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Review Article

PHYSIOTHERAPEUTIC MANAGEMENT OF ADHESIVE CAPSULITIS: A REVIEW OF LITERATURE

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ABSTRACT

Background and Objective: Adhesive Capsulitis (also known as frozen shoulder) is a painful and disabling disorder of unclear cause in which the shoulder capsule, the connective tissue surrounding the glenohumeral joint of the shoulder, becomes inflamed and stiff, greatly restricting motion and causing chronic pain. Adhesive Capsulitis has an incidence of 3–5% in the general population and up to 20% in those with diabetes.

Methodology: Lot of research papers, articles, books, etc. was referred in order to review literature about physiotherapeutic management of Adhesive Capsulitis.

Conclusion: For many years much of the literature has referred to frozen shoulder as a self-limiting disease but the duration and severity may vary greatly. However there were few authors who have suggested that this condition lasts up to 6 months. But over a period of time many authors argued this old belief and found that the condition actual remains for 2 to 3 years. During last 40-50 years, many authors have conducted various studies on this condition and its physiotherapeutic management. But it is finally concluded that exercise is undoubtedly an important adjunct to treatment, its effectiveness as a sole treatment for frozen shoulder has not been thoroughly evaluated.

KEY WORDS: Physiotherapeutic Management, Adhesive Capsulitis.

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INTRODUCTION

Adhesive Capsulitis (also known as frozen shoulder) is a painful and disabling disorder of unclear cause in which the shoulder capsule, the connective tissue surrounding the glenohumeral joint of the shoulder, becomes inflamed and stiff, greatly restricting motion and causing chronic pain. Pain is usually constant, worse at night, and with cold weather. Certain movements or bumps can provoke episodes of tremendous pain and cramping. The condition is thought to be caused by injury or trauma to the area and may have an autoimmune component.

Risk factors for frozen shoulder include tonic seizures, diabetes mellitus, stroke, accidents, lung disease, connective tissue diseases, thyroid disease, and heart disease. Treatment may be painful and taxing and consists of physical therapy, occupational therapy, medication, massage therapy, hydr od-ilatation or surgery. A physicianmay also perform manipulation under anaesthesia, which breaks up the adhesions and scar tissue in the joint to help restore some range of motion. Pain and inflammation can be controlled with analgesics and NSAIDs.

Adhesive Capsulitis has an incidence of 3–5% in the general population and up to 20% in those with diabetes. This disorder is one of the most common musculoskeletal problems seen in orthopaedics. Although some have described adhesive Capsulitis as a self-limiting disorder that resolves in 1–3 years, other studies report ranges of between 20 and 50% of patients with adhesive Capsulitis which suffer long-term ROM deficits that may last up to 10 years.

Movement of the shoulder is severely restricted, with progressive loss of both active and passive range of motion. The condition is sometimes caused by injury, leading to lack of use due to pain, but also often arises spontaneously with no obvious preceding trigger factor (idiopathic frozen shoulder). Rheumatic disease progression and recent shoulder surgery can also cause a pattern of pain and limitation similar to frozen shoulder. Intermittent periods of use may cause inflammation. In frozen shoulder, there is a lack of synovial fluid, which normally helps the shoulder joint, a ball and socket joint, move by lubricatingthe gap between the humerus (upper arm bone) and the socket in the shoulder blade. The shoulder capsule thickens, swells, and tightens due to bands of scar tissue (adhesions) that have formed inside the capsule. As a result, there is less room in the joint for the humerus, making movement of the shoulder stiff and painful. This restricted space between the capsule and ball of the humerus distinguishes adhesive Capsulitis from a less complicated, painful, stiff shoulder.

Physical therapy or physiotherapy (often abbreviated to PT) is a physical medicine and rehabilitation specialty that remediates impairments and promotes mobility, function, and quality of life through examination, diagnosis, prognosis, and physical intervention (therapy using mechanical force and movements). It is performed by physical therapists (known as physiotherapists in many countries).

OBJECTIVE

The study aims to investigate the effectiveness of physiotherapeutic treatment i.e. Manual Mobilization & Active Exercises in the treatment of Adhesive Capsulitis.

REVIEW OF LITERATURE

For years, much of the literature has referred to frozen shoulder as a self-limiting disease but the duration and severity may vary greatly [1]. Even these studies describe the process as lasting a minimum of 12-18 months, before resolution. However there are those who suggest that it can last for as little as 6 months [2]. Binder et al (1984) [3] described frozen shoulder classically lasting for 18-24 months. Other studies have however challenged this popular belief. Reeves (1975) [4] and Shaffer et al (1992) [5] agree that it can last two to three years, although report significant numbers of people have residual clinical detectable restriction of movement and smaller numbers have residual disability (at seven years 50%had mild pain, stiffness or both). The clinical picture seen commonly by physiotherapists is characterized by this spontaneous onset of shoulder pain and progressive global stiffness of the gleno-humeral joint, accompanied by decreased function and significant disability [6]. The presence of night pain leads to disturbance of sleep and often difficulty lying on the affected shoulder. As the restriction in the motions increases, more difficulties are encountered with activities of daily living [7]. Routine radiographs are typically normal. These are important to rule out serious pathology, abnormalities in the bone, joint or in the local soft tissues e.g. calcific deposit and are a prerequisite to a definitive diagnosis of frozen shoulder [8].

Despite considerable research in the last century, the etiology and pathology of frozen shoulder remain enigmatic [7]. The prevalence is found to be approximately 2-3% of adults in the general population [3,7], and is thought to develop between the ages of 40 and 70 [3,6]. It rarely recurs in the same shoulder unless an injury or disease process predisposes the joint to repeat episodes of stiffness [3]. It is generally agreed that the non-dominant arm appears more likely to be involved [9]. However, Bunker (1998), reports that the condition occurs with equal frequency in the left and right shoulders. With regard to gender, Neviaser and Neviaser 1987; Stam 1994 and Hand et al 2008, found that there is a greater occurrence in women. Bunker (1998) also disputes this reporting that there is equal

prevalence between both genders; more recent studies showed a ratio of 1:1 male to female (Bunker 2009).Frozen shoulder usually presents unilaterally and the incidence of subsequent involvement on the contra lateral side is 20% [10]. It affects 20% of people with diabetes and has been described as the most disabling of the common musculoskeletal manifestations of diabetes. Although the aetiology of frozen shoulder remains elusive, the understanding of its pathogenesis is increasing. Generally, three schools of thought have emerged: an inflammatory process. A fibrotic process and an inflammatory process with subsequent reactive capsular fibrosis [11]. Duplay (1872) theorized that the pathologic condition of frozen shoulder was found in the subacromial bursa but later [1] related the disorder to calcific tendonitis. Neviaser (1945) discovered a tight, thickened capsule that adhered to the humeral head. He described an inflammatory reaction that led to adhesions, specifically in the axillary fold and in the attachment of the capsule at the anatomic neck of the humerus. On biopsy and histological examination, he identified perivascular infiltration, capsular thickening, contracture and fibrosis. He proposed that the pathology primarily involved the shoulder capsule, suggesting the term "adhesive capsulitis" as a better name for the disease. However, Lundberg (1969) [10], Wiley (1991) [12] and Bunker et al (1994) [13], found no adhesions in their arthroscopic studies. Simmonds (1949) [14] agreed with Neviaser (1945) [15] and specula-ted that a loss of motion at the glenohumeral joint was because of degenerative changes and secondary inflammation of the supraspinatus tendon. Lundberg (1969) [16] also observed an inflammation of the capsule as a precursor of the process leading to stiffness, pain and capsular fibrosis but no significant number of inflammatory cells. Significant evidence exists in support of the hypothesis that the underlying pathological changes are synovial inflammation with subsequent reactive capsular fibrosis, making adhesive capsulitis an inflammatory and a fibrosing condition, dependent on the stage of the disease [17]. Several investigators have proposed an autoimmune basis for frozen shoulder [18]. However, specific immunological studies reveal no evidence of any specific autoimmune or arthritic process [18].

This fact is used in the differential diagnosis of frozen shoulder. There is general agreement that the pathology affects the glenohumeral capsular tissue and is particularly localized to the coracohumeral ligament in the rotator interval [19]. Neer et al (1992) [19] also postulated that the coracohumeral ligament was contracted and Ozaki et al (1989) [20] stated that the release of this ligament was curative and this was confirmed by Bunker et al (1994) [13]. Bunker et al (1994) [13] observed that thickening and contracture of the glenohumeral ligament and rotator interval, acts as a check rein which prevents external rotation and causes global loss of active and passive movements. The contracture also causes superior translation of the humeral head leading to impingement and pain [21].

Bunker and Anthony (1995) [8] likened the changes of the glenohumeral capsule to Dupuytrens contracture in the palm. They reported that the pathological process is active fibro plastic proliferation, accompanied by some transformation to a smooth muscle phenotype (myofibroblasts). The fibroblasts lay down collagen that appears as a thick nodular bond or fleshy mass. They further noted that in the shoulder capsule the inflammatory component was absent or localized to the synovial and sub synovial layers. The tissue observed was highly cellular with cells identified as fibroblasts and myofibroblasts and this has been confirmed by Killian et al (2001) [22]. The findings by Hand et al (2007) [23] confirm these results and support the theory that frozen shoulder is an inflammatory condition that progresses in a continuum to a fibrosing condition. In conclusion, all the histological evidence to date shows that this is a capsular contracture of the shoulder [24]. Characteristically, pain precedes stiffness in frozen shoulder which suggests an evolution from inflammation to fibrosis. Many studies have attempted to establish the most effective treatment for frozen shoulder but much debate still remains. Currently there is no agreement on the standard management of this condition [25]. The lack of consensus on diagnostic criteria and concordance in clinical assessment

complicates treatment choices. The controversy is due in part to a failure of many authors to precisely define and accurately identify frozen shoulder among other causes of shoulder pain and stiffness [26]. Orthopedic and physiotherapy interventions or treatment modalities have been advocated in the management of frozen shoulder in the past thirty years, to alleviate the signs and symptoms and aid recovery. There is a considerable body of work devoted to the orthopedic management of this condition but the aim of this study is to focus on the conservative physiotherapy management. Therefore, only a concise review of orthopedic management follows. Initially, treatment is directed at pain relief. Non-steroidal anti-inflammatory drugs (NSAID's) are traditionally given but there are no randomized control trials that confirm the effectiveness of these. Oral corticosteroids have been recommended but little evidence exists to support their routine use [27]. Suprascapular nerve block [28] and steroid injection have been suggested by some authors [29]. However, this approach alone has not been shown to improve the range of shoulder motion [10,29,30]. Orthopedic interventions that have been shown to produce successful outcomes in restoring function include; distension arthrography, manipulation under anesthetic (MUA) and arthroscopic release. Distension arthrography was described by Andren and Lundberg as early as 1965 and has been advocated as a means of expanding the contracted capsule. Rizk et al (1994) [31] promoted it as a promising treatment. They performed a study of 16 patients and found that 13 experienced immediate pain relief and increased shoulder mobility. This was also found by Buchbinder et al (2004) [32] who demonstrated a significantly greater improvement in pain, function and active range of movement (ROM) in the group that received distension at three and six weeks. Manipulation under anesthetic (MUA) is the established form of treatment [33].

It results in a rapid return of shoulder motion, although some authors disagree about whether it shortens the disease course [10-34]. Bunker and Anthony (1995) showed that 75% of their patients attained a near normal range of movement, 79% were relieved of their pain and 75% returned to normal within nine weeks. Some authors consider manipulation an effective intervention, whereas others claim that it is traumatizing and may even exacerbate pain [35]. Bunker (2005) [8] suggests that arthroscopic release has transformed the management of this disease and recently in 2009 reports that it is still delivering relief of pain, undisturbed sleep and improved function in the majority of people with frozen shoulder. Ogilive-Harris et al (1995) [36] compared the results of MUA versus arthroscopic release. Although both groups gained the same improvement in ROM, the arthroscopic group had significantly better pain relief and function. Harrymann et al (1997) [37] demonstrated excellent results. The ROM went from 41% of the opposite side to 78% on the first day following surgery and 93% at the end of the study. Berghs et al (2004) [38] demonstrated that 36% of patients experienced pain relief and reduced stiffness after one day following an arthroscopic release and 80% within two weeks. Physiotherapy is often the first line of management for shoulder pain, yet its efficacy has not been established (Lynch 2002) [39]. In the review conducted by Cleland and Durall (2002) [40] twelve papers met the inclusion criteria and were split into prospective (n=9), retrospective (n=1) and randomized clinical trials (n=2). Their methodological scoring criteria included points for identifying the stage of pathology, whether the frozen shoulder was primary or secondary, duration of symptoms prior to intervention, and number of treatments. Due to the self-limiting nature of the condition these are important aspects to consider when reviewing the efficacy of treatment and are frequently omitted; this is therefore strength of their work. However, this study is limited as it only searched two databases and the reviewer was not blinded to the aim of the study and was therefore a threat to the internal validity. They only found two randomized controlled trials (RCT s) and therefore highlighted the need for more prospective RCT s using a standardized outcomes assessment to judge the efficacy of various physiotherapy interventions on frozen shoulder. The results revealed many inconsistencies. There was considerable variation in intervention strategies, duration of treatment and

outcome measures between the studies and lack of rigor and poor standardization of terminology. This made it difficult to compare relevant published research and determine the effectiveness or economic efficiency of treatments. As most of the studies used complex interventions and combined treatment modalities, they argued that it was difficult to determine which elements of physiotherapy were efficacious. Green et al (2003; 2009) [41] also highlighted this in their Cochrane review of physiotherapy interventions for shoulder pain. This review has been updated in 2009; however there was no change to the conclusions. They stated that it is unusual for shoulder disorders to receive a single treatment in isolation, demonstrating a conflict between validity and clinical practice. Green et al (2003; 2009) [41] reviewed twenty-six trials that met their inclusion criteria and were concerned by the low number of single modality studies. They identify this as one of the key areas to improve future research, along with larger trials of higher methodological quality, well-defined interventions and a validated inclusion/exclusion criterion. They concluded that there was no evidence that physiotherapy without concurrent interventions, such as corticosteroids, was of benefit for frozen shoulder. They stressed the need for trials of physiotherapy interventions for specific clinical conditions associated with shoulder pain. Four RCT s have been published since Green et al (2003; 2009) [41] published this systematic review: Guler-Uysal and Kozanoglu (2004) [42]; Buchbinder et al (2007) [43]; Vermeulen et al (2006) [44] and Johnson et al (2007) [45]. These authors examined different types and combinations of treatments over different time periods and used a variety of self-report instruments to assess pain, function and quality of life. It is still unclear from these papers which interventions may be most effective. Both the number and diversity of treatments, which have been recommended, reflect the extremely general nature of the physiotherapy treatment for frozen shoulder. Therefore, there is yet no definitive agreement on the most effective form of treatment [46]. Physiotherapy management aims to relieve pain, promote healing, reduce muscle spasm, increase joint range of motion and strengthen weakened muscles and ultimately to prevent and treat functional impairment [41]. These include: heat or ice applications; Ultrasound; Interferential therapy; Transcutaneous Electrical Nerve Stimulation (TENS); pulsed electromagnetic field therapy; active and passive ROM exercises; Proprioceptive Neuromuscular Facilitation (PNF) techniques; manual physical therapy and laser therapy [41], concluded that vast range of recommended treatment, coupled with a lack of many conclusive studies in this area, means that there is little guidance for today's physiotherapist with patients with a diagnosis of frozen shoulder. Currently there is no robust evidence on the superiority of any one treatment modality compared to another [47].

Modern literature commonly recommends the use of multiple modalities which precludes the effectiveness of individual treatment [40]. The Chartered Society of Physiotherapy has completed a project on the management of frozen shoulder [48]. Conclusions drawn from these evidence-based clinical guidelines suggest that future researchers should report their physiotherapy interventions in sufficient detail to remove ambiguity consider multi-center trials and focus on specific stages of frozen shoulder. Research demonstrates considerable variability in methods of treatment; however, it has been shown for some time that virtually all of them advocate some form of exercise to restore movement [30,49,50]. In clinical practice, exercises are almost always incorporated into the physiotherapy management of a patient with frozen shoulder. Whilst exercise is undoubtedly an important adjunct to treatment, its effectiveness as a sole treatment for frozen shoulder has not been thoroughly evaluated. Diercks and Stevens (2004) [51] performed a randomized prospective study of 77 patients with idiopathic frozen shoulder to compare the effect of intensive physical rehabilitation treatment. The patients were divided into two groups. All patients had more than 50% motion restriction for a period of three months or more. One group involved passive stretching and manual mobilization (stretching group) with supportive therapy and the second with a regime including active and auto-assisted exercises, within the pain limits (supervised neglect group).

All patients were followed-up for 24 months after the start of treatment. In the patients treated with supervised neglect, 89% had normal or near- normal painless shoulder function (Constant score >80) at the end of the observation programme 64% reached this result within 12 months. In contrast, in the group receiving intensive physiotherapy treatment, only 63% received a score of 80 or more after 24 months. The authors concluded that supervised neglect yields better outcomes than intensive physiotherapy and passive stretching in patients with frozen shoulder. However, they do not state where or how the sample was obtained, the frequency of treatment sessions or the compliance of patients. The study does not describe the validity or reliability of the measurement tools, which carried out the assessment and whether they were blinded to the intervention the patient received. One of the key findings was that intensive stretching prolonged the course of the disease and increased pain levels. This may be due to stretching into the painful range and could aggravate the symptoms and therefore increase the pain. Jurgel et al (2005)[52] found that a four week course of physiotherapy treatment resulted in significant improvements in the range of movement (ROM), pain levels, muscle strength and endurance in 10 patients with idiopathic frozen shoulder, who had pain for two weeks to three months duration. However, they continued to have reduced ROM and strength compared with their unaffected side or controls. Positions were standardized for measuring ROM and muscle strength and endurance. Unfortunately, only endurance in flexion was measured which was performed using a prolonged static posture. This does not relate to many functional activities. Pain was measured using a 10 point visual analogue scale (VAS).

However it was unclear if this was at rest or during activity, or over what time period e.g. past day, past week. It was also unclear if pain was measured or whether this was asked pre or post assessment of ROM, muscle strength and endurance. They used a questionnaire to determine difficulties in activities of daily living (ADL). However a standardized validated questionnaire was not used and it was uncertain which activities were assessed. Additionally the ADL questionnaire was not repeated at the final assessment, thus giving no information on whether the improvement in ROM, pain, muscle strength and endurance relate to an improvement in the ability to carry out ADL. The sample was very small therefore affecting the generalizability of the findings to the general population. Moreover, recruitment and the inclusion and exclusion criteria were not stated. No power calculations were mentioned and it is questionable if 10 patients provide enough power to enable robust statistical analysis. Only one physiotherapist assessed muscle strength and endurance, one physiotherapist assessed ROM and one physiotherapist performed the therapeutic interventions. This does reduce error due to inter-rater variability and also reduces the variability in personal interaction between the treating physiotherapist and the patients in the study. However, it is unclear whether the assessing and treating physiotherapists were the same or different people. Though the participants achieved good results in this study it was through quite intensive input of up to an hour per day of mixed therapeutic modalities. A combination of pool and gym exercises, various electrotherapies and massage therapy were used and tailored to each individual participant. Whilst this is how patients tend to be treated in the clinical setting it is difficult to ascertain which intervention was beneficial and as none of the interventions are described it would be difficult to apply this to clinical practice. There are no clear indications in the literature concerning the optimal treatment frequency and duration, but the trial physiotherapist hypothesized that the regime used would be intensive and long enough to induce changes. Moreover in the absence of scientific evidence regarding the effectiveness of multimodal physiotherapy programmes, this programme was developed in light of results obtained in studies of other groups with musculoskeletal disorders, in animal studies, and from the best available evidence [53,26]. In reviewing the studies presented, it is evident that more research is needed in order to draw conclusions and establish an efficacious and clinically valid treatment method. As identified, this evidence needs to come from

robust randomized clinical trials. Such trials need to clearly define the methodological approach used and include sufficient follow up length, with use of clinically valid and reliable outcome measures.

CONCLUSION

It is concluded from the study that for many years much of the literature has referred to frozen shoulder as a self-limiting disease but the duration and severity may vary greatly. However there were few authors who have suggested that this condition lasts up to 6 months. But over a period of time many authors argued this old belief and found that the condition actual remains for 2 to 3 years. Hannafin & Chiaia, 2000 mentioned very clearly that despite considerable research in the last century, the etiology and pathology of frozen shoulder remain enigmatic. Different researchers have different views regarding the pathologic condition of the Frozen Shoulder. Duplay (1872) theorized that the pathologic condition of frozen shoulder was found in the subacromial bursa but later Codman (1934) related the disorder to calcific tendonitis. Neviaser (1945) discovered a tight, thickened capsule that adhered to the humeral head. Orthopedic and physiotherapy interventions or treatment modalities have been advocated in the management of frozen shoulder in the past thirty years, to alleviate the signs and symptoms and aid recovery Orthopedic interventions that have been shown to produce successful outcomes in restoring function include; distension arthrography, manipulation under anesthetic (MUA) and arthroscopic release. Distension arthrography was described by Andren and Lundberg as early as 1965 and has been advocated as a means of expanding the contracted capsule. Rizk et al (1994) promoted it as a promising treatment. They performed a study of 16 patients and found that 13 experienced immediate pain relief and increased shoulder mobility. Physiotherapy management aims to relieve pain, promote healing, reduce muscle spasm, increase joint range of motion and strengthen weakened muscles and ultimately to prevent and treat functional impairment (Green et al 2003; 2009).

The Chartered Society of Physiotherapy has completed a project on the management of frozen shoulder (Hanchard et al 2011). Conclusions drawn from these evidence-based clinical guidelines suggest that future researchers should report their physiotherapy interventions in sufficient detail to remove ambiguity consider multi-center trials and focus on specific stages of frozen shoulder. Research demonstrates considerable variability in methods of treatment; however, it has been shown for some time that virtually all of them advocate some form of exercise to restore movement (Lee et al 1973; Neviaser and Neviaser 1987; O Kane et al 1999). It is finally concluded that in clinical practice, exercises are almost always incorporated into the physiotherapy management of a patient with frozen shoulder. Whilst exercise is undoubtedly an important adjunct to treatment, its effectiveness as a sole treatment for frozen shoulder has not been thoroughly evaluated.

Conflicts of interest: None

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