(vii) Patellofemoral instability

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Abstract
The accurate assessment and management of the unstable patellofemoral joint depends on knowledge of the anatomy and stabilizing structures. Surgery should not be considered until non-operative interventions have failed and the recurrent nature of the disease has resulted in functional impairment. The surgical strategy should, whenever possible, aim to restore normal anatomy rather than introduce new abnormalities. Often a combination of surgical techniques is required.

Keywords patellar dislocation; patellofemoral dysplasia; patellar instability; patellofemoral malalignment

Introduction

The rate of first-time dislocation is highest among female adolescents. Following a first event, 17% of patients will experience subsequent instability. The younger a patient is at the time of first dislocation and the more severe the dislocation, the greater the risk of subsequent dislocation. If the patient has an established history of subluxation or dislocation the risk of subsequent episodes rises to 50%.[1,2]

Stable tracking of the patella relies on complex orchestration of static ligament and capsular constraints with balanced dynamic forces from muscle actions working within the anatomical constraints of the joint. Disorders in any of these relationships results in maltracking or instability of the patellofemoral joint. Patients with patellar instability will have either objective instability (true atraumatic dislocation with an anatomical abnormality) or potential instability (patellar pain, a feeling of the knee giving way or locking, with an associated underlying anatomical abnormality).[3]

A number of morphological, static and dynamic factors predispose the patellofemoral joint to instability. Trochlear dysplasia and patella alta are the most common morphological abnormalities, with lateral condylar hypoplasia and torsional abnormalities being occasionally present. Abnormalities in static structures, including the medial patellofemoral ligament (MPFL), lateral retinaculum and iliotibial band, or the dynamic soft-tissue forces may lead to symptoms.

Malalignment of the patellofemoral joint does not necessarily equate to functional or symptomatic instability. Many patients will function without problems with simple, non-operative interventions. When non-operative treatments have failed and the recurrent nature of the disease has resulted in functional impairment, the goals of re-alignment surgery are to stabilize patellar tracking and optimize load transmission within the joint. Thus, the challenge is not only to understand the different anatomical abnormalities but also the interplay of the components described above, before considering corrective measures.[4]

Anatomical and physiological considerations

Morphology of the trochlea and patella

The trochlea is a concave trough, its shape dependant on the contour of the distal femur and depth of overlying articular cartilage. The depth and steepness of this grove influence the stability of articulation. The lateral facet of the trochlear groove is highest on the anterior aspect of the femur and decreases in height more distally and posteriorly, giving some osseous constraint to the patella in extension and early flexion. Dysplasia of the lateral femoral condyle reduces the osseous protection conferred to the patella.

The role of patellofemoral dysplasia has only recently been fully appreciated and implicated in patellar dislocation. Trochlear dysplasia is flattening or elevation of the central area of the trochlear groove, rather than deficiency of the lateral facet of the groove (Figure 1). Dysplasia of the femoral trochlea has been found to correlate most strongly with objective patellar instability.[3] The cartilaginous surface geometry of the patella and trochlea has been shown to differ from that of the underlying osseous morphology in patients with trochlear dysplasia.[5,6] The overlying cartilage exacerbates the abnormal shape.

Figure 1 Axial MR scan demonstrating flat dysplastic trochlea with associated tilted patellar tendon. As can be seen this patient has patella alta (only patellar tendon visible).

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Patella alta is associated with instability since the patella lies superior to the lateral femoral condyle and the trochlear groove (Figure 2). This results in a greater arc of knee flexion during which the patella is not engaged within the trochlea and is therefore at increased risk of instability. The length of the patellar tendon has been shown to be equal to the diagonal length of the patella. Variations of more than 20% are considered abnormal.[7] A number of established indices have been used to define patellar height, although the authors find the more recently described patellotrochlear index is more useful.[8]

Static soft-tissue constraints

Static soft-tissue constraints are most important before the patella is fully engaged in the trochlea. The primary static restraint preventing lateral subluxation or dislocation of the patella is the medial patellofemoral ligament (MPFL). This extra-synovial ligament runs transversely from the medial femoral condyle, deep to vastus medialis, inserting into the proximal two-thirds of the patella (Figure 3). The MPFL is the primary passive soft-tissue restraint to lateral patellar displacement, reported to provide greater than 50% of lateral restraint from 0° to 30° of knee flexion.[9,10]

The lateral sided fascial system has three layers that contribute to a lateral static soft-tissue constraint. The superficial layer is confluent with the iliotibial band. The intermediate layer extends from the deep layer of the iliotibial band to the mid-lateral aspect of the patella. The deep layer is confluent with the knee capsule.[11]

Other stabilizers of the patellofemoral joint include the patellomeniscal and patellotibial ligaments, which attach on the medial and lateral sides respectively. At present little consideration has been given to assessment of these ligaments and management of dysfunction.

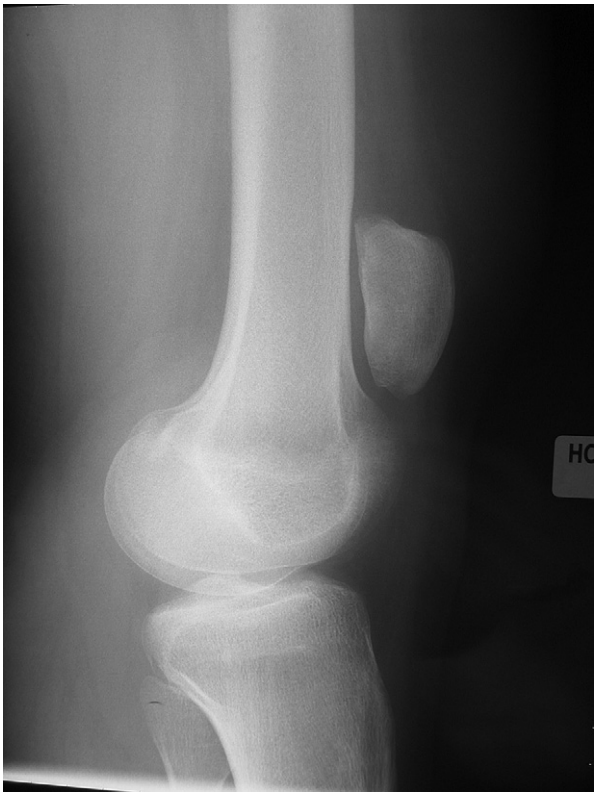

Figure 2 Lateral radiograph demonstrating patella alta.

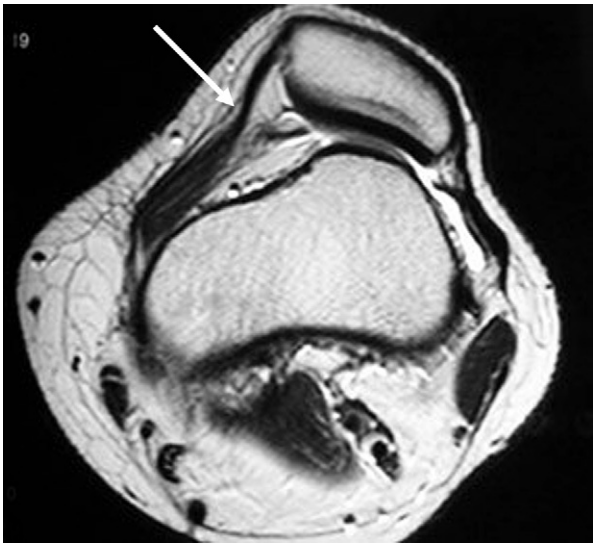

Figure 3 Axial MR demonstrating a grossly stretched MPFL (arrow) associated with patella alta.

Dynamic soft-tissue constraints

The rectus femoris, vastus lateralis, vastus intermedius and vastus medialis insert in a layered arrangement onto the proximal patella, creating different vectors of force on the patella.[12,13] An imbalance of strength may lead to instability, although this influence on the patellofemoral joint remains controversial.

The lower part of the vastus medialis, known as the vastus medialis obliquus (VMO), may be important in patellar instability.[13,14] There is attachment of the deep fascia of the VMO to the MPFL, and they probably act together as a combined dynamic complex.[15] The VMO is the first part of the quadriceps to weaken and the last to strengthen when function is inhibited.[16]

The vastus lateralis is variable in length, cross sectional area and orientation and its role in patellofemoral instability remains uncertain.[4]

Coronal and rotational alignment of the lower limb and the attachment points of the extensor mechanism result in lateral force vectors through the patellofemoral joint. The direction of pull of the quadriceps mechanism relative to the patellar tendon is known as the quadriceps angle (Q angle) (Figure 4). The Q angle is greater in females than males (15° *vs.* 10°) as a result of a wider pelvis and an associated incidence of valgus knee alignment. The Q angle can be estimated but is inaccurate. An objective measurement of the tibial tubercle to trochlea groove offset (TTTG) from MRI or CT scans is an equivalent, accurate alternative.

An increased Q angle results in greater lateral displacing forces acting on the patella. The effect is a more laterally placed patella; the patella tends to tilt laterally and there is a rise in patellar contact pressure laterally.[17] The overall effects from this depend on the angle of knee flexion and the competence of the trochlea and soft-tissue restraints.[18]

The Q angle is greatest in full extension as the tibia rotates externally (screw-home mechanism) moving the tibial tubercle