SciMedCentral

Annals of Sports Medicine and Research

Review Article

Adhesive Capsulitis: Current Concepts

Scott M. Feeley[1]*, Eric S. Larson[2], and David R. Diduch[2]

[1]School of Medicine, University of Virginia, USA

[2]Department of Orthopaedic Surgery, University of Virginia, USA

*Corresponding author

Scott M. Feeley, School of Medicine, University of Virginia, P.O. Box 800793, Health Sciences Center, Charlottesville, VA 22908, USA, Email: smf3ab@virginia.edu

Submitted: 01 October 2017

Accepted: 12 October 2017

Published: 13 October 2017

ISSN: 2379-0571

Keywords

- Adhesive capsulitis
- Frozen shoulder
- Diabetes mellitus
- Corticosteroid injection

Abstract

Adhesive capsulitis is a common disorder of the shoulder with unknown etiology. It presents with insidious onset of pain accompanied by progressive stiffness and loss of both active and passive range of motion. Adhesive capsulitis is a diagnosis of exclusion and can only be made once other pathologies have been ruled out. The progression and risk factors associated with the disease necessitate individualized patient approaches for pain reduction and functional improvements. This review outlines what is currently understood about the progression of the disease and what treatments have been shown to be efficacious. Despite decades of research into the underlying causes, progression, and treatment, much is still unknown about the condition. The pathology and risk factors for adhesive capsulitis should continue to guide further research to better understand the triggers for adhesive capsulitis and its treatment.

ABBREVIATIONS

ROM: Range of Motion; MRI: Magnetic Resonance Imaging; Nsaids: Nonsteroidal Anti-Inflammatory Drugs; PT: Physical Therapy; US: Ultrasound; MUA: Manipulation Under Anesthesia

INTRODUCTION

Adhesive capsulitis, initially termed frozen shoulder in 1934 by Codman, is a condition of the shoulder with insidious onset of pain that progresses to stiffness. Neviaser described the condition as adhesive capsulitis in 1945 to emphasize the underlying pathologic fibrosis that leads to stiffness. Adhesive capsulitis has two forms: primary/idiopathic and secondary. While common in the general population, its incidence is greatly increased in patients with diabetes mellitus. Adhesive capsulitis was considered self-limiting with a time course under two years until recent studies showed persistent limitation in ROM and function for a large percentage of patients receiving minimal treatment. While much research has gone into the condition, the etiology of the disease and definitive clinical identifiers for its progression remain unclear.

Anatomy

Adhesive capsulitis primarily affects the capsule of the rotator interval, a triangular area that is bounded by the coracoid process, the anterior margin of the supraspinatus tendon, and the superior margin of the subscapularis tendon. The rotator interval capsule is reinforced by the coracohumeral and superior glenohumeral ligaments. Additionally, the long head of the biceps tendon travels through the rotator interval [1].

Progression

While the etiology of adhesive capsulitis is not well understood, pathology has shown the progression of the disease to begin with an inflammatory condition of the synovium followed or combined with fibrosis of the capsule [2]. Pathologically, this is exhibited by capsular thickening leading to adhesion of the capsule, synovium, and surrounding ligaments. When the coracohumeral ligament stiffens, external rotation at the glenohumeral joint becomes constrained [1]. Pain and muscular inhibition contribute to further dysfunction of the shoulder as disuse exacerbates the existing loss of ROM and ultimately immobilizes the joint [3].

Hannafin et al., categorized the progression of adhesive capsulitis into 4 stages as exhibited under arthroscopy. Stage 1 lasts from 0-3 months with diffuse, hypervascular synovitis of the capsule. Pain accompanies limited active and passive ROM that normalizes under anesthesia. Stages 2-4 describe the clinical presentation of the disease and were termed the "freezing," "frozen," and "thawing" stages. Stage 2 occurs from 3-9 months with continued synovitis and the beginning of scar formation. Chronic pain and significant limitation of ROM exist, even under anesthesia. Stage 3 occurs from 9-15 months with notable absence of hypertrophy or hypervascular synovitis. Scar formation and capsular thickening are present. Stage 3 is also notable for reduced pain with increased stiffness in the joint. Stage 4 occurs from 15-24 months with further reduction in pain and improvements in ROM [3].

Adhesive capsulitis can be self-limiting, but symptoms often persist for years with and without treatment [3,4]. Shaffer et al., found that 50% of patients with adhesive capsulitis had persistent symptoms and 60% had remaining signs after an average follow-up of seven years [5].

Epidemiology

Adhesive Capsulitis is found in approximately 3-5% of the general population; however, some propose that the disorder is over diagnosed due to a lack of definitive diagnostic criteria [6,7]. Adhesive capsulitis affects women more than men and most often presents between 40-70 years of age, with most of these patients presenting in their sixth decade [4,5] up to 70% of those affected are women [3].

Adhesive capsulitis' incidence is increased in insulin-dependent, non-insulin dependent, and prediabetes patients [4]. Incidence is roughly 10% in Type 1 diabetics and approaches 29% in Type 2 diabetics [7]. Overall, it has been proposed that the increased incidence in diabetics is due to the formation of certain end products that contribute to fibrosis [8]. Other factors that have been shown to contribute to secondary adhesive capsulitis include surgery or fracture involving the affected joint, prolonged immobilization, thyroid disease, and various autoimmune diseases [3]. Additionally, while presentation is usually unilateral, 5-34% of individuals with adhesive capsulitis developed the condition at some point in the contralateral shoulder and this is especially true in diabetics [3,5,9,10].

Diagnosis

Adhesive capsulitis is a clinical diagnosis that can only be made once all other shoulder pathology has been ruled out [1]. Differential diagnosis can include acromioclavicular arthropathy, autoimmune disease, biceps tendinopathy, osteoarthritis, rotator cuff tendinopathy or tear, and subacromial and subdeltoid bursitis [4]. It is best distinguished from other shoulder disorders by the limitation of both active and passive ROM, particularly in external rotation, then abduction, and followed by internal rotation. This loss of passive ROM separates stiffness from weakness, which can be associated with other pathologies [3]. Hannafin et al.,' arthroscopic findings can be supportive of the clinical diagnosis of adhesive capsulitis if aggressive treatment is pursued in cases of refractory symptoms [3].

Diagnosing adhesive capsulitis in the early stages can be difficult. Walmsley et al., assessed eight clinical identifiers commonly used for early stage adhesive capsulitis and were not able to validate any of them. Despite consensus on the stages of adhesive capsulitis, how to appropriately distinguish them is still debatable [7].

Efforts have been made to use radiography and MRI to identify diagnostic criteria for adhesive capsulitis and its stages. Nonetheless, imaging modalities are currently not considered diagnostic for adhesive capsulitis, but can be useful to rule out other disorders and support the clinical diagnosis. Conversely, MRI can show distractors that further confound the clinical picture. Radiographs are typically negative but can show osteopenia from disuse of the joint, especially considering the tendency of the disease to affect women from age 40-70 [3].

Park et al., evaluated the MRIs of patients with clinical findings consistent with adhesive capsulitis and concluded that MRI could help predict the clinical stage of adhesive capsulitis. The study focused on joint capsule edema, obliteration of subcoracoid fat triangle by fibrous tissue, and joint capsule thickness and assessed how these MRI findings correlated with disease stage [2]. All were shown to decrease throughout the progression of the disorder from the inflammatory to fibrotic stages. Additionally, pain correlated with degree of capsular thickening and ROM correlated with edema [2].

Petchprapa et al., conducted a meta-analysis of MRI performed for adhesive capsulitis and found that the thickness of the capsule-synovium in the axillary recess was significantly increased, but that other signs were not significant. They concluded that while MRI can support diagnosis of adhesive capsulitis, it is not yet diagnostic in nature [1].

Future diagnosis and staging of adhesive capsulitis may involve evaluating serum levels of certain molecules whose expression is increased with the condition. Kim et al., discovered that ICAM-1, a molecule contributing to fibrosis when expressed, was significantly increased in the capsular tissue, synovial fluid, and serum of patients with adhesive capsulitis. Additionally, ICAM-1 activity was found to be more robust in the setting of hyperglycemia and could help explain the increased prevalence of adhesive capsulitis in diabetics [11].

Treatment

Despite decades of research and application, uncertainty still remains in the treatment of adhesive capsulitis. No gold standard currently exists, largely due to the lack of understanding surrounding the underlying triggers for the condition. Additionally, the vast heterogeneity in treatment protocols and their application within different stages of the progression of the condition have led to conflicting results or weakened conclusions in studies [12,13]. While particular treatment methods are more commonly used, the literature reveals limited consensus on treatment modalities. Additionally, distinctions must be made between treatment of idiopathic and secondary adhesive capsulitis. Diabetics in particular have been shown to have reduced improvement from treatment [12].

The disease stage dictates which form of treatment is most appropriate and it can impact the outcomes. The progression can be broken down broadly into an initial inflammatory stage accompanied by pain followed by a fibrotic stage accompanied by stiffness. Considerable overlap may occur between these two stages and treatment focuses on improving these underlying conditions [3].

In the setting of extended and refractory symptoms, the previous treatment received is an additional factor that should be assessed when considering treatment for adhesive capsulitis. Initial treatment has focused on conservative methods, to include supervised neglect, physical therapy, NSAIDs, and corticosteroids. Following 6-12 weeks of conservative treatment with intolerable symptoms, patients should be referred to an orthopedic surgeon [4]. Refractory symptoms are treated with more aggressive approaches, to include hydrodilatation, MUA, and capsular release of the involved joint.

Conservative approaches

Due to adhesive capsulitis' potential for self-limiting progression, supervised neglect was once seen as an option for treatment. However, because of the percentage of patients with

persistent symptoms long term, as shown by Shaffer et al., other forms of conservative therapy have become more common [5]. These findings, combined with the significant improvements in function and pain following newer treatments have led to the current conservative methods for treatment.

PT has been utilized extensively in the treatment of adhesive capsulitis. Adjunctive therapies have been utilized both prior to and during PT in an effort to improve overall outcomes. PT methods vary widely, but have included US, heat, ice, transcutaneous electrical nerve stimulation (TENS), ROM exercises, stretching, proprioceptive neuromuscular facilitation techniques, strengthening, and mobilization [6,14,15].

Noten et al., conducted a systematic review that concluded that patients in the stiff phase of the disease showed improvement following mobilization techniques. Timing is important for this aspect of PT [10]. Vermeulen et al., went further to compare high-grade mobilization techniques with low-grade ones and found that high-grade techniques were more effective, but the differences were small. The most improvement was seen in the first three months from mobilization; it is proposed that these improvements occur due to increased joint mobility and mechanical changes [6].

Others have made efforts to quantify the effect of pain on the efficacy of PT. The suprascapular nerve innervates the joint capsule involved with adhesive capsulitis [16]. Several methods have focused on this nerve as an adjunctive treatment to PT with the belief that reduction in pain would lead to improved outcomes. Some PT interventions are painful and limit the patient's ability or willingness to complete them [16]. Kilic et al., compared the effect of a suprascapular nerve block prior to PT with PT alone and found that the block significantly reduced pain [15]. Additionally, Wu et al., compared the effect of PT alone to pulsed radiofrequency lesioning of the suprascapular nerve prior to PT. Although the mechanism is uncertain, nerve lesioning has been shown to reduce chronic pain without neural injury and was performed in this study with US guidance for a duration of three minutes. The nerve lesioning group had significantly faster and better improvement compared to PT alone for up to 12 weeks [16].

Decreasing inflammation and pain in the joint has been approached in several ways. NSAIDs are recommended for all stages of adhesive capsulitis and are a mainstay of treatment [3]. Separately, administration of hyaluronate has yielded varying results. In a comparison of PT alone to PT co-administered with intra-articular injections of hyaluronate, Hsieh et al., found no significant difference between the randomized groups [14]. However, the injections in this study were made with a blind technique which has been shown to be less effective than when US-guided. In another study comparing capsular distension with US-guided intra-articular hyaluronic acid injection to intra-articular corticosteroid injection, the hyaluronic acid showed similar improvement to corticosteroids, without the risk of some of the rare but significant complications associated with corticosteroid use [17,18].

While hyaluronic acid injections have shown promising therapeutic benefit, corticosteroids have shown more consistent results in the conservative treatment of adhesive capsulitis. Corticosteroids reduce the inflammation and pains associated with this condition and have been helpful in determining the stage of disease when combined with a local anesthetic. Patients that regain ROM following the anesthetic are likely in the early stages where ROM is limited more by inflammatory pain and less by structural changes caused by fibrosis [3,7]. We have found intra-articular steroid injections to be the most helpful intervention, leading to a rapid reduction in pain and subsequent improvement in range of motion and ability to comply with physical therapy. Not infrequently, a second (or occasionally third) injection is required at 4-6 week intervals to fully reverse the process. The injections seem to be most effective during the early inflammatory or freezing phases.

Sun et al., conducted a systematic review comparing steroid injection to PT and found that each had similar safety and effect in improving function, passive external rotation, and reducing pain [12]. This suggests that patients in whom corticosteroids are contraindicated may be better off with PT. With the increasing prevalence of diabetes combined with the high incidence of adhesive capsulitis in diabetics, it is important to recognize the serum glucose level changes that can result from corticosteroid administration [12]. Choudhry et al., conducted a review of studies measuring blood glucose levels following intra-articular corticosteroid injection and found that all showed a rise in blood glucose levels. The blood glucose peak was variable in level and timing, but occurred between 1-3 days after injection for most diabetic patients receiving injection. They recommended blood glucose monitoring for up to a week, especially for Type 1 diabetics [19].

Griesser et al., conducted a systematic review of the effectiveness of intra-articular corticosteroid injections and found that compared to the control group, those receiving steroid injections experienced significant improvements in passive ROM and pain in the short term, but did not yield significant differences compared to controls in the long term. They found most patients achieved their end improvements as late as 2-4 years after treatment [13]. A review by Wang et al., found similar results with significant improvements in pain in the short term and passive ROM up to 24 weeks compared to control [20].

Yoon et al., found that no significant difference existed between high and low doses of corticosteroid injection and that 20mg triamcinolone is the preferred dose for inflammatory adhesive capsulitis. This dose provided significant improvement while minimizing complications from higher steroid doses [21]. Additionally, while both oral and intra-articular steroids yield significant improvements in adhesive capsulitis, improvements from intra-articular steroids were significant when compared to oral [13]. Oral steroids are also contraindicated in certain patients [22].

The technique used to inject the corticosteroid has also been shown to influence outcome in adhesive capsulitis. Although US-guided injection takes significantly longer, Patel et al., found they were significantly more accurate than a blind technique by palpation with a posterior approach conducted on cadavers [23]. Other studies have shown similar improvements with utilization of US. Lee et al., further investigated outcomes with the use of a blind technique compared to US-guided intra-

articular corticosteroid injection for adhesive capsulitis patients. They found that US-guided injections improved pain significantly over the blind technique for the first two weeks and significantly improved ROM and function over the first three weeks [24].

Refractory approaches

Aggressive treatment of adhesive capsulitis is reserved for persistent and refractory symptoms that tend to occur beyond the early stage of progression. These approaches are aimed at countering the remaining fibrotic tissue in an effort to reduce stiffness and restore function and ROM.

Manipulation under anesthesia is performed under general anesthesia with the surgeon taking the glenohumeral joint through flexion, external rotation, abduction, internal rotation, and then cross-body abduction in order to disrupt the adhesions and restore ROM. Some surgeons have proposed starting with MUA due to its success and then proceeding to capsular release if restrictions in movement still exist [25]. However, complications during MUA include soft tissue injury, proximal humeral neck fracture, dislocation, and brachial plexus palsy [26]. The high incidence of humeral neck fracture and increased costs associated with MUA have led to utilization of other methods to improve joint mobility [9].

One such outpatient procedure that also reduces cost is injection of fluid into the joint capsule for distension or to the point of rupture. These methods have been utilized in Stages 2 and 3. Vad et al., used capsular distention to rupture the capsule and found that all patients in Stage 2 adhesive capsulitis had significant improvement, while a small sample size of Stage 3 patients did not. Despite it being an aggressive approach, they suggested that capsular distention with rupture may not be appropriate for patients with severe restriction of ROM [27].

Quraishi et al., compared hydrodilatation with capsular rupture to MUA in Stage 2 patients and found significantly improved pain and scores with improved satisfaction in the hydrodilatation group compared to the MUA group. The hydrodilatation group also had significant ROM improvements over MUA in short-term follow up [9]. Surprisingly, the capsule tended not to rupture in the area of fibrosis, but through the subscapularis bursa or down the biceps sheath [9]. Due to this, outcomes are improved by capsular distension without rupture as opposed to hydrodilatation with rupture [28].

Others have examined the effect of distension combined with other conservative treatments. Lee et al., compared treatment of refractory Stage 2 adhesive capsulitis with US-guided corticosteroid injection alone versus US-guided corticosteroid with capsular distension. Unexpectedly, joint distension did not significantly improve outcomes when compared directly [28]. However, Buchbinder et al., found that joint distension with saline and corticosteroid led to significant improvements over the first three weeks compared to a placebo arthrogram [29]. In a follow-on study, Buchbinder et al., also found that limited additional benefit was exhibited from PT added to joint distension [30].

While hydrodilatation with and without capsular rupture has shown significant improvements for patients - mainly those in Stage 2 - it is unclear at this time how they compare to alternatives due to heterogeneity of studies and varying results.

Arthroscopic capsular release has been an effective treatment for refractory adhesive capsulitis. Like hydrodilatation, arthroscopic capsular release reduces the risk of fracture associated with MUA and provides faster improvement. It does, however, have complications of infection and axillary nerve injury during release of the inferior portion of the capsule. Improvements from arthroscopic capsular release have been shown to maintain or improve further up to seven years after their operation [31].

Hannafin et al., recommended arthroscopic inspection of the joint before any MUA, to ensure that the disease has actually progressed beyond synovitis and that the restrictions are from contracture of the joint. Any remaining synovitis can be removed, or if none is found, the capsule can be partially released to allow for easier MUA [3]. However, despite a lack of studies directly comparing outcomes in MUA to arthroscopic capsular release, treatment has trended towards capsular release as an alternative or adjunct therapy to MUA in order to decrease the risk of complications associated with MUA [32]. Capsular release is performed by releasing the rotator interval, superior glenohumeral ligament, and the coracohumeral ligament. Then the anterior superior and inferior aspects of the capsule can be released followed by the posterior superior and inferior portions [25]. Holloway et al., compared arthroscopic capsular release in idiopathic frozen shoulder with release in frozen shoulder secondary to previous surgery or fracture. They found that release was effective for all three groups to improve ROM, but the results were significantly worse in terms of pain, satisfaction, and functional activity for patients with post-surgery frozen shoulder [25].

Mehta et al. examined differences in outcome for arthroscopic release in patients with and without diabetes. They found that while both groups had significant improvement with release, non-diabetics improved significantly more. Additionally, diabetics were more likely to have refractory symptoms even two years after operation [8].

DISCUSSION

While much effort has gone into investigating adhesive capsulitis, its etiology still needs to be more clearly elucidated in order to better identify, diagnose, and treat the disease. Several therapies are currently being researched to determine if other approaches would be effective and help to further identify underlying mechanisms of the disease.

Badalamente et al., theorized that collagen may be affected in adhesive capsulitis in a similar fashion to Dupuytren's contracture and that selective lysis of collagen fibers might improve adhesive capsulitis symptoms. They administered collagenase clostridium histolyticum with extra-articular injections, which are used to treat Dupuytren's contracture, to patients with adhesive capsulitis to determine potential outcomes. These patients had reduced pain and improved ROM and function in a shortened time, but future studies are needed to further validate these results [33].

In another approach, Rouhani et al., investigated the efficacy of intranasal calcitonin administration for the treatment of adhesive capsulitis. Calcitonin is a peptide produced by the

parafollicular cells of the thyroid and it has been shown to exhibit analgesic effects for other diseases. Compared to placebo, they found that calcitonin was significantly more effective for pain reduction and improvements in ROM and function following six weeks of daily administration [22].

CONCLUSION

The progression of adhesive capsulitis and the risk factors associated with the disease necessitate individualized patient approaches for pain reduction and functional improvements. Conservative treatment modalities shown to have efficacy include NSAIDs, PT with preparatory pain reduction, and low-dose US-guided intra-articular corticosteroid injections. Aggressive treatment approaches with efficacy include hydrodilatation and arthroscopic capsular release. Diabetic patients, in particular, should be cautioned on the outcomes of all treatments and the associated complications. The understanding of the underlying inflammatory and fibrotic processes requires further exploration.

REFERENCES

1. Petchprapa CN, Beltran LS, Jazrawi LM, Kwon YW, Babb JS, Recht MP. The rotator interval: a review of anatomy, function, and normal and abnormal MRI appearance. AJR Am J Roentgenol. 2010; 195: 567-576.
2. Park S, Lee D, Yoon S, Lee HY, Kwack K. Evaluation of Adhesive Capsulitis of the Shoulder With Fat-Suppressed T2-Weighted MRI: Association Between Clinical Features and MRI Findings. AJR Am J Roentgenol. 2016; 207: 135-141.
3. Hannafin JA, Chiaia TA. Adhesive capsulitis. A treatment approach. Clinical Orthopaedics & Related Research. 2000; 372: 95-109.
4. Ewald A. Adhesive capsulitis: A review. Am Fam Physician. 2011; 83: 417-422.
5. Shaffer B, Tibone JE, Kerlan RK. Frozen shoulder. A long-term follow-up. J Bone Joint Surg Am. 1992; 74: 738-746.
6. Vermeulen HM, Rozing PM, Obermann WR, le Cessie S, Vliet Vlieland TPM. Comparison of high-grade and low-grade mobilization techniques in the management of adhesive capsulitis of the shoulder: randomized controlled trial. Phys Ther. 2006; 86: 355-368.
7. Walmsley S, Osmotherly PG, Rivett DA. Clinical identifiers for early-stage primary/idiopathic adhesive capsulitis: are we seeing the real picture. Phys Ther. 2014; 94: 968-976.
8. Mehta SS, Singh HP, Pandey R. Comparative outcome of arthroscopic release for frozen shoulder in patients with and without diabetes. Bone Joint J. 2014; 96: 1355-1358.
9. Quraishi NA, Johnston P, Bayer J, Crowe M, Chakrabarti AJ. Thawing the frozen shoulder. A randomised trial comparing manipulation under anaesthesia with hydrodilatation. J Bone Joint Surg Br. 2007; 89: 1197-1200.
10. Noten S, Meeus M, Stassijns G, Van Glabbeek F, Verborgt O, Struyf F. Efficacy of Different Types of Mobilization Techniques in Patients With Primary Adhesive Capsulitis of the Shoulder: A Systematic Review. Arch Phys Med Rehabil. 2016; 97: 815-825.
11. Kim Y, Kim J, Lee Y, Hong O, Kwon H, Ji J. Intercellular adhesion molecule-1 (ICAM-1, CD54) is increased in adhesive capsulitis. J Bone Joint Surg Am. 2013; 95: 181-188.
12. Sun Y, Lu S, Zhang P, Wang Z, Chen J. Steroid Injection versus Physiotherapy for Patients With Adhesive Capsulitis of the Shoulder: A PRIMSA Systematic Review and Meta-Analysis of Randomized Controlled Trials. Medicine (Baltimore). 2016; 95: 3469.
13. Griesser MJ, Harris JD, Campbell JE, Jones GL. Adhesive capsulitis of the shoulder: a systematic review of the effectiveness of intra-articular corticosteroid injections. J Bone Joint Surg Am. 2011; 93: 1727-1733.
14. Hsieh L, Hsu W, Lin Y, Chang H, Chen C, Huang V. Addition of intra-articular hyaluronate injection to physical therapy program produces no extra benefits in patients with adhesive capsulitis of the shoulder: a randomized controlled trial. Arch Phys Med Rehabil. 2012; 93: 957-964.
15. Kilic Z, Filiz MB, Cakr T, Toraman NF. Addition of Suprascapular Nerve Block to a Physical Therapy Program Produces an Extra Benefit to Adhesive Capsulitis: A Randomized Controlled Trial. Am J Phys Med Rehabil. 2015; 94: 912-920.
16. Wu Y, Ho C, Chen Y, Li T, Lee K, Chen L. Ultrasound-guided pulsed radiofrequency stimulation of the suprascapular nerve for adhesive capsulitis: a prospective, randomized, controlled trial. Anesth Analg. 2014; 119: 686-692.
17. Park KD, Nam H, Lee JK, Kim YJ, Park Y. Treatment effects of ultrasound-guided capsular distension with hyaluronic acid in adhesive capsulitis of the shoulder. Arch Phys Med Rehabil. 2013; 94: 264-270.
18. Cuni B, Ozcakar L. Ultrasound-guided capsular distension in adhesive capsulitis: the hyaluronic acid or the local anesthetic. Arch Phys Med Rehabil. 2013; 94: 2309.
19. Choudhry MN, Malik RA, Charalambous CP. Blood Glucose Levels Following Intra-Articular Steroid Injections in Patients with Diabetes: A Systematic Review. JBJS Reviews. 2016; 4: 51-56.
20. Wang W, Shi M, Zhou C, Shi Z, Cai X, Lin T, et al. Effectiveness of corticosteroid injections in adhesive capsulitis of shoulder: A meta-analysis. Medicine (Baltimore). 2017; 96: e7529.
21. Yoon SH, Lee HY, Lee HJ, Kwack KS. Optimal dose of intra-articular corticosteroids for adhesive capsulitis: a randomized, triple-blind, placebo-controlled trial. Am J Sports Med. 2013; 41:1133-1139.
22. Rouhani A, Mardani-Kivi M, Bazavar M, Barzgar M, Tabrizi A, Hashemi-Motlagh K, et al. Calcitonin effects on shoulder adhesive capsulitis. European Journal of Orthopaedic Surgery & Traumatology. 2016; 26: 575-580.
23. Patel DN, Nayyar S, Hasan S, Khatib O, Sidash S, Jazrawi LM. Comparison of ultrasound-guided versus blind glenohumeral injections: a cadaveric study. J Shoulder Elbow Surg. 2012; 21: 1664-1668.
24. Lee H, Lim K, Kim D, Lee K. Randomized controlled trial for efficacy of intra-articular injection for adhesive capsulitis: ultrasonography-guided versus blind technique. Arch Phys Med Rehabil. 2009; 90: 1997-2002.
25. Holloway GB, Schenk T, Williams GR, Ramsey ML, Iannotti JP. Arthroscopic capsular release for the treatment of refractory postoperative or post-fracture shoulder stiffness. J Bone Joint Surg Am. 2001; 83: 1682-1687.
26. Magnussen RA, Taylor DC. Glenoid fracture during manipulation under anesthesia for adhesive capsulitis: a case report. J Shoulder Elbow Surg. 2011; 20: e23-26.
27. Vad VB, Sakalkale D, Warren RF. The role of capsular distention in adhesive capsulitis. Arch Phys Med Rehabil. 2003; 84: 1290-1292.
28. Lee D, Yoon S, Lee MY, Kwack K, Rah UW. Capsule-Preserving Hydrodilatation With Corticosteroid Versus Corticosteroid Injection Alone in Refractory Adhesive Capsulitis of Shoulder: A Randomized Controlled Trial. Arch Phys Med Rehabil. 2017; 98: 815-821.
29. Buchbinder R, Green S, Forbes A, Hall S, Lawler G. Arthrographic joint distension with saline and steroid improves function and reduces pain in patients with painful stiff shoulder: results of a randomised, double blind, placebo controlled trial. Ann Rheum Dis. 2004; 63: 302-309.

30. Buchbinder R, Youd JM, Green S, Stein A, Forbes A, Harris A, et al. Efficacy and cost-effectiveness of physiotherapy following glenohumeral joint distension for adhesive capsulitis: a randomized trial. Arthritis Rheum 2007; 57: 1027-1037.

31. Le Lievre HMJ, Murrell GAC. Long-term outcomes after arthroscopic capsular release for idiopathic adhesive capsulitis. J Bone Joint Surg Am. 2012; 94: 1208-1216.

32. Grant JA, Schroeder N, Miller BS, Carpenter JE. Comparison of manipulation and arthroscopic capsular release for adhesive capsulitis: a systematic review. J Shoulder Elbow Surg. 2013; 22: 1135-1145.

33. Badalamente MA, Wang ED. CORR ORS Richard A. Brand Award: Clinical Trials of a New Treatment Method for Adhesive Capsulitis. Clin Orthop. 2016; 474: 2327-2336.

Cite this article

Feeley SM, Larson ES, Diduch DR (2017) Adhesive Capsulitis: Current Concepts. Ann Sports Med Res 4(6): 1124.