REVIEW ARTICLE

OSTEOARTHRITIS DISEASE PROGRESSION THROUGH THE LOWER EXTREMITY: A REVIEW

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It has long been felt that osteoarthritis is the result of wear and tear. Many physicians are not aware that biological science has now validated that this increased "wear and tear" on the joint is the result of loading-induced stresses that undermine cell function and aging. The loading creates shear forces on the cartilage that leads to increased oxidants. These oxidants then cause chondrocyte premature aging, leading to the development of what we know as osteoarthritis.

Studies have also found a significant association between foot pain and knee and hip pain. Likewise, many physicians are not aware of the relationship between the hip, knee and foot are a result of the kinetic chain. The kinetic chain being the dynamic transfer of forces during ambulation of the foot, ankle, knee, and hip. The kinetic chain explains how the body's joints and segments affect one another during movement, and so play a role in pain. The pain results from transferred mechanical stress from one joint to another. The same stress and shear forces that leads to osteoarthritis.

This article involves a literature of the connection of osteoarthritis and the kinetic chain in the lower limb and highlights the need to consider related joints in the kinetic chain when addressing and injury in one joint to address and perhaps delay progression of osteoarthritis in related joints.

INTRODUCTION

Osteoarthritis (OA) is the leading arthritic condition worldwide.1 Nearly 1 million people were hospitalized for osteoarthritis in the United States in 2011.[1] The cost to care for these patients—at almost $15 billion—elevated it to the position of second most expensive disease in the country.[1] In 2013, OA was the second most costly health condition treated in U.S. hospitals, in that it accounted for $16.5 billion.[2] OA is most often found in the knee joint, while the hand and the hip are the next most common areas to be affected.[3] The authors questioned if the osteopathic tenets would explain progression and perhaps management of OA. This article will review the results of the Medline literature search and discuss possible application of osteopathic manipulative treatment (OMT).

METHODS

Applying osteopathic tenets of body connectedness, one would expect that OA of one joint in the lower extremity would transfer forces to the opposite extremity as a compensatory mechanism. Therefore, a Medline/PubMed literature search was performed using the search phrase "association between knee and contralateral hip osteoarthritis." The search returned 68 articles between 1981 and 2021. These were then reviewed for relevance to the question. Those that addressed association or disassociation of OA and ipsilateral joint are included in the discussion.

Because association is not causation, a separate, second PubMed search was performed looking for a biochemical/physical force cause for OA. The intent of the search was to identify a biochemiocal explanation for the association found in the first search. The search returned 538 articles between 2001 and 2021. These were also reviewed for relevance to the question of causation. Of note, most of the research has been related to risk factors and genetic predisposition. Those articles addressing biochemical or biomechanical causes were included in the discussion.

RESULTS/DISCUSSION

It has long been believed that OA is the result of wear and tear on a joint.[4] Research into occupations that include strenuous physical labor suggests an association between repeated and intense episodes of joint loading with the early onset of joint degeneration. Additionally, an increased risk for the development of OA has been associated with sports that involve repeated exposures to high joint-loading activity.[5] Newer studies have demonstrated that the pathophysiology of OA is somewhat more complex and other considerations must be taken into account. A myriad of factors contributes to the destruction of joint cartilage,

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including inflammatory, metabolic and mechanical etiologies.[3] However, it appears that all factors contribute to mechanical forces on joint surfaces.

In evaluating the mechanical forces, cellular studies have demonstrated the association of shear force on the chondrocyte with the release of multiple biochemical factors that can lead to oxidation, inflammation, decreased metabolic activity, early aging and chondrocyte cell death.[5] Thus, at the cellular level, it is the development of shear forces on the joint that appears to lead to the development and progression of OA.

For osteopathic clinicians, it is also helpful to consider how these processes could impact the patient, beyond just a single joint. The motion paradigm of the kinetic chain can provide insight into this bigger picture. The kinetic chain concept, specifically the kinetic chain reaction, has been attributed to Franz Reuleaux (1829–1905). He proposed a system of overlapping rigid segments connected by pin joints whereby, fixed at each end, application of an external force results in a chain reaction, with each segment receiving and transmitting force to the next segment in the system.[6] The concept has been adapted over time to the human body, where it has been described as "a sequencing of individual body segments and joints to accomplish a task."[7]

In the gait cycle, contact with the ground begins at heel strike,[8] beginning the process of directing forces through the lower extremity. When looking at the lower extremity, the relationship between the toes, foot, ankle, lower leg, knee, upper leg, hip, pelvis and spine comprises the kinetic chain. To evaluate the secondary biomechanical effects of knee gait mechanics, investigators studied ipsilateral hip and ankle joint motion among patients with knee OA against a control group. This would be a marker for potential transfer of forces onto the follow-on affected joint in OA patients. Results showed that the range of motion (ROM) of the hip and ankle joints were significantly smaller in the OA group and were associated with limited ROM of the knee joint (both $p<0.001$). The authors concluded that OA of the knee has a negative effect on the ROM of both the hip and ankle.[9]

Another study looked at the pattern of evolution of end-stage lower extremity OA in a clinical cohort of 5894 patients. The study used total joint replacement as the marker of end-stage OA. Investigators selected patients who underwent hip or knee replacement and evaluated the relative likelihood those patients would have a subsequent total joint replacement. Since mechanical processes are not the primary driving force behind the pathology seen in rheumatoid arthritis (RA), RA patients were used as a control population. In OA patients undergoing a second joint replacement, these individuals were significantly more likely to have contralateral limb joint affected (hip to knee $p<0.001$; knee to hip $p= 0.013$), whereas the RA patients did not show any pattern of laterality.[10] A subsequent study expanded on the concept, investigating 85,616 patients who had either a total knee, total hip, or total shoulder arthroplasty. The authors found that 23.6% of the patients went on to have the contralateral joint replaced 5–8 years later, while 3.7% had a different joint replaced in that same timeframe.[11] Thinking of these results in the context of the kinetic chain, it is understandable that injurious forces causing damage at one joint may be distributed throughout the chain, causing damage to other joints.

In follow-up to the findings that the contralateral knee was more likely to require knee replacement after total hip replacement, investigators examined whether the finding was related to asymmetries in dynamic loading at the knee joint. Specifically, the investigators assessed increases in the peak external knee adduction moment, as this has been associated with disease progression in knee OA. Fifty individuals had their gait analyzed prior to the total hip replacement, and 22 of them were reevaluated 10–23 months after surgery. At each evaluation, dynamic joint loads were compared between the contralateral and ipsilateral knees. Peak external knee adduction moment and peak medial compartment load were increased in the contralateral knee, persisting post-operatively.[12] This further exemplifies the concept of the kinetic chain and force distribution throughout the different elements of that system.

CONCLUSION

Typical management of OA has involved symptomatic treatment of pain with non-steroidal anti-inflammatory drugs (NSAIDs) until total joint replacement was deemed medically necessary. Exercise is encouraged, but is difficult to implement, as insufficient exercise is not helpful and too much increases pain.[13] It has been suggested that the standard recommendation of resistance and weight-loaded exercises (such as walking and exercise bicycles) that involve repeated short-arc motions could actually decrease joint mobility and continue to propagate the gait disturbances noted around the kinetic chain, as they would be similar to the repetitive motions seen in work- and sport-related activities implicated in the type of joint damage, which leads to OA.[14] Given the biochemical nature of OA and the impact of the kinetic chain on connected joints, an argument could be made for enhanced physical therapy after joint injuries (for example, ankle sprains or strains) to correct or prevent establishment of compensatory but destructive gait disturbances that perpetuate the progression of OA. In response to clinical findings of gait dysfunction and OA, it makes sense that physicians should ensure that there is a focus on restoration of the gait, and not just ROM and strengthening of the affected joint, in physical therapy efforts. Considering these kinetic chain mechanics, there is logic to the addition of manual therapy, including the use of OMT, to reduce shear forces applied to the joints to perhaps reduce or delay the progression of OA.[14]

The interrelationship of structure and function in the human body is a central osteopathic tenet.[15] Along with the concept of somatic dysfunction,[16] these foundational principles of osteopathic medicine correlate with the concepts discussed above—mechanical stressors lead to joint damage leading to further joint damage. Using OMT to treat arthrodial and myofascial somatic dysfunctions will, by definition, improve positional asymmetry and remove restrictions to joint range of motion. These improvements in joint function translate to a reduction of strain/shear forces on the joint, the type that the literature discussion above states will lead to joint damage and progression of OA. Specific techniques to address these types of somatic dysfunction include muscle energy, counterstrain, myofascial release, high-velocity, low amplitude (HVLA), and articulatory techniques. These technique methods are components of a comprehensive osteopathic principles and practice/osteopathic manipulative medicine (OPP/OMM), and the majority are required to be taught in the osteopathic medical student curriculum.[17] Therefore, these techniques are in the wheelhouse of most osteopathic physicians and are easy to apply in outpatient primary care, as well as other clinical settings. Studies looking at knee OA, for example, have shown improvements in range of motion, joint function, and pain after manual manipulation to the knee (including bony articular, axial traction, oscillatory mobilization and fascial/vascular manipulative techniques).[18] Given the impact of OA on the lower extremity, there is room for the physician to make significant improvement on patient morbidity and quality of life, while potentially reducing financial healthcare burdens related to this disease process.

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