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Review article

Acute traumatic patellar dislocation

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ABSTRACT

Inaugural traumatic patellar dislocation is most often due to trauma sustained during physical or sports activity. Two-thirds of acute patellar dislocations occur in young active patients (less than 20 years old). Non-contact knee sprain in flexion and valgus is the leading mechanism in patellar dislocation, accounting for as many as 93% of all cases. The strong displacement of the patella tears the medial stabilizing structures, and notably the medial patellofemoral ligament (MPFL), which is almost always injured in acute patellar dislocation, most frequently at its femoral attachment. Lateral patellar glide can be assessed with the knee in extension or 20° flexion. Displacement by more than 50% of the patellar width is considered abnormal and may induce apprehension. Plain X-ray and CT are mandatory to diagnose bony risk factors for patellar dislocation, such as trochlear dysplasia or increased tibial tubercle-trochlear groove distance (TT-TG), and plan correction. MRI gives information on cartilage and capsulo-ligamentous status for treatment planning: free bodies or osteochondral fracture have to be treated surgically. If patellar dislocation occurs in an anatomically normal knee and osteochondral fracture is ruled out on MRI, non-operative treatment is usually recommended.

1. Introduction

Acute traumatic patellar dislocation is the second most frequent cause of traumatic hemarthrosis of the knee, after anterior cruciate ligament tear, and accounts for 3% of all traumatic knee lesions [1,2].

It is usually due to trauma sustained during physical or sports activity. In two-thirds of cases, it concerns young active subjects under 20 years of age [3–5].

It may have long-term consequences: instability of patellar origin, pain, recurrent dislocation and patellofemoral osteoarthritis [6].

2. Anatomy

The patellofemoral joint is complex; stability depends on osteoarticular conformation and static and dynamic stabilization structures. Any change in anatomy, such as extensor apparatus alignment defect, patellofemoral dysplasia or trauma, can induce patellar instability.

The embryonic patella develops during the 7th week of gestation. Cells develop in the deep layer of the patellar tendon, forming a nascent cartilage structure, which ossifies only at the age of 4–6 years [7]. Six centers of patellar ossification develop then fuse to form a single nucleus of ossification.

Normally, the two patellar joint surfaces, medial and lateral, are symmetric and congruent with the femoral trochlea. Trochlear morphology remains relatively constant but, with growth, the thick trochlear cartilage thins in the middle, creating an illusion of a trochlear "hollow" [8]. The peripatellar soft tissue and particularly the medial patellofemoral ligament (MPFL) and vastus medialis obliquus (VMO) muscle contribute significantly to joint stability [9]. The MPFL inserts to the femur between the medial epicondyle and the adductor tubercle, and to the superomedial edge of the patella (Fig. 1).

3. Biomechanics

The MPFL provides 50–80% of the mechanisms counteracting lateral patellar glide [10,11]. Lateral patellar dislocation renders it incompetent, promoting recurrence of dislocation [12].

Neighboring structures, such as the patellomeniscal or patellotibial ligaments and the superficial medial retinaculum, make lesser contributions to patellar stability.

The VMO muscle is very important, acting as a dynamic stabilizer, and is intimately related to the MPFL [8].

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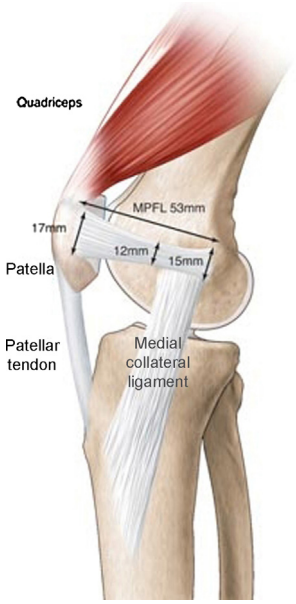

Fig. 1. Anatomic diagram showing MPFL position.
From Maîtrise orthopédique *No. 186–2009.*

Patellar tracking and patellofemoral stability result from highly complex interactions between these static and dynamic stabilizers. Patellar trajectory in the femoral trochlea is not straight, but includes tilt, glide and rotation.

With the knee in complete extension, the patella lies above and beyond the femoral sulcus. It enters the trochlea when the knee is in 10–30° flexion, depending on patellar tendon length: trochlear entry is later in case of patella alta or of "short" trochlea, in which case osteoarticular stabilization of the patellar is lacking when the knee is in extension or slight flexion [8,13].

4. Definitions

Patellar dislocation, if not otherwise specified, is lateral dislocation; medial dislocation, which is exclusively iatrogenic, and intra-articular dislocation are very rare, but exist and need to be known.

There is an important distinction between inaugural traumatic dislocation (single), recurrent dislocation (several) and habitual dislocation, as definitions and treatments differ. Inaugural traumatic dislocation ruptures previously intact medial para-patellar structures [5,14,15], and may remain single. In common to all inaugural traumatic dislocations is knee hemarthrosis, due to medial para-patellar stabilization structure rupture. Dislocation becomes "recurrent" if repeated several times following the inaugural traumatic dislocation. Habitual dislocation is a complex entity defined by patellar dislocation on each movement of flexion of the knee.

5. Incidence and risk factors

There have been several studies of incidence of inaugural patellar dislocation in adults [3–5,14]. Mean annual incidence of patellar dislocation varies with age group: it is between 5.8 and 7.0 per 100,000 person-years in the general population, but 29 per 100,000 in 10–17 year-olds; It reaches 69 per 100,000 person-years in military personnel undergoing aptitude testing and very demanding training [14].

Women are at greater risk than men, as are young subjects, whether military or in the general population; risk decreases with age [3–5,14]. This may be due to young subjects' more intense physical activity and/or to morphologic and tissue-related factors that make some adolescents more vulnerable.

6. Traumatic mechanism and predisposing factors

The typical mechanism underlying patellar dislocation is a movement of the knee in flexion and valgus without direct contact, accounting for 93% of traumatic patellar dislocations [3]. Most patients report a sensation of slippage, intense pain and secondary effusion, often suggestive of knee sprain. True traumatic dislocation, caused by direct tangential shock dislocating the patella laterally, also occurs. History taking can determine the trauma mechanism.

Whatever the mechanism, according to Sillanpaa et al., almost all patients with traumatic patellar dislocation show hemarthrosis, MPFL lesion and medial patellar wing fracture. Osteochondral fracture occurs in 25% of traumatic patellar dislocations [3].

Recurrence risk is increased 6-fold in case of history of ipsi- or contralateral patellar dislocation [4]. Dejour and Walch, in 1987, attributed patellar predisposition toward dislocation and instability with recurrence to four principal and certain secondary factors.

6.1. Principal predisposing factors

6.1.1. Trochlear dysplasia

Trochlear dysplasia [16,17] is a determining factor, consisting in progressive filling of the trochlear floor, resulting in greater or lesser smoothing out of the trochlear groove, which becomes flat or convex. There are several associated signs on strict lateral knee X-ray (Fig. 2):

- *crossing sign*: crossing between the deep line of the trochlea and the anterior edge of the two condyles (which would normally be more anterior), at and below which point the trochlea is completely flat;
- *supra-trochlear spur*: a spur above the trochlea, resulting from overall trochlear pre-eminence;
- *double contour*: projection of the subchondral bone of the hypoplastic medial edge of the trochlea;
- *trochlear projection*: measured with respect to the tangent to the last 10 cm of the anterior femoral cortex; the floor of the trochlea may lie forward, flush or backward with the cortex (positive, zero or negative projection, respectively);
- *trochlear depth*: mainly of prognostic value. Maldague and Frot [18] measured trochlear depth at 1 cm from the summit. Dejour et al. [16,19] suggested a variant: a tangent to the last 10 cm of the posterior femoral cortex and a perpendicular through the summit of the posterior condyles cross at a point through which a line is drawn subtending an open angle of 15° forward and downward; this line crosses the line of the floor of the trochlea at point B and the bicondylar line at point A. It is the distance AB that is to be measured, with a threshold of 4 mm, values less than which signifying pathology.

Dejour and Lecoultre classified trochlear dysplasia in 4 grades [20] (Fig. 3):

- *grade A*: crossing sign with normal morphology of the sides of the trochlea on CT;
- *grade B*: crossing sign, supra-trochlear spur, trochlea flat on CT;
- *grade C*: double contour ending below the crossing sign, medial hypoplasia and lateral convexity on CT;
- *grade D*: double contour ending below the crossing sign, supra-trochlear spur, medial hypoplasia and lateral convexity on CT, with the two sides joining in a "cliff".