The Role of Trochlear Dysplasia in Patellofemoral Instability

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The Role of Trochlear Dysplasia in Patellofemoral Instability

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Abstract
Trochlear dysplasia is characterized by abnormal trochlear morphology and a shallow groove. It is associated with recurrent patellar dislocation, but it is unclear whether the dysplasia is congenital, the result of lateral tracking and chronic instability, or caused by a combination of factors. Lateral radiographs elucidate the crossing sign and characteristic trochlear prominence. Recurrent patellofemoral instability is multifactorial, and each component must be considered in determining treatment. Managing other factors associated with recurrent instability may compensate for a deficient trochlea and provide stability. Medial patellofemoral ligament reconstruction is recommended for patellofemoral instability in the presence of trochlear dysplasia in patients without patella alta or increased tibial tubercle–trochlear groove distance. Trochleoplasty should be reserved for severe dysplasia in which patellofemoral stability cannot otherwise be obtained.

Recent patellar instability has been reported in 15% to 44% of patients following nonsurgical treatment of initial patellar dislocation. Fithian et al reported recurrence in 49% of patients with at least two prior instability events. Recurrent patellar dislocation is caused by deficiency in static soft-tissue constraints (eg, medial capsular restraints, medial patellofemoral ligament [MPFL]), dynamic constraints (eg, vastus medialis obliquus [VMO], inadequate core stability), and osseous structures (eg, patella alta, increased distance of the tibial tubercle–trochlear groove [TT-TG], rotational malalignment, trochlear dysplasia).

Trochlear dysplasia is seen as a shallow, flattened trochlear groove. In the early 1960s, Brattstroem observed an association between trochlear dysplasia and patellar instability. Trochlear morphology and its role in patellofemoral pathology has since been studied in many European centers. However, trochlear dysplasia has received little attention in the United States.

Trochlear dysplasia is estimated to occur in <2% of the population; however, it is present in up to 85% of patients with recurrent patellar instability. Dejour and Le Coultre reported that 96% of patients with a history of patellofemoral dislocation had radiographic evidence of trochlear dysplasia. Despite the association of trochlear dysplasia with patellofemoral instability, there are few reports regarding its etiology, assessment, and management.

Anatomy and Biomechanics
The trochlear groove has a complex three-dimensional shape that varies...
from proximal to distal. The lateral wall is highest proximally on the anterior surface of the femur, and it decreases in height distally as the trochlear groove deepens. Considerable individual variability exists with regard to the orientation, length, and curvature of the trochlear groove. The appearance of the sulcus angle on axial radiographs and CT is different from the appearance of the same angle on MRI. In an MRI study of 23 patients with trochlear dysplasia, van Huyssteen et al reported a mean bony sulcus angle of 168° and a mean cartilaginous sulcus angle of 187°. Shih et al found that in healthy knees, articular cartilage is usually thicker in the center of the groove. Patellofemoral joint stability is dependent on coordinated interaction between static, dynamic, and osseous structures to keep the patella centered within the trochlea during knee range of motion (ROM). This interdependence was highlighted in a recent MRI study of MPFL injury. The authors reported that in patients with acute patellofemoral dislocation, the location of the MPFL injury is determined by the extent of trochlear dysplasia, patellar height, and TT-TG distance.

During full extension, the patella is not engaged in the trochlear groove, and it is dependent on soft-tissue restraints for stability. The medial knee retinacular structures, including the MPFL and the medial patellotibial ligament, provide static restraint to lateral patellar translation. The MPFL guides the patella into the trochlear groove during the first 20° of knee flexion. In early knee flexion, the distal aspect of the patella makes contact with the superior aspect of the trochlea. Correct patellar centering within the groove during early knee flexion is essential to maintaining patellofemoral stability. Correct patellar positioning is largely determined by the dynamic and static soft-tissue structures. At 30° of knee flexion, the patella should be centered in the trochlear groove, with <1 cm lateral translation. The depth and slope of the trochlea influence patellar stability as the knee continues to flex. The role of soft-tissue constraints is minimized with flexion >30°, at which point the trochlea becomes the most important patellar stabilizer. The height and slope of the lateral trochlear facet provide the primary resistance to lateral patellar translation as knee flexion progresses. Senavongse and Amis reported that lateral patellar translation is more likely with a flattened lateral trochlear wall than with either a ruptured medial retinaculum or a released VMO. Dynamic constraints have a more variable and inconsistent role in patellar stability than do static restraints and trochlear geometry. Trochlear dysplasia results in decreased resistance to lateral patellar translation and increased stress on the medial soft tissues as they assume a greater role in the prevention of lateral patellar dislocation.

**Etiology**

Patellofemoral instability has been extensively studied, but most re-
search consists of retrospective reviews and observational cohort series. Patellar instability may be acute or chronic. Specific anatomic characteristics are associated with recurrent patellofemoral instability.\textsuperscript{21-23} Bony and soft-tissue deficiencies diminish the ability of the patella to remain centered within the trochlear groove during knee ROM. Cash and Hughston\textsuperscript{22} reported a 43% rate of redislocation following nonsurgical treatment in patients with underlying predisposing factors (eg, trochlear dysplasia, VMO dysplasia, hyperlaxity) compared with a 20% rate of redislocation following nonsurgical treatment in patients without predisposing factors. Other predisposing factors include lower extremity rotational malalignment, increased TT-TG distance, and patella alta. Each of these factors may be present in isolation; however, a combination of predisposing factors is more common.

An association between trochlear dysplasia and recurrent patellar instability has been established.\textsuperscript{2} However, no prospective developmental study has demonstrated patellar instability secondary to trochlear dysplasia. It is not known whether dysplasia leads to instability or whether inborn abnormalities in patellofemoral tracking and alignment create dysplasia.

Glard et al\textsuperscript{24} reported that trochlear shape appears to be the same in fetuses as in adults. The authors suggest a genetic origin of trochlear dysplasia. However, they examined only sample means from each group. Pre-disposing factors (eg, hyperlaxity, rotational malalignment, lateral extensor mechanism vector) are known to cause recurrent patellar subluxation or dislocation, a lateralized patellar resting position, and distortion of trochlear morphology. In the pediatric hip, a reduced femoral head is necessary for normal acetabular development (ie, depth, shape). Subluxation or dislocation of the femoral head can result in a dysplastic, shallow socket.\textsuperscript{25} Lateral patellar positioning may limit the development of normal trochlear depth and morphology. Persistent lateral patellar tracking can result in a flattened lateral trochlea and can indirectly create a shallow groove. This scenario is suggestive of a developmental process. Patellar dislocation typically occurs in persons with several anatomic risk factors. Patellar dislocation likely is caused by a combination of congenital and developmental factors.

**Classification**

Traditional trochlear dysplasia classification systems are associated with poor interobserver and intraobserver reliability.\textsuperscript{5,26} Dejour and Le Coultre\textsuperscript{5} developed a classification system based on combined evaluation of axial and lateral radiographs. This classification has been found to be more reproducible than previous systems\textsuperscript{26} (Figure 2). Type A dysplasia is characterized by a shallow trochlea, a crossing sign on the lateral view, and a shallow sulcus angle >145° on the axial view. Type B dysplasia is characterized by the appearance of a flat trochlea on axial radiographs and a supratrochlear spur on lateral images. Type C dysplasia is characterized by a shallow sulcus angle >145° on the axial view and by a shallow trochlea on the lateral view. Type D dysplasia is characterized by a combination of factors, including a supratrochlear spur on lateral images and an asymmetry of the trochlear facets on the axial view.
well as medial hypoplasia and lateral convexity on axial radiographs. Type D dysplasia is the most extreme form. It is characterized by asymmetry of the trochlear facets as well as a cliff between the medial and lateral facets on the axial radiographic view. The crossing sign, double contour, and supratrochlear spur are seen on the lateral view.

**Clinical Evaluation**

The first step in the evaluation is to determine whether instability or pain is the primary complaint. Giving way secondary to pain must be differentiated from episodic pain caused by patellofemoral instability. Patients with patellofemoral instability describe pain during and after subluxation or dislocation; however, these patients may be relatively asymptomatic between episodes. A thorough history is required, including the mechanism of injury and leg position during the initial dislocation. Substantial trauma sustained during the first episode of instability can result in osteochondral fracture or disruption of proximal medial soft-tissue restraints. When possible the number and severity of subsequent instability events should be determined. The patient with recurrent instability may report giving way and apprehension during routine activities of daily living or minor twisting injuries. Predisposing factors should be identified, such as increased TT-TG distance (eg, lateralized tibia tubercle, excessive femoral anteversion), patella alta, trochlear dysplasia, poor core stability, and soft-tissue hyperlaxity. Trochlear dysplasia is suspected in patients with failed medial imbrication and in those who report many dislocations within a short period.

Pain, guarding, and swelling make physical examination difficult in patients with acute patellofemoral dislocation. However, determining the type of instability (ie, medial, lateral) and assessing lower extremity alignment and the status of soft-tissue constraints can facilitate management. In the patient with chronic or recurrent patellofemoral instability, a full patellofemoral examination should be performed to assess lower extremity alignment, core stability, Q angle, patellar tilt, crepitus, tracking, periarticular tenderness, generalized laxity, apprehension, muscle strength, and flexibility. The patella should be centered in the trochlear groove during the first 10° to 20° of knee flexion. Patellar mobility is evaluated with the knee in full extension and in flexion. Patellar translation should measure ≤1 cm (ie, approximately two quadrants) in 30° of knee flexion, and it should be symmetric medially and laterally. Laxity testing is limited by its reliance on the degree of knee flexion, depth and length of the trochlea, and the integrity of the retinacular restraints. Despite these limitations, increased laxity and lack of a firm end point are suggestive of MPFL deficiency.

Apprehension and guarding with lateral patellar translation are pathognomonic for patellofemoral instability. Patellar apprehension is not always present, however. Sallay et al27 found a positive apprehension sign in only 9 of 23 patients following patellar dislocation. In the moving patellar apprehension test, the patella is pushed laterally as the knee is flexed. Apprehension or quadriceps activation denotes a positive test. This test of patellar instability was found to have 100% sensitivity, a 100% negative predictive value, 88% specificity, and 94% accuracy.14

**Imaging**

Standard radiographs, including a weight-bearing PA view as well as notch, lateral, and Merchant axial views, are essential in the evaluation of patellofemoral instability. Initial radiographic analysis is done to assess trochlear morphology and patellar positioning. Patella alta and trochlear dysplasia are important risk factors for recurrent instability.28 The Merchant view is useful for the assessment of patellar tilt, subluxation, and shape. The normal sulcus angle measures 138° ± 6°; the angle is measured from the highest point on the condyles to the lowest point in the intercondylar sulcus.5 A sulcus angle measuring >145° is indicative of trochlear dysplasia.5,4 However, the sulcus angle can be normal when measured at 45° of knee flexion, and it can have a flattened appearance proximally at the entrance to the trochlear groove. Trochlear dysplasia is more common proximally, and ≤35% of dysplastic trochleae are missed on a 30° axial view.29

Given the limitations of the axial view, a true lateral radiographic view (ie, medial and lateral posterior condyles overlapping) may be used to assess the morphology of the trochlear groove. The trochlear floor is visualized radiographically as a radiodense line (Figure 3). In the healthy knee, this line should not pass anterior to a tangential line contiguous with the anterior femoral cortex. Dejour et al25 reported that the crossing sign and a trochlear prominence were consistently demonstrated on lateral radiographs in patients with patellar instability.4 The crossing sign, a primary radiographic indication of trochlear dysplasia, is visualized when the line representing the trochlear floor crosses the anterior border of the femoral condyles (Figure 3, C). The crossing sign is pathognomonic for trochlear dysplasia. Specifically, the crossing sign indicates a deficient proximal trochlea with decreased relative height of the lateral trochlear wall. The more distal the location of
the crossing sign, the more severe the dysplasia. The crossing sign is highly sensitive in determining the presence of trochlear dysplasia. However, rotational deviation of as little as 5° while obtaining a lateral radiograph can cause a false-positive or a false-negative result. A perfect lateral view of the knee (ie, symmetric overlap of medial and lateral posterior condyles) at 30° of knee flexion is essential to allow calculation of patellar height, as well.

Figure 3

A, Axial radiographic view of the knee. The sclerotic line (+) correlates with the floor of the healthy trochlea. The arrows indicate the condyles. B, The lateral view shows that the trochlear floor (+) does not cross the condyles (arrows). C, The crossing sign (arrowhead) is visible on the lateral view as the trochlear floor crosses the anterior contour of the lateral femoral condyle. D, The trochlear prominence or spur (arrows) is measured from the trochlear floor (dashed line) and a line extending from the anterior cortex of the distal femur (solid line). (Reproduced with permission from Fithian DC, Nyret P, Elvire S: Patellar instability: The Lyon experience. Current Orthopaedic Practice 2008;19:328-338.)

The trochlear prominence or spur is an extension of the trochlear groove above the projection of the anterior cortex of the femur (Figure 3, D). The trochlear prominence is the distance between the anterior femoral cortex and the most anterior point of the trochlear floor. As the patella enters the trochlear groove, it must pass over this prominence. Elevation of the trochlear floor, as represented by the trochlear prominence, leads to diminished relative height of the lateral wall and resistance to lateral patellar dislocation. Prominence >4 mm is associated with trochlear dysplasia and patellar dislocation; the degree of dysplasia increases with increased prominence.

Three-dimensional visualization of the patellofemoral joint is useful in the assessment of patellar instability and trochlear dysplasia as well as in surgical planning. CT delineates trochlear morphology up to 30° of knee flexion, when dysplasia is most likely to occur, and it enables more accurate measurement of patellar tilt, using the posterior femoral condyles as reference points. MRI is another valuable tool for the assessment of patellofemoral instability. In patients with acute instability, MRI delineates disruption of the proximal medial soft-tissue restraints (ie, MPFL) and classic medial patellar facet. Lateral femoral condyle bone bruise patterns also can be seen on MRI. TT-TG distance can be assessed on CT or MRI with the use of overlapping axial images to measure the distance between the center of the trochlear groove and the center of the tibial tubercle. TT-TG distance >20 mm is indicative of patellofemoral malalignment.

Surgical Management

Osseous, static, and dynamic structures work together to maintain patellar stability during knee ROM. When managing patellar instability, the selected surgical approach must address each of these pathoanatomic factors. Trochlear dysplasia is a common finding in persons with patellofemoral instability, and it is an important risk factor in recurrent dislocation and subluxation. Other risk factors include a TT-TG distance >20 mm, patella alta, and deficient medial soft-tissue restraints. Each component that may contribute to instability should be consid-
ered preoperatively. For example, a TT-TG distance >20 mm is indicative of external tibial torsion and/or excessive femoral anteversion. In such cases, malalignment should be addressed with tibial tubercle transfer or femoral derotation osteotomy to restore proper alignment between the trochlea and the tubercle. Correction of malalignment can compensate for a deficient trochlea and can provide patellofemoral stability.

One of two approaches may be used in the skeletally mature patient with substantial trochlear dysplasia, with recurrent patellofemoral instability, and with normal TT-TG distance but without patella alta. The first approach involves bony procedures that include direct reconstruction of the dysplastic trochlea. These include the Albee trochleotomy, deepening trochleoplasty, and rotational trochleoplasty.5,32-39 Bony procedures may be performed alone or in combination with other procedures. The second approach addresses associated factors that compensate for the dysplastic trochlea, such as TT-TG distance, patella alta, and deficient medial soft-tissue restraints. Compensatory surgical procedures that address these factors can provide patellofemoral stability without the need for direct reconstruction of the dysplastic trochlea.

In the Albee opening wedge osteotomy, the lateral trochlear facet is elevated, and a bone graft is placed to increase the height of the lateral wall. This procedure was described in the early 1900s and is still used to manage significant trochlear dysplasia.19 However, the Albee osteotomy creates an incongruent patellofemoral joint, which can lead to overloading on the lateral side. Kuroda et al40 reported that raising the lateral condyle as little as 3 mm can lead to increased patellofemoral contact pressures.

The term trochleoplasty is used to describe any procedure that reshapes the trochlea to improve patellofemoral stability. Indications for trochleoplasty include recurrent patellofemoral instability with radiographic high-grade trochlear dysplasia.35,36 Patellofemoral degenerative joint disease and arthritis are contraindications to trochleoplasty. Many different trochleoplasty procedures exist, and no consensus exists regarding an ideal technique. Most reports involve deepening the groove by removing subchondral bone and compressing the overlying cartilage into the defect as described by Dejour and Saggin16 (Figure 4). Open and arthroscopic techniques have been described. Goutallier et al41 described resection trochleoplasty, in which the trochlear groove is flattened to the level of the anterior femoral cortex without deepening.

Recurrent dislocation is rare following trochleoplasty. However, persistent pain is common, and articular cartilage damage is a concern. Zaki and Rae48 reported on 27 trochleoplasties to address trochlear dysplasia in 25 patients with patellar instability. No recurrence was reported as of a mean 54-month follow-up. Average Lysholm scores improved from a mean of 54 preoperatively to >83 postoperatively in 19 patients. However, 33% of knees had residual symptoms, including pain, swelling, and crepitus.

Utting et al17 reported on 54 patients who were treated with Bereiter
Trochlear dysplasia has been shown to prevent recurrent patellofemoral instability, improve radiographic signs of trochlear dysplasia, maintain cartilage viability, and result in increased biomechanical stability. However, many patients have continued pain and symptoms, some have evidence of patellofemoral joint incongruity, and all patients have damage to the articular cartilage and underlying subchondral bone that may predispose them to late arthritis. Trochleoplasty should be reserved for cases of extreme dysplasia in which other options cannot provide patellofemoral stability and in salvage situations in which no significant degenerative changes are present.

Extensor mechanism alignment and soft-tissue constraints play a greater role in maintaining a centered patella in the setting of trochlear dysplasia. Trochlear dysplasia is associated with recurrent dislocation. However, other risk factors (eg, hyperlaxity, deficient medial soft-tissue constraints, increased TT-TG distance, patella alta) are generally present as well. Even in the presence of significant trochlear dysplasia, procedures to address associated factors are usually recommended as first-line treatment instead of trochleoplasty. Correction of these other factors can compensate for a deficient trochlea and can provide stability.

In patients with a normal TT-TG distance, MPFL reconstruction can provide patellofemoral stability in the presence of trochlear dysplasia (Figure 5). Steiner et al examined 34 patients who underwent MPFL reconstruction to manage chronic patellar instability and trochlear dysplasia. Significant improvement in Tegner and Lysholm scores was shown at a mean follow-up of 66.5 months (3.1 to 5.1, P < 0.001, and 52.4 to 92.1, P < 0.001, respectively). No patient experienced recurrent dislocation. The authors concluded that MPFL reconstruction provided excellent long-term pain relief and function and prevented recurrent dislocation even when the deficient bony constraints were not addressed.

Open or arthroscopic medial imbrication does not provide adequate stability in the presence of underlying trochlear dysplasia in patients with normal patellofemoral alignment. Schottle et al performed arthroscopically assisted medial retinacular reefing on 91 patients with and without underlying trochlear dysplasia. Patients with trochlear dysplasia had a higher incidence of postoperative patellofemoral dislocation. When a proximal soft-tissue procedure is indicated, it is essential to assess tissue quality, degree of hyperlaxity, and underlying bony deficiencies. In the presence of trochlear dysplasia, the medial soft-tissue constraints play a greater role in preventing lateral patellar translation. MPFL reconstruction with a tendon graft is recommended rather than imbrication or reefing in patients with significant trochlear dysplasia or generalized hyperlaxity and a normal TT-TG distance.

Summary

Trochlear dysplasia is defined as a shallow, flattened groove with inadequate bony resistance to lateral patellar translation. Osseous, static, and dynamic structures interact to provide patellofemoral joint stability. Osseous constraint is particularly important after the first 20° of knee flexion, when the soft tissues play a lesser role in maintaining a centered patella. Trochlear dysplasia has been clearly linked to recurrent patellar dislocation. However, it is unclear whether this dysplasia is congenital, develops as a result of lateral tracking and chronic instability, or is
caused by a combination of factors. Procedures that address other factors associated with recurrent instability (eg, medial soft-tissue deficiency, patella alta, increased TT-TG distance) can compensate for a deficient trochlea and provide stability. Trochleoplasty involves reshaping a dysplastic trochlea and should be reserved for cases of extreme dysplasia in which other options are insufficient to provide patellofemoral stability.

References

Evidence-based Medicine: Levels of evidence are described in the table of contents. In this article, no level I, II, or III references are cited. References 2-4, 7, 9, 10, 12, 14, 21-23, 26-30, 34, 35, 37, 40, 41, and 45 are level IV studies. References 1, 5, 6, 8, 11, 13, 15-20, 25, 31-33, 36, 38, 39, and 42-44 are level IV expert opinion.

Citation numbers printed in bold type indicate references published within the past 5 years.


