelve months after surgery) with patients who experienced cartilage loss over an extended time period (i.e., multiple years after surgery). Difficulties differentiating osteonecrosis and rapidly progressing arthritis from chondrolysis were notable in the hip. Patients with osteonecrosis ranged in age from twenty-three to seventy-four years, and those with osteoarthritis ranged from twenty-nine to ninety-five years. Although older age should not rule out the diagnosis of chondrolysis, a biological diathesis at a younger age may be a factor associated with an accurate diagnosis of chondrolysis. The age of the patients with chondrolysis of the hip appeared to have a bimodal distribution, with younger patients presenting with chondrolysis secondary to slipped capital femoral epiphysis.

Potential Etiologic Contributors to Joint Chondrolysis

A myriad of potential etiologic contributors to chondrolysis have been reported. The majority of these etiologies can be classified into three categories: (1) mechanical, (2) chemical, and (3) thermal. A summary depiction of the commonalities and differences in suspected etiologies associated with chondrolysis across various joints is provided in Table I. Clearly, the etiology of chondrolysis is complex and multifactorial.

Cases of chondrolysis after the placement of implants for periarticular soft-tissue or fracture fixation, a history of joint trauma, and surgical insult with or without improper techniques have been reported across all joints[1-3,13,14-27,31-35,45-48,51,53-61,63-66,69,70,72-80,82,84,85,87,89,92-94,96-103,105,106,108-132]. In the shoulder, knee, and ankle, the infusion of local anesthetics through intra-articular pain pumps has been widely reported[13,108,109,113,121-123,125-129,131]. For the hip and shoulder, the literature also implicates thermal sources (e.g., radiofrequency devices) as potential etiologic contributors to chondrolysis[13,98,100,103,113,114,116-120,122-125]. Infection is another associated etiologic factor documented globally in several reports of chondrolysis in the knee, shoulder, hip, and ankle[3-6,13,20,24,29-31,34,35,40-45,47,50,52,57,59,66,67,71,81,88,91,97,98,102,109,117,118,121-123,128,129,131,132].

TABLE I Potential Etiologic Contributors to Joint Chondrolysis in the Literature from 1930 to 2010

Potential Etiologic Contributor	Joint*									
	Hip		Shoulder		Knee		Ankle		Elbow	
	U.S.	Internat.	U.S.	Internat.	U.S.	Internat.	U.S.	Internat.	U.S.	Internat.
Chlorhexidine						✓				
Gentian violet		✓		✓						
Hardware (e.g., suture materials, anchors, pins, or screws)	✓	✓	✓	✓	✓	✓	✓			✓
History of traumatic injury to joint	✓	✓	✓	✓		✓	✓			✓
Immobilization	✓	✓								
Infection	✓	✓	✓	✓	✓	✓	✓			
Infused local anesthetics			✓	✓	✓		✓			
Intra-articular bone cement leak		✓								
Intra-articular pain pump catheter			✓	✓	✓		✓			
Irrigation fluids (e.g., lactated ringers, normal saline solution, or other)			✓							
Manipulation and/or reduction	✓	✓								
Radiofrequency or intra-articular electrocautery devices		✓	✓	✓		✓				
Severity of slipped capital femoral epiphysis	✓	✓								
Surgical insult or instrumentation	✓	✓	✓	✓	✓	✓	✓			✓

*U.S. = reports in U.S. studies, and Internat. = reports in international studies.

Some etiologic factors are unique to specific joints. Holmium:YAG laser[100,103] and chlorhexidine[6,99,101,102] have only been associated with chondrolysis in the knee. In the shoulder, gentian violet[110,111] and irrigation fluids[114,123] have been hypothesized as contributors to chondrolysis. The hip has several unique potential etiologies associated with chondrolysis, including the severity of slipped capital femoral epiphysis[15-24,26,27,31-35,38,51,54-56,59,60,62-64,70,72,75-77,79,82,84,85,87,89,93,96], immobilization[1,13,16,17,20-23,26,37,54-56,62,74,77,78,80,84], manipulation or reduction procedures[1,2,16-19,21-24,27,32-34,38,46,51,56,61,72,74,77,94,96,97], and one case of an intra-articular leak of bone cement[80]. Overall, the shoulder is associated with the greatest number of potential etiologic contributors to chondrolysis.

Comparative analysis of the cumulative frequency of presumed etiologic contributors is documented by joint and geography in Figure 2. For the hip, chondrolysis was most often reported to be idiopathic (14% [thirty-nine] of 287 U.S. cases versus 46% [157] of 339 international cases; $p < 0.001$) or believed to be a complication of slipped capital femoral epiphysis (83% [239] of 287 U.S. cases versus 40% [137] of 339 international cases; $p < 0.001$). Although many hip articles lacked adequate specificity with regard to etiology, mechanical factors predominated in reports that considered etiologic contributors. Over one-third (35%; 125) of the 355 cases were reported to have mechanical contributors to chondrolysis, with a significantly higher fraction of patients from the U.S. (73% [seventy-two] of ninety-nine patients) compared with international patients (21% [fifty-three] of 256 patients; $p < 0.001$). By comparison, chemical and thermal factors were rarely reported for the hip. A single case of chondrolysis associated with an intra-articular leak of methylmethacrylate[80] and another case associated with electrocautery[98] were also reported.

For the shoulder, U.S. studies were significantly more likely than international studies to link chondrolysis to exposures related to thermal devices (3% [one] of thirty international studies versus 47% [forty-six] of ninety-eight U.S. studies; $p < 0.001$); noxious chemical agents such as continuous infusion of local anesthetics (30% [twenty-two] of seventy-three international studies versus 66% [sixty-five] of

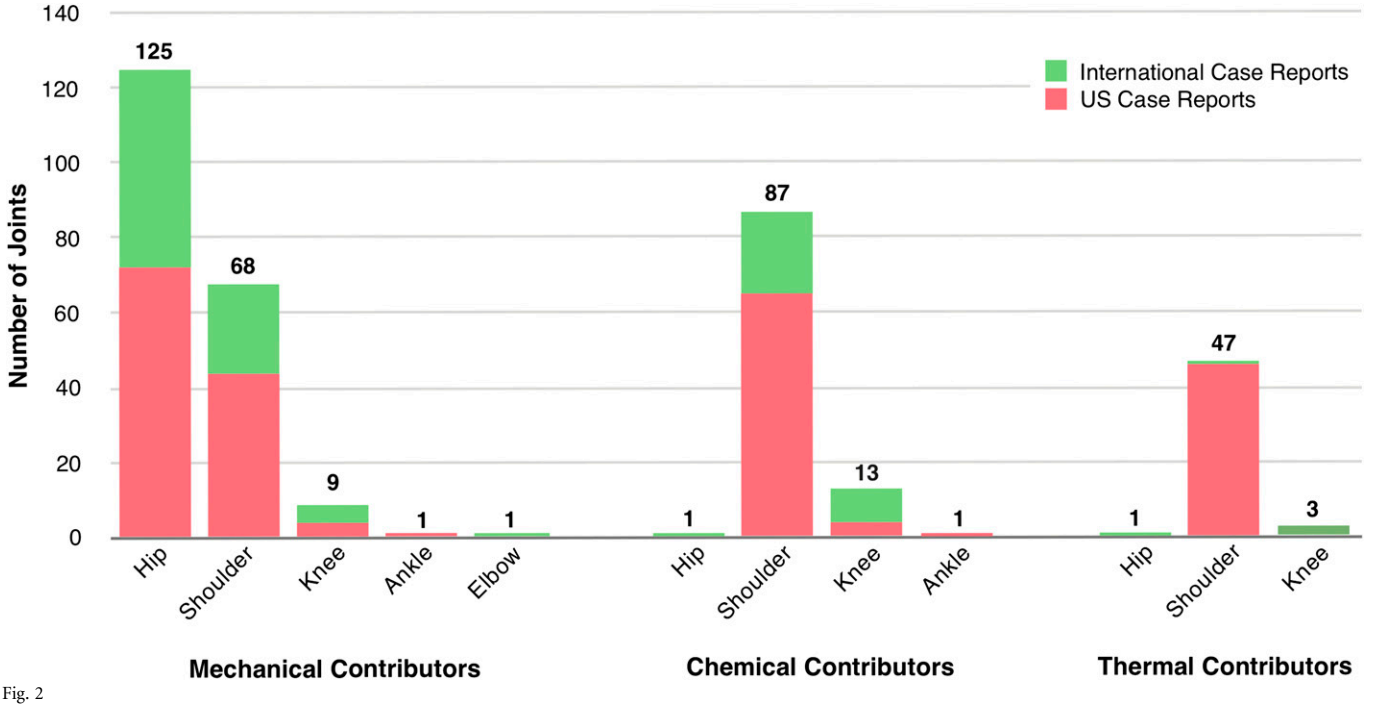

Fig. 2

A global comparison of potential etiologic contributors to joint chondrolysis. On the basis of aggregate case reports, the greatest number of potential etiologic contributors to chondrolysis relate to mechanical and chemical factors. For the hip, both U.S. and international reports place the emphasis on associations between mechanical factors and chondrolysis. By contrast, in the shoulder, chemical agents are the most frequently reported potential contributor, followed by mechanical and thermal factors.

ninety-eight U.S. studies; $p < 0.001$); but less likely to link chondrolysis to exposures related to mechanical insults (45% [forty-four] of ninety-eight U.S. studies versus 80% [twenty-four] of thirty international studies; $p < 0.001$). The proportion of studies on shoulder chondrolysis in which two or more etiologic contributors were indicated was significantly higher in the international literature (80% [twenty-four] of thirty studies) than the U.S. literature (53% [forty-six] of eighty-seven studies; $p < 0.05$).

In studies on the knee, chemical agents were identified as noxious stimulants significantly more often in the U.S. literature (four of five reports) than in the international literature (38% [nine] of twenty-four reports; $p < 0.001$). The nine international studies on the knee noted only chlorhexidine usage, whereas the four U.S. studies focused on the adverse role of local intra-articular anesthetic infusions for pain management. Mechanical factors associated with knee chondrolysis included anchors, screws, and other implants. Potential mechanical contributors to knee chondrolysis were far more likely to be reported in the U.S. literature (four of five studies) than the international literature (21% [five] of twenty-four studies; $p < 0.001$). Thermal devices were reported to be associated with knee chondrolysis only in international studies (three of twenty-four international studies versus none of five U.S. studies; $p < 0.05$).

For the ankle, only U.S. studies noted chemical toxicity due to infusion of local anesthetics[131] and mechanical insult as potential etiologic contributors[132]. For the elbow case, mechanical damage associated with traumatic injury and implants were implicated as potential etiologic contributors[8].

Treatment Implications

Optimal treatment of chondrolysis continues to evolve[10,88,90,108,109,113,116,119,122,123,125,128,129,132]. Nonarthroplasty options for the treatment of chondrolysis are somewhat limited but may be reasonable for those with chondrolysis at a young age[13,122,123,125]. While technically an entire joint surface can be replaced biologically, diffuse cartilage loss is associated with a hostile biologic and mechanical environment, leading to suboptimal outcomes[13,122,125]. Across all joints, total joint arthroplasty remains the gold standard for the treatment of extensive articular cartilage damage. However, in patients with chondrolysis, a younger age and an inflamed synovial environment in association with high physical demands complicates decision making.

In contrast to patients with osteoarthritis, those with chondrolysis often have levels of pain and dysesthesia that require narcotic medication[125]. This finding is not commonly seen among patients in whom the joint space improves with treatment (i.e., osteotomy of the hip, knee, and ankle) or in patients who have chondrolysis associated with slipped capital femoral epiphysis[23,39,49]. In addition, patients who have prolonged periods of time to adapt to the progressive loss of cartilage, such as after a meniscectomy in the knee, present with a more indolent clinical course as they adapt to the relatively slow biologic response and remodeling. By comparison, patients with diffuse acute cartilage demise present with severe pain and

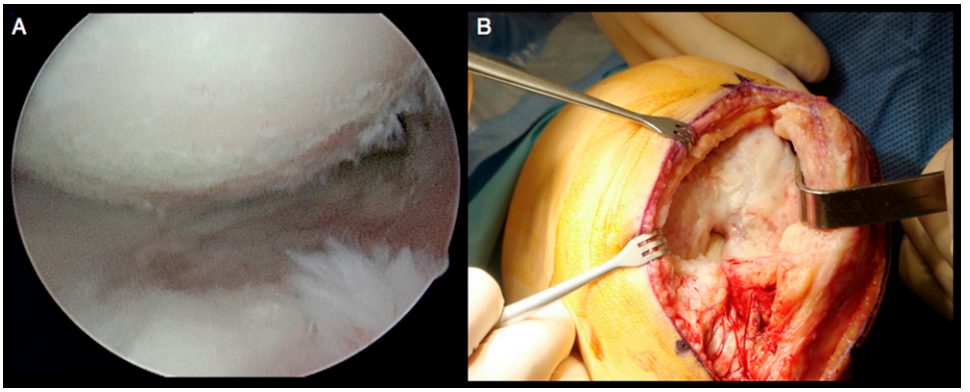

Fig. 3

Arthroscopic (**Fig. 3-A**) and open (**Fig. 3-B**) images of the medial knee compartment in a seventeen-year-old female patient who presented with knee pain four months after anterior cruciate ligament reconstruction with hamstring autograft[109]. The visibly diffuse, severe nature of the chondral damage demonstrates chondrolysis of the knee. (Figure 3-A is reprinted from Slabaugh M, Friel N, Cole B. Rapid chondrolysis of the knee after anterior cruciate ligament reconstruction: a case report. J Bone Joint Surg Am. 2010;92:186-9.)

rapid functional loss. Hence, time to presentation may define which joints are irrevocably damaged compared with joints that have some recovery of function.

Implications for Orthopaedic Practice

Within the last five years, chondrolysis has increasingly become a challenging clinical problem. While some patients with chondrolysis have relatively clear documentation of the insult, etiology, treatment, and outcome, most lack such clarity. Semantically, the degree of cartilage loss as a defining factor for the diagnosis of chondrolysis is too narrow to consider in isolation. Much like difficulties in grading chondromalacia, in which an area of articular cartilage loss may have an intralesional severity ranging from grade I to IV[135], the exact amount of articular cartilage loss necessary to diagnose chondrolysis remains ill-defined (Figs. 3 and 4).

This review identified many cases with insufficient details about the articular surface, leading to inadvertent misclassification (i.e., osteoarthritis or focal cartilage loss) as chondrolysis (Fig. 5). Understanding definitional differences has substantial clinical and treatment implications. For example, biologic or nonarthroplasty solutions with less favorable outcomes for chondrolysis can often be implemented for the treatment of localized articular cartilage loss due to trauma, prominent suture anchors (Fig. 6), or localized degenerative lesions presenting with less intense symptoms (Fig. 7).

A proper history and clinical examination will assist in the differentiation of focal articular cartilage damage from chondrolysis. For example, focal cartilage damage of the glenohumeral joint leads to pain and mechanical symptoms over a very narrow range of motion (when the focal defect is engaged or

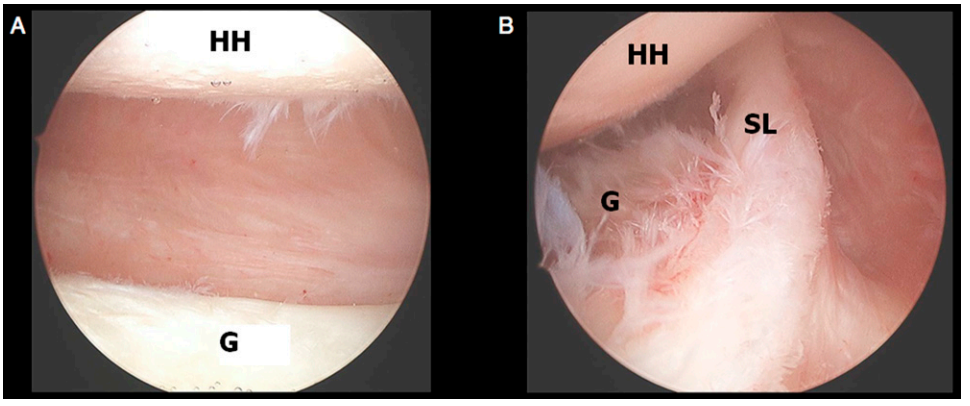

Fig. 4

Figs. 4-A and 4-B Arthroscopic images of the left shoulder of a twenty-five-year-old man who presented with glenohumeral chondrolysis eight months after the index arthroscopic surgery to repair a superior labral anterior-posterior (SLAP) tear. **Fig. 4-A** Complete loss of humeral head (HH) articular cartilage was evident near the glenoid (G). **Fig. 4-B** There was fraying of the superior labrum (SL) along with full-thickness loss of cartilage on the adjacent humeral head (HH) and glenoid (G). The patient failed to have relief after debridement and capsular release, and a resurfacing arthroplasty was performed with initial good results at the eighteen-month follow-up evaluation.

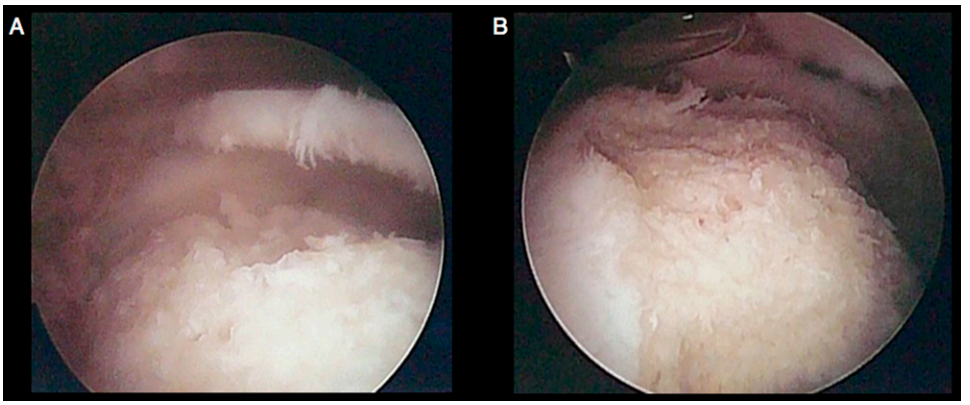

Fig. 5

Figs. 5-A and 5-B Images made at the time of presentation of a thirty-nine-year-old man who fell onto his left hip six weeks after the onset of vague pain in the anterior aspect of the hip after he had resumed jogging for two to three miles daily. He had anterior hip catching and limitation of hip motion on flexion, internal rotation, and external rotation at 100°, 10°, and 35°, respectively. Radiographic findings were normal; however, magnetic resonance imaging (MRI) findings were suggestive of a femoral head fracture. **Fig. 5-A** Arthroscopic image reveals delamination of the femoral head cartilage and subchondral osseous collapse. **Fig. 5-B** The rapidly progressive osteonecrosis following trauma as seen on this arthroscopic image could easily be misconstrued as chondrolysis on a standard preoperative MRI evaluation.

loaded)[9,10,13,113,122,125,127,129]. In contrast, patients with glenohumeral chondrolysis present with severe pain, markedly diminished shoulder motion, crepitus, and mechanical catching throughout the limited motion. Patients with chondrolysis often complain of pain out of proportion to the clinical findings, leading to a misdiagnosis of complex regional pain syndrome. The same examination findings in other joints are somewhat more difficult to ascertain, given their inherent motion limitations. Patients who have had a surgical procedure and present with persistent, unrelenting, postoperative pain recalcitrant to therapy and standard postoperative medications should be closely scrutinized for a diffuse articular cartilage disease process.

On a global level, a lack of consistent criteria to facilitate diagnostic and etiologic differentiation of chondrolysis has resulted in a pooling effect, whereby physicians combine diffuse with focal cartilage damage, and rapid with prolonged timing to symptom onset. On an international level, investigators have focused primarily on the recognition and differential diagnosis of chondrolysis. In contrast, in the U.S., a greater emphasis has been placed on the etiologic determinants of chondrolysis. Nonetheless, without evidence-based definitional guidelines to support physicians with proper diagnostic criteria, we believe that establishing predictable nonsurgical and surgical disease management will most likely remain challenging. Thus, we recommend that the term *chondrolysis* be applied to patients who are seen within twelve months after an operative intervention or potential cartilage insult, with pain, stiffness, limited joint motion, and severe diffuse articular cartilage loss evidenced by radiographs, magnetic resonance imaging, or arthroscopic evaluation. Furthermore, the clinical symptoms should generally exceed a comparable amount of joint destruction in an otherwise chronic condition, for which the patient has likely had more time to adapt to this pathoanatomic deterioration.

Future Directions

The specific pathophysiology of chondrolysis remains elusive. The condition seems clinically and histopathologically most similar to so-called acute cartilage necrosis and differs dramatically in its

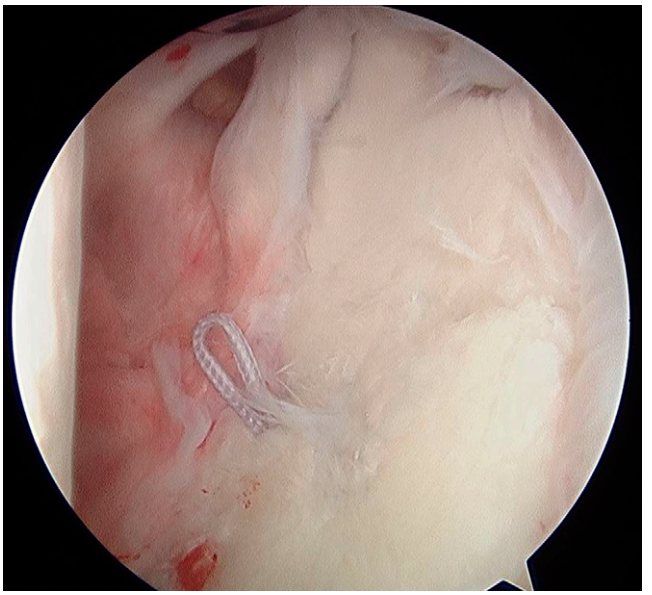

Fig. 6

Arthroscopic image of the left shoulder of a twenty-five-year-old woman made two years after an arthroscopic repair of a superior labral anterior-posterior (SLAP) tear and Bankart lesion. The anchors are positioned substantially on the face of the glenoid, with focal complete loss of glenoid cartilage and partial-thickness loss of the humeral head cartilage. The image reveals several full-thickness regions of glenoid cartilage loss superiorly and anteriorly, coinciding with anchor and suture locations. The image represents focal mechanical damage of cartilage—not diffuse cartilage loss, which is indicative of chondrolysis.

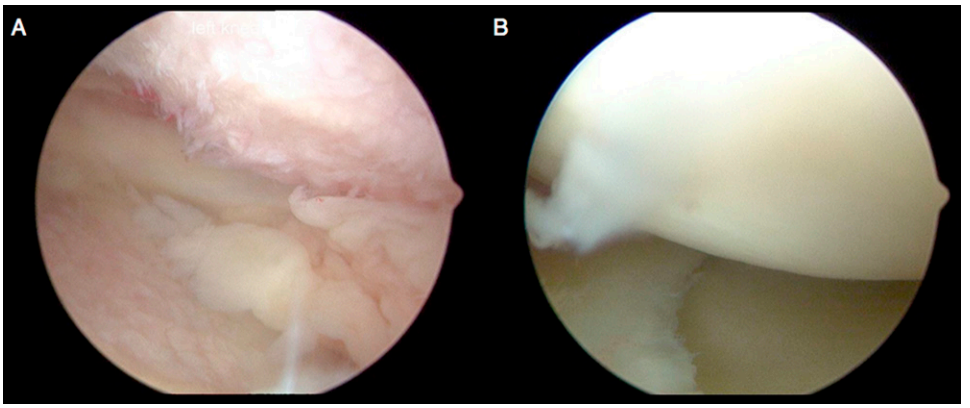

Fig. 7

Arthroscopic images of the lateral (**Fig. 7-A**) and medial (**Fig. 7-B**) compartments of the knee in a twenty-five-year old man initially treated with a subtotal lateral meniscectomy. Six months postoperatively, he presented with slowly increasing pain in the lateral part of the knee and activity-related swelling. Repeat arthroscopy revealed a grade-IV defect of the lateral femoral condyle (20 x 20 mm) and the absence of a functional lateral meniscus. Despite nearly complete cartilage destruction on the lateral side, the medial compartment was well preserved. This case is similar to those from previous reports in which secondary osteoarthritis that developed following lateral meniscectomy was inadvertently diagnosed as chondrolysis because the image does not reveal diffuse tricompartmental chondrolysis. The asymmetric pattern of chondral damage, with complete lateral compartment chondral loss and normal cartilage on the medial side of the knee, shows that this case was not global knee chondral destruction but rather was lateral articular cartilage damage due to a localized process.

presentation compared with osteoarthritis. The most fundamental differences are that it remains most commonly found in younger patients and generally presents following surgical intervention of a diarthrodial joint with a progressive and rapid clinical course. Given the variability across joints with respect to clinical presentation, it is likely that there are yet unidentified patient-specific and pathology-specific factors that contribute to the development of catastrophic joint destruction.

To substantially diminish the possibility of misdiagnosis, an evidence-based diagnostic algorithm that includes greater specificity related to the patient's history, examination findings, imaging studies, and surgical findings is essential. Remaining mindful that chondrolysis is an acute or subacute process with the pathologic changes developing over months, rather than years, will help to differentiate it from osteoarthritis. Imaging and surgical findings should be associated with a global process occurring so rapidly that the joint does not respond as would be expected from a slower process such as rheumatoid arthritis or osteoarthritis. Future bench, clinical, and epidemiological research will enhance our understanding of the many relevant factors leading to chondrolysis. Although numerous potential contributing factors lead to substantial chondral damage, isolating the singular cause of chondrolysis is much more challenging in a patient than in a laboratory setting. As our understanding of this complex disease evolves, identifying the reasons why some patients are more susceptible than others to developing chondrolysis can become a reality. At present, as practical solutions to these challenging cases remain elusive, moving toward the adoption of a more cohesive and standardized definition of chondrolysis will likely yield missing answers as to who is truly at risk for this pathologic condition and why. ■

NOTE: The authors thank Christopher B. Dewing, MD, from the Naval Medical Center San Diego, San Diego, California, for supporting clinical evaluations. They also thank Mark S. Lawler, MD, from Marin Orthopedics and Sports Medicine, Novato, California, for sharing selective arthroscopic images from his archival collections. In addition, they acknowledge Vasili Karas, BS, from Rush University Medical Center as well as Diane Blyler, MA, PhD, Emily A. Chavez, MPH, and Leighla Sharghi, BA, MPA, from Advance Health Solution, LLC, for their support with article retrieval, review, and synthesis.

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