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General

Adhesive Capsulitis of the Hip: A Case Presentation and Review

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There remains a paucity of literature addressing adhesive capsulitis of the hip (ACH), making the diagnosis and treatment a continued challenge for healthcare providers. ACH encompasses restricted hip range-of-motion and pain that progresses through analogous *Stages (1-4)* to adhesive capsulitis of the shoulder. We report a case presentation of a middle-aged man that illustrates the significance of certain patient factors and provide a review of current literature to aid in the diagnostic evaluation and treatment for addressing ACH. Initial conservative treatment of ACH includes the appropriate management of associated comorbidities, oral and/or injectable pharmacologics, and physical therapy. While frequently resolving with time, refractory cases of ACH may require more aggressive approaches including pressure dilation, manipulation under anesthesia, synovectomy, capsular release and, for select patients, total hip arthroplasty. Given the limited available literature addressing ACH, healthcare providers may be forced to rely on a small number of published case reports and outdated review articles to guide their diagnostic evaluation and treatment approaches. Thus, this case presentation and review provides an updated approach to better diagnose and manage patients with ACH.

INTRODUCTION

In 1963, Caroit et al[1] was the first to report idiopathic capsular restriction of the hip. Clinically, these two cases presented analogous to adhesive capsulitis of the shoulder (ACS), with acute painful active and passive range-of-motion (ROM) progressing to painless, restricted ROM across multiple planes. However, since the article was originally published, there remains a paucity of impactful literature addressing the prevalence, etiology, diagnosis, and management of adhesive capsulitis of the hip (ACH).[2]

Notably, in 2006, Byrd et al[3] reported a case series of 9 patients who had capsular fibrosis without concomitant pathology upon arthroscopic visualization. These findings propelled the authors to conclude that ACH may be an underdiagnosed condition, likely because ACH is clinically diagnosed without explicit parameters.[2] While some studies have analyzed isotope bone scans, synovial fluid volume, and intracapsular pressure as tools to assist in making a diagnosis,[4–6] it remains difficult to justify these potentially invasive and expensive procedures without evidence-based research to support their diagnostic utility. Moreover, ruling out concomitant hip pathology is a demanding task, as standard radiographs do not eliminate the possibility of cartilage injury or femoroacetabular impingement as the primary source of painful and restricted ROM.[7] Additionally, common hip pathologies may further complicate the diagnostic workup, as labral tears can occur in up to 69% of asymptomatic individuals[8,9] and extraarticular soft tissue dysfunction can contribute to myofascial pain and restriction of motion without definitive imaging correlates.[10,11] Consequently, healthcare providers may be challenged with managing a patient who presents with suspected ACH without definitive evidence proving the absence of concomitant pathology, thus resulting in an exhaustive and costly diagnostic evaluation to exclude secondary contributors.

Given the limited available literature addressing ACH, healthcare providers may be forced to rely on a small number of published case reports, outdated review articles, and/or principles to treating ACS to guide the diagnosis and management of ACH.[2,4,5,12–15] Thus, we describe a case presentation of ACH and review the literature to provide an updated approach to the management of ACH.

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CASE PRESENTATION

A 43-year-old male with a past medical history of insulin-dependent diabetes mellitus (DM), psoriasis and seasonal allergies originally presented to a Physical Medicine & Rehabilitation sports medicine clinic for evaluation of right shoulder pain. The patient endorsed diffuse pain along with limited ROM and was diagnosed with right shoulder adhesive capsulitis. The patient underwent a protracted recovery course, and his treatment consisted of two ultrasound-guided glenohumeral joint corticosteroid injections and a course of physical therapy, per his request.

On a subsequent follow-up, the patient noted improvement in right shoulder pain and ROM; however, he endorsed a several month history of left anterolateral hip pain, especially with external rotation. The patient denied associated locking, clicking, catching, and instability. Physical examination was consistent with tensor fascia lata myofascial pain and concerns for intraarticular pathology, including adhesive capsulitis of the hip caused by restricted motion (full ROM with hip flexion and internal rotation, limited/painful with external rotation). Pelvic radiographs demonstrated minimal degenerative changes of the hips (**Figure 1**).

The patient received tensor fascia lata and gluteus medius/maximus trigger point injections with lidocaine that resulted in minimal relief of symptoms. Therefore, to rule out intraarticular pathology, the patient underwent a magnetic resonance arthrogram (MRA) of the left hip, which revealed an anterosuperior acetabular labral tear without evidence of femoroacetabular impingement or any findings associated with septic arthritis (synovial enhancement, perisynovial edema, joint effusion). Additionally, the left hip articular cartilage was noted to be intact, with no evidence of soft tissue pathology. (**Figure 2**).

Medications included insulin via pump (DM managed through endocrinology), cetirizine, cholecalciferol, ibuprofen PRN, magnesium, and garlic capsules. Lab tests demonstrated normal thyroid function, along with an elevated glucose and hemoglobin A1c (8.2%). Moreover, the patient's complete blood count, complete metabolic panel (besides elevated glucose), and urinalysis were unremarkable. Overall, clinical evaluation raised concerns for both intra- and extra-articular contributors to his hip pain. Therefore, the patient underwent an ultrasound-guided left hip joint injection [5.0mL mixture of 40mg triamcinolone (1.0mL) and 1% lidocaine (4.0mL)], a course of physical therapy, and was referred to orthopedic surgery for further assessment.

On subsequent evaluation 3 months later, the patient revealed approximately 1-1.5 years of left hip pain, followed by a recent, dramatic decrease in ROM with no specific inciting event. The patient endorsed diffuse hip region pain, worse with activities, especially squatting and movements involving external rotation. The patient noted small improvements with physical therapy and modest relief with the previous hip injection. On examination, the patient was nontender to palpation, neurovascularly intact with 5/5 strength bilaterally (flexion, abduction, and adduction),

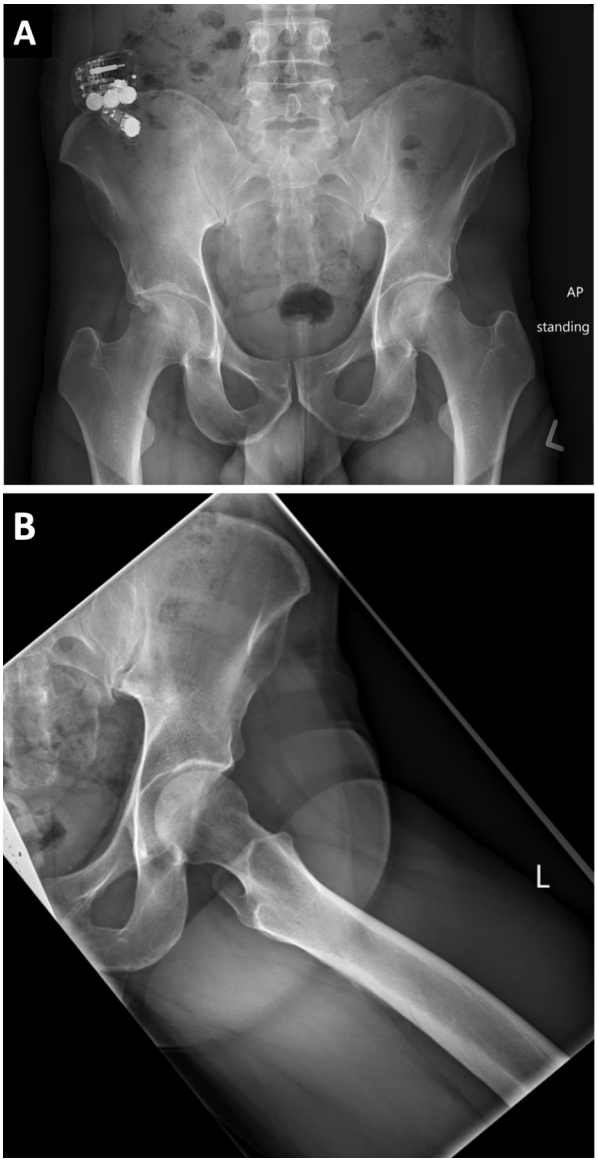

Figure 1. Pelvic Radiographs

A) Anterior-Posterior B) Left Dunn View

and a normal lumbosacral spine examination. The patient demonstrated reduced left hip ROM (FF:85°, IR:10°, ER:15°) compared to the contralateral side (FF:115°, IR:15°, ER:55°), especially in forward flexion and external rotation. Functional assessment maneuvers [i.e., FABER (flexion, abduction, and external rotation) and FADIR (flexion, adduction, and internal rotation)] were unable to be performed due to limited hip ROM, however resisted straight leg raise and log roll tests were negative. Overall, given the patient's medical history (DM and previous ACS), laboratory results, physical exam findings and functional limitations, the patient was diagnosed with adhesive capsulitis of the hip. The patient subsequently underwent a second image-guided intraarticular hip injection [5.0mL mixture of 40mg methylprednisolone (1.0mL) and 0.2% ropivacaine (4.0mL)].

At 6-week follow-up, the patient endorsed subjectively improved left hip ROM and decreased pain. He was nontender to palpation and demonstrated increased forward flexion and external rotation (FF:110°, IR:10°, ER:30°; ΔFF:

Figure 2. Magnetic Resonance Arthrogram Demonstrating an Anterosuperior Acetabular Labral Tear

A) Sagittal T1 FS B) Coronal T1 FS

+25°, ΔER: +15°) compared to the prior clinic visit. Furthermore, the patient had 5/5 strength (flexion, abduction, and adduction), bilaterally. Given the resolving symptoms following the second hip injection, the patient was recommended continued observation with regular clinic follow-up. At 4-months follow-up, the patient continued to endorse subjectively improved left hip ROM, decreased pain, and improved function in daily activities. Physical exam revealed further improvement in forward flexion and external rotation (FF:115°, IR:10°, ER:40°; ΔFF: +30°, ΔER: +25°) compared to his initial presentation.

Ethical Considerations: Case reports are deemed to be exempt by our Institutional Review Board. The patient was informed that data concerning the case would be submitted and he provided written consent for this publication.

PREVALENCE AND REVIEW OF THE LITERATURE

Since Caroit et al[1] originally reported ACH, the literature has continued to be sparse with mostly case reports and small case series. Specifically, fourteen years after ACH was described, Murphy et al[16] reported three patients who presented with chronic hip pain and restricted ROM, along with normal joint space and regional osteopenia noted on standard radiographs. While all these patients were clinically diagnosed with ACH, two subsequently underwent total hip arthroplasty (THA) after they were identified to have filling defects and cartilage excoriation, and the remaining patient's symptoms were partially relieved with an L5-S1 partial laminectomy and disc excision.[16] Thus, underlining the importance of ruling out secondary contributors prior to diagnosing ACH.

Four years later, Lequesne et al[6] became the first to utilize contrast arthrography to both quantify the articular capacity of the hip and serve as a method of treatment for ACH by expanding the capsule. In these patients who were absent of concomitant cartilage pathology, the contrast injection was noted to significantly increase their articular capacity and provide symptom improvement.[6] Although the period of pain relief was highly variable for study subjects (ranging from none to 18 months of relief), the study remains the first to utilize contrast injections to prove that there is a significant reduction in hip volume in patients with idiopathic ACH.

Throughout the 1980s, there were only two case series featuring ACH.[4,15] Griffiths et al[4] became the first to report on low-velocity, post-traumatic ACH, which is a well-established etiology for ACS.[17] The four included patients presented with unremarkable standard radiographs, and the diagnosis was made following arthrography that identified relatively low synovial fluid volume (<8mL) and high intracapsular pressure.[4] Secondly, Chard et al[15] reported three cases of idiopathic ACH, each underwent initial diagnostic testing, including hip aspiration, but were subsequently found to have resolution of symptoms after 6-12 months with no further interventions.

ACH continued to be an underreported condition in the 1990s, with only two additional case reports published.[18,19] However, Byrd et al[3] provided a breakthrough study in 2006 with the first clinical case series featuring arthroscopic examination along with intervention to manage patients with ACH. Like ACS, Byrd et al[3] visualized joint capsule thickening, fibrosis, and adherence of the joint capsule to itself. The included patients subsequently underwent manipulation under anesthesia, which allowed them to regain a mean of 25° of rotational motion.[3] Moreover, for patients without baseline degenerative changes, their functional outcomes significantly improved by nearly 32 points (modified Harris Hip Score).

Notably, since the aforementioned publication, the presence of ACH in the literature has gradually increased.[8,13,20,21] From 2008-2012, four case reports were published featuring patients who were clinically diagnosed with ACH and managed with conservative therapy, which included anti-inflammatory medications, corticosteroid injection(s), and physical therapy.[5,22–24] Patients who failed a 12-month course of conservative treatment were then treated with manipulation under anesthesia, possible pressure dilation (infusion of isotonic sodium chloride and contrast medium under fluoroscopy), and an additional course of physical therapy. While providers could rely on patterns

of diagnosis and treatment from prior case reports and small case series, the literature remained limited with respect to prospective studies evaluating the optimal management of these patients.

Lee et al[25] published the first large cohort study in 2019 addressing the diagnostic challenge of ACH. By utilizing computerized tomography arthrography (CTA), the authors demonstrated that hips of ACH patients have a lower anterior-posterior filling-ratio and smaller anterior joint cavity when compared to a cohort of age and sex-matched controls. These findings were likely a result of joint contracture and adhesions, which is analogous to the loss of axillary recess that can be found in many patients with ACS.[17]

Kim et al[26] reported the first large case series aimed at treatment strategies for ACH by evaluating 50 hips treated nonoperatively with or without an active stretching exercise regimen. While an active stretching regimen may have accelerated recovery in certain patients, Kim et al[26] found that a majority of ACH patients will recover to their baseline within 2 years irrespective of stretching regimen. However, the external validity of this study remains in question, as 22% of included patients had osteoarthritic changes, which may invalidate the diagnosis of ACH in nearly one quarter of the cohort.

Conversely, Yoon et al[21] investigated the utility of ultrasound-guided intra-articular hydrodilation with a mixture of 0.5% lidocaine (25mL) and triamcinolone (40 mg; 1 mL). Patients experienced significant improvements in patient-reported outcome measures (PROMs) and ROM across multiple planes at 6-month interval follow-up. However, 7 of the 93 patients reported recurrence of symptoms within a mean of 4.1 months, which lends credence to the previous notion that hydrodilation may just serve as a temporary solution.[2]

Lastly, Lim et al[20] reported 2-year follow-up results for conservative management (physical therapy and NSAIDs +/- muscle relaxants) versus arthroscopic surgery (capsulotomy +/- debridement and/or synovectomy) to address ACH. The surgical group reported a significantly greater mean reduction in VAS pain scores at two-week, six-week and 24-month follow up ($p = 0.001$, $p = 0.023$, and $p = 0.041$, respectively), along with improvement in hip external rotation at six weeks postoperatively ($p<0.05$). While these results are promising, the findings are susceptible to significant selection bias as groups were not randomized, VAS pain scores were significantly different only at certain timepoints, and no significant difference in follow-up University of California, Los Angeles (UCLA) activity scale or modified Harris Hip Score were found between cohorts.

PATHOPHYSIOLOGY AND CLINICAL PRESENTATION

Due to the paucity of ACH in the literature, few studies have been able to evaluate the definitive pathophysiology. Nevertheless, due to the similarities between ACH and ACS in terms of clinical presentation, findings upon arthroscopic visualization, and prognosis, many have suggested that the two conditions share a common pathophysiology.[2,14]

Four stages of ACS have been well described in the literature, each of which lie on a spectrum between an inflammatory synovitis without adhesions to substantial fibrosis with an abundance of adhesions.[27] Specifically, *Stage 1*, colloquially referred to as the "painful" stage, features a hypervascular response with perivascular lymphocyte infiltration.[2] At this point, the capsule is grossly inflamed and begins to undergo hypertrophy. Clinically, patients in this stage will experience nonspecific pain without any loss of motion. Patients with *Stage 1* ACS will commonly report pain with overhead movements and while sleeping on their affected shoulder at night, while those with ACH have been documented to report difficulty sitting on the floor, painful limitations with pivoting and sudden rotations of the hip, and an unclear association with nighttime symptoms.[26] Additionally, in a case report of a patient with ACH and ACS, the patient hypothesized that their hip symptoms were better tolerated overall– potentially a result of the smaller hip ROM required for walking/sitting versus the larger shoulder ROM needed to perform normal activities of daily living.[22]

Patients with capsulitis will often progress from *Stage 1* to *Stage 2*, the "freezing" stage, within 3 months.[2,14] *Stage 2* typically lasts from 4 to 9 months and pathologic features include continued synovitis with disorganized collagen deposition that forms early adhesions.[14] For ACS, these early adhesions cause obliteration of the inferior capsular fold, which decreases the amount of contrast that can be injected into the joint. Patients in *Stage 2* will continue to endorse pain as their main complaint; however, they will start to notice decreased ROM as the capsule begins to contract. In ACS, contraction of the coracohumeral ligament commonly leads to loss of ROM first affecting external rotation and later abduction.[28] Conversely, when examining for ACH, the findings will often be nonspecific as the loss of ROM could be confounded by secondary osteoarthritis or femoroacetabular impingement.[2,17,27] Thus, it is essential to corroborate physical examination findings with an accurate history, as timing of symptoms and progression over months may allude to the current stage of pathology.

Stage 3 is termed the "frozen" stage and is defined by substantial fibrosis with declining synovitis relative to *Stage 2*. With dense and hypercellular collagenous tissue throughout the capsule, patients will report significant stiffness with minimal pain and examination would likely reveal limitations in ROM in all directions. However, stretching the joint beyond the limits of the scarred capsule may result in significant, sudden pain, which could be a common complaint of patients who are trying to regain function in physical therapy. Lastly, *Stage 4*, the "thawing" stage, is marked by the absence of synovitis and the presence of fully mature adhesions.[17,29] Eventually, breakdown of the fibrotic tissue occurs with or without specific intervention. The natural history of ACS has been well described from *Stage 1* to *Stage 4* and may take anywhere from 6 months to 18 months in most patients; however, those with insulin-dependent DM, non-insulin-dependent DM, or thyroid disease may take as long as 24 months.[30,31] Conversely, the natural history of ACH has yet to be defined

Figure 3. Coronal T2 FS Magnetic Resonance Imaging Demonstrating a Thickened Hip Joint Capsule (red arrows)

in current literature and while parallels exist to the pathophysiology of ACS, further studies are needed to understand the etiology and disease progression within the hip.

Of note, a significant association between Dupuytren's disease and adhesive capsulitis led many to question the role of inflammation in ACS.[32] However, biopsy specimens at various stages of ACS have demonstrated a clear progression from perivascular mononuclear inflammatory infiltrates to reactive capsular fibrosis, which is likely driven by increased levels of transforming growth factor-beta and other proinflammatory cytokines that have been found in capsular tissue.[33,34] However, analogous findings have yet to be reported in ACH.

DIFFERENTIAL DIAGNOSIS

Based on the existing literature, ACH is a relatively rare condition and remains a diagnosis of exclusion. Moreover, the clinical evaluation of ACH may be further supported by the incorporation of radiographs, MRI, and laboratory tests (**Figure 3**). The differential diagnoses for hip pain and restricted ROM can be broadly divided into infectious, inflammatory, mechanical, and neoplastic conditions.[35]

Infectious causes may include septic arthritis, Lyme disease, osteomyelitis of the femoral head or pelvis, psoas abscess, and referred pain from appendicitis or an abdominal abscess. While most of these conditions will present acutely, healthcare providers should avoid overlooking the possibility of indolent infections as they can have devastating downstream effects. Inflammatory causes may include transient synovitis, systemic arthropathies, idiopathic chondrolysis of the hip, and chronic recurrent multifocal osteomyelitis. Importantly, laboratory tests such as complete blood counts, C-reactive protein, erythrocyte sedimentation rate, rheumatoid factor, and chemistry panels can aid in the diagnosis or exclusion of the infectious and inflammatory causes of hip pain. In patients with fevers/chills, elevated inflammatory markers and/or evidence of joint effusion on imaging, an arthrocentesis of the affected hip with subsequent cultures and cytology should be considered to rule out cases of indolent, sub-clinical infections. Mechanical conditions may include femoroacetabular impingement, developmental dysplasia of the hip, acetabular labral tears, avascular necrosis, stress fractures, Legg Calvé-Perthes, and slipped capital femoral epiphysis, all of which can be diagnosed with either standard radiographs and/or magnetic resonance imaging (MRI). Lastly, neoplastic conditions may include osteoid osteoma, primary or metastatic tumors, leukemia, and pigmented villonodular synovitis.

In addition to many of the abovementioned pathologies, adult hip pain should also raise suspicion for other causes such as osteoarthritis, referred pain from sacroiliac osteoarthritis or lumbar spine degenerative disk disease, muscle strains, trochanteric bursitis, intraarticular loose bodies, complex regional pain syndrome, and hernias.[36] Additionally, in female patients, ovarian cysts and ectopic pregnancies can present with referred hip pain.

However, many conditions present with nonspecific hip pain, offering a unique challenge for healthcare providers as ACH can present with varying levels of pain and ROM restrictions depending on the maturity of the disease. While previous studies have utilized arthrography to both diagnose ACH as well as eliminate other causes of hip pain, it can be difficult to justify using an invasive and costly procedure that may not ultimately alter the course of management. Thus, a proper diagnosis of ACH hinges on the ability of healthcare providers to gather an accurate history featuring acute non-specific hip pain that progresses to restricted ROM, typically most prominent in external rotation.

TREATMENT AND MANAGEMENT

Once a diagnosis of ACH is established, patients should be thoroughly educated regarding disease progression, pathophysiology, and treatment timeline. Clinicians should emphasize that treatment often does not involve surgical intervention and symptoms are likely not representative of joint damage, but rather an inflammatory process. Additionally, for patients with comorbidities that have been associated with ACH, such as thyroid dysfunction, DM, or the utilization of certain medications, clinicians should diligently address the patients' underlying medical conditions before entering nonoperative therapy. For example, in a case report of an HIV-positive patient with simultaneous ACH and bilateral ACS[12] that was being treated with elvitegravir and cobicistat (anti-retroviral medications that have been previously associated with ACS[37]), cessation of both medications led to rapid improvement of symptoms without the need for further treatment.

For patients with ACH, conservative management is often in the form of physical therapy combined with a home exercise program, although the efficacy, timing, and mode of manual therapies remains controversial.[38] While many reported cases of ACH have spontaneously resolved with physical therapy, an evidence-driven rehabilitation protocol or home stretching regimen has not been universally established.[15,23,39] Rather, many providers utilize the prin-

ciples of ACS therapy, which rely on anti-inflammatory modalities for patients in *Stage 1* and emphasize a transition to therapeutic exercises and mobilization in *Stages 2* and *3* to improve pain, ROM and overall function.[40] Additionally, targeted stretching exercise programs have shown to be an effective treatment during *Stage 2* and may contribute to subsequent adhesion breakdown for patients transitioning through the "frozen" and "thawing" stages.[2,17,41] However, it is important to note that avoiding aggressive stretching in the acute stages is imperative to prevent further inflammation that may provoke pain and contribute to further functional limitations. Thus, it is our recommendation that patients should avoid physical therapy and ROM exercises in the acute, 'painful' stages of ACH, and implement manual modalities of treatment once the acute, inflammatory phase has subsided.

NSAIDs are recommended in all stages of disease to treat daily pain and inflammation for patients without medical contraindications. Additionally, intraarticular corticosteroid injections may be used as part of conservative therapy for ACH (precautions for patients with DM and glaucoma). While corticosteroid injections have not been found to effect long-term outcomes, they have been associated with clinically significant pain relief when compared to placebo in the acute stages of disease progression.[42,43] Moreover, intraarticular corticosteroid injections combined with local anesthetic may serve a diagnostic purpose, as some studies have shown post-injection pain relief to be correlated with intraarticular pathology rather than referred pain from the lumbar spine or adjacent musculature.[2,14,44–46] The authors recommend offering an image-guided injection with the lowest effective dose of corticosteroid combined with local anesthetic in the early, painful stages to disrupt the inflammatory process, along with assisting in discriminating between *Stage 1* and *2* of the disease process.[45] Importantly, no consensus has been reached on the ideal dosage or volume for intraarticular injections of the hip.

The indications for surgical intervention are patient specific and no clear guidelines exist within available literature. However, patients who fail to improve with conservative therapy may undergo more aggressive interventions including pressure dilation, manipulation under anesthesia, synovectomy, and capsular release.[2,14,24] Pressure dilation is both diagnostic and therapeutic and has been shown to significantly decrease pain and increase ROM.[21] Similarly, manipulation under anesthesia improved rotational motion and functional outcomes for patients without degenerative changes.[3] For elderly patients with refractory symptoms following minimally invasive treatment, total hip arthroplasty (THA) is a strategy that has been successfully implemented in a small number of patients.[14]

SUMMARY AND CONCLUSION

As a rarely reported and likely underdiagnosed condition, ACH continues to pose a noteworthy challenge for healthcare providers. Since originally being described in 1963,[1] the literature has continued to be sparse, leading to inconclusive diagnostic parameters and treatment guidelines that have largely been extrapolated from widely available, yet controversial ACS literature. To date, ACH has largely been established as a mimic of ACS with a disease progression that occurs in defined phases, ultimately resulting in restricted ROM of the hip. *Stages 1* and *2* represent the acute and most painful phases of the disease, with the later *Stages 3* and *4* representing the progression to chronic ACH. As demonstrated by the included case presentation, ACH is a diagnosis of exclusion after effectively ruling out other intraarticular pathology including infectious, inflammatory, mechanical, and neoplastic conditions.[1–3,14] Moreover, at this point, the clinical workup may be further supported by the incorporation of radiographs, MRI, and laboratory tests.

Initial conservative treatment of ACH commonly includes appropriate management of related comorbidities, pharmacologic interventions, and physical therapy. While etiology of ACS has been strongly linked to thyroid dysfunction and DM, our case presentation adds to the slowly growing body of evidence that endocrine pathologies may contribute to the ACH disease process.[31,47] Specifically, the relationship to DM was demonstrated in that our patient had concomitant ACS with his presentation of ACH. Going further, while frequently resolving with time, refractory ACH may be addressed with more aggressive approaches, including pressure dilation, manipulation under anesthesia, and, for select patients, THA.

Given the outdated and overall paucity of literature regarding the diagnosis and treatment of ACH, this review provides healthcare providers with an updated approach for the management of ACH. Furthermore, the case presentation illustrates the significance of certain patient factors, along with outlining the diagnostic evaluation process and treatment strategies for addressing ACH. As the true prevalence of ACH becomes further elucidated, future high-level studies are needed to create specific diagnostic parameters and evidence-based treatment methods.

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