PATELLOFEMORAL PAIN SYNDROME IN ATHLETES

Written by Robbart van Linschoten, Qatar

Patellofemoral pain is seen frequently in young and physically active people. The incidence in primary care for the young adult age is about 10 to 12 new cases per 1000 patients per year, while in sports medicine practice, its prevalence may well be around 25%.

The palette of complaints is varied, from mild short-term pain to long-term and load-dependent pain, leading to functional restriction. Patellofemoral pain has been considered as a separate entity for decades, although various names have been used for the condition. Chondromalacia patellae, patellar chondropathy and anterior knee pain have been used interchangeably. Over the last 10 years there has been consensus to refer to these complaints as ‘patellofemoral pain syndrome’ (PFPS). The condition is characterised by peri- or retro-patellar pain which occurs during loaded bending of the knee (like in climbing stairs, cycling or other activities), during prolonged sitting with bended knees or kneeling. Specific causes of knee pain such as patellar tendinopathy, Osgood Schlatter’s disease, intra-articular injuries, osteoarthritis are to be ruled out in this clinical picture.

It is assumed that patellofemoral pain has a favourable prognosis. However, in a sporty and active population, this assumption is to be questioned. A number of studies show that even after several years, 30 to 50% of patients with patellofemoral pain are not free of symptoms.

ETIOLOGY AND PATHOPHYSIOLOGY

For many years, the majority of research into the origin of PFPS has focused on the biomechanics of the patellofemoral joint, in relation to the position and anatomy of the lower extremities (mechanical model) and on the movement of the patellofemoral joint (neuromuscular model).

Original assumptions in the mechanical model considered that a deviation of position (valgus position of the knee [Q-angle] or anteversion of the hip) and incongruence between patellar surface and the trochlea femoris (patellar dysplasia, patella alta) leads to malalignment of the patellofemoral joint. This would predispose to a lateral malalignment of the patella in which ultimately the patella can dislocate. In prospective studies however, only an association between hypermobility of the patella and the development of patellofemoral pain complaints has been established. Nowadays, in the mechanical model the emphasis is put more on anterior knee pain associated with patellar dislocations in conjunction with objective radiological abnormalities.

In many cases the absence of a direct relationship between pain and mechanical-anatomical abnormalities have led to the
idea that the influence of the quadriceps muscles might be of greater importance to patellar tracking at the trochlea than the anatomical structures alone. This neuromuscular model (‘maltracking’) became an important concept in the 1980s and 1990s. The idea was fuelled by initial research that linked patellofemoral pain to an altered activation pattern of the medial (vastus medialis obliquus [VMO]) and lateral (ventral lateral [VL]) part of the quadriceps muscle. Interpretation of the various studies subsequently showed that there is no convincing evidence for the proposition that the VMO contracts later than the VL in patients with PFPS compared to pain-free control patients.

Since there is no conclusive evidence for the mechanical model or the neuromuscular model to date, a biological model is proposed to explain the symptoms. This biological model assumes that the tissue around the patella can be loaded physiologically within a pain-free zone. Outside this physiological zone (‘envelope of function’) overloading of structures can occur. In this situation tissue can literally fail (rupture or break) or cannot sufficiently recover from repetitive overload. This could eventually lead to permanently reduced capacity and therefore a smaller ‘envelope of function’.

Although the model incorporated both a mechanical component and a dynamic component, it offers no explanation for the origin of the pain. Several structures around the patella include pain receptors (lateral retinaculum, infrapatellar fat pad, joint capsule and subchondral bone) but none of them have been found as a single cause for the pain. It is therefore currently unclear what the genesis of the patellofemoral pain syndrome is or which structures cause pain.

**PFPS is in fact still a diagnosis ‘by exclusion’ and less formally formulated a ‘wastebasket diagnosis’**

Although the model incorporated both a mechanical component and a dynamic component, it offers no explanation for the origin of the pain. Several structures around the patella include pain receptors (lateral retinaculum, infrapatellar fat pad, joint capsule and subchondral bone) but none of them have been found as a single cause for the pain. It is therefore currently unclear what the genesis of the patellofemoral pain syndrome is or which structures cause pain.

**DIAGNOSIS AND ADDITIONAL INVESTIGATIONS**

As shown above, PFPS is in fact still a diagnosis ‘by exclusion’ and less formally formulated a ‘wastebasket diagnosis’. The history of the patient and the physical examination along with the exclusion of other diagnoses are sufficient in order to outline the diagnosis.

Patellofemoral pain syndrome is primarily about pain around the kneecap. The symptoms are mainly insidious – often a period of excessive load like increased sports activities may be associated with the pain and sometimes a mild direct trauma to the kneecap appears to be the beginning of the complaints. Known symptoms are pain during squatting and prolonged sitting with a bended knee (‘movie sign’). Crepitus of the knee is a variable finding and occasionally (pseudo) giving way is reported. When examining the knee, effusion is rarely found and actually indicates another pathology. Pain is mostly prominent at the medial and lateral borders of the patella (patellar facets) and sometimes also at the inferior pole of the patella. Provocative tests for the patella, proximal to distal movement while compressing the patella on the femur (Rabot sign, Clarke’s test) are often painful and not very specific.

Radiological investigations should be done only based on the presumption of more specific causes of peripatellar pain such as patellar dislocations.

Figure 1: The ‘envelope of function’ (according to Dye, 2005): increase in activities (both frequency and intensity) leads to tissue loading outside the zone of physiological homeostasis.
(instability), malalignment, patellofemoral osteoarthritis or osteochondritis dissecans. A simple X-ray (anteroposterior, lateral and skyline view and a notch view) for a suspected osteochondritis dissecans of medial femur will render sufficient information about anatomical-structural defects. More extensive investigations (computed tomography scan, magnetic resonance imaging) are to be considered by the orthopaedic surgeon for further analysis of patellar malalignment and patellar dislocations.

TREATMENT OPTIONS AND PROGNOSIS

The majority of patellofemoral complaints will be diagnosed and dealt with by primary care and sports physicians. The general approach for patients with PFPS is to inform the patient about the favourable course and good prognosis of the complaints, to prescribe rest and instruct on avoidance of activities which are provocative for pain. The application of isometric quadriceps exercises may be suggested. Despite this cautious approach about 30% of patients are referred to physiotherapy.

Physiotherapy is a broad concept and treatment for PFPS often consists of combinations of ‘treatment modalities’ like massage, muscular electrostimulation, exercise therapy or patellar taping and orthotics.

Exercise therapy is an intervention for which much research has been done. The origin of exercising for PFPS can be traced back to the 1970s when it became clear that surgical intervention for patellofemoral pain had a very small indication area. Since then, exercise therapy became an alternative treatment originally involving simple isometric quadriceps contractions followed by ‘closed kinetic chain’ and ‘open kinetic chain’ exercises as well as eccentric quadriceps exercises. During recent years, much interest has focussed on the (additional) effect of muscle strengthening exercises for the hip and gluteal muscles combined with quadriceps strengthening.

Although widely used, the effectiveness of exercise therapy remained unclear mainly because published studies had significant flaws in the methodology. Many studies did not meet the characteristics of good research such as:

- lack of randomisation,
- unclear outcome measures,
- short follow-up duration and
- the use of small study groups.

The results of a randomised clinical trial – the PEX study – on the effects of supervised and protocolised exercise therapy for patellofemoral pain syndrome have been published in 2009. In this study, patients followed a daily exercise programme for 3 months (initially under the supervision of a physiotherapist) focused on muscle strengthening for knee and hip muscles and co-ordination training. The control group received the usual advice according to the Dutch guidelines for primary care physicians. This intervention study (n=131) shows that exercise therapy is more effective on pain and functional improvement than the current guideline of primary care physicians. The effect sizes of exercise versus ‘usual care’ ranged from 0.35 for
function to 0.60 for pain at 3 and 12 months follow-up. The level of subjective recovery of the patients was also studied. Although a higher proportion of patients in the exercise group considered themselves ‘recovered’ (62 vs 50%), the difference between the two groups after 3 and 12 months was not significant.

The overall effectiveness of exercise for patellofemoral pain syndrome is reported in a recent systematic review. This review shows evidence for the effectiveness of exercise with respect to pain on short- and long-term recovery and evidence regarding functional improvement in the short term. However, the effects of exercise therapy on the outcome ‘recovery’ are not clear. Other interventions, additional to or compared with exercise such as taping, bracing, insoles and manual therapy, could not be demonstrated to be more effective than exercise alone.

Although there is a general agreement that primarily a conservative strategy for PFPS should be followed, there are specific cases in which surgical intervention may be considered. In these cases, evident patellar malalignment, anatomical ‘maltracking’ problems and recurrent patellar dislocations are addressed. Only after very careful consideration – both objectively and subjectively – are there a number of surgical techniques that can be used to correct an anatomical disorder. Re-attachment of the medial patellofemoral ligament and a medial transposition of the patellar tendon give good results.

CONCLUSION

Patellofemoral pain remains a common problem in primary care and sports medicine. The diagnostic and therapeutic approach to the complaints has been changed during the last few decades. The initial dominance of the anatomical-mechanical model has been replaced by a functional approach. It has become clear now that there is no direct relationship between pre-existing anatomical or radiological abnormalities and patellofemoral pain. The primary treatment for PFPS comprises a conservative approach and one should be very cautious with respect to surgery.

Based on recent research, an active approach to primary care through intensive (supervised) exercise therapy is shown to be more effective for patients with patellofemoral pain syndrome than a ‘wait and see’ management.

Although patellofemoral pain can be prolonged and sometimes seriously debilitating, it remains important to be cautious with regard to surgery and to emphasise the natural history of the complaints, even though that may be less favourable than originally assumed.

In patients with recurrent patellar dislocation, clear instability or protracted patellofemoral pain, a thorough analysis into ‘maltracking’ may be executed. With the demonstration of anatomical abnormalities surgery may be necessary.

References