Osteoarthritis

Abnormal biomechanics: a precursor or result of knee osteoarthritis?

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Biomechanical studies are required to differentiate between the causes and results of knee osteoarthritis

Although osteoarthritis (OA) is a common cause of disability in people over 65 years,[1] the causes and pathogenesis of knee OA remain largely unknown. In addition to biological studies, there is increasing interest in the contribution of biomechanical variables in the pathogenesis and management of this disease.[2–6]

OA of the knee occurs most commonly in the medial tibiofemoral compartment,[3] and increased regional load across this compartment's articular cartilage is believed to be an important factor in the pathogenesis of the disease.[2–6] The external knee adduction moment is argued to distribute 60–80% of total intrinsic knee compressive load to the medial tibiofemoral compartment,[3] and people with medial tibiofemoral OA tend to walk with larger knee adduction moments than normal subjects, resulting in increased medial compartment pressure.[2 7] Despite this, there is no clear evidence to suggest whether biomechanical abnormalities such as increased knee adduction moments cause or occur as a result of OA because all previous studies have examined people with established disease.

The knee adduction moment is generated by the combination of the ground reaction force, which passes medial to the centre of the knee joint, and the perpendicular distance of this force from the centre of the joint.[3] Given that varus alignment of the lower limb theoretically increases the perpendicular distance of the ground reaction force from the centre of the knee joint, it is not surprising that radiographic varus alignment is associated with the magnitude of the peak knee adduction moment in subjects with healthy and osteoarthritic knees.[5 6 8] However, only 50% of knee adduction moment variability in subjects with medial tibiofemoral OA is accounted for by the mechanical axis of the lower limb, emphasising the need for dynamic evaluation of the knee joint loading environment.[5] Preliminary studies have shown a moderately strong and significant association between both the line of progression and degree of foot rotation during gait and the magnitude of the knee adduction moment.[5 8] Further examination of dynamic factors associated with the knee adduction moment is required to help better understand the biomechanical pathogenesis of knee OA.

Although biomechanical factors are likely to contribute to the causes and pathogenesis of knee OA, their effect on joint morphology is unknown. Whereas increased mechanical load at the knee results in increased bone mineral density,[9 10] little is understood about cartilage response to repetitive altered load. Previous studies on people with knee OA showed that a larger knee adduction moment was associated with greater medial joint space narrowing.[2 6] However, because knee joint space consists of other structures such as menisci, joint space narrowing is not always a valid indicator of articular cartilage volume.[11] Although there is emerging evidence that cartilage volume will be a useful measure in studies of the pathogenesis of OA,[12–15] future work must examine how human tissues, including hyaline cartilage, respond to altered biomechanical variables such as the knee joint loads experienced during locomotion.

> "Quadriceps weakness has long been anecdotally recognised as a feature common to knee OA."

While joint compressive forces such as the knee adduction moment have received considerable attention in recent times, the relation between muscle weakness and knee OA is also becoming better understood. Quadriceps weakness has long been anecdotally recognised as a feature common to knee OA. Although longitudinal studies have shown that quadriceps weakness is a characteristic of people with established knee OA, weakness is also likely to be a risk factor for the development of disease.[16 17] A previous study showed that baseline knee extensor strength was lower in women without radiographic knee OA at initial examination who later developed OA changes, compared with unaffected women.[18] It may be that weak quadriceps strength during gait reduces the net extensor moment, which may help to counteract the lateral knee joint opening and medial compression that would occur if the knee adduction moment acted as an unopposed force. Although it may be useful to determine whether there is an association between quadriceps strength and the knee adduction moment during gait, further longitudinal studies are required to substantiate whether quadriceps weakness is a major cause or effect of knee OA.

Knee joint laxity, which is defined as displacement or rotation of the tibia with respect to the femur,[18] is another biomechanical variable argued to contribute to the pathogenesis of OA. One study showed that varus-valgus laxity is greater in the unaffected knees of patients with unilateral OA than in healthy control subjects,[19] suggesting that knee joint laxity may predispose to disease. However, such presumptions rely on the subjects with unilateral knee OA developing bilateral disease, which may not transpire. Nonetheless, it has been shown that varus and valgus alignment of the lower limb is associated with the progression of medial and lateral compartment knee OA, as determined by joint space narrowing and deterioration of physical function.[20] Moreover, changes resulting from the relation between alignment and disease progression can be detected after only 18 months of observation.[20] This suggests that over a relatively short time frame of intervention, the correction of biomechanical variables in people with established knee OA may delay the progression of disease.

Recently, a study combined several of the previously discussed variables and examined the role of quadriceps strength in the progression of knee OA using subjects with malaligned and lax knee joints.[21] Although earlier results suggested that women with reduced quadriceps strength have a greater risk of developing knee OA,[16] Sharma *et al*[21] concluded that greater quadriceps strength at baseline was associated with increased likelihood of OA progression in malaligned and lax knees. Although these results infer that strong quadriceps reduce the risk of developing knee OA, they also suggest that strong quadriceps are a risk factor for the progression of disease in people with malaligned and lax arthritic knees.

Biomechanical factors are increasingly being recognised as potential contributors to the causes and pathogenesis of knee OA. Until recently most studies on the biomechanics of knee OA have tended to be cross sectional rather than longitudinal, making it difficult to differentiate between the factors that are a cause or result of the disease. Longitudinal studies in normal subjects are required to determine whether biomechanical variables, such as the knee

adduction moment, predate the onset of OA or occur after disease is present. Other studies in subjects with OA will be required to clarify the role of biomechanical variables in disease progression, to identify potentially modifiable factors to alter the course of disease. Once these data are available, simple interventions such as gait re-education or orthoses may provide a future strategy for modifying the biomechanical risk factors associated with the onset or progression of knee OA.

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