# ADHESIVE CAPSULITIS OF HIP: AN ENIGMA

# CAPSULITA ADEZIVĂ A ȘOLDULUI: O ENIGMĂ

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**Key words: a**dhesive capsulitis of hip; frozen hip; physiotherapy; surgery; arthroscopy; pressure dilatation

**Abstract**

Adhesive capsulitis of the hip (ACH) is considered an uncommon clinical condition but it is not so. In ACH a characteristic decrease in hip active and passive range of motion is observed; which is painful. In acute stage focus should be on decreasing inflammation by means of steroid injections, NSAIDs and physiotherapy. Also biomechanical dysfunction should be corrected for joints proximal and distal to affected hip. In chronic stage, treatment should focus on regaining range of motion through vigorous physiotherapy. In medical literature treatment described for chronic ACH include manipulation under anesthesia; pressure dilatation; and lysis of adhesions, open or arthroscopic synovectomy, and capsular release etc. A minimum of 3-months duration of conservative treatment should be tried before considering surgical option.

**Cuvinte cheie:** capsulită adezivă a șoldului, sold înghețat; fizioterapie; intervenție chirurgicală; dilatație de presiune

**Rezumat**

Capsulita adezivă a șoldului (ACH) este considerată o afecțiune mai puțin întâlnită, dar nu este adevărat. În ACH se observă o reducere caracteristică a mobilității active și pasive a șoldului, care devine dureroasă. În stadiul acut tratamentul se concentrează pe reducerea inflamației prin injecții cu antiinflamatoare steroidiene, nesteroidiene și fizioterapie. Se vor corecta disfuncțiile biomecanice ale articulațiilor porximale și distale ale șoldului afectat.

În stadiul cronic, tratamentul se va concentra pe recâștigarea amplitudinii de mișcare prin fizioterapie. În literature medicală, tratamentul descris pentru ACH cronic include manipulări sub anestezie, dilatare prin presiune, listeza aderențelor, sinovectomie deschisă sau artroscopică, și decoaptări capsulare etc. Înainte de a aborda o intervenție terapeutică în manieră cgirurgicală, se va urma un minim de tratament conservativ de 3 luni.

## Introduction

Adhesive Capsulitis of shoulder (ACS) or frozen shoulder is a well recognized clinical disorder. Surprisingly, it seems to be exclusive to the shoulder joint. The cases described by Chard and Jenner suggest that an analogous disorder may also affect the hip and is probably under-diagnosed. [1] Chard and Jenner have named this condition as frozen hip.

Adhesive capsulitis of the hip (ACH) is due to constriction of the fibrous joint capsule of the hip. Though ACH was first described by Caroit *et al* in 1963, [2] there have been very few publications on the condition since then [1, 3, 4, 5], and its exact cause still remains unknown.

Lequesne *et al* described two kinds of ACH: idiopathic ACH (e.g. due to diabetes) and secondary ACH (e.g. due to osteochondromatosis). [3, 5] The clinical assessment of ACH is similar to that for adhesive capsulitis of the shoulder (ACS) and features a combination of pain and restricted active and passive joint motion. The treatment can be by drugs mainly oral NSAIDs, intra-articular steroid and analgesic injections; physiotherapy involving mobilization and stretching; or surgical means: MUA in conjunction with pressure dilatation, open synovectomy, or arthroscopic surgery. In all cases, physiotherapy is essential for limiting functional deficits and functional impairments.

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## Aim

The purpose of this article is to review case studies or case series presented in the literature for the diagnosis of this underestimated condition [5] and examine therapeutic strategies. It is asserted that ACH is more common than suggested in the published literature, which describes only a few stray cases which may occur with or without associated intra-articular pathology.

## Aetiology, classification and pathophysiology

ACS is a common pathology, affecting between 2% and 5% of the general population. In contrast, the incidence of ACH is unknown but is probably higher than is generally believed. The condition may preferentially affect women between the ages of 35 and 50. [1, 6, 5] McGrory and Enddrizzi [4] & Griffin *et al.* [7] suggested that ACH is probably often undiagnosed or under-diagnosed because its impact on functions of an individual is less disabling than ACS. Both conditions can be idiopathic or secondary to medical or orthopedic pathologies. However, the ACS is the only one of the two that can be caused by neurological pathologies (parkinsonian syndrome, hemiplegia), cardiac pathologies (myocardial infarction) or lung pathologies (pneumonia). However, synovial chondromatosis is unique to ACH.

Lequesne *et al* proposed classifying ACH into idiopathic and secondary types: idiopathic ACH is uncommon but may follow on from diabetes mellitus or prolonged phenobarbital use; secondary ACH has five different causes, the most common of which is synovial chondromatosis. The other causes are primary osteoarthritis, osteoid-osteoma, acetabular labrum lesions and ligament lesions. [5, 6] Although many probable explanations for the pathophysiology of ACH have been proposed, (e.g. capsular fibrosis, abnormal nonenzymatic glycooxidation of proteins in diabetes), the underlying cause remains unknown.

## Clinical Presentation & Assessment

Adhesive capsulitis of the hip is characterized by progressive clinically evidenced pain and restricted passive and active joint motion. This limitation is in all three planes (flexion-extension, internal-external rotation and abduction-adduction) [5]. Shoulder arthroscopy and biopsies of the synovium and capsule have given clinicians the ability to diagnose ACS and its corresponding stage (Stage: 1-4). [8, 9] 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).

**Table 1**
Clinical characteristics of ACH by stage.

| | Symptoms | Irritability | Range of motion |
|---|---|---|---|
| Stage 1<br>Initial Stage | 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. | High | When range of motion (ROM) is performed when the patient is under anesthesia there is no, or very little, range of motion deficit. |
| Stage 2<br>Freezing Stage | Pain is constant and can be more severe, particularly at night. | High | 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. |
| Stage 3<br>Frozen Stage | Primary complaint is stiffness. Pain occurs at end of motion. | Moderate | Significant range of motion limitation. When ROM is performed under anesthesia there is no improvement with testing. |
| Stage 4<br>Thawing Stage | Patients report minimal pain. Patients can have a gradual return to functional activities. | Low | Can have a gradual improvement in range of motion. |

## Biomechanical Consideration

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. [10] Evidence in the research shows interrelationships between the hip and the lumbar spine. [11, 12]

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. [13]

**Diagnosis**

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. 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. [5]

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 at the histological level. Presence of cytokine levels in synovial fluid aspirations also confirms the presence of ACH. [9]

Adhesive capsulitis of the hip can be suspected in the absence of any acetabulo-femoral pathologies (such as osteonecrosis or severe osteoarthritis) or if there is a contrast between the most prominent symptoms and a reassuring radiological assessment. Lequesne et al. propose that radiological assessment with acetabulo-femoral injection of iodine-containing contrast medium can reveal a decrease in joint volume (below 12 ml), since the normal range is between 12 ml and 18 ml (with an average of 15 ml). [3]

Thomas Byrd and Jones consider that radiological assessment with acetabulo-femoral injection of iodine-containing contrast medium only demonstrates the presence of ACH when the joint volume is below 5 ml. When the volume is between 5 ml and 12 ml, these latter authors recommend performing a volume comparison with the contralateral acetabulo-femoral joint. If the difference between the two is at least 25%, ACH can be suspected. Thomas Byrd also suggest performing MRI of the hip in order to detect potential bone or cartilage pathologies, followed by either a therapeutic hip arthroscopy (that is to treat the causal affection identified by the MRI) or a diagnostic therapeutic hip arthroscopy (for difficult cases with normal imaging results). Arthroscopy enables confirmation of ACH associated with fibrinous and hemorrhagic fragments in the capsular recess and/or acetabular fossa. [5] Griffiths *et al* described 4 cases of ACH confirmed by arthrography. [14]

McGrory & Endrizzi reported a case of ACH in a patient with hypothyroidism and previous ACS who was receiving thyroid-hormone replacement. The orthopedic surgeons should be aware of this diagnosis and its association with ACS and thyroid dysfunction, so that they can recognize it and provide early intervention. [4]

**Differential diagnosis**

Cases of inflammatory and tuberculous hip joint arthritis are easy to diagnose with standard laboratory and radiological assessments. Complex regional pain syndrome type 1 can be suspected in case of painful joint stiffness, notably with severe osteoporosis of the upper femoral epiphysis or signs of associated neurotrophic damage. It is bit difficult to say that mild to moderate osteoarthritis is the cause of ACH or presence of ACH leads to progression of osteoarthritis of hip due to reduced acetabulo-femoral joint capacity.

A controlled, prospective study with systematic hip joint radiological assessment via intra-articular injection of iodine-containing contrast medium should be performed, in order to evaluate the joint volume in mild to severe osteoarthritis. One possible consequence of such a trial might be early implementation of physiotherapy intervention for mild hip joint osteoarthritis where the symptoms are essentially because of ACH.

**Treatment**

According to earlier reports it is resistant to conventional treatment but spontaneous resolution can be expected in periods varying from 3 to 18 months [1, 2, 3, 15].

Idiopathic ACH usually responds favorably to maintenance treatment. This treatment consists in physiotherapy and radiologically-controlled intra-articular corticosteroid injections performed once or twice a month. According to Lequesne's guidelines, the total number of injections varies between five and 12. Even though NSAIDs and corticosteroid intra-articular injections can relieve pain in cases of idiopathic ACH, long-term physiotherapy is the only way to recover joint amplitude, may go on for between five and 24 months. There is a lack of medical literature on this condition.

Mont *et al* (1999) mentioned that there were only four reports of ACH in the literature and all of them were post-traumatic. Treatment methods usually include analgesics and physiotherapy. However, resolution has been unpredictable, requiring more than 1 year in most cases. He described a case of ACH in which the patient's symptoms resolved only after open surgery [16].

The gross outline of the **physiotherapy approach of ACH** is the similar to the ACS:

(a) therapeutic ultrasound, pulsed/continuous shortwave diathermy, transcutaneous electrical stimulation etc;
(b) progressive eccentric manual stretching, depending on the pain threshold and eccentric muscular strengthening;
(c) auto-mobilization, yoga and Tai-chi for maintaining good mobility;
(d) hydrotherapy can be useful for better movement awareness.
(e) proprioceptive exercises for improving postural control and avoiding joint over-use;

The treatment is prescribed once a day for the first four weeks and then three times a week for at least two months. On average, follow-up lasts for nine months after the onset of symptoms. Secondary ACH requires immediate capsulotomy or synovectomy to treat the underlying cause. Luukkainen *et al* described a case of frozen hip that was treated with manipulation and pressure dilatation [17].

In acute stages of ACH, physiotherapist focuses on decreasing pain and inflammation via anti-inflammatory techniques, functional adaptations, correction of hip and other associated joint mechanics, and correction of muscle imbalances or deficits. Information on self-treatment and a home program are also provided. Aggressive ROM exercises should be avoided in acute stages of ACH because they tend to flare up inflammatory symptoms. Chronic ACH requires more aggressive physiotherapy techniques to improve joint mechanics and reduce the effects of capsular fibrosis. These techniques include increasing ROM through joint mobilization, soft-tissue mobilization, and stretching; strengthening of specific muscles (typically the hip abductors and extensors); and instruction on a home program of aggressive ROM, stretching, strengthening, and self-treatment techniques.

The goal of physiotherapy for chronic ACH is a return to prior functional and recreational activities. Evaluation and treatment of joints adjacent to the spine is important. If the mechanics of the hip, lumbar spine, sacroiliac joint, and lower limb joints are not evaluated, treated, and supplemented according to ACH stage-specific stretching and strengthening exercises and a home physiotherapy program, there is very less chance for improvement of joint mechanics and reduction in the progression of capsular fibrosis.

Physiotherapy can reach its maximum benefit during chronic stages of the disease. If no demonstrable improvement is achieved with the application of NSAIDs, corticosteroid injections, and physiotherapy, surgery can be considered. Surgery should be avoided in the acute stages of ACH and should be considered only after failure of a 3-month course of conservative or nonsurgical treatment. This time frame has been derived from common treatment recommendations for ACS described in the literature. The definition of failure should also be adjusted to the individual patient. As the hip tolerates decreased ROM; therefore, restoration of full ROM may not be necessary for nonsurgical management to be considered as successful.

Refractory idiopathic ACH can also benefit from arthroscopic capsular release. According to Thomas Byrd and Jones, the average time between the initial appearance of symptoms and surgery is 12 months (range: 4 to 21 months), whereas the average time between the beginning of physiotherapy and surgery is 7.4 months (range: 2 to 18 months) [5]. Thomas Byrd and Jones also recommend a combination of surgery and manipulation under general anesthesia.

If arthroscopy is required (for secondary ACH or refractory idiopathic ACH), the above-mentioned physiotherapeutic approach remains valid and should be started on the second day post-surgery. The use of crutches is recommended for five to seven days, in order to help restore normal gait.

## Conclusion

Adhesive capsulitis of the hip is probably more common than that the paucity of literature leads us to believe. This situation should alert physiotherapists to the possible diagnosis of a poorly recognized condition. Also in physiotherapy curriculum hardly any mention is there about this condition that also may be a reason for physiotherapists to remain ignorant about this condition. In acute stage effort should be on containing the inflammation; and in chronic stage our aim should be to regain the range of motion of the hip joint and strength of related muscles. Surgery would be the last option when all other conservative options are exhausted. It is not necessary to regain full ROM in the hip joint. Human body can tolerate some amount of loss of ROM in the hip because it is not so disabling for the hip for daily activities.

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