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Osteoarthritis Pain and Muscle

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Abstract

Osteoarthritis, a chronically painful debilitating joint disease affecting many aging adults, is not always amenable to, or improved by current pharmacologic and surgical approaches. In light of the contribution of peri-articular structures to the osteoarthritic pain cycle, this exploratory overview and opinion piece was designed to examine if there is sufficient evidence in favor of treating muscle both as the sole means of reducing osteoarthritic pain or as a supplementary strategy for minimizing joint pain and further joint damage. To this end, research that focused on the sources of osteoarthritis pain, especially those detailing some aspect of neuromuscular derived pain was assessed. As well, research examining the outcome of treating muscle as regards osteoarthritis pain was explored. The results show that muscle can be deemed to play a key role in the osteoarthritis pain cycle. Moreover, treatments directed towards improving muscle function in some way tend to yield pain relief, when used alone, or in combination with other approaches, regardless of joint or method examined. It is concluded more work to better understand the muscle pain linkages in osteoarthritis will produce both a better understanding of the pathology associated with this disease, as well as its amelioration.

Introduction

Osteoarthritis, a prevalent chronic disease causes appreciable disability in a high percentage of older adults [1-5]. Strongly associated with progressive destructive changes in the articular cartilage and underlying bone structures of one or more freely moving joints, as well as pathological changes of surrounding joint structures, such as the ligaments and muscles [6-10], persons with this health condition commonly experience various degrees of unrelenting pain both at rest and on motion [11] that is somewhat resistant to simplistic pain relieving approaches.

For example, pharmacologic strategies directed towards ameliorating osteoarthritic pain, are not always efficacious [12-14], do not always treat the underlying cause of the problem [14], can hasten articular cartilage degeneration [15], produce systemic toxic side effects [16], or result in excess joint destruction because there is no 'warning' sign in potent pain relief situations to prevent further joint damage [12]. Moreover, origins of osteoarthritis pain are complex (see Box 1), and no single drug has been developed that can reverse or adequately delay the disease progression [17]. In addition, total joint replacement surgery is not always indicated as a pain relieving strategy, and may not be accompanied by anticipated improvements in pain, especially if associated muscle dysfunction remains untreated. In light of these facts, and current research implicating muscle in the disease process, it is hypothesized that if left untreated, this important protective component surrounding the freely moving joint can contribute towards exacerbating the disease and with this the related degree of osteoarthritic pain and disability. In addition, this author believes the nature of the sources of muscle pain, if not clearly understood and delineated might similarly produce little or no response to treatment, or may even evoke pain, or further destructive processes, inadvertently. Conversely, if muscle – which is amenable to intervention, is responsible for producing or heightening osteoarthritic pain, muscle may prove to be a potent target for improving upon standard practices to alleviate osteoarthritis pain as suggested by findings of Wang, et al. [18].

To this end, this brief examines, a) whether muscular features of osteoarthritis can produce pain or exacerbate prevailing pain, and, b) whether treatment of muscle in any form yields substantive reductions in osteoarthritic pain. It was hypothesized that the literature would reveal a wide variety of muscular mechanisms with the potential to impact osteoarthritic pain both directly and indirectly. It was also believed that if this muscle contributes to pain in a significant way, a variety of muscle-related treatment approaches would be found to reduce osteoarthritis pain.

Methods

Works in the PUBMED and ACADEMIC SEARCH COMPLETE data bases extending from 1980-to the present, using key words such as: management of osteoarthritis pain and muscle, osteoarthritis pain and muscle; muscle pain and muscle afferents; muscle dysfunction and osteoarthritis; muscle spasm and osteoarthritis were sought. Only articles focusing on both muscle and pain as related to osteoarthritis were selected for review. Described in narrative and tabular forms, first evidence supporting an association between muscle factors and osteoarthritis pain is discussed, followed by reports highlighting results of therapeutic studies directed towards

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Box 1: Possible Sources of Pain in Osteoarthritis.

Peri-articular
Tendons, fascia
Bursitis
Excess body fat
Muscle spasm, damage
Nerve pressure
Psychosocial
Anxiety
Depression
Poor self-efficacy
Intra-articular
Periosteum, osteophyte formation
Subchondral bone, micro-fractures, engorgement
Intra-articular ligament degeneration
Capsule distension
Synovitis
Comorbid health conditions
Cardiovascular disease
Diabetes
Neurological disorders
Obesity

treatment of osteoarthritic muscle. The specific topic of interest, pain, was chosen, as this is the most common complaint of people with osteoarthritis.

Results

Basic research linking muscle and osteoarthritis pain

A variety of studies confirm symptomatic osteoarthritis is associated with varying degrees of articular cartilage, bone, and ligamentous tissue damage [19-20], as well as joint inflammation [21-23] that produces pain. Another sub-group of studies implicates muscle-related structural and functional changes in the osteoarthritis pain cycle [20, 23-30].

As outlined in (Box 2), some of these muscle abnormalities may stem directly from the abnormal forces placed on osteoarthritic muscle; others may stem from the exposure of osteoarthritic muscle to persistent abnormal sensory inputs from one or more of the surrounding tissues of a diseased joint, such as the ligaments [31], and all can provoke pain, including muscle contractile abnormalities, deforming contractures, varying degrees of muscle spasm, and muscle neurogenic and myogenic abnormalities [24,25].

Others include, muscle weakness, poor muscle endurance, diminished joint range of motion, as well as joint stiffness, especially on movement [32,33]. In addition, associated changes in changes in motor unit recruitment [31], muscle morphology and/or abnormal agonist/antagonist strength ratios consequent to unremitting pain, may induce a situation of joint instability [32] that can potentiate osteoarthritic damage and pain [34-37] as depicted in (Figure 1).

Box 2: Diverse Array of Muscle Problems Encountered in Osteoarthritis that Can Produce Pain.

Abnormal flexor/extensor torque ratios [87]
Altered muscle contractile properties [77]
Alterations in muscle activity [82]
Muscle atrophy or wasting [25,28,52,68,79]
Muscle biochemistry [91]
Muscle co-contraction [97]
Muscle contractures [32]
Muscle degeneration [80]
Muscle fat infiltration [76,80]
Muscle fatigue [18,83,85,86]
Muscle fiber pathology [78]
Muscle inflammation [84,85,91]
Muscle mass declines [81]
Muscle quality alterations [81]
Muscle inhibition [75,88]
Muscle power declines [26]
Muscle rate of force development changes [34]
Muscle spasm [23,38]
Muscle strength declines [93]
Muscle size [18]
Muscle thickness declines [95]
Muscle trigger points [40,73]
Muscle weakness [42,88,92]
Pain during muscle contraction [88]
Poor muscle endurance [85]
Signs of muscle degeneration [52]
Soft tissue changes including fibrosis [52]
Tendinous lesions [90]

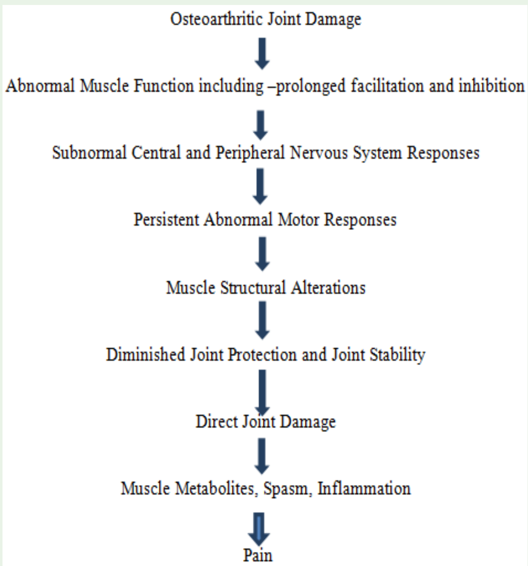

Figure 1: Hypothetic relationship between osteoarthritic pain and muscle factors.

Table 1: Summary of Outcomes of Treatments Directed Towards Improving Muscle Function for Symptomatic Relief of Osteoarthritis Pain.

Authors	Sample	Intervention	Outcome	Comments
Aguiar et al. [56]	22 adults with knee OA	3-80 min sessions of flexibility training and muscle strengthening for 12 weeks	Pain was reduced and function increased	Exercise therapy is effective for treating pain associated with knee OA
Al-Khlaifat et al. [97]	19 patients with knee OA	A 6 week exercise program was integrated with self-management education	A significant decline in pain was observed post-intervention	The improvements were attributed to associated changes in vastus lateralis and biceps femoris co-contraction
Cherian et al. [99]	18 patients with knee OA randomized to unloader brace or standard treatment	Unloader brace	Unloader brace had same impact as standard treatment as regards pain	Unloading the knee can be useful as a strategy for reducing knee OA pain
Hendriksen et al. [107]	60 knee OA cases	Patients were randomized to 12 wk supervised ex or no attention control grp	Pain was more favorably reduced in ex grp	Pressure-pain sensitivity, and self-reported pain are reduced after a 12-wk supervised ex program compared to a control grp
Imoto et al. [103]	100 knee OA cases were randomized to 2 groups	One group received neuromuscular stimulation, the other did not	The experimental group showed a greater pain improvement than control group	Electrical muscle stimulation is effective for reducing knee osteoarthritis pain
Ju et al. [64]	14 knee OA cases	Two grps were examined: a proprioceptive circuit ex grp; and control grp	In the proprioceptive grp pain was significantly reduced	A proprioceptive circuit ex routine may strengthen knee muscles and reduce pain of patients with knee OA
Ito et al. [106]	30 patients with knee OA	One of 2 randomized grps received 5 acupuncture sessions at traditional points, one grp at trigger points, and one received sham treatment	After treatment, those receiving trigger point acupuncture reported less pain	Trigger point acupuncture may be more effective than standard acupuncture for pain relief in knee OA
Knoop et al. [02]	169 knee OA cases,	One group received supervised ex for 12 wk, the other did the same but joint stabilization ex was added	Both groups attained significant pain reduction	Knee stability ex do nor add to the benefits of muscle strengthening ex and daily training of activities
Laufer et al. [100]	63 participants with knee OA	Groups received either exercise or exercise and neuromuscular electrical stimulation	A greater reduction of knee pain was observed in the electrically stimulated group	The effect was maintained for up to 12 weeks
Lee et al. [78]	30 patients with knee OA-15 actively treated; 15 controls	Kinesiology taping 3 x week for 4 weeks	Pain was significantly relieved	Kinesiology taping is a positive cost-effective safe strategy for decreasing pain
Nejatie et al. [58]	56 knee OA cases randomized to exercise or acupuncture and physiotherapy	Patients in experimental group exercised and received anti-inflammatory drugs or 10 sessions of acupuncture and physiotherapy modalities	Patients in exercise group had significant reductions in pain	Exercise can augment medical and other therapies for knee OA
Oliveira et al. [104]	100 cases of knee OA	Patients were randomized to receive exercise versus instruction	The ex group improved their pain levels more than the instruction grp	Knee extensor strengthening ex for 8 wk effectively improved knee OA pain
Rabini et al. [108]	50 knee OA cases	Patients were randomized inro focal muscle vibration or a sham grp	At 3 and 6 months, pain assays favored the experimental grp	Focal muscle vibration may improve physical function in knee OA
Salacinski et al. [109]	37 cases with knee OA	27 cases were assigned to a cycling grp; 18 to a control grp	After 12 wk, cycling grp showed greater improvements in pain on 3 different pain scales	Stationary cycling is an effective option for adults with mild-moderate knee OA and can reduce pain
Sayers et al. [101]	33 cases with knee OA	12 underwent high-speed power training, 10 underwent slow-speed strength training, and 11 served as control subjects	Pain improved in both exercise groups compared to controls	Both forms of exercise were effective, but high-speed may encourage high-speed task efficiency during daily activities
Vas et al. [61]	Knee OA	Pulsed radiofrequency of the knee joint nerves	Pain relief was reported and appeared to be sustained up to 6 months	Knee pain is a product of neuromyopathy and reducing the pain reduces peripheral and central sensitization
Vaz et al. [105]	20 women with knee OA, 10 healthy women-phase 1 12 OA cases, healthy cases-phase 2	8 weeks of neuromuscular stimulation aimed at strength training was undertaken	Initially knee muscles were weaker and less thick than those of health subjects; the training increased the thickness of vastus lateralis as well as fasicle length, and reduced joint pain	Neuromuscular training benefits the structure of the knee muscles of knee OA cases, as well as pain

This hypothetical model linking a series of adverse interactions, between the joint pathology and the muscular elements of a joint including heightened fatigue ability [38] and others shown in Box 2, is strongly associated with joint biodynamics abnormalities [39], including the ability to attenuate excess load [40,41], that can independently heighten the patient's pain experience [43-45]. Other research suggests persistent muscle spasm resulting from excessive stretching of diseased tissues or abnormally stimulated muscle nociceptors from accumulation of metabolites or myopathic alterations may produce ischaemic pain [46-49] as observed in basic studies of muscle fatigue [47-49]. Moreover, muscle inflammation [32], due to muscle fiber damage may sensitize group III and IV muscle afferents and related trigger points that conduct pain [30,50-51], and an associated rise in intra-articular pressure as a result of this process, may heighten pressure on the subjacent bone that results in venous congestion and bone pain.

Research also shows that abnormal neuronal inputs from muscle may combine with abnormal input from joints to produce referred pain [40]. As a result, there may be a general unwillingness on the part of the patient to move their joints, along with more extensive cartilage deterioration and pain, as well as joint stiffness [32]. In addition, with marked or complete stiffening, the abnormal and awkward movements used by the patient to avoid pain in one or more periarticular joint[s] may throw strain on other joints, causing pain, and/or muscle imbalances that enhance the risk of further joint pathology.

Systematic electrophysiological investigations performed in polyarthritic rats have demonstrated inputs from inflamed joints also reach the upper levels of the nervous system, and it is possible some of this input occurs directly as a result of muscle nociceptive stimulation. As well, it is hard to resolve this pain, because the responsiveness of the central receptors is commonly increased, and their discharge can outlast by several folds (2-12 times) that of their stimulation. Another striking feature is that in a large population of thalamic neurons, responses of long duration are elicited not only as a result of gentle movement, they also occur readily without stimulation as demonstrated by Gilbaud [40] or in response to innocuous inputs from other sites [42].

Alternatively, the widespread reflex organization found to occur in the presence of a persistent noxious chemical or mechanical stimuli may impair the body's ability to protect articular cartilage from impact, thus fostering possible generalized osteoarthritis and increased pain. It is possible too that the ability to absorb abnormal damaging impact forces which produces painful osteoarthritis is further reduced in overweight osteoarthritic cases due to an associated predominance of intra muscular fat content [76]. Moreover, where muscular protective reflexes are completely abolished, the risk of further joint destruction and pain is increased significantly.

In sum, osteoarthritic pain may originate in several articular or para articular structures supplied by sensory nerves (see Box 1). In turn, as outlined in (see Box 2), pain may be associated with muscle atrophy [25], a possible cofactor in the progression of osteoarthritis [52], muscle spasm, muscle contractures, muscle fibrosis, muscle inflammation [53] and muscle dysfunction [54], among other factors.

In other related work, van der Esch et al. [55] proposed that the tendency towards avoidance of pain-related activity among people with painful osteoarthritis, may produce more pain, rather than relieve pain, because it can enhance muscle weakness and joint instability, and as discussed above muscular factors may alter central processing mechanisms which amplify the pain attributable to the local condition. Suboptimal muscle function may also mediate cartilage damage of the affected or unaffected joint directly, thus supporting the use of timely and efficacious efforts to optimize local and system wide muscle function.

Research examining the effect of treating muscle on pain

Among studies that have attempted to treat muscle and have simultaneously assessed the impact of this approach on osteoarthritic pain, are a variety of exercise regimens, studies that employ heel wedges that alter muscle force generation, patellar taping, electrical muscle stimulation, and supportive aids, bandages and sleeves. As outlined in the table below, which represents a good cross-section of the recent literature, most are efficacious, regardless of research design or treatment approach employed, and the outcomes can be attributed to changes in muscle structure and related physiological changes. For example, Aguiar et al. [56] reported positive pain reductions among individuals with knee osteoarthritis after employing a protocol, which consisted of flexibility training and muscle strengthening over 12 weeks, three times a week. In addition to showing a decrease in pain perception using two different scales, interleuken-6 levels too, related to the pain cycle, also appeared to be affected favorably. In addition to exercise, which is generally helpful for alleviating the magnitude of osteoarthritis pain, Goryachev et al. [57] who applied foot center of pressure manipulation and gait therapy to help improve the status of patients with knee osteoarthritis, produced changes in the electromyography pattern of the lower leg muscles that were associated with a reduction in pain. As mentioned above, the type of exercise applied to relieve pain in osteoarthritis does not seem to play a large role in the positive outcomes observed as far as reducing pain goes, as demonstrated by Nejati et al. [58] who found that non aerobic exercises applied to people with osteoarthritis was accompanied by a significant degree of pain reduction, and by Sayers et al. who used power training. Other forms of intervention that appear effective for reducing osteoarthritis pain are neuromuscular stimulation and exercise, strength training [59], massage therapy, yoga, and tai chi [60] (Table 1).

In terms of other treatment options, Vas et al. [61] reported good results after applying a new pulsed radiofrequency form of intervention to the knee joint, which improved both pain and fostered muscle relaxation. After that patients were able to more readily participate in endurance exercise training to further reduce pain and improve function. This approach appears to have a sound basis as discussed above, and appears highly promising. Similarly, pain was more effectively reduced after a well-designed neuromuscular training program compared to a reference group [37], as well as after leg-press training with moderate vibration [62]. Treatment of active and latent trigger points in muscles surrounding osteoarthritic joints may also prove beneficial [30,51], as may non-invasive biomechanical therapy [63] and proprioceptive circuit exercise [64]. Gait modification also appears promising for reducing pain in medial compartment knee osteoarthritis [65], as does bracing [66], and a multipoint coupling dynamic technique [67]. Muscle power training, moderate pressure massage, yoga, and tai chi is also predicted to impact osteoarthritic pain positively [68]. Promoting optimal muscle coordination, flexibility, strength, and endurance, and balancing rest and activities, while minimizing joint effusion which can cause muscle inhibition and associated cartilage damage [19,73] is likely to be helpful. The same applies to functional exercises, interventions to enhance muscle control, efforts to maintain or normalize joint range of motion [74], and modalities to treat muscle and joint alignment problems.

Conclusion

Although osteoarthritis is currently deemed a chronic disabling disease with no cure [28], research indicates an array of muscle related factors can contribute to the osteoarthritis pain cycle [See Box 2]. Conversely, a diverse array of intervention approaches that focus on muscle are found to reduce the degree of pain encountered by this population, regardless of joint site, and disease severity or duration. Hence, as outlined by Dubin [2], recognition of the possible role of muscle as a contributing factor to the pain experience, plus the application of carefully construed interventions undertaken at the earliest possible time [14], is likely to prove more advantageous than not for alleviating osteoarthritis pain, even in the more advanced disease stages [14]. Moreover, these approaches, which include, but are not limited to electrical muscle stimulation, massage, exercise, yoga and tai chi, are safe and cost-effective and can be integrated with pharmacologic and surgical measures.

To achieve optimal results, care must be taken however, to avoid overexertion, which can heighten muscle nociceptive afferent inputs [71] and accelerate cartilage destruction [69], as can the complete elimination of pain [12]. Additional care and careful monitoring to avoid excess exercise activity is advocated in the presence of acute inflammation [14], severe overweight, and in situations where there has been a prior joint injury, a surgical procedure [41,53,84], or a prolonged period of immobilization found to hasten cartilage degeneration [72]. In addition, preventing obesity is crucial, given its effect on muscle function, as well as on joint and inflammatory disease responses [96].

In short, while the disease remains incurable [16,28], it is this author's view that significant improvements in the patient's wellbeing can be anticipated by the application of carefully integrated interventions to offset the varied muscular deficits that may accompany osteoarthritis. In particular, to avert rapid or excess disease progression and disability, and its association with pre-frailty and frailty [3], as well as work-related losses [4], deconditioning, disuse atrophy, increased falls risk [98], and comorbid illnesses [5] such as depression and obesity, identification of muscle impairments, and their treatments is of paramount importance in efforts to minimize osteoarthritic joint pain and dysfunction. However, since this is by no means a universally accepted idea or practice, more studies that tease out the possible relationship between muscle factors and osteoarthritic pain along with central factors that affect pain would clearly be beneficial. Carefully controlled intervention studies with larger samples with similar muscular and disease related characteristics conducted over extensive time periods utilizing variety of possible interventions could prove insightful as well.

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