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Patellofemoral joint osteoarthritis: An individualised pathomechanical approach to management

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A B S T R A C T

Patellofemoral joint integrity is maintained by an optimal interaction of passive, dynamic and structural restraints. Disruption of these mechanics can lead to structural joint damage and subsequent patellofemoral osteoarthritis, which is a prevalent and disabling condition with few effective conservative management strategies. Due to the influential role of biomechanics in this disease, targeting the specific pathomechanics exhibited by an individual is logical to improve their likelihood of a positive treatment outcome. This review summarises the effect of different pathomechanical factors on the presence and progression of patellofemoral osteoarthritis. It then presents a synthesis of mechanical effect of treatment strategies specifically addressing these pathomechanics. Identifying the pathomechanics and clinical characteristics of individuals with patellofemoral osteoarthritis that respond to treatment may assist in the development of individualised treatment strategies that alleviate symptoms and slow structural damage.

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Introduction

It is increasingly acknowledged that osteoarthritis (OA) is a complex and multidimensional disease. OA has the fastest growing prevalence of all musculoskeletal diseases and of these conditions was recently found to attract the greatest indirect health costs in terms of years of healthy life lost due to disease and disability adjusted life years [1]. Of the weight-bearing joints, the knee is the most commonly affected by OA [2] and is characterised by joint space narrowing, loss of articular cartilage, osteophyte formation, subchondral bone cysts and synovitis. While the majority of prognostic and intervention studies have focused on the medial tibiofemoral (TF) joint, OA of the patellofemoral (PF) joint, either in isolation or combined with TF OA, is reported to be more prevalent [3]. This is concerning as PF OA is a significant source of knee pain and disability [4,5].

Older age, female gender, high body mass index and previous anterior cruciate ligament injury are risk factors of both PF and TF OA [6,7]. However, PF OA has unique clinical characteristics that differentiate it from TF OA, such as difficulty descending stairs and pain on compression of the PF joint [6] (Fig. 1). It is also independently associated with lower self-perceived functional scores [8]. This unique disease burden could be due to the unique mechanics of the PF joint. Unlike the TF joint, the PF joint is not loaded during level walking. Rather, the PF joint reaction forces gradually increase up to 90° of knee flexion and can reach up to 8 times body weight depending on the type of activity (i.e. stair climbing, squatting etc.) [9]. During loaded activities, the PF joint shows maximal contact area and maximal cartilage thickness between 20° and 90° of knee flexion, where the compressive loads are highest [10]. This balance is dependent on optimal interaction of passive, dynamic and structural restraints [11] and can be easily disturbed, resulting in structural joint damage [12]. Thus, PF OA is largely biomechanically mediated and it seems logical that the specific biomechanical factors that are disrupted in a particular individual need to be addressed when designing a treatment strategy.

Designing treatment strategies that are individualised to target patients' specific pathomechanics follows current recommendations for the management of PF OA [4,13]. Due to the diversity of knee OA with respect to aetiology, clinical and radiographic presentation, one-size-fits-all treatment approaches are suboptimal. Rather, tailoring management to the individual is preferred in order to

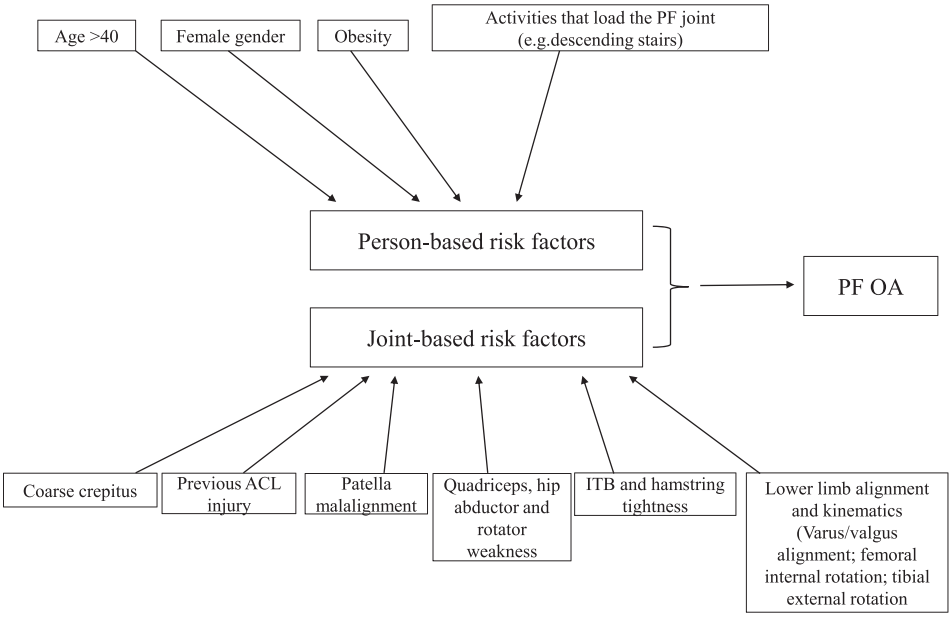

Fig. 1. Risk factors for the presence and progression of PF OA at an individual level and at a joint level.