Adhesive capsulitis of the hip: A case report: An entity in question

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ABSTRACT

Very little has been discussed in the medical literature concerning adhesive capsulitis of the hip (ACH). There are no articles to date in the physical therapy literature regarding ACH and only a dozen or so in medical journals. Evidence suggests ACH may present in a similar progression through four stages as adhesive capsulitis of the shoulder (ACS) (from synovial inflammation to capsular fibrosis). Consensus does not exist for management of ACS or ACH. However, most clinicians agree that treatment should be guided by the stage of the disorder, whether medically, surgically, or through physical therapy. A large part of the confusion for management of adhesive capsulitis (ACS and ACH) is due to the many studies that have not reported their findings by stage. Arthroscopy and synovial/capsular biopsy can confirm the presence and stage of adhesive capsulitis. Primary (idiopathic) ACH is proposed to be caused by biomechanical dysfunction in the hip or other joints related to the mechanical function of the hip. The treatment for stages 1 and 2 consists of using techniques to reduce inflammation and correct biomechanical faults that affect the hip. In stages 3 and 4 treatment focuses on the biomechanical dysfunction of the spine, hip(s), pelvic ring, and lower limb, if needed. In this case, the 55-year-old female patient presenting with probable stage 3 adhesive capsulitis, responded well to manual therapy and has been able to return to functional activities and maintain them with a home program.

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1. Introduction

Adhesive capsulitis has been defined as a condition that begins with synovial inflammation and ends in capsular fibrosis (Rodeo et al., 1997; Kabbabe et al., 2010; Neviaser and Hannafin, 2010; Neviaser and Neviaser, 2011). It is also described as a continuum of four stages (Hannafin and Chiaia, 2000). Hsu et al. (2011) reports the association between ACS and other diseases/conditions. This has not been discussed in the literature for the hip.

Caroit et al. (1963) introduced the concept of ACH. Since then only a dozen or so articles have been published referring to this diagnosis (Murphy et al., 1977; Lequesne et al., 1981; Griffiths et al., 1984; Chard and Jenner, 1988; Luukkainen and Asikainen, 1992; Modesto et al., 1995; Mont et al., 1999; McGrory and Endrizzi, 2000; Byrd and Jones, 2006; Joassin et al., 2008; Luukkainen et al., 2008). Adhesive capsulitis of the hip has also been referred to as "frozen hip" (Chard and Jenner, 1988) or "capsular constriction" (Lequesne et al., 1981). Two cases of adhesive capsulitis of the ankle have been reported (Goldman et al., 1976; Griffiths et al., 1984), as well as one in the wrist (Hanson et al., 1988). Many articles have addressed this condition in the shoulder with recent reviews by Neviaser and Hannafin (2010), Hsu et al. (2011), and Neviaser and Neviaser (2011). Hannafin and Chiaia (2000) and Kelley et al. (2009) discuss treatment approaches to ACS that include physical therapy and medical/surgical interventions based on the stage of the condition.

Shoulder arthroscopy and biopsies of the synovium and capsule have given physicians the ability to diagnose ACS and its corresponding stage (1–4) (Rodeo et al., 1997; Sakai et al., 2001; Oehler et al., 2002; Mullet et al., 2007; Kabbabe et al., 2010; Neviaser and Hannafin, 2010; Hsu et al., 2011; Neviaser and Neviaser, 2011). Arthroscopic appearance and biopsy have also been used to diagnose ACH (Byrd and Jones, 2006). Similar to ACS, the synovium and capsule of ACH have either a classically inflamed or fibrosed appearance depending on the stage of the disorder which can be observed during hip arthroscopy or open surgery. Biopsy of the synovium and capsule can show the presence and concentration of cytokines revealing inflammation and fibrosis on the histological level. Cytokine levels in synovial fluid aspirations have also been shown to confirm the presence of ACS (Mullet et al., 2007). A detailed discussion of the arthroscopic appearance and histological findings of ACS per stage can be found elsewhere (Hannafin and Chiaia, 2000; Kelley et al., 2009; Neviaser and Hannafin, 2010; Hsu et al., 2011; Neviaser and Neviaser, 2011).
In ACH, testing often reveals osteopenia of the hip. Other diagnostic tests are most often negative, unless other pathology is present such as labral tears, chondral injury, and tears of the ligamentum teres (Byrd and Jones, 2006). Unless ACH is diagnosed through surgery or biopsy, clinicians must rely on the patient’s history and clinical findings to diagnose ACH and aim treatment at the underlying pathology or pathologies. The literature for ACH does not discuss the findings by stage, nor does it report the natural progression of the disorder. However, clinical characteristics are proposed to be similar to those of ACS and are listed by stage (see Table 1).

The medical literature discusses differentiation between primary (idiopathic) and secondary ACS (Hannaﬁn and Chiaia, 2000; Neviaser and Hannaﬁn, 2010; Hsu et al., 2011). Hsu et al. (2011) deﬁne primary ACS as adhesive capsulitis that is present without known etiology or concomitant pathology. If adhesive capsulitis is present and a second pathology is already known, then the clinician should treat both diagnoses appropriately. The complex presentations of secondary ACH can be challenging and may require further diagnostic testing.

The literature of ACS and ACH does not discuss biomechanical dysfunction as a possible etiology, although one article on ACH reports the presence of back pain in 3 case studies (Joassin et al., 2011; Hsu et al., 2011; Hsu et al., 2011). Evidence in the research shows interrelationships between the hip and the lumbar spine (Ellison et al., 1990; Sims, 1999; Lee and Wong, 2002; Shum et al., 2005; Hwang and Kim, 2009; McGregor and Hukins, 2009; Carvalhais et al., 2011). Primary ACH is proposed to be caused by biomechanical dysfunction in the hip and/or in the joints related to the mechanical function of the hip, including the spine, sacroiliac joint, and lower limb joints. It would be interesting and informative to evaluate joint biomechanics, muscle weakness/muscle imbalances, pain or reﬂex inhibition, and leg length discrepancies when considering the etiology of any adhesive capsulitis. In addition to these, immobility should also be considered as a factor, as it has been shown to lead to joint contractures produced by the same cytokines that lead to capsular ﬁbrosis in adhesive capsulitis (Hagiwara et al., 2008).

1.1. Case history

At the initial visit in October 2009 the patient was a 55-year-old female who gave a ﬁve-year history of right hip pain that progressed to bilateral hip pain (see Fig. 1). The patient’s right lateral hip pain began suddenly while the patient was sleeping. The patient described the initial pain as a constant throbbing pain s that was felt only at night, but it gradually began to include times while driving and sitting. At some point the pain progressed to include the left hip and to include episodic sharp pain. The patient described the pain as being worse in sitting and driving and lessened with standing. The patient’s pain location began at her lateral hip region and progressed to anterior hip region. Several months prior to initial consultation with the author the patient’s pain had changed again, but to the groin region (see Fig. 1). This included episodes of sharp pain with certain hip movements.

The patient’s past medical history included an episode of low back pain 20 years prior to initial consultation. The patient had been reaching for her young son when she felt immediate low back pain. She was diagnosed with a “pulled muscle” and prescribed valium and bed rest. The patient had only one recurrence of low back pain before she began swimming, bicycling, and walking for exercise. When she was 26-years-old and working as a nurse, the patient started wearing orthotics for foot pain. A podiatrist prescribed a 1/3 inch heel lift for her left lower extremity shortly after the patient developed the hip pain. Over-the-counter Ibuprofen was ineffective for her hip pain. Diagnostic testing by her physician included X-rays and bone density testing. The X-rays of her hips showed “normal wear-and-tear” and the bone density testing was “within normal limits.” The patient had seen a different physical therapist several years after initial onset. The treatment from the prior physical therapist consisted of stretching of her hips and knees and performing leg lifts which had minimal effect.

1.2. Objective examination

The patient was 5’4” and weighed 155 pounds. Observation of the patient in standing demonstrated moderate spinal lordosis with convexity to the right in the thoracic spine and convexity to the left in the lumbar spine. The patient also had increased lumbar lordosis and increased right foot pronation. The patient’s scoliosis was diminished with a heel lift under her left foot.

Observation of the patient’s gait revealed increased internal rotation of both femurs throughout the gait cycle accompanied by circumduction of the hips. Neurological testing, which included sensory, motor, reﬂex, and Babinski/Ankle Clonus testing was negative. The range of motion of the lumbosacral spine demonstrated signiﬁcantly impaired ﬂexion with an inability to ﬂatten the lumbosacral lordosis.

The range of motion of the patient’s hips was tested using combined movement tests. The patient’s hip range of motion was signiﬁcantly reduced bilaterally in combined extension/external rotation and ﬂexion/adduction/external rotation (see Table 2). The strength of the right hip abduction/extension was 4/5, compared to the left which was 4+/5.

Table 1
Clinical characteristics of ACH by stage.

<table>
<thead>
<tr>
<th>Stage</th>
<th>Symptoms</th>
<th>Irritability</th>
<th>Range of motion</th>
<th>End feel</th>
</tr>
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<tbody>
<tr>
<td>Initial Stage</td>
<td>Gradual onset of pain, achy at rest and often sharp with movement. Night pain is common. Often patients unable to sleep on affected side. Patients describe limitation of motion or limitation of functional activities.</td>
<td>High</td>
<td>When range of motion (ROM) is performed when the patient is under anesthesia there is no, or very little, range of motion deficit.</td>
<td>Empty</td>
</tr>
<tr>
<td>Freezing Stage</td>
<td>Pain is constant and can be more severe, particularly at night.</td>
<td>High</td>
<td>Moderate range of motion limitation. When ROM is performed under anesthesia it is the same or almost the same as when the patient is awake.</td>
<td>Pain is felt prior to resistance or end feel.</td>
</tr>
<tr>
<td>Frozen Stage</td>
<td>Primary complaint is stiffness. Pain occurs at end of motion.</td>
<td>Moderate</td>
<td>Significant range of motion limitation. When ROM is performed under anesthesia there is no improvement with testing.</td>
<td>Abnormally firm capsular end feel. Resistance met prior to pain.</td>
</tr>
<tr>
<td>Thawing Stage</td>
<td>Patients report minimal pain. Patients can have a gradual return to functional activities.</td>
<td>Low</td>
<td>Can have a gradual improvement in range of motion.</td>
<td>Similar to Stage 3.</td>
</tr>
</tbody>
</table>
Joint mobility testing showed decreased arthrokinematic glides in multiple joints including the thoraco-lumbar junction and segments L3-S1. The patient’s hips had decreased joint glides, particularly in the posteromedial direction.

Palpation of the lumbar spine and hips was significant for tightness and tenderness of the paravertebral muscles, the adductor muscles, the anterior hip musculature, and the iliopsoas muscles bilaterally.

The following tests were negative: provocative sacroiliac joint stress tests, dural testing – slump and SLR, posterior-to-anterior pressure on the spinous processes, and axial compression/distrac-

tion of the spine.

1.3. Treatment

The patient was seen 9 times per year over a two-year span. The following is a list of the exercises and techniques used for her treatment.

1. Joint mobilization of the spine and hips. The mobilization of the spine using grade 4/4+ techniques focused on reducing the patient’s scoliosis. The mobilizations of the hips were performed using a sustained technique for 5–7 min for the posterior and posteromedial glides, to the patient’s tolerance. While this technique was being held the patient was instructed to “make her leg longer” and then relax. Combined range of motion testing was repeated at the end of each treatment to note the improvement in range of motion and symptom reduction.

2. Soft tissue mobilization of the paraspinals, the hip muscula-

ture, and the iliopsoas muscles.

3. Strengthening exercises including core strengthening and specific hip strengthening exercises. The hip strengthening exercises consisted of standing hip extension/abduction with a circle of grey theraband around the ankles. The patient was instructed to perform these exercises on both sides daily.

4. Hip stretching exercises were given to the patient to perform at home. These included stretching into hip abduction/external rotation, extension/external rotation.

5. The patient was instructed in self-massage techniques to perform daily to address muscle tightness.

6. Posture instruction was given as well, focusing on minimizing the lumbar hyperlordosis.

By the end of the second course of treatment the patient had a good understanding of how to self-manage her condition and was performing her home program consistently. Even so, she continues to need further manual therapy treatment approximately once every 3 months when the sharp pain returns. The patient has been

<table>
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<th>Passive range of motion</th>
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<tr>
<td>Flexion/adduction/internal rotation</td>
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<tr>
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<td>Extension/external rotation</td>
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<td>Flexion/extension</td>
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<tr>
<th>End feel</th>
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<tbody>
<tr>
<td>Capsular</td>
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<tr>
<td>Abnormally firm capsular</td>
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<tr>
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<td>Capsular</td>
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<table>
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<tr>
<th>Symptoms</th>
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<tbody>
<tr>
<td>Pinching in groin</td>
</tr>
<tr>
<td>Pain in groin and anterior hip</td>
</tr>
<tr>
<td>Pain in groin and anterior hip</td>
</tr>
<tr>
<td>No symptoms</td>
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able to continue with ADLs without problems as long as she stretches 2×/day and performs exercises as given by the therapist. The patient swims 3×/week, uses a stationary bike for 30 min 3×/week, and limits walking to 15–20 min/day. She has pain only after prolonged sitting that she rates as 5/10 on a pain scale and describes as “being aware of soreness.”

2. Discussion

The patient in this case was treated for Stage 3 ACH, as determined by the following clinical signs and symptoms: pain history indicating low irritability, significantly reduced range of motion (particularly into combined flexion/abduction/external rotation and extension/external rotation), and the end feel. The end feel was abnormally firm and pain was produced after resistance was met. Since the ACH was in the fibroed stage with low irritability and inflammation, the hip was managed biomechanically including treatment for the hips, the spine, and the pelvic ring. The patient had already received a heel lift and orthotics to address leg length discrepancy and right foot over-pronation.

It is theorized that if the patient had received earlier intervention for her leg length discrepancy and scoliosis she may not have developed hip dysfunction. Or, if she had received biomechanical treatment earlier, the patient may have had full recovery or at least not required the same level of maintenance at home to prevent recurrence.

3. Summary

This case discusses adhesive capsulitis of the hip, an under-recognized entity. There has been enough evidence to support ACH including the clinical presentation, observation of the capsule on open reduction or hip arthroscopy, and through synovial/capsular biopsy. In this case report, the patient had sufficient clinical findings to support the diagnosis of stage 3 ACH and was treated biomechanically, which resulted in successful management of her symptoms and return to functional activities.

The literature is missing a discussion of the different stages and a description of the findings and treatment for each stage. However, by looking at the research on ACS, therapists can make comparisons and apply evaluation and treatment principles accordingly until the research can confirm our suppositions more definitively.

Current studies of ACS, ACH, osteoarthritis, and immobility report the presence of the same cytokines that result in fibrosis of the joint capsule. It is proposed that there are different levels of the cytokines found in these different conditions.

If future studies examine the biomechanics of the affected joint, and those joints related mechanically to the affected joint, the author believes the research will confirm that biomechanical dysfunction is the etiology in otherwise unexplained ACH or ACS (primary or idiopathic adhesive capsulitis).

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References