

Clinical Review of Adhesive Capsulitis of the Ankle: An introductory article and clinical review

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Adhesive capsulitis is a well known ailment that most often affects the shoulder, but can occur in the hip, wrist, and the ankle. As it relates to the ankle joint, the condition is commonly referred to as 'frozen ankle' and presents as a challenge in both its diagnosis and treatment. Although there is much literature regarding the etiology, pathology, and treatment of 'frozen shoulder', there is little with regards to the ankle, with most being case reports. The purpose of this article is to provide a clinical review of the concept of adhesive capsulitis, its impact as it relates to the ankle, diagnostic criteria, and current treatment modalities.

Key words: Adhesive capsulitis, frozen ankle, ankle pain, adhesions.

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Adhesive capsulitis is a broad term that can be confusing at times since it is often used synonymously for "frozen shoulder". In order to better understand adhesive capsulitis of the ankle, we begin with a brief review of a better documented phenomenon concerning the shoulder.

Adhesive capsulitis of the shoulder is a common condition that will cause pain and restrictions during active and passive range of motions (ROM).

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Though motion loss is global, a typical capsular pattern is usually present with motion into external rotation being most limited followed by abduction and finally internal rotation.¹

Frozen shoulder can be categorized into two categories: primary, or idiopathic, and secondary. Primary adhesive capsulitis presents without any underlying cause, while secondary adhesive capsulitis occurs as a result of other causative factors, such as trauma to soft tissue and bone. Pre-disposing factors to developing idiopathic frozen shoulder include diabetes, female, and being in the fifth or sixth decade of life. Incidence is reported as being 10-36% in the diabetic population, with a 40% chance of developing the condition if an individual is Type I diabetic.^{1,2,5}

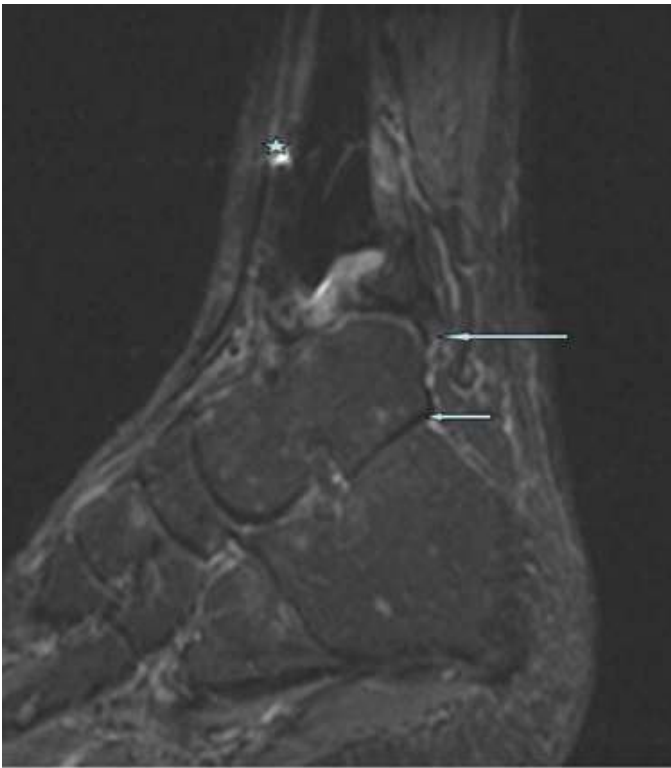


Figure 1 Sagittal FSE T2 weighted image in a patient with post traumatic and postsurgical ACA showing complete paucity of synovial fluid in the ankle joint (long arrow) and in the posterior subtalar joint (short arrow). Note magnetic susceptibility artifact (star) from surgical hardware.

For the glenohumeral joint, 3 distinct phases have been described in staging this condition. The first is an early painful phase, or “freezing stage,” with a duration of 2 to 9 months. This is followed by an intermediate stiffening or adhesive phase, which has duration of 4 to 12 months. In this phase, patients typically experience increasing stiffness, but less pronounced pain. The final phase is known as the recovery, or “thawing” phase, which lasts anywhere from 5 to 24 months. Here, patients display a gradual return of movement.^{3,4}

Adhesive Capsulitis of the Ankle (ACA) is infrequently reported in the literature, but the few reports that do exist suggest that it occurs secondary to trauma such as ankle or pilon fractures or chronic ankle sprains.

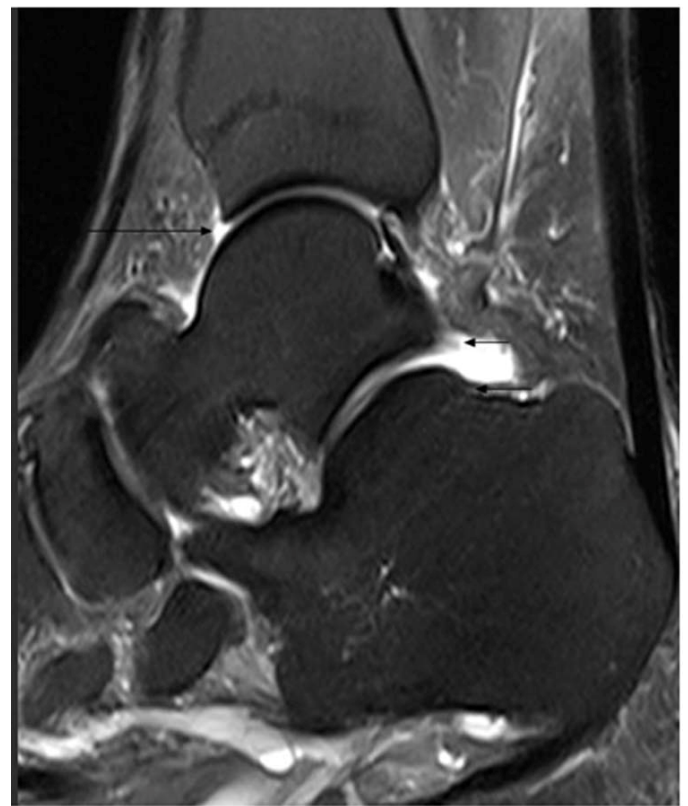


Figure 2 Normal ankle with normal fluid in the ankle (long arrow) and posterior recess of the posterior subtalar joint (short arrow).

Some cases are thought to be idiopathic, or to have some underlying association with diabetes, inflammatory arthropathies, connective tissue disease, heart disease, infection, or autoimmune processes.^{5,6,7}

Pathophysiology

With regards to pathophysiology, the literature between adhesive capsulitis of the shoulder and that of the ankle appears to be synchronous. Not only is the site of injury affected, but the entire joint capsule is disturbed, resulting in a global loss of motion.

Initially, there is proliferation of synovial inflammatory cells and infiltration of lymphocytes. Within 3 to 4 weeks, the capsular fibrous layer becomes thickened, destroying the integrity of the anterior and posterior joint recesses, and the presence of new collagen inhibits the availability of joint ROM.^{1,6,8}

This proliferation of inflammatory infiltrate and the subsequent ankle joint contracture that follows is thought to be the result of cytokines, which allow for initiation and progression of the fibrous thickening process. Patients will often report a tight, sharp stretching sensation, and they may report a cracking, or popping sound with motion of the joint.⁷

Diagnosis

Currently, there exists no clinical criteria for diagnosing ACA; however, the literature does show some similarities with respect to patients presenting with frozen shoulder. Patients usually complain of ankle pain, swelling, and difficulty walking. The onset of symptoms is variable and may present anywhere from immediately after an incident to weeks later, when the patient resumes weight bearing and ambulation after having been immobilized for an extended amount of time.^{5,6}

Observation of significant deficits of ankle ROM is the key to further investigation into a possible diagnose of adhesive capsulitis. A study by Lui, et al., recorded ankle dorsiflexion and plantarflexion in patients with diagnosed ankle adhesive capsulitis and found that ankle dorsiflexion was limited to an average of 1°, while plantar-flexion averaged 16° pre-op. In addition to limited ankle ROM, calf atrophy may also be present if there was prolonged immobilization.⁵

Currently, conventional arthrography is the most accepted diagnostic tool. In 1976, Goldman, et al., proposed arthrography criteria for diagnosing adhesive capsulitis of the ankle, which included the following:

- 1) decreased joint space volume ($n = 10-25$ mL fluid, pathologic accepts $< 3-5$ mL)
- 2) obliterated ankle recesses (anterior & posterior)
- 3) resistance with injection of contrast
- 4) backflow of contrast medium⁸

Due to convenience, and the invasive nature of the procedure, arthrography is not typically the diagnostic tool of choice amongst physicians, but has demonstrated value in evaluating shoulder adhesive capsulitis.⁹ Radiographs are nonspecific and are, therefore, of no benefit in diagnosing adhesive capsulitis.⁵

Although there is no established criteria for diagnosing ACA on magnetic resonance imaging (MRI), in the appropriate clinical setting a paucity of ankle joint fluid and thickening of the joint capsule on MRI (Figs. 1 and 2), as seen in conventional ankle arthrography and non-contrast shoulder MRI in shoulder adhesive capsulitis, will raise the suspicion for ACA. Since there is often a communication between the posterior subtalar joint and the ankle joint, there can also be a paucity of fluid in the posterior recess of the posterior subtalar joint. (Figs. 1 and 2) Further work is required to establish the role of ankle MRI in the diagnosis of ACA.^{8,9}

Additionally, a hallmark technique in the diagnosis and treatment at the level of the shoulder is the feeling of a capsular end feel with passive range of motion testing, or in stage 3; a rigid end point.¹⁰ This diagnostic exam technique along with acknowledging restricted joint play on attempted anterior, posterior, and distraction joint mobilization can be applied in the diagnostic approach to the ankle as well.

Treatment

In order to understand the treatment of the ankle, it is necessary to review how treatment is based on staged criteria for frozen shoulder. In stage 1, which involves no capsular tightness but is progressively painful, the treatment strategy consists of soft tissue mobilizations, with an emphasis on reducing inflammation as no adhesions have yet formed. In the adhesive stage 2, there is an emphasis on reducing inflammation, and minimizing capsular adhesions through active and passive range of motion exercises as well as joint mobilization techniques. In stage 3, there is an eventual slow and steady recovery of motion, with the course of disease taking anywhere from one to three years to resolve fully.^{10,11}

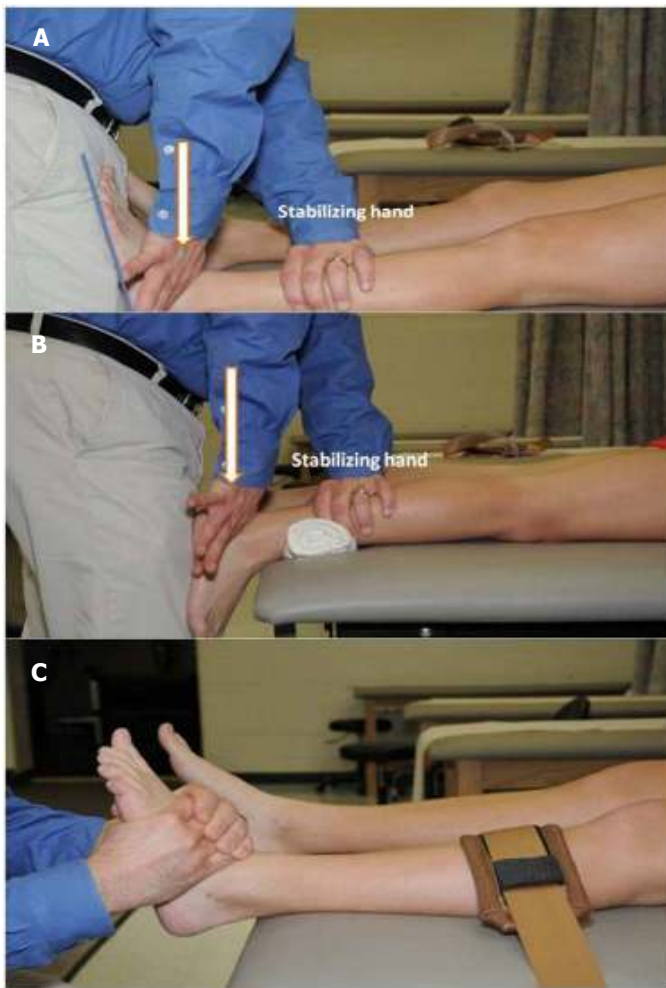


Figure 3A, 3B and 3C From top to bottom: Posterior talo-crural glide mobilization for dorsiflexion range of motion return. (A) Anterior talo-crural glide mobilization for plantarflexion range of motion return. (B) Talo-crural joint distraction mobilization. (C)

Currently, treatment options for the ankle include physical therapy focusing on active and passive range of motion as well as ankle joint mobilization techniques. (Figs. 3A, 3B and 3C) Shaffer, et al., showed that after 8 weeks of immobilization post-ankle fracture, 10 weeks of mobilization with physical therapy can successfully restore ROM, strength, and function of the ankle joint.¹² If conservative care is unsuccessful, arthroscopic debridement may be necessary. Lui, et al., found that ankle ROM may improve up to 18° with DF and up to 23° with PF post-arthroscopic debridement.

They also showed that corticosteroid injections were ineffective alone.⁵ Based on current understanding of the pathological process of ACA, Cui, et al., proposed a diagnostic and treatment algorithm which we have produced here.(Fig. 4)

Conclusion

Adhesive capsulitis of the ankle is a difficult condition to diagnose and manage. Careful review of existing literature reveals limited research on the subject, with most studies being directed towards adhesive capsulitis of the shoulder. Such shoulder pathology is host to wide range of etiologies, resulting in decreased shoulder ROM (External rotation > Abduction > Internal rotation) and increased pain along all aspects of the shoulder joint capsule.

Though adhesive capsulitis of the ankle appears to present in a similar fashion, there is currently no clinical criteria to properly diagnose or stage ACA. Today, radiographic arthrograms remain the standard for definitive diagnosis, although their use is limited due to the invasive nature of the procedure. Goldman, et al., defined ACA based on similar arthrogram findings between 3 patients suspected of ACA.

With ACA presenting similarly to the pathology seen the shoulder (decreased ROM and increased pain around the involved joint), current treatment options have followed similar suit. Currently, physical therapy, focusing on active and passive range of motion, as well as ankle joint mobilization techniques, is showing to be the most beneficial for patients identified early on.

Further controlled studies are needed in order to better properly diagnose and treat ACA. Given the serious lack of literature regarding this condition, more studies are needed and perhaps more rigorous diagnostic criteria to clinically diagnose ACA with more modern radiographic techniques.

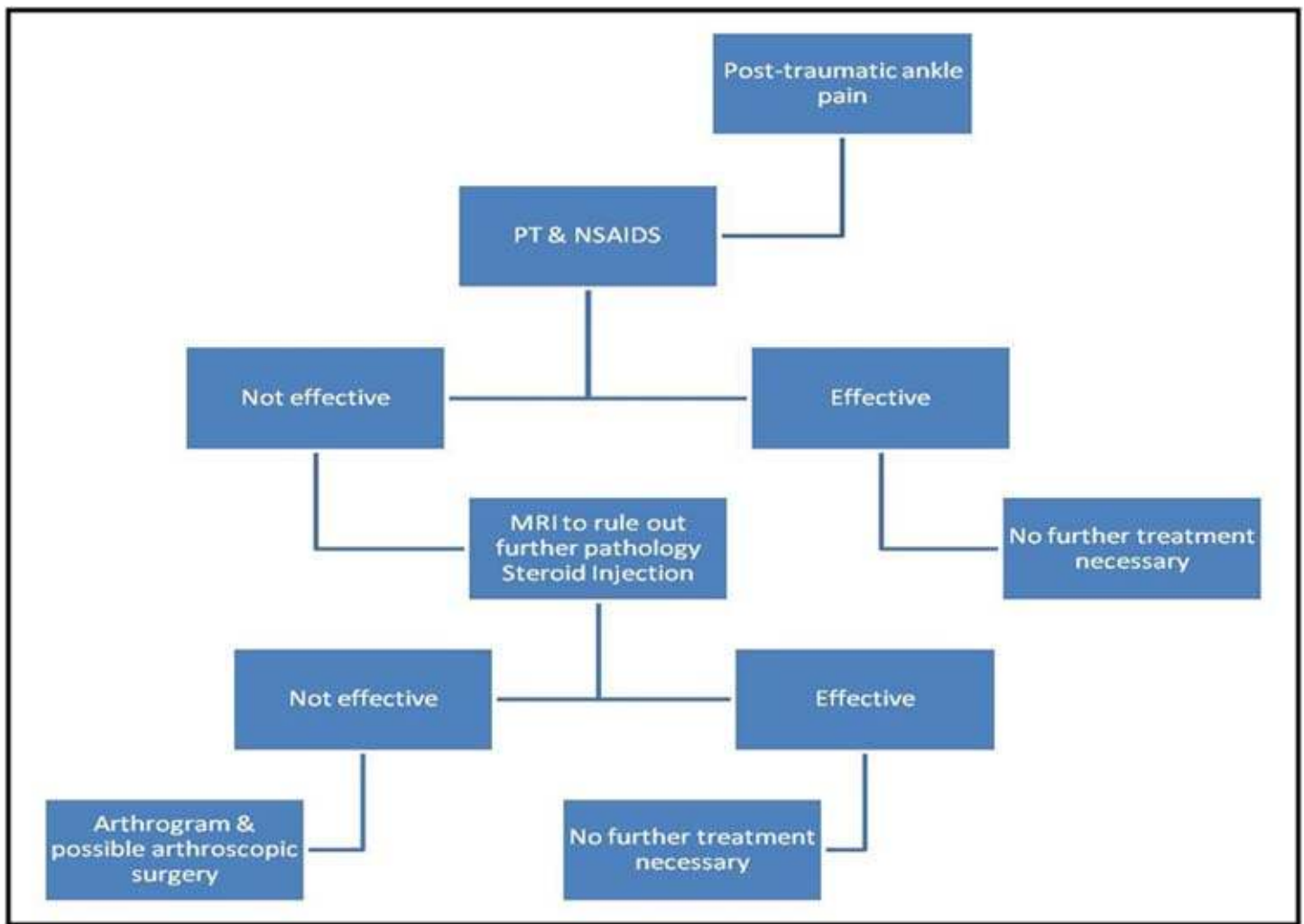


Figure 4 A diagnostic and treatment algorithm adapted from Cui, et al., Copyright © 2011 by the American Orthopaedic Foot and Ankle Society, Inc., originally published in *Foot & Ankle International*, 26(8):606 and reproduced here with permission.

More importantly, better treatment protocol can be implemented and the possibility of staging the condition would aid physicians in earlier intervention, thereby preventing progression.

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