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REVIEW ARTICLE

Current evidence on physical therapy in patients with adhesive capsulitis: what are we missing?

Filip Struyf · Mira Meeus

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Abstract Adhesive capsulitis is, in most cases, a self-limiting condition of poorly understood etiology that results in shoulder pain and large mobility deficits. The socio-economic burden will increase as with continuous aging of our population. In addition, both prevalence and incidence figures of adhesive capsulitis are increasing. No literature overview solely focuses on the physiotherapeutic options in patients with adhesive capsulitis and their scientific evidence. Moreover, although some physiotherapeutic interventions show evidence regarding reducing pain or increasing mobility, there is little evidence to suggest that the disease prognosis is affected and this raises the need for new, innovative research in the area of adhesive capsulitis and its treatment. By presenting its current evidence, we hope to retrieve several gaps in the present management of adhesive capsulitis by physiotherapists and provide us with new insights for improving the physiotherapists' policy in treating adhesive capsulitis patients, e.g., continuously increasing nociceptive impulse activity, as in early stages of adhesive capsulitis, could lead to peripheral and subsequently long-lasting central sensitization, as well as to an increased activity of the sympathetic nervous system. But up to now the involvement of central sensitization in adhesive capsulitis has not been studied yet and remains speculative. Finally, when selecting a physical treatment method for adhesive capsulitis, it is extremely important to consider the patient's symptoms, stage of the condition, and recognition of different patterns of motion loss. Guidelines for clinical assessment will be presented in this scoping review.

Keywords Adhesive capsulitis · Frozen shoulder · Physical therapy · Review

F. Struyf (✉) · M. Meeus
Department of Rehabilitation Sciences and Physiotherapy, Faculty of Medicine and Health Sciences, University of Antwerp, Universiteitsplein 1, 2610 Wilrijk, Belgium
e-mail: Filip.struyf@uantwerpen.be

F. Struyf · M. Meeus
URL: www.paininmotion.be

M. Meeus
Department of Rehabilitation Sciences and Physiotherapy, Ghent University and Artevelde University College, Ghent, Belgium

Introduction

Although frozen shoulder and adhesive capsulitis (AC) are frequently used as synonyms, AC is a distinct pathological entity, while frozen shoulder solely refers to any condition that restricts active or passive glenohumeral motion [1], e.g., conditions such as subacromial bursitis and calcific tendonitis were previously termed as frozen shoulder as they could lead to a stiff and painful shoulder [2]. Although these conditions cause a clear limitation of active range of motion, they lack capsular contracture and restriction in passive range of motion. Therefore, these conditions should not be labeled as AC [2]. AC was recently defined as range of motion (ROM) loss of greater than 25 % in at least two movement planes, together with at least 50 % loss of passive external rotation in comparison to the uninvolved shoulder [3]. However, many randomized controlled trials (RCTs) define AC as motion loss of more than 50 % of passive movement of the shoulder joint in one or more directions and a duration of complaints for more than 3 months, which simplifies the inclusion criteria. To complete the diagnosis, the pain and restricted movement should be present for at least 1 month and has either reached a plateau or worsened [3]. A prevalence rate of primary adhesive capsulitis 2 % up to 5.3 % in the general population has previously been described, mainly affecting women (70 % of the AC patients), those with sedentary jobs, the non-dominant shoulder, and patients older than 40 years of age [1, 3–5]. Diabetes patients

have an increased risk (up to 40 % chance) of developing AC in their lifetime [5, 6]. In addition, having AC on one side places an individual at risk (5–34 %) for opposite arm involvement in the future [3].

The literature mainly describes two types of AC, the idiopathic or primary form and the acquired or secondary form [7, 8]. Whereas no specific cause can be identified in the primary form of AC, several review articles [3–5, 7, 8] describe possible associations in secondary AC to be of systemic, extrinsic, or intrinsic nature. Systemic causes include diabetes mellitus, thyroid dysfunction, and hypoadrenalism. Extrinsic associations include cardiopulmonary diseases, cervical spine pathology, stroke, Parkinson's disease, and humerus fractures. In addition, possible intrinsic factors are rotator cuff pathologies, biceps tendinitis, calcific tendinitis, and AC joint arthritis. Likewise, the presence of recent surgery, immobilization, trauma, and even Dupuytren disease has also been associated with the development of secondary AC [9]. AC is often described as a continuum of four stages [1, 3–5, 10]: The first stage (approximately the first 10 weeks) is the so-called "inflammatory stage," in which hypervascular synovitis is seen. During the "freezing stage" (stage 2; lasting approximately 10 to 26 weeks following stage 1), glenohumeral motion is starting to restrict progressively. During stage 3 (5–12 months), the "frozen stage," hypervascularity reduces and the joint capsule thickens. Finally, during stage 4 (from 12 months after onset), the "thawing stage," the symptoms start to resolve. As primary AC tends to resolve over the course of 1 to 3 years, it is often described as self-limiting. However, it appears that a large percentage of patients will still have some residual movement limitations and residual pain after many years (more than 2 years). The presence of secondary comorbidity such as diabetes and a severely restricted glenohumeral joint at baseline is potentially associated with poor prognosis [11].

Several review articles provided recommendations for physical therapy in patients with AC. Overall, therapy is controversial and depends on the stage of the disease. However, most systematic reviews advise that treatment of AC should primarily be conservative, including physical therapy, patient education, intra-articular steroid injections, and nonsteroidal anti-inflammatory drugs (NSAIDs) [1, 4, 5, 12–16]. A recent systematic literature review reported that the majority of studies are in favor of the role of physical therapy for improving pain, functionality, and range of motion [15]. However, it was also suggested that physical therapy may not alter disease progression, particularly if the therapeutic regime is aggressive, potentially increasing inflammation [15]. Concerning physical therapy, the majority recommends the use of manual mobilizations and stretching (supervised and home exercises) in all stages of AC and high grade mobilizations in the last stages of AC [2, 5, 17].

To our knowledge, no literature overview solely focuses on the physiotherapeutic options in patients with AC. Moreover, although some physiotherapeutic interventions show evidence regarding reducing pain or increasing mobility, there is little evidence to suggest that the disease prognosis is affected and this raises the need for new, innovative research in the area of AC and its treatment. By presenting its current evidence, together with the mentioned associations of AC with other pathologies or diseases and the new insights in central pain mechanisms, we hope to retrieve several gaps in the present management of AC by physiotherapists and provide us with new insights for improving the physiotherapists' policy in treating AC patients. However, before we can present the therapeutic modalities of AC, we need to know how to assess and classify the patient.

Clinical examination of the adhesive capsulitis

The diagnosis of AC should be mainly based on anamnesis and clinical examination with limited dependence on specific radiological of laboratory findings [4]. The latter examination tools are beyond the scope of this review. Anamnestic history analysis and physical examination allow clinicians to discriminate primary AC from these other conditions. Soft-tissue contracture creates both active and passive limitation of motion, resulting in mechanical restriction of movement [7]. This contracture may occur in combination with e.g., rotator cuff tears or degenerative arthritis [7]. Finally, other pathologies such as glenohumeral or acromioclavicular osteoarthritis, rotator cuff disease, cervical radiculopathy, or locked posterior dislocations should be excluded [2, 17].

Anamnesis

AC refers to a cluster of symptoms, which mainly originate from synovial inflammation, fibrosis, and chondrogenesis of the shoulder joint capsule, denoting painful and gradual active and passive motion loss [18]. The symptoms most often described are stiffness and painfulness of the shoulder. However, one should be cautious in generalizing this pathology to stiffness and painfulness, as one could miss other more serious disorders [4]. Other symptoms that can aid diagnostic decision making are the slow onset of pain, originating at the deltoid insertion, the inability to sleep on affected side, and atrophy of the supra- and infraspinatus muscles [4, 5]. Anamnestic features depend on the stage the patient is in. During the inflammatory stage, patients mainly complain of aching pain at rest and sharp pain during active and passive motion. Patients also report the inability to sleep on the affected side. During the freezing stage, the patient reports a more chronic nagging pain, which worsens at night. Next (frozen stage), pain reduces and is mainly present at the end range of movement. However, a large motion limitation is

present. Finally, during the thawing stage, range of motion starts improving and pain is just minimal.

Assessing participation and activity limitations

Although many guidelines focus on physical impairment measures, physical therapists should also use easily reproducible activity limitation and participation restriction measures to assess the changes in the patient's level of shoulder and consequent general function [3]. In addition, recent clinical practice guidelines stated with strong evidence that physical therapists should use validated functional outcome measures, such as the Disabilities of the Arm, Shoulder and Hand (DASH) or the Shoulder Pain and Disability Index (SPADI) [3, 17]. It is recommended to use these questionnaires before and after interventions.

Pain assessment

As pain is a major symptom in patients with AC, it is highly recommended to properly assess pain. As in many shoulder disorders, the pain provoked by AC is mediated by the peripheral α-adrenoreceptor hyperresponsiveness in the somatosensory neurons of both nociceptive and proprioceptive fibers [5]. Together with increased capillary growth, there is evidence of new nerve growth in the capsuloligamentous complex, which also may explain the heightened pain response [3]. Most studies analyzing treatment effects make use of a visual analog scale (VAS) [13]. Physical therapists should also screen for the presence of psychosocial factors, potentially influencing pain perception. Elevated scores on the Tampa Scale of Kinesiophobia or the Fear–Avoidance Beliefs Questionnaire have been associated with a longer recovery, chronic symptoms, and work loss in patients with shoulder pain [3]. And although the role of psychosocial factors is not extensively studied in AC, it is well-known from other chronic pain populations that personality traits, cognitions, and behaviors such as catastrophizing, maladaptive coping, hypervigilance, external locus of control, and chronic stress are yellow flags, important therapy mediators/barriers, and related to poor recovery and higher pain scores [19–24]. Therefore, assessing these psychosocial factors by using questionnaires (e.g., Pain Catastrophizing Scale, Pain Coping Inventory, Pain Vigilance, and Awareness Questionnaire) is warranted.

Palpation

During the first two phases of AC, the patient will report pain on palpation of the anterior and the posterior capsule and describe pain radiating to the deltoid insertion [4]. These symptoms can extend to the trapezius and rhomboid region [4]. In addition, Carbone et al. described the coracoid pain test as a pathognomonic sign in the physical examination of patients with AC [25]. The coracoid pain test is performed by performing manual (one finger) pressure on the area of the coracoid process. For comparison, pressure should be carried out on the acromioclavicular joint and the anterolateral subacromial area. The test is considered positive when a score on a visual analog scale was three points or higher on pressure in the coracoid area compared with the other two areas [25]. However, in order to standardize this test, we recommend using some type of dynamometer, in order to put the same pressure on the three different sites. This test is based on both characteristic magnetic resonance arthrographic findings of thickening of the coracohumeral ligament and of the joint capsule in the rotator cuff interval and based on a clinical trial with an ultrasound-guided corticosteroid injection into the rotator interval that resulted in a significant improvement in SPADI and active range of motion [25, 26]. In addition, Carbone et al. reported very high specificity (98 %) and sensitivity (99 %), making it possible to respectively rule in or out the diagnosis of AC [25]. Although promising, some methodological issues remain, such as its unknown reliability and the fact that only one (not blinded) assessor was used in this study.

Assessing glenohumeral range of motion

As glenohumeral global motion loss is one of the main symptoms of a patient suffering from frozen shoulder, clinicians are recommended to assess glenohumeral motion capacity in different directions in order to obtain an accurate and objective analysis of treatment efficacy. As the disease progresses, motion loss will be more prominent. Up to 80 % of shoulder motion can be lost [5]. It has previously been shown that a large loss of external rotation is a pathognomonic sign of frozen shoulder [4, 5, 14, 17]. AC least affects extension and horizontal adduction motion [4]. When the external rotation restriction resolves after 45° up to 90° of abduction, motion loss was likely due to subscapularis restriction [3].

One must be cautious when evaluating active glenohumeral mobility. On the one hand, increased scapulothoracic upward rotation together with some rotation in the thorax can potentially give rise to a false pattern of sufficient shoulder mobility. On the other hand, co-existing disorders, such as rotator cuff tears, can result in a large limitation of active shoulder motion, despite the absence of any capsular restrictions. Thus, if passive external rotation is full but active external rotation is limited, a possible rotator cuff tear should be considered. In addition, pain may result in poor active movement. Therefore, it has previously been suggested by Warner [7] that an analgesic injection could reveal true active motion by eliminating pain. Each physical therapist should therefore identify and control these compensatory movements [7].

Active shoulder flexion should be measured with the patient seated, in the scapular plane and relative to the thorax [7]. In addition, active external rotation and internal rotation are measured in both neutral (shoulder in adduction) and 90° of abducted position. In order to reduce muscle activity during passive movement measurements, the patient is placed supine. This position also restricts excessive scapulothoracic movement through the patient's weight on the scapula [27].

Physical therapy

In general, the principles for treating patients with AC are defined by the ability of the tissue to cope with physical stress, often described as "tissue irritability." Tissue irritability will guide physical therapists in deciding whether a specific therapeutic modality is appropriate at that time, what kind of intensity to apply, and how long and how often a specific technique can be performed. Kelley et al. [3] defined three levels of irritability: high (≥7/10 on a VAS, consistent night or resting pain, high disability, pain before end range, less active ROM than passive ROM due to pain), moderate (4–6/10 on a VAS, intermittent night or resting pain, moderate disability, pain occurs at end range, active ROM=passive ROM) and low (≤3 on a VAS, no night or resting pain, low disability, pain at overpressure end range, active ROM=passive ROM) [3]. In patients with a high irritability level, treatment should emphasize on pain reduction, patient education, and pain-free active or passive exercises (e.g., low-intensity joint mobilizations in a pain-free zone). Together with the lowering of the tissue irritability, the intensity of the active and passive exercises increases. Finally, at the lowest levels of tissue irritability, physical therapists can apply more intense stretching and strengthening exercises.

Patient education

Physical therapist treating patients with frozen shoulder are encouraged to start their treatment protocol by educating their patients in order to reduce frustration and increase compliance [3, 4]. Explaining the pathology can also reduce possible fears. The focus should be placed on the self-limiting character of the condition. However, it is advisable to acknowledge that full range of motion might never be restored. In addition, the therapist should promote activity modifications to encourage functional, pain-free range of motion [3]. Patients should also be aware that they need to match the intensity of stretching to his current level of irritability [3]. However, patient education should be presented in moderation. We do not recommend informing the patient that there is a large chance of developing AC on the contralateral side, as it could alter their activity pattern and consequently induce AC.

Glenohumeral mobilizations and stretching

Limitations in glenohumeral movements are often associated with the location of a capsular contracture [7]. For example, limitation of internal rotation is associated with contracture of the posterior capsule, limitation of external rotation of the adducted shoulder is associated with contracture in the anterosuperior capsular region, and limitation of external rotation when the shoulder is abducted is associated with contractions in the anteroinferior region of the capsule. Glenohumeral mobilization techniques should therefore be focused on the affected area. In contrast, Johnson et al. reported that posterior glide mobilizations, thus focusing on the posterior capsule contractures, demonstrated greater improvement in external rotation range of motion compared to the patients treated with anterior glide mobilizations [28]. Likewise, Placzek et al. [29] performed glenohumeral manipulations to patients under anesthesia and concluded that posterior translations restored both external and internal rotation motion. They hypothesized that posterior translations stretch both the posterior capsule and the rotator cuff interval. Finally, they also concluded that inferior translation techniques stretch the adhesions within the inferior fold, leading to greater elevations of motion.

Few mobilization concepts are described in the literature, such as Mulligan's technique. This technique combines sustained manual application of a "gliding" force to the shoulder joint in accordance with the normal arthrokinematic pattern of shoulder motion. Both Mulligan's technique and passive stretching reduced pain and restored range of motion and function in a randomized controlled study [30]. In addition, better improvements in terms of pain, range of motion, shoulder scores, and patient and physiotherapist satisfaction were achieved in favor of Mulligan's technique [30]. In addition, Gaspar et al. concluded that the use of the a dynamic splinting mechanism may be an effective adjunct as home stretching exercise for stage 2 AC patients, recommending 60 min per day of low-load, prolonged-stretch [31]. However, their outcome measure only included external rotation range of motion.

Stretching techniques can influence pain and improve range of motion [3]. However, these improvements may not exceed the efficacy of other interventions. Still, no strong evidence exists in order to guide physical therapists in defining the optimal therapy frequency, or the number of repetitions and duration of stretching. However, stretching intensity that matches the given level of tissue irritability is indicated [1, 3].

Pain management

Pain management has made great progress during the last decades. Physical therapy is often perceived as painful, while

the major aim of the therapy is in fact to alleviate pain. Gleyze et al. examined whether the treatment options respect the pain threshold or not and whether this influences experienced pain and clinical outcome [32]. They concluded that sub-threshold supervised rehabilitation (VAS <6/10) provided progressive results but limited in time, whereas suprathreshold self-rehabilitation (VAS >6/10) provided reduced pain from the first days, including reduced pain at night. On the one hand, this emphasizes the strength of home exercise programs, which place the locus of control within the patient. On the other hand, this requires sufficient adherence and compliance of the patient. These results may indicate that the patients in this study were already more self-efficacious, being compliant with exercise therapy and showing a high degree of internal locus of control, which is beneficial for the prognosis of chronic pain patients [33, 34].

Discussion

AC is a relatively common problem seen by different medical specialists, including general practitioners, orthopedic surgeons, rheumatologists, physiatrists, and physiotherapists [8]. Patients with AC are frequently referred to as rheumatologists, as they are specialists in inflammatory joint diseases and pain disorders affecting joints in general. In addition, rheumatologists are well trained to make differential diagnosis (e.g., with rheumatoid arthritis). This scoping review can help these clinicians in their evaluation and treatment policy or guide them for appropriate referral.

When selecting a physical treatment method for AC, it is extremely important to consider the patient's symptoms, stage of the condition, and recognition of different patterns of motion loss. A proper clinical assessment (as described above) is clearly warranted. It is obvious that AC is, in most cases, a self-limiting condition of poorly understood etiology. The socio-economic burden will increase as with continuous aging of our population; the prevalence rates of AC will increase. However, also the incidence of AC increases [35]. For every 10 years, the incidence rate is estimated to increase by 8 % in women. No increase in the incidence of AC is predicted for men.

Blanchard et al. [13] and Favejee et al. [12] systematically summarized the effectiveness of corticosteroid injections compared with physiotherapeutic interventions for AC. They indicated that the treatment of AC with corticosteroid injections is more effective than physical therapy in the short term and to a lesser extent in the longer term. Six weeks up to 4 months follow-up, all included trials in the systematic review of Favejee et al. [12] showed a significant benefit of intra-articular injections over physiotherapy alone or placebo on pain. However, it must also be mentioned that both interventions (physical therapy & injection) were more effective than the control groups at both 6 and 12 weeks [13]. But the combination of corticosteroid injections and shoulder mobility and stretching exercises is also more effective in providing short-term (4–6 weeks) pain relief and improved function compared to shoulder mobility and stretching exercises alone [3, 12]. Finally, Robinson et al. reported that no prospective comparative studies have been performed on interventions such as ultrasound, acupuncture, laser, pulsed electromagnetic field therapy, bipolar interferential current, or transcutaneous electromagnetic stimulation [36]. Nevertheless, in the systematic review of Favejee et al., strong evidence was found for the effectiveness of laser therapy in the short term, while moderate evidence was found for mobilization techniques in the short and long term [12]. In conclusion, although some physiotherapeutic interventions show evidence regarding reducing pain or increasing mobility, innovative research in the area of AC and its treatment is highly warranted.

However, we need to acknowledge several limitations while performing this scoping literature overview. Firstly, there is a lack of a clear definition for AC. Many studies use different inclusion criteria, such as the duration of injury and the magnitude of the ROM loss. Secondly, from several randomized clinical trials, it is difficult to take into account the self-limiting aspect of AC. As mentioned above, symptoms of AC can last up to several years, whereas the follow-up period of many RCTs is often limited to 3 months. Thirdly, most studies did not clarify whether total range of motion was presented, or only glenohumeral. Scapulothoracic movements are known to alter when pain or disorders are present [37]. External glenohumeral motion may be compensated by external scapulothoracic rotation, and elevation may be compensated by early and excessive scapular upward rotation. More specifically, Yang et al. [38] were the first to create prediction rules for identifying the kinematic features of patients with frozen shoulder who are more likely to respond to physical therapy. By means of tracking shoulder motion during three tests (abduction in the scapular plane, hand-to-neck and hand-to-scapula), a prediction method with two variables was identified. They stated that scapular tilting of more than 8.4° during arm elevation, together with external rotation of more than 38.9° (during hand to neck), increased the probability of improvement with treatment from 41 to 92 % (positive likelihood ratio=15.71). It appears that shoulder kinematics may predict improvement in subjects with AC [38]. Although they stated that scapular mobilization should be applied in subjects with a stiffness stage AC [38], we believe that the benefits of scapular mobilization are localized within the glenohumeral movement that is created during this type of mobilizations, rather than the movements that are created within the scapulothoracic region. This latter region is probably responsible for the remaining shoulder movement and may subsequently be overloaded to compensate glenohumeral mobility restrictions.

Finally, it is hypothesized that the pain experienced in those with AC is associated with the pathogenesis of inflammation evolving into fibrosis of e.g., subacromial bursa [39]. The origin of pain is inflammation and the inflammatory response. Activation of pain receptors, transmission and modulation of pain signals, neuroplasticity, and central sensitization are all one continuum of the inflammation and the inflammatory response [40]. Up to now, pain in AC is considered as nociceptive pain, resulting from peripheral abnormalities in ligaments, bursae, capsular structures, etc. However, it is well-known that inflammatory mediators—like cytokines, neuropeptides, growth factors, and neurotransmitters— may play a direct role in the process of central sensitization as well. Cytokines related to acute or repetitive tissue injuries may be responsible for long-term activation of spinal cord glia and dorsal horn neurons, thus resulting in central sensitization [41]. Also peripheral sensitization is mediated by local components of the local inflammatory reaction released by the microenvironment of the inflamed and injured tissue [42]. In a state of inflammation-evoked neuroplasticity, sensory stimuli such as light pressure or movements in the working range of the joint may evoke much larger responses in the peripheral nervous system than under normal conditions [43, 44]. Continuously increased nociceptive impulse activity, as in early stages of AC, could lead to peripheral and subsequently long-lasting central sensitization, as well as to an increased activity of the sympathetic nervous system [44, 45]. But up to now, the involvement of central sensitization in AC has not been studied yet and remains speculative. If central sensitization is present in a subgroup of patients with AC, as it is in many other arthritic and rheumatic conditions [46], this could explain why some patients are therapy resistant and this would mean that this subgroup requires a completely different approach. The latter may partly explain why results are not univocal and therapeutic approach of these patients is that challenging.

Research agenda

This overview showed that physical therapy interventions are more effective than a 'wait-and-see' policy, but to date, there is little evidence to support the superior efficacy of joint mobilizations over other interventions [3, 47]. In addition, a successful therapy outcome can be defined as a significant reduction of pain and improved function together with high levels of patient satisfaction [3]. But what about disease progression? There is little evidence suggesting that physical therapy or other therapy modalities can alter disease progression. Further study is warranted in order to establish the most effective physical therapy modality for those suffering of AC. More high-quality clinical trials could allow more definitive conclusions about long-term outcomes. Patients should be grouped together according to the stage of their disease, in order to determine whether certain treatments are more effective at specific times in the disease process [13]. Together with these recommendations, a strong cost effectiveness analysis may be of benefit in order to determine whether the effect of any treatment approach would be large enough to warrant the extra resources required [47].

Furthermore the biochemical background of AC remains unclear. The high incidence of AC in e.g., diabetic is peculiar. It is suggested that excessive glucose concentration could lead to a faster rate of collagen glycosylation and cross-linking in patients with diabetes mellitus, leading to restriction of the joint capsule [2]. This could lead to the assumption that patients with AC could benefit from low-sugar intake or even a specific training that focuses on e.g., cardiorespiratory functions. To our knowledge, no clinical trials have investigated the impact of sugar restriction or cardiovascular training on the disease progression of AC. In addition, as obesity is related to the overproduction of inflammatory cytokines, a recent prospective clinical study suggested that being overweight (BMI >25 kg/m^2) resulted in a significantly lower improvement in the constant score after physical therapy and intra-articular injections were applied [48]. In addition to the relevance of cytokines in this condition, the possible involvement of abnormal central pain mechanisms in subgroups of AC patients needs to be studied, as pain mechanisms are becoming more and more important in the rehabilitation of chronic pain patients. Understanding the exact pain mechanism is essential to steer treatment, since the approach of nociceptive pain is completely different from that of central sensitization pain. In the case of central sensitization, therapy should account for altered central pain processing. This means that the approach should definitely be biopsychosocial and consist of elements like pain education and stress management, while the manual and exercise components should be adopted to account for inappropriate cognitions, impaired pain inhibition, and enhanced nociceptive bottom-up transmission [49, 50]. Concretely, this means that attention should be distracted from the biomedical problems in terms of damage and injury and away from the pain, as pain is no longer a valid sign in the case of central sensitization. Therefore, physical therapists should be careful with adopting manual techniques, not only because this may cause a supplemental nociceptive barrage on the central nervous system, but also because this may strengthen the patient's beliefs of a clear biomedical problem. Regarding the exercise component, exercise should be cognition targeted and it might be beneficial to start with graded exposure and graded activity, rather than with strenuous exercise therapy. But before considering this approach, central sensitization should be documented at least in a subgroup of patients, and subsequently, therapists should be able to assess signs and correlates of central sensitization.

Conclusion

We believe that the management of patients with AC requires a multidisciplinary approach involving physical therapists, general practitioners, orthopedic surgeons, and rehabilitation specialists. Further study is warranted on the presence of central pain mechanisms, the possibility of using the present association with diabetes by interfering accordingly in non-diabetic patients, and subclassifying patients based on strong clinical parameters.

Disclosures None.

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