Is patellofemoral osteoarthritis a common sequela of patellofemoral pain?

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Patellofemoral pain (PFP) remains one of the most common conditions encountered in sports medicine. Characterised by anterior knee pain that is aggravated by activities such as running, squatting and stair ambulation; PFP generally reduces or restricts physical activity. While PFP may subside with activity reduction, the natural history of this common condition is not one of spontaneous recovery. Indeed, PFP is often recalcitrant and can persist for many years. In a prospective study of people with PFP, symptoms persisted in 25% of people up to 20 years.[1] Despite considerable evidence for the efficacy of conservative interventions for PFP, such as multimodal physiotherapy,[2] these interventions do not appear to have long-lasting effects.[2] Compounding the management of PFP is that surgery for PFP is widely considered to have poor outcomes.

ARE PFP AND OSTEOARTHRITIS ON A DISEASE CONTINUUM?

As mentioned in the 2014 consensus statement from the International Patellofemoral Pain Research Retreat[3] there is speculation that PFP may be a prelude to degenerative joint changes and ultimately the development of patellofemoral osteoarthritis (PFOA).[4 5] While no current studies have prospectively studied people with PFP through to the development of PFOA (and thus verify this relationship), a recent systematic review[4] observed that individuals undergoing arthroplasty for PFOA were more than twice as likely (OR=2.31, 95% CI 1.37 to 3.88) to report having had PFP as an adolescent than patients undergoing an arthroplasty for isolated tibiofemoral OA.

In the absence of rigorous research in this field, we can infer a relationship between PFP and PFOA from the similarities in impairments between people with each condition. Cross-sectional studies of people with PFOA reveal impairments similar to those observed in individuals with PFP, such as patellar malalignment,[6–11] quadriceps dysfunction[12–17] and hip abductor dysfunction.[15] Furthermore, the pain patterns[18] and self-reported difficulties with stair ambulation[14 19] in people with PFOA are also similar to those with PFP.

Is PFOA a common sequela of PFP? Clearly not all patients with PFP will suffer PFOA. If PFP is a major risk factor for PFOA, or if chronic PFP is a sign of early PFOA, then the condition of PFP would likely be the subject of more research and taken more seriously by clinicians, than if it were considered a "self limiting" disease. Recent reports using newer imaging sequences report early degenerative changes (increased T1ρ relaxation times) in the patellar cartilage of patients with PFP that correlate strongly with the severity of patellar tilt.[20] Also, Farrokhi *et al*[21] identified elevated patellofemoral stress in people with PFP, a potential mechanism underpinning a link between PFP and structural joint disease. Further evidence for this link may come from our evaluation of adults aged >40 years with chronic PFP, where radiographic PFOA was present in 69% of people, and was more prevalent than tibiofemoral OA (45%).[22] Thus, the best available evidence suggests that PFP and PFOA exist along a continuum of disease.

TIME FOR A CHANGE IN OUR MANAGEMENT OF PFP?

In light of the potential disease continuum between PFP and PFOA, do we need to change the way we assess and treat people with PFP? PFOA imposes a considerable personal and societal burden.[18 19] Perhaps most importantly, but not surprisingly due to its intimate relationship to structures such as the infrapatellar fat pad, PFOA is a potent contributor to pain and dysfunction in people with knee OA.[18 19] Therefore, while we continue to employ the interventions with the best efficacy for reducing pain and improving function[2] for PFP alone, perhaps it is time to expand our treatment options to include education about OA and secondary prevention measures, such as weight management, increasing muscle strength and neuromuscular control.

WHAT CHANGES COULD WE MAKE?

Patients should be advised of the long-term implications of PFP. Avoid expectations of a 'cure' and educate patients to recognise and monitor their joint health status to effectively manage their joint loading and symptoms. The decision to return to sport must consider that higher joint loads are a risk factor for accelerated knee OA when the joint is abnormal,[23] yet no studies have evaluated whether resuming sports or physical activity in people with PFP will hasten the development of PFOA. Furthermore, the psychological and physical benefits of regular physical activity should not be underestimated. While the safe recommendation would be to undertake activities with less impact loading (ie, water-based activities or cycling), this suggestion may not be accepted by patients, and may not change the natural course of PFOA. A patient eager to return to higher impact activities can try to avoid other known factors for OA progression, including weight gain (see below) and lower limb muscle weakness (see below). The final decision must involve an informed dialogue between clinician and patient. It is imperative that if a person is advised not to return to his/her desired activities, the sports medicine team works to find alternatives that can promote psychological and physical well-being.

For many young adults who develop PFP, physical activity is the major means of weight control. When this is no longer possible, through protracted periods of rehabilitation, weight gain can ensue. This sets off a vicious cycle, whereby increasing weight increases joint loading, making return to physical activity even more painful and difficult. This weight creep can exacerbate the psychological factors (frustrations, grief, depression, etc) associated with exercise restriction, which further feeds the vicious cycle. Additionally, higher body mass index or body weight is associated with greater loss of patellar cartilage volume over time.[24 25] Patients need help if they are to avoid weight gain associated with PFP.

Quadriceps muscle function is integral to optimise patellofemoral joint loads and hence, joint health. Impaired strength and neuromuscular control of the quadriceps are frequently observed in people with PFP,[26–29] which likely reflects adaptations to reduce patellofemoral stress and pain. However, the persistence of such adaptations may increase susceptibility of the patellofemoral joint to further joint damage over time, through mechanisms such as altered sensorimotor function,[30] reduced shock absorption[31] and/or joint stiffening. Emerging evidence from cross-sectional[12 15 17] and prospective[32] studies support a potentially harmful relationship between relative quadriceps weakness and PFOA. Hip muscle weakness and poor motor control are also observed in people with PFP,[33 34] and poor control of hip extension, external rotation and abduction

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has potential to adversely influence patellofemoral joint stress.[35 36] Therefore, optimising quadriceps and hip muscle function may be required, not only over a short time period in order to effect changes in patellofemoral symptoms, but also for a lifetime. Strategies to maintain neuromuscular function over a long time period may need to be developed and evaluated.

Are PFP and PFOA on a disease continuum? Is it time for a change in our management of PFP? These are currently areas of intensive research. Until we have definitive answers regarding the relationship between PFP and PFOA (ideally from prospective controlled studies), sports medicine clinicians who are treating people with PFP should consider the possibility that their patients may be at risk of degenerative joint changes.

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Notes