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Review Article

Overview of Congenital Patellar Dislocation in Children

Nguyen Ngoc Hung*

Surgical Department, Vietnam National Hospital for Pediatric, Ha Noi Medical University, Vietnam

***Corresponding Author**: Nguyen Ngoc Hung, Surgical Department, Vietnam National Hospital for Pediatric, Ha Noi Medical University, Vietnam.

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Abstract

Congenital patellar dislocation can occur as a persistent lateral patellar dislocation manifested by knee flexion contractures and the patella being forced externally onto the femoral condyle or as an intermittent patellar dislocation. In the latter syndrome, the patella dislocates completely with each cycle of knee flexion and extension and is best referred to as an obligatory patellar dislocation, as the patient has no control over the patella dislocating as child moves the knee. The first type of congenital patellar dislocation, which is fixed, is often associated with syndromes such as arthrogryposis and should be corrected surgically by lateral release and realignment of the patella. Obligatory patellar dislocation tends to be an isolated dysplastic deformity and can be tolerated relatively well. Patellar realignment is usually performed at an older age because it has less impact on function. The present author describes the natural history of patellar dysplasia, details the pathologic changes present, and recommends surgical techniques for the correction of both types of congenital patellar dislocation.

Keywords: *Congenital Patellar Dislocation; Children; Knee Flexion*

Introduction

Patellar dislocation is common in children and many surgical procedures have been described for its treatment [1-3]. Surgical treatments include realignment of the extensor mechanism both proximally and distally, and most are associated with significant surgical trauma, large scars, and prolonged rehabilitation. However, no single procedure is appropriate for all cases because of patient variability. Multiple factors contribute to patellar dislocation, including: (1) congenital mechanisms (i.e. generalized ligament laxity, patellar or femoral condyle dysplasia, genu valgum, tight lateral ligaments, and high patella [1-3]), and (2) secondary mechanisms, due to iatrogenic quadriceps fibrosis following intramuscular antibiotic injections, altered quadriceps traction and vastus lateralis fibrosis, vastus intermedius, and lateral patellar retinaculum; and degeneration and contracture of the rectus femoris and vastus medialis muscles.

Predisposing factors include ligament laxity, lateral patellar soft tissue contracture, high patella, and lateral femoral condyle dysplasia. More than 100 different surgical techniques for the treatment of patellar instability have been described in the past 100 years [4].

Several surgical procedures have been described for the treatment of this condition [3-7]. These surgeries involved realignment of the extensor mechanism both proximally and distally, and most were associated with significant surgical trauma, extensive scarring, and prolonged rehabilitation. In 1961, Hněvkovský [8] first aroused interest with his report of progressive fibrosis of the vastus intermedius muscle in young children. Gunn (1964) [9] later demonstrated a causal relationship between quadriceps contracture and intramuscular injections, and Garcia Novales (1959) [10] suggested a relationship with common patellar dislocation. Although all the conditions that we now associate with this spasticity are well known, the importance of the underlying muscle condition in each case has not been emphasized.

There are many proposed mechanisms for this disorder such as patellar mechanism, distal femoral dysplasia, imbalance between the lateral and medial patellar luxation (such as quadriceps femoris fibrosis due to intramuscular antibiotic injection). A large number of surgical procedures have been described to realign the patellar mechanism. Proximal realignment procedures alter the medial and lateral position of the patella by balancing the soft tissues near its inferior pole. Release of the lateral patella, folding of the medial patella or capsule, oblique vastus medialis muscle advancement, and repair or reconstruction of the medial patellar ligament are included in this category. However, no single procedure is suitable for all cases due to inter-patient variability [11,12]. Soft tissue surgeries are recommended during development to avoid secondary deformities, such as valgus that can occur when the tibial tuberosity1 is dislocated in children [7]. Lateral patellar dislocation or subluxation in children is described as recurrent when the dislocation is episodic, habitual when the dislocation occurs with each knee flexion, and permanent when the dislocation persists in all knee positions. Monotraumatic non-recurrent dislocations are excluded by these definitions.

In recurrent dislocations, medial patellar stability is impaired due to vastus medialis weakness, dysplasia, generalized joint laxity, or post-traumatic medial capsular laxity.

Permanent dislocations or subluxations may be congenital and due to muscular dysplasia [13] or acquired due to progressive contracture of the extra-lateral muscle. In both types, contracture is the primary pathology, whether idiopathic or due to injection-induced fibrosis [9,14]; medial laxity or weakness of the medial patellar stabilizing muscles is secondary.

Some bony deformities may be associated with patellar dislocation, but may not be the true cause, since in such cases, patellar osteotomy without quadriceps reconstruction may result in recurrent patellar tendon dislocation [6]. This is not seen after patellar osteotomy for patellar fractures. Revision osteotomy, for genu valgum associated with lateral patellar dislocation, often fails to control the dislocation [15]; and many patients with severe genu valgum do not dislocate the patella. Bony factors probably play only a minor role in dynamic patellar stability.

A number of reconstructive procedures for recurrent patellar dislocation have been described. Most of them focused on medial stabilization of the patella or its tendon: the tibial tuberosity displacement of Roux (1888) [16] was popularized by Hauser (1938) [5]; the medial implantation of the lateral half of the patellar tendon was described by Goldthwait (1904) [4]; and the semitendinectomy of Galeazzi (1922) [17] was popularized by Baker., *et al*. (1972) [2]. Medial capsulotomy and creation of a suprapatellar check ligament were recommended by Campbell (1930) [18]; patellar osteotomy and quadriceps realignment were recommended by West and Soto-Hall (1958) [19]; reconstruction of the extensor mechanism by Hughston (1972); and vastus medialis muscle implantation distally and laterally anterior to the patella by Madigan, Wissinger, and Donaldson (1975) [3]. There is also the Elmslie-Trillat procedure for lateral retinaculum release, medial retinaculum flexion, and medial tibial tuberosity transfer reported by Cox (1976) [20], and endoscopic lateral release with or without medial retinaculum flexion [21,22]. In 1981, I reported on the transposition of the pes annulus to restore dynamic stability in all types of patellar dislocation [23]. The need to release any external contractures in patients with frequent or permanent dislocations has been emphasized, but this is not considered necessary for recurrent dislocations without contractures. This article will report on long-term follow-up of the above procedure over a period of 3 to 18 years.

Anatomy of patella

Structure and function

the patella lies deep within the vastus lateralis fascia and rectus femoris tendon, anterior to the knee joint. The upper third of the patella serves as the attachment point for the vastus lateralis and vastus medialis muscles. The vastus medialis and lateral muscles attach to the medial and lateral borders of the patella, respectively. The quadriceps tendons unite at their distal attachment points, superficially crossing the anterior surface of the patella to form the vastus lateralis fascia, which attaches to the lateral epicondyle of the tibia. The patellar ligament encases the lower third of the patella and attaches the bone to the tibial tuberosity. The patella enhances the effect of the quadriceps, acting as a fulcrum that increases the moment arm of the extended knee. In physics, moment refers to the rotational effect of a force on an object about a particular point or axis. Moment is the product of a force and the moment arm of that force—the perpendicular distance from the line of action of the force to the axis of rotation. The force required to extend the knee, or torque, is proportional to the moment arm of the knee joint, which is the perpendicular distance between the patellofemoral ligament and the knee [24].

As the knee extends from a fully flexed position, the patella initially connects the quadriceps tendon and the patellofemoral ligament, allowing the quadriceps to generate torque on the tibia. However, twice as much torque is required for the final 15° before full extension as it does initially. The patella helps achieve this by increasing the moment arm during extension. Displacing the quadriceps tendon-patellofemoral ligament connection away from the axis of rotation increases the effective moment arm of the knee, thus generating 60% more torque for the final 15° of knee extension.

Static and dynamic patellar alignment explain the causes of patellofemoral pain.

Static patellar alignment depends on the depth of the femoral groove, the height of the lateral femoral condyle, and the shape of the patella. Rough patellar alignment is usually assessed in the supine position, with the knee fully extended. In this position, the femur and patella have minimal contact, and the patella is most mobile. When the knee is fully extended, the patella will be above the trochanter and between the two condyles, sometimes slightly to one side.

Slightly flexing the knee 30° from full extension will place the patella at or near the joint line. In this position, the ratio of patellar ligament length to patellar height should be approximately 1.0. A ratio significantly lower or greater than 1.0 may indicate a baja patella or a high patella. People with high patellas are at increased risk of patellar dislocation.

Each patellar rim should be equidistant from the femur. Anterior or posterior tilt describes the position of the inferior pole of the patella in the vertical plane. Inferior tilt occurs when the inferior pole is depressed, while superior tilt occurs when the inferior pole is elevated. Inferior tilt can compress or irritate the patellar fat pad deep within the patellofemoral ligament and cause pain.

Lateral tilt occurs when the lateral border of the patella is depressed in the horizontal plane. Medial tilt refers to the position where the medial border of the patella is depressed in the horizontal plane. Lateral tilt can lead to patellar impingement syndrome.

Patellar rotation is described by the direction of rotation of the inferior pole of the patella. External or internal rotation of the patella can suggest an underlying tibial torsion [25].

The following factors influence dynamic motion:

- Active quadriceps contraction.
- Extensibility of the connective tissue surrounding the patella.
- Shape of the patella and trochlear groove.

In tibiofemoral motion, the patella acts as a gliding joint and can move in multiple planes. Upward gliding occurs during knee extension when the quadriceps contract and pull the patella upward. Conversely, downward gliding occurs during knee flexion.

Lateral and medial gliding refer to the lateral and medial tracking of the patella. During normal patellar tracking, there is some medial or lateral gliding. However, the patella deviates slightly to one side when the knee is fully extended due to external tibial rotation.

The articular surface of the patella changes as the knee completes its range of motion. As the knee flexes, the patellar contact point moves inferiorly and posteriorly along the femoral condyle and closer to the patella. Initially, during flexion, the lateral surface of the patella is the first surface to contact the superior aspect of the lateral femoral condyle. However, at 30° of flexion, the contact surface is evenly distributed on both sides of the patella and femoral condyle. The contact surface of the patella also expands during knee flexion, increasing from approximately 2.0 cm at 30° to approximately 6.0 cm at 90°. This distributes joint forces over a larger surface area and helps prevent the potentially deleterious effects of repeated exposure to high compressive loads. At 90° of knee flexion, the superior surface of the patella abuts a region of the femoral groove above the femoral notch. During deep flexion, the patella crosses the intercondylar notch, with contact occurring only at its medial and lateral edges. When the knee is in full flexion, the only point of contact is between the odd side of the patella and the lateral surface of the medial femoral condyle [25].

Embryology

The patella develops from a continuous fibrous connective tissue band in the mesenchymal interosseous zone along the knee joint surface at the distal femoral margin. Around the ninth week of gestation, chondrogenesis of this fibrous band begins, giving rise to the quadriceps tendon above and the patellar ligament below. The patella becomes fully cartilaginous by the 14th week. The medial and lateral patellar facets are initially of equal size, but the lateral facet becomes larger than the medial facet by the 23rd week of gestation. Primary ossification of the patella usually does not occur until age 5 or 6, but radiographic evidence of ossification may be present by age 2 or 3. Multiple small foci of ossification appear initially, which later fuse and spread toward the margin of what will eventually become the mature bone. Periosteum forms early on the anterior patellar surface. However, other patellar margins retain the cartilage-bone interfaces that exist throughout adolescence, leaving them susceptible to fracture until skeletal maturity [24].

Pathologic anatomy

All structures derived from the embryonic mesoderm are abnormal in patellofemoral dysplasia, including bones, muscles, and ligaments. Pathologic changes in each of these tissue components must be evaluated to restore normal alignment and function of the extensor mechanism. The term dysplasia may be applied to an individual cell or to an organ. Patellofemoral dysplasia refers to abnormal development and growth of the anterior portion of the knee and associated structures [26]. It is characterized by abnormal lateral deviation of the patella and the associated quadriceps mechanism. Cases of medial dislocation have been reported to occur after overdissection of the quadriceps mechanism. The most consistent pathologic finding in patellofemoral dysplasia is lateral soft tissue contracture, which results in lateral deviation and malalignment of the quadriceps mechanism, patella, and quadriceps tendon. This contracture is most severe in persistent lateral patellar luxation, where a thick fibrous band tethers the patella to the lateral intermuscular septum.

In obligatory patellar luxation, lateral fibrous tethering of the joint capsule and retinaculum occurs, but to a lesser extent. In the more common recurrent patellar luxation or subluxation, contracture limits the patella's ability to move medially away from the lateral femoral condyle, but the patella remains more or less in the trochlear groove, although it may be tilted or displaced laterally from its true central position.

The degree of abnormality of the medial capsule and retinaculum is directly correlated with the degree of lateral abnormality. In congenital patellar luxation, the medial retinaculum is thin and extends anteriorly over the joint like a hood over the distal femur. In most of these cases, the joint capsule and medial periosteum are firmly attached to the articular surface of the distal femur and must be dissected to medially reposition the patella. In milder dysplasias, the medial muscles are underdeveloped or weak, requiring some type of graft or augmentation to support the patella in the middle of the trochlear groove.

In severe dysplasias such as congenital patellar luxation, the pathological features of the joint capsule are similar to those of ankylosis in that the capsule is contracted and tightened, thus reducing the volume of the joint.

The quadriceps femoris is often shortened. In severe dysplasias, the quadriceps femoris mechanism is not long enough to center the patella in its normal position. With severe lateralization, as in congenital patellar luxation, the quadriceps femoris acts as a flexor and external rotator of the tibia, contributing to the bowing and valgus deformity commonly seen in older individuals when the malalignment is not corrected.

In terms of skeletal malformations, the patella is small and underdeveloped. The distal end of the femur may be completely absent from the trochlear groove in congenital patellar luxation. When the distal femur is exposed, it appears smooth and rounded. In milder dysplasias, the trochlear groove is underdeveloped and is characterized by a low lateral femoral condyle. The lateral femoral condyle is usually the highest part of the distal femur seen in cross-section and resists lateral displacement of the patella. When the lateral femoral condyle is underdeveloped, the bone is less able to resist lateral displacement of the patella. However, the development of the trochlear groove appears to be in response to normal quadriceps muscle action. As the patella realigns in infancy, the development of the trochlear groove occurs during further growth and development. This reciprocal relationship between the patella and the trochlear groove appears to be similar to the relationship between the femoral head and the acetabulum in that the hip develops normally when the femoral head is in the middle of the acetabulum.

The superior tibia is abnormal in patellofemoral dysplasia in that the tibial tuberosity tends to be displaced laterally. This displacement may be associated with external tibial torsion, which may be a clinical clue to the diagnosis. Lateral displacement of the tibial tuberosity may be a distinct feature from normal tibial torsion of the leg.

Anatomists consider the patellar ligament to be an extension of the quadriceps tendon that continues distal to the patella to insert on the tibial tuberosity [27,28]. The lateral position of the tibial tuberosity is associated with the abnormal lateral insertion of the patellofemoral ligament. This displacement creates a lateral vector that should be considered and is often surgically corrected in cases of patellofemoral dysplasia.

The quadriceps femoris is weak and often shortened. The hamstrings may be shortened and the posterior capsule of the knee may be contracted. Flexion forces also arise from the displaced lateral quadriceps muscle acting posterior to the center of rotation of the joint. Recognizing that this pathologic feature contributes to knee flexion contracture is an important part of correcting the deformity in congenital patellar dislocation and allowing full extension and normal walking.

In cases of skeletal dysplasia such as spondyloepiphyseal dysplasia or Ellis-van Creveld syndrome, the degree of genu valgum that develops due to abnormal bone growth can create a lateral vector so strong that patellar dislocation occurs either permanently or intermittently. The main component of correction in these cases is osteotomy, which realigns the quadriceps femoris vectors and is the most important factor in establishing normal patellofemoral joint function.

Aetiology

Epidemiology

Congenital patellar luxation is rare and its incidence is unknown. Although some studies have grouped it into a spectrum of conditions that includes irreversible dislocations acquired before age 10 [29] and patellar instability, most agree that it is a distinct entity [13,30-33]. Congenital patellar luxation can be associated with a variety of conditions, including diastrophic dysplasia, arthrogryposis, Down syndrome, Rubinstein-Taybi syndrome, nail-patella syndrome, Larsen syndrome, and Ellis-van Creveld syndrome. Acute and chronic trauma, chronic abnormal loading of the joint, and hemarthrosis have been implicated in the development of osteoarthritis. Patellar instability with acute and recurrent patellar dislocations provides all of these components. It is therefore not surprising that patellofemoral arthritis is ultimately a common sequela of patellar dislocation. Furthermore, although our surgical procedures are often most successful in preventing recurrent dislocations, they have not been shown to reduce the incidence of patellofemoral osteoarthritis. Clark [34] has summarized the possible causes of patellofemoral arthritis and has provided some evidence to support the hypotheses that excessive joint loading and increased duration of joint loading are possible contributing factors. Patients with recurrent patellar dislocations often have anatomical variations around the knee that predispose to patellar instability. These anatomic pathologies may involve variations in lower limb alignment, patellofemoral alignment, or trochlear configuration.

The two most common anatomic pathologies are trochlear dysplasia and high patellae, which are associated with the development of patellofemoral arthritis, with or without patellar dislocation. Further damage to the articular cartilage, resulting from the acute trauma of patellar dislocation, sometimes leads to osteochondral fractures, hemarthrosis, and chronic patellar dislocation; this is followed by patellofemoral osteoarthritis.

Epidemiology and natural history

In a prospective population study of the Kaiser Health Plan, individuals in their second decade of life had the highest incidence of acute patellar dislocation, with 69% of all dislocations occurring in a year affecting these individuals. The overall risk for members of this health plan at all ages was 7 per 100,000 person-years, but the risk for those aged 10 to 19 years was 31 per 100,000 person-years, with a nearly equal distribution between girls and boys according to the American Academy of Orthopaedic Surgeons (33 per 100,000 person-years versus 30 per 100,000 person-years, respectively).

Female sex, a family history of patellar instability, and a history of patellar dislocation or subluxation were associated with a higher risk of subsequent dislocation. Furthermore, the degree of trauma associated with the first dislocation is an important predictor of subsequent dislocations [35]. Fithian., *et al*. noted that in patients with medial retinaculum and medial patellofemoral ligament (MPFL) lesions documented on MRI, the rate of subsequent patellar dislocations was lower than in patients without retinaculum lesions. This finding is understandable if one considers that patellar dislocations in the absence of MPFL injury may indicate concomitant patellofemoral dysplasia. Crosby and Insall [36] reported that episodes of patellar subluxation/dislocations decreased over time and that the rate of patellofemoral osteoarthritis was low and unrelated to the frequency of dislocations.

Biomechanics of patellar instability

Patellar motion is influenced by a complex interaction of muscles, ligaments, bone morphology, and lower limb alignment. The patellar tendinous ligaments are important stabilizers of the patella, and the MPFL in particular is the primary soft tissue constraint to lateral patellar translation during the initial 20° to 30° of knee flexion. This ligament is most stretched in full extension, with the quadriceps femoris contracting and assisting in guiding the patella to the pulley during the early phase of flexion. Amis., *et al*. [37] and Senavongse., *et al*. [38] demonstrated that the least resistance to lateral patellar translation occurs at 20° of flexion, with resistance increasing with further extension and flexion. Once engaged in the pulley, the patellofemoral joint compressive forces generated by the ascending force vectors of the quadriceps and patellar tendon, combined with the patellofemoral joint geometry, exert a major influence on stability as knee flexion progresses. As the patella moves within the pulley, the slope of the pulley lateral aspect provides the major resistance to lateral patellar translation. Studies have been conducted on the influence of the musculature and the vastus medialis obliquus (VMO) muscle in particular on knee stability [39]. The evidence supporting the VMO as a primary determinant of patellofemoral stability remains controversial, but like the patellofemoral retinaculum, the VMO has the greatest influence on patellar alignment during the early stages of knee flexion. Few studies have examined the influence of lower limb alignment on patellofemoral instability. However, Fithian., *et al*. [35] demonstrated that the lower limb and patellofemoral alignment cannot by themselves cause a patellar dislocation without concomitant soft tissue restraint deficits due to increased elasticity or trauma.

Many factors contribute to patellar dislocation, including generalized ligamentous laxity, patellar or femoral condyle dysplasia, genu valgum, tight lateral bands, and a high patella. Several surgical procedures have been described to treat this condition [1-4,6]. These surgeries involve realignment of both the proximal and distal extensor mechanisms, and most are associated with significant surgical trauma, extensive scarring, and prolonged rehabilitation.

The degree of disability resulting from recurrent patellar dislocation is variable, and postoperative morbidity may be higher. Complications of surgery include osteoarthritis of the patellofemoral joint [39], loss of flexion, pain with screw or staple fixation, patellar sprain [40], and knee flexion after distal realignment before full development [41,42].

Release of the lateral extensor mechanism, whether by open or arthroscopic surgery, results in faster rehabilitation, less surgical trauma, and fewer complications. However, not all outcomes are favorable; therefore, we attempted to identify clinical and imaging features associated with poor outcomes. All reported surgeries were performed for patellar stabilization, not for relief of nonspecific anterior knee pain.

Abnormal attachment of the ilio-tibial tract

Abnormal attachments of the iliotibial tract are a recognized cause of recurrent patellar dislocation [43,44]. However, this condition is uncommon. Apart from being mentioned in passing in standard works as a possible cause of recurrent dislocation, it has received little attention in the literature.

The iliotibial tract is usually inserted into one side of the lateral epicondyle of the tibia. Through this insertion, the gluteus maximus and the vastus lateralis fascia stabilize the slightly flexed knee during weight bearing. But the iliotibial tract is simply a thick band of the femoral fascia. This fascia forms partitions between the muscles of the quadriceps group and on both sides of the patella is reinforced by fibers associated with the fascia of the vastus lateralis and vastus medialis. These fibers are stronger on the lateral side, where they arise from the iliotibial tract. The special thickening of the fascia, which attaches to sites other than the classical aspect of the tibia, but occurs in tissues with a continuous structure, cannot be considered a true congenital anomaly. Normal histology and no history of trauma exclude any acquired cause of this anomaly. The term “congenital attachment defect of the iliotibial tract” by Ober (1939) [44] is descriptive and not specific. The iliotibial tract lies anterior to the axis of rotation of the knee joint and passes posterior to that axis when the knee is flexed. Therefore, if the patella is attached to the tract in any way, it must be pulled laterally during knee flexion. If the patella in such a case is held in a central position in the intercondylar groove, the knee can only flex at an angle where the iliotibial tract passes posterior to the axis of rotation.

Clinical

The main physical finding is lateral patellar dislocation with each knee flexion. With the patella anchored in the femoral groove, we found knee flexion limited to between 10 and 20 degrees and 30°. With the patella dislocated, full flexion was possible. Palpable bands attached to the lateral border of the patella were noted in all knees.

Clinical and imaging examinations

A complete history of each patient’s dislocation was obtained prior to surgery. The affected knee was evaluated preoperatively and postoperatively based on symptoms, signs, and imaging findings (X-rays/MRI, CT, and ultrasound). Patellar dislocation was determined based on clinical examination and imaging findings as described below.

Clinical examination

Clinical examination was performed in the standing, sitting, and supine positions. Patellar compression testing was performed with knee flexion and extension to assess for joint crepitus or pain.

The thigh circumference of the injured leg was measured and compared with the contralateral thigh, with the distance from the distal third of the thigh to the superior border of the patella being approximately 10 cm. For dislocations, we expected to find the following clinical signs: (a) knee deformity; (b) thigh atrophy compared with the contralateral healthy thigh; (c) protrusion of the contralateral knee when the knee was flexed; and (d) the patient was unable to run. We also tested for the following factors.

Figure 1: *Patient had left habitual dislocation of the patella.*

Figure 2: *Knees with tight lateral band.*

The Q-angle

Q angle: This is the angle between the line connecting the anterior superior iliac spine and the center of the patella and the line from the center of the patella to the tibial tuberosity. A Q angle greater than 16 degrees is abnormal and leads to patellar instability (Figure 3) [45].

Patella glide test

By determining the medial and lateral patella mobility, tightness or insufficiency of MPFL and lateral retinaculum can be estimated.

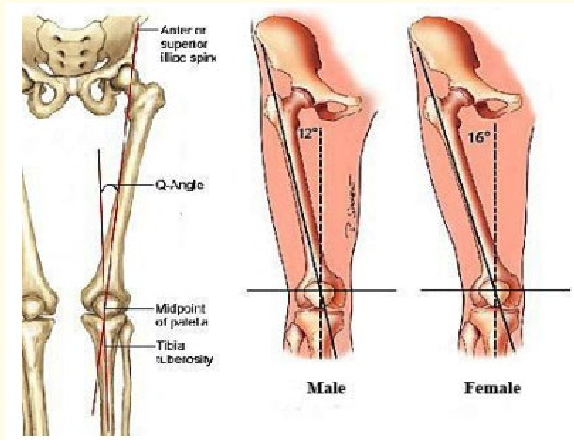

Figure 3: *Q angle.*

The knee is in 0-20 degrees of flexion and the examiner pulls the patella medially and laterally and measures patellar mobility. Normally the patella slides laterally, but not more than two-quarters of the patella width. If lateral mobility is increased, MPFL injury is suspected. Excessive lateral patellar flexion is indicated by limited patellar medial glide (Figure 4).

Figure 4: *Patella glide test.*

Check the "J sign"

This test is useful for assessing dynamic patellar tracking. The knee is first in flexion and then in full extension. When the knee is near full extension, the patella is significantly dislocated laterally and its movement shows a reverse J sign. Normally, the patella moves more superiorly than laterally (Figure 5).

Figure 5: *"J sign" test. A positive j sign indicates abnormal lateral patella tracking.*

Genu valgum is determined with a goniometer while the patient is standing. Range of motion is estimated using the contralateral (healthy) knee as a control, unless both knees are affected, in which case the range of 0 to 150 degrees is used to estimate normal range of motion (Figure 6).

Figure 6: *The genu valgum.*

Ligament laxity

Ligamentous laxity is determined by examining the patient's other joints and demonstrating any excessive range of motion, without associated discomfort.

Patellar instability

Patellar instability refers to a spectrum of clinical manifestations, ranging from abnormal medial or lateral displacement to dislocation or subluxation of the patella. Patellar instability is often multifactorial but is primarily due to anatomical and mechanical imbalances in the patellofemoral joint. These imbalances result in chronic instability and secondary flattening of the lateral aspect of the femoral trochanter. The patella then slides laterally during flexion and either completely dislocates or snaps medially into place with progressive flexion.

After recovery from the acute injury, patellar instability can be treated non-surgically with immobilization and weight bearing. Physical therapy is usually sufficient to correct the mechanical imbalance. However, tissue damage often accompanies dislocations. Recurrent dislocations are common because the patella may become less stable after healing. Repeated patellar dislocations require surgery to correct the underlying problem, usually with arthroscopic reconstruction of the patellar ligaments [24].

Chondromalacia patellae

Sex differences exist in patellar depth, lateral patellar contact angle, and lateral patellar tilt angle [46]. These differences may explain why chondromalacia patellae-or patellar chondromalacia-is much more common in women than in men [46].

Trochlear dysplasia

Trochlear dysplasia, a common cause of recurrent patellar instability, refers to anatomical defects in the femoral trochanter that interfere with the normal movement of the patella. Such defects include:

- Decreased height of the medial femoral condyle
- Decreased trochanteric depth
- Increased groove angle
- Decreased depth of the lateral trochanteric surface, resulting in a flat or convex surface.

Tubercular dysplasia can be identified on radiographs by the "crossover sign," defined as the convergence of the deepest part of the femoral groove with the most prominent aspect of the lateral trochanteric surface. The crossover sign is seen in 96% of individuals with objective patellar dislocation and 85% of individuals with recurrent patellar instability. Trochanteric dysplasia is treated conservatively as is patellar instability, and surgical intervention is reserved for those with recurrent dislocations. Surgical options include medial patellar ligament reconstruction, tibial tubercle osteotomy, and tracheoplasty [24].

Patella alta

High patella is characterized by the patella protruding above the trochanteric groove of the femur. As a result, the patella does not articulate with the trochanteric groove until later in flexion. High patella increases the risk of patellar dislocation and is associated with other patellofemoral abnormalities, including:

- Chondromalacia patellae
- Dysplastic condyle
- Dysplastic trochanter
- Micropatella
- Excessive patellar tilt
- Joint effusion
- Ligamentous laxity.

The Insall-Salvati index is the ratio of the length of the patellar ligament to the greatest diagonal length of the patella on a flexed knee X-ray. This index is commonly used to diagnose high patella. An Insall-Salvati value greater than 1.2 may be diagnostic of high patella.

Conservative treatment of the patella may include manual sliding to adjust the resting height of the patella before knee extension. Banding to correct patellar misalignment is an alternative.

Surgically, patellar luxation can be treated with a tibial tuberosity osteotomy. The patellar ligament attachment is moved inferiorly to the tibia [24]. Surgery improves patellar instability in patients with patellar luxation, resulting in better functional outcomes.

Patella alta (Roentgenogram/X-ray)

The lateral view is important to determine the patellar height. For that purpose, a lateral view with 30 degrees of knee flexion is performed and the Insall-Salvati index [47] (the ratio between the length of the patellar tendon and the diagonal length of the patella) is determined (Figure 7). If the Insall-Salvati index is greater than 1.2, the patient has a high patella and if it is less than 0.8, the patient has a low patella.

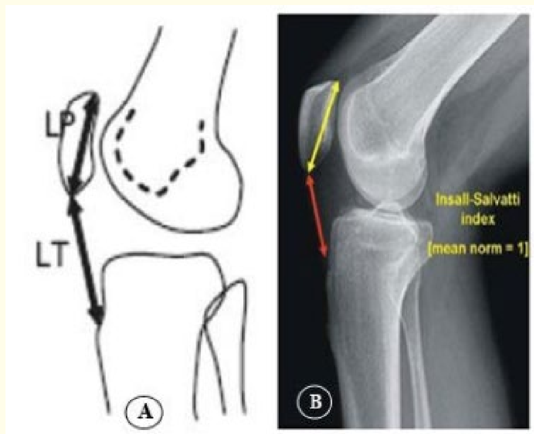

Figure 7: *Measurements diagnose patella alta. Lateral radiograph with 30 degrees of knee flexion. Insal-Stavati index (red line/yellow line).*

Patella baja

Patella baja, or low patella, is a condition in which the lower end of the patella moves too close to the articular surface of the tibia. This condition can occur when the patella is positioned too low on the distal aspect of the femoral trochanter or when the patellar tendon is abnormally short. Patella baja can cause the following:

- Anterior knee pain
- Stiffness
- Changes in joint mechanics
- Decreased lever force
- Lag in the extensor muscles
- Decreased range of motion.

Like a high patella, patella baja can be identified using the Insall-Salvati index. A ratio of 0.8 or less is diagnostic of patella baja.

In normal individuals, the patella does not articulate with the trochanter when the knee is fully extended. In contrast, in patella baja, the patella is always in contact with the trochanter, even when fully extended.

Baja patella is commonly seen after repair of a torn patellar ligament, a fracture of the patella, or a high tibial osteotomy, all of which reduce the upward pull of the quadriceps muscle on the patella. Treatment of symptomatic baja patella typically involves surgical intervention to bring the patella closer to the patella. There is no gold standard treatment for baja patella. However, surgical techniques have been used, such as tibial tuberosity transfer to improve patellar height and patellar tendon lengthening using autografts or xenografts [24].

Imaging findings

Each patient underwent radiographs (performed according to Hugston [48]), MRI, CT, and ultrasound of the knee and thigh. We expected to see the following findings in cases of dislocation: high patella and lateral patellar tilt; small and high patella; shallow patellar groove and flattening of the distal femoral condyles. We examined radiographic abnormalities of lateral femoral dysplasia, high patella, and flat or convex trochlea by CT and MRI. The trochlea depth was assessed on MRI and flattening of the distal femoral condyles by CT. We also found fibromuscular fascia in the distal thigh on ultrasound in all patients.

Lateral patellar tilt (Roentgenogram/X-ray)

On the X-ray film, the patient is lying on his back, the knee is bent at an angle of 20 to 30 degrees, the foot is pointed straight up, the X-ray tube is held between the ankles and the tape support is kept in place.

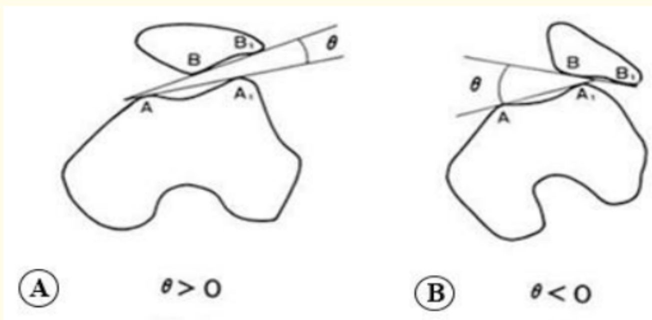

Figure 8: *Measuring lateral patellofemoral angle to diagnose patellar tilt.*

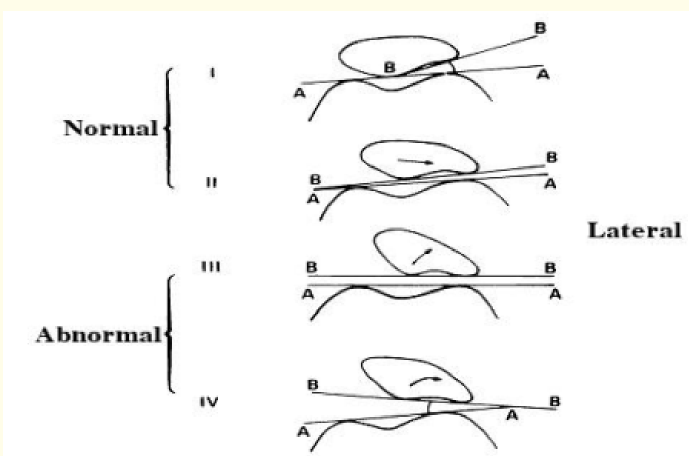

Figure 9: *Normal and abnormal lateral patellofemoral angles. In normal individuals, the patellofemoral angle is usually laterally open and never medially open. However, in patients with recurrent patellar dislocation, the parallel lines or the contralateral patellofemoral angle is medially open.*

The cassette is placed near the knee, perpendicular to the X-ray beam, with the lower border of the film parallel to the ground. It is seen that a line between the femoral condyle (A-A) and a line between the edges of the lateral patellar notch (B-B) form the lateral patellar angle. Measurement of the lateral patellar angle is performed as Laurin., *et al*. and the angle is determined to be positive when extended to the side [49] (Figure 8).

Abnormal tilt: The examiner looks for evidence of lateral deviation and tilt in this way: In patients with knee extension, the patella may be pulled laterally more than 2 cm. It is uncertain whether the tight collateral ligament is the cause or the result of the tilt. On the X-ray, the tilt is calculated as described above; a line along the subchondral bone of the anterior patella may replace the line from edge to edge. We considered it an abnormal tilt angle if it was greater than 15º based on Grelsamer and Proctor [50].

Congruence angle (Figure 10)

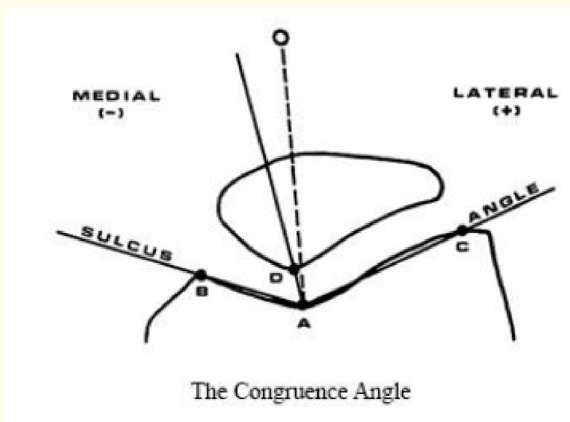

Figure 10: *To measure the congruence angle: Find the highest point of the medial (B) and lateral (C) condyles and the lowest point of the intercondylar notch (A). (A clear plastic straight edge is helpful.) Angle BAC is the notch angle. Bisect the notch angle to establish the zero reference line, AO. Find the lowest point on the articular ridge of the patella (D). (A straight edge held parallel to the transverse axis of the patella is helpful.) Projection line AD. Angle DAO is the congruence angle. All values midway between the zero reference line AO are designated as negative and values lateral to it as positive. Mean = -6 degrees; standard deviation = 11 degrees.*

Shallow patellar groove (MRI)

Pulley depth was assessed on MRI images according to Seil., *et al*. [51]. A line drawn parallel to the posterior surface of the femoral condyle served as a reference line (Figure 11, line D, green). Lines drawn perpendicular to the reference line indicated the largest anteroposterior diameter of the lateral (line A) and medial (line C) pulley surfaces and the deepest point of the groove (line B). Pulley depth was calculated as follows: (A + C/2) − B. A pulley depth of 3 mm or less was considered to indicate dysplasia.

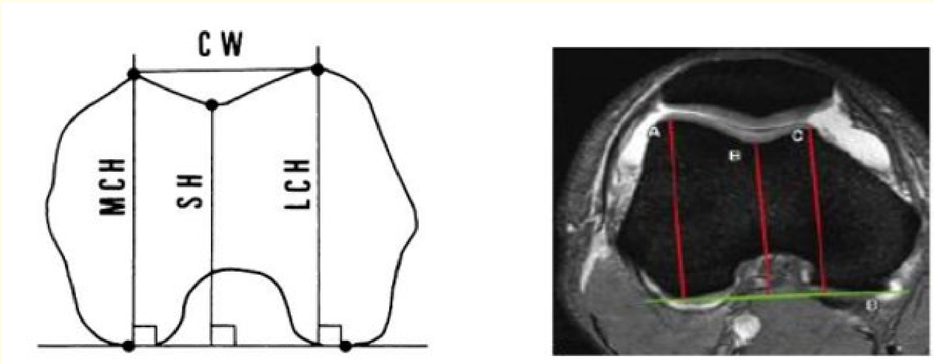

Figure 11: *Measurements used to calculate trochlear depth.*

Trochlear dysplasia (Figure 12 and 13)

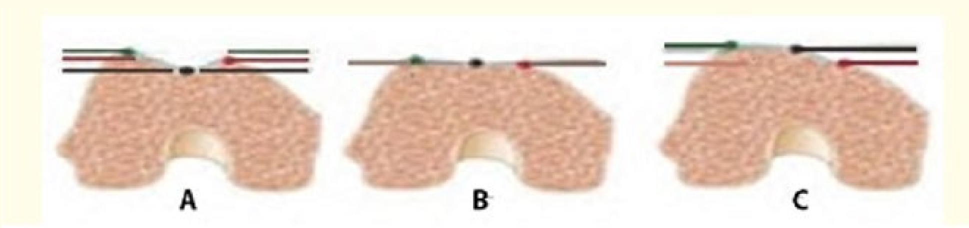

Figure 12: *A: Normal pulley, with a pulley depth of 12 mm. B: Dysplastic pulley with marked flattening, indicated by a pulley depth of 1.5 mm. C: Lateral convexity (double contour): apex of lateral surface = groove > apex of medial surface [52].*

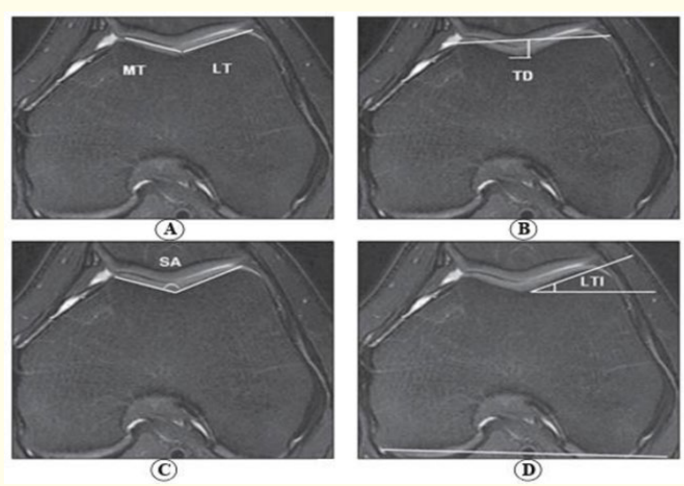

Figure 13: *MR Images show axial measurements in 16 years girl who underwent knee MRI. A: Trochlear facet asymmetry. AT= Lateral Trochlea. B: Trochlear depth (TD). C: Sulcus angle (SA). D: Lateral trochlear inclination (LT).*

Classification of dysplasia

A pulley depth of 3 mm or less is considered to indicate dysplasia, as mentioned above. We used the classification of pulley dysplasia by Dejour., *et al.* [53]:

(1) Grade A: Crossing sign and shallow pulley.
(2) Grade B: Crossing sign; superior pulley spur; flat pulley.
(3) Grade C: Crossing sign; double contour ("double appearance"); asymmetry of pulley surfaces.
(4) Grade D: Grade B + C, asymmetry of pulley surfaces and cliff-like pattern.

We found a shallow patellar groove in 28.6% of patellar dislocations and no shallow patellar groove in 71.4% of patellar dislocations, compared with normal pediatric anatomy.

Patients with a shallow patellar groove may perform more active movements than other patients.

Flattening of the distal lateral femoral condyles (CT scan)

We performed CT scans of the distal femur of each patient on the injured side. The grooves were visualized in both the transverse and sagittal planes. We used the following definitions:

- Normal pulley: lateral end > groove < medial end
- Flat pulley (crossing sign): lateral end = groove = medial end
- Lateral convexity (double contour): lateral end = groove > medial end [53].

Patellar dislocation classification

Our patients were classified according to the criteria of Bensahel., *et al*. [54]:

(1) Type 1: Patellar dislocation without major radiographic abnormalities; and
(2) Type 2: Patellar dislocation with major femoro-patellar dysplasia, namely a high patella and a flat or convex pulley.

Treatment - Surgery

Paletta, in 1820, reported the first case of congenital patellar dislocation, followed by Wuhzer. 1835; Lelius. 1840 and Michaelis. 1854. In 1959, Cotta [55] listed 137 surgical approaches to address patellar instability, however, differences in reporting and study design make comparisons between these studies almost impossible.

Hauser (1938) [5], in his classic paper, described six different surgical approaches for recurrent dislocations, and Marion and Barcat (1950) [56] described over 100 surgical techniques. However, there were only three basic goals in all of these procedures: (1) release of the tight patellar ligament and capsule; (2) to correct any abnormal patellar path caused by excessive lateral patellar ligament attachment; and (3) to tighten the patellar retinaculum and medial patellar capsule.

Tibial tuberosity transfer is contraindicated in children because of the risk of premature closure of the anterior aspect of the proximal tibial epiphysis with the development of a retroflexion deformity [57].

The three most common surgeries are the Hauser procedure, the Roux-Goldthwait procedure for children [4,58] and the semitendinectomy procedure [59]. All three are major surgical procedures, involving a long skin incision, weeks of immobilization in a cast, and extensive rehabilitation.

Several different surgical techniques have been described [13,17,20,31,33] but all follow the same principles:

- Extensive lateral release to allow patellar and quadriceps centralization:
- Release of the iliotibial ligament.
- Lateral capsulotomy.
- Biceps femoris lengthening if there is a subluxation of the tibia.
- Posterior capsular release may be considered if there is significant knee flexion contracture.
- V-Y quadriceps plasty, z-lengthening or femoral shortening may be required if the extensor mechanism is short and cannot be centralized.
- Medial capsular grafting to stabilize the patella.

Consideration may be given to reconstructing the medial patellofemoral ligament to complement the medial interposition. If the patellar tendon insertion is excessively lateral, distal alignment can be achieved by Roux-Goldthwait transfer or by completely medializing the patellar tendon [60].

Madigan., *et al*. 1975 [3]

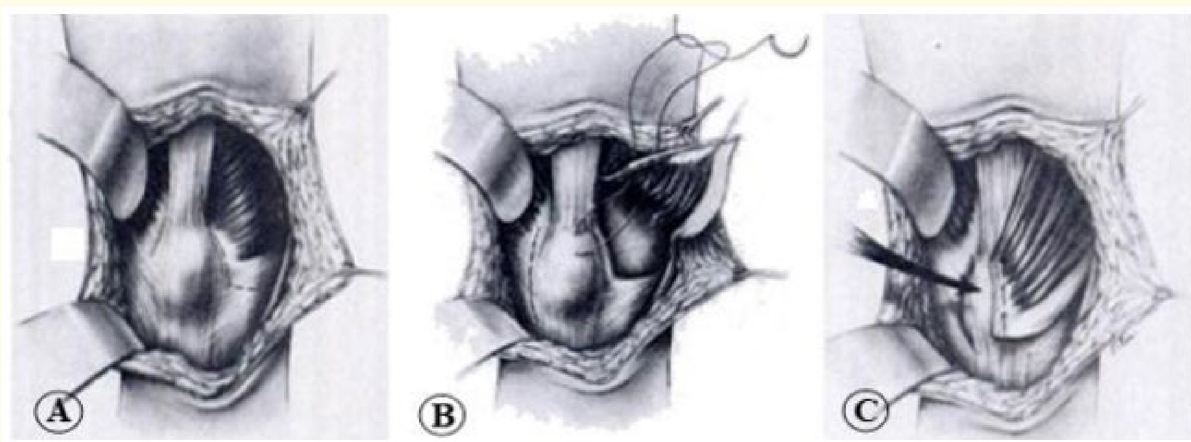

Figure 14: *Technique of quadricepsplasty. A: Interrupted line indicating capsular incision to mobilize vastus medialis obliquus. B: Interrupted line indicating lateral retinacular relaxing incision. C: Insertion of vastus medialis obliquus is transferred laterally and distally.*

West-Soto Hall 1958 [6]

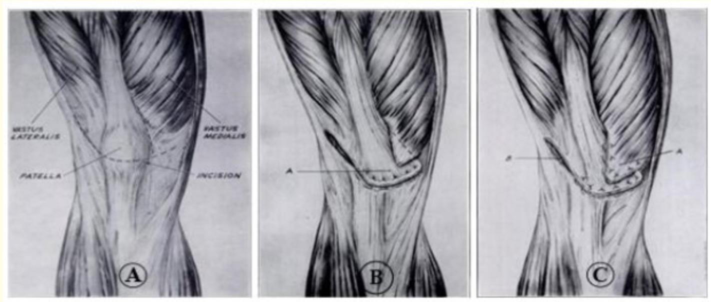

Figure 15: *West-Soto Hall's surgical technique.*

Conn Harold R., *et al*. 1925 [31]

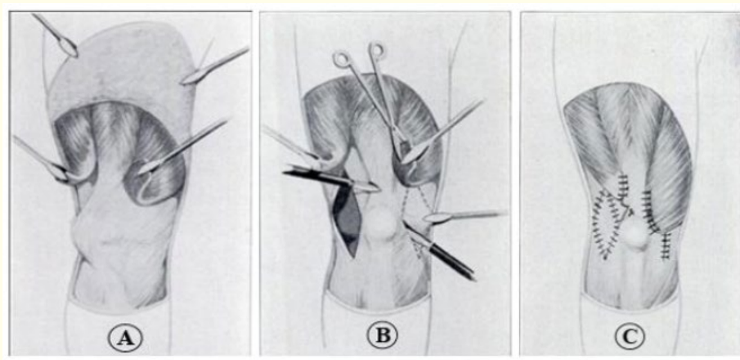

Figure 16: *Conn Harold's surgical technique.*

Roux 1888 [16]

Figure 17: *Modified Roux - Goldthwaite Technique patellar tendon split and transferred medially onto the tibial insertion of the sartorius muscle (1) modification of the classical Roux-Godthwait operation by adding a lateral release (2); and medial half of patellar tendon.*

Other surgical techniques for dislocation of the patella

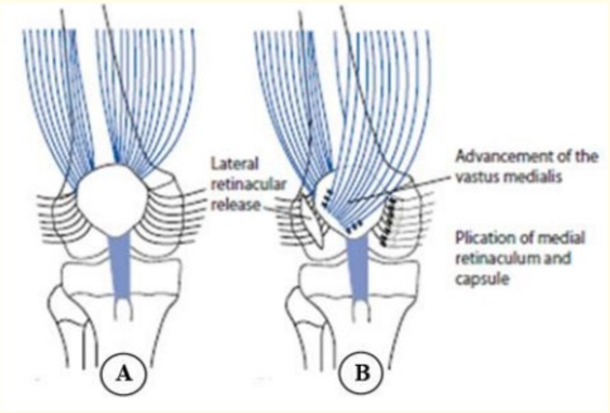

Figure 18: *Diagram showing the technique of advancement of the vastus medialis to restore dynamic stability. Release of the lateral retinaculum and placation of the medial capsule and retinaculum are also shown.*

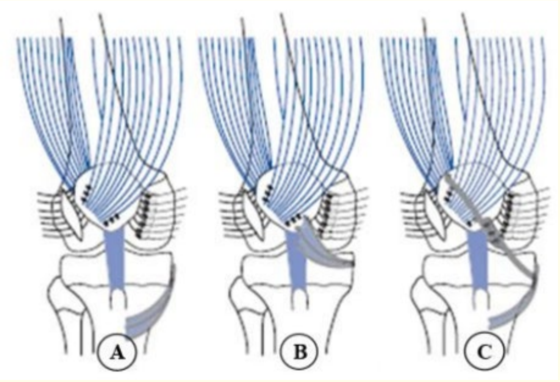

Figure 19: *Diagram illustrating the technique of performing the pes anserinus transfer to augment the dynamic forces stabilizing the patella (B) and the technique of performing a semitendinosus tenodesis (C).*

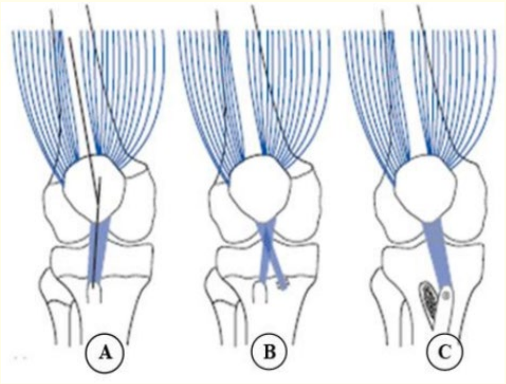

Figure 20: *Diagram illustrating how an abnormally high Q-angle (a) can be effectively reduced by transferring the lateral half of the patella tendon medial (the Roux-Goldthwait procedure) in a skeletally immature child (b) or by transplanting the tibia tuberosity laterally in the skeletally mature child.*

Hung NN 2008 have reported [61,62]

The iliotibial tract passed through patella [61]

The iliotibial tract is usually inserted into one side of the lateral epicondyle of the tibia. Through this insertion, the gluteus maximus and the vastus lateralis fascia stabilize the slightly flexed knee during weight bearing. However, the iliotibial tract is simply a thick band of the vastus lateralis fascia. This fascia forms a septum between the muscles of the quadriceps group, and on both sides of the patella is reinforced by fibers associated with the vastus lateralis and vastus medialis fascias. These fibers are stronger on the lateral side, where they originate from the iliotibial tract. The iliotibial tract lies anterior to the axis of rotation of the knee joint and passes posterior to that axis when the knee is flexed. Therefore, if the patella is bound to the tract in any way, it must be pulled laterally during knee flexion. The iliotibial tract is attached to the vastus lateralis distal. If the vastus lateralis is shortened, lateral patellar dislocation will occur when the knee is flexed. The vastus lateralis tendon and the iliotibial tract must be bisected so that the patella can lie in the intercondylar groove when the knee is fully flexed. However, no one has ever thought of using the bisected iliotibial tract to stabilize the patella.

The author used the iliotibial tract to dislocate the patella by passing the iliotibial tract through a tunnel created between the anterior surface of the patella and the patellar fascia, without drilling the patella, so that the patella was not fractured. The iliotibial tract generated forces to stabilize the patella, medially aligning the quadriceps femoris patellofemoral mechanism, and providing a medial balancing vector and distal realignment of the extensor mechanism. Our surgical technique met all the requirements of Hinton [63] and Stanitski [64]: (1) release of an abnormal tethering vector, (2) balancing the medial vector, and (3) aligning the quadriceps femoris patellofemoral mechanism.

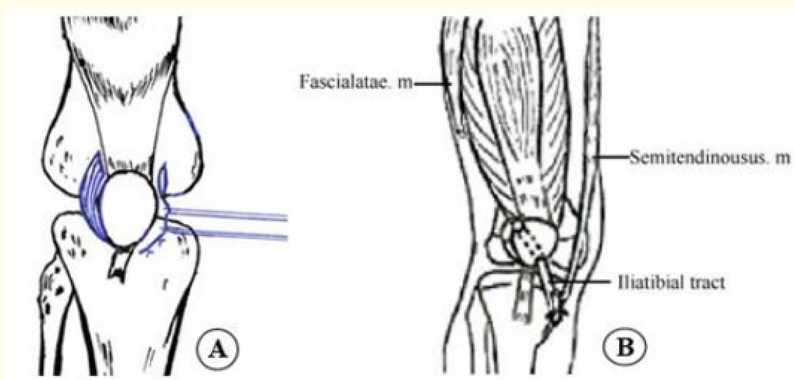

Figure 21: *(1) In the first stage, the lateral patellar tendon is released and the medial tendon tension is restored to maintain the patella in a lower position and allow 25% lateral displacement with the knee flexed at 45 to 60 degrees with a No. 3 Ethibond suture used; (2) In the second stage, the free tendon is cut approximately 3 inches from the iliotibial line of the superior lateral border of the patella, the remaining proximal end is sutured to the femoral fascia; a tunnel is made under the periosteum of the patella from the superior lateral border to the inferior medial border, the iliotibial line is passed through the tunnel, the patella is displaced medially and laterally, the iliotibial line is sutured to the patella, and the free end of the iliotibial line is secured to the semitendinous attachment with the knee flexed at 45 to 60 degrees with a No. 3 Ethibond suture.*

Figure 22: *Free tenotomy of iliotibial tract.*

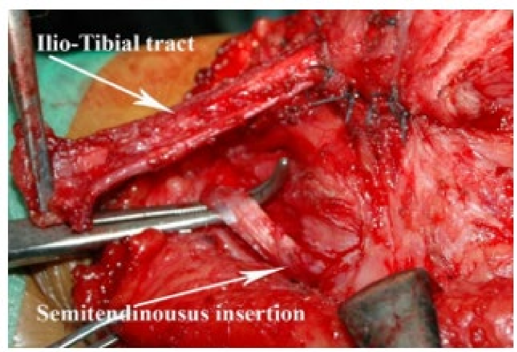

Figure 23: *The tunnel is beneath periosteum of the patella. The Ilio-Tibial tract is suture at semitendinousus insertion. The patella is translated medially and distally.*

Transfer vastus medialis muscle to superior border patella [62]

The operation is performed in 3 stages.

The first stage: Lateralis vastus medialis from the patella and restoring medial retinaculum.

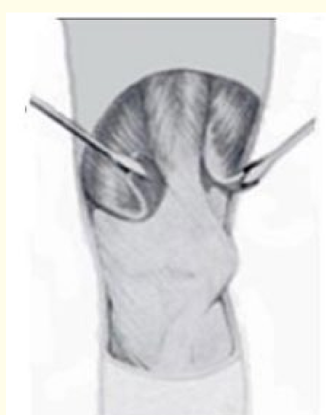

Figure 24: *Detach the insertion of vastus.*

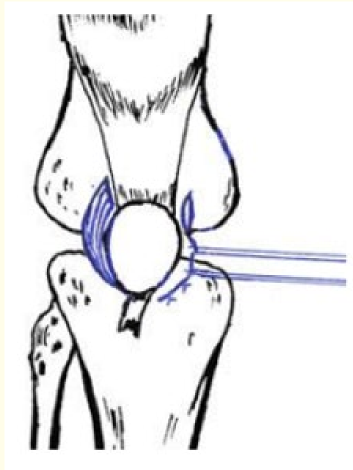

Figure 25: *Releasing lateral retinacular.*

Second stage

This stage for lengthening rectus femoris and vastus intermedius separating the adherence between vastus intermedius tendon and rectus femoris tendon.

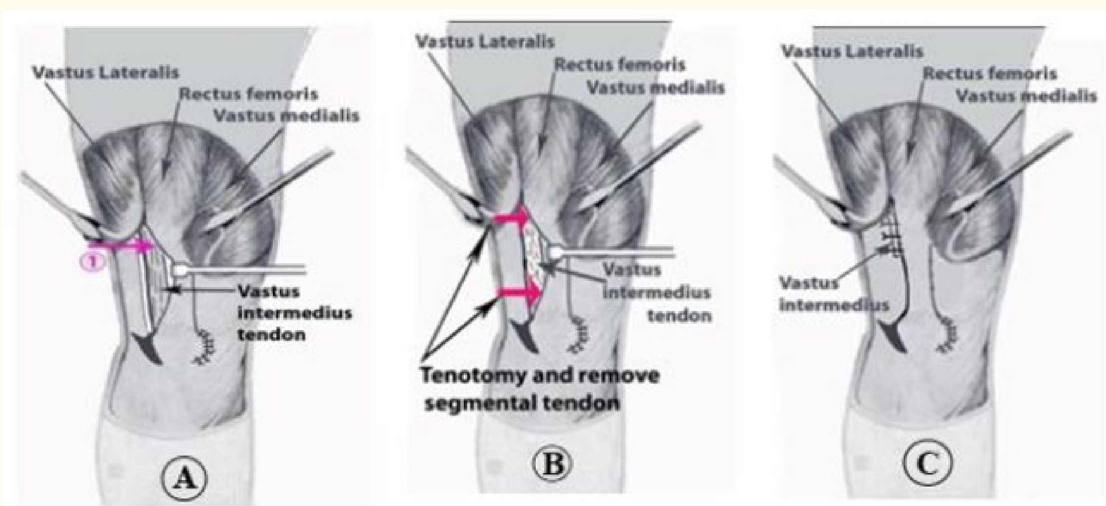

Figure 26A-26C: *A: Separating vastus intermedius tendon and rectus femoris tendon, 1. Positional tenotomy at the musculo-tendinous junction one 2 inches above the patella; B: Tenotomy and remove segmental tendon of vastus intermedius; C: The remaining vastus intermedius is sutured to beneath rectus femoris.*

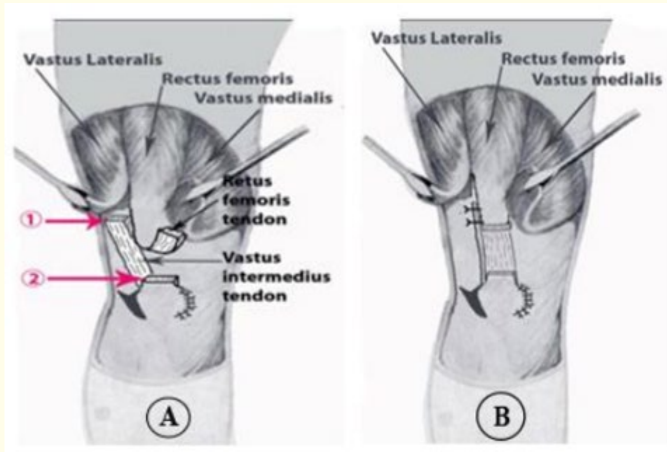

Figure 27A and 27B: *A: 1. Positional tenotomy at the musculo-tendinous junction of vastus intermedius 2 inches above the patella, 2. Positional tenotomy of rectus femoris 5 cm above the patella; B: Two tendons Vastus intermedius and Rectus femoris were sewn together with knee flexion 60º.*

Third stage: The transfer of vastus medialis oblique to superior border patella. The vastus lateralis is sutured to the lateral side of the rectus femoris (Figure 28A) or to joint tendon (rectus femoris and vastus intermedius) (Figure 28B).

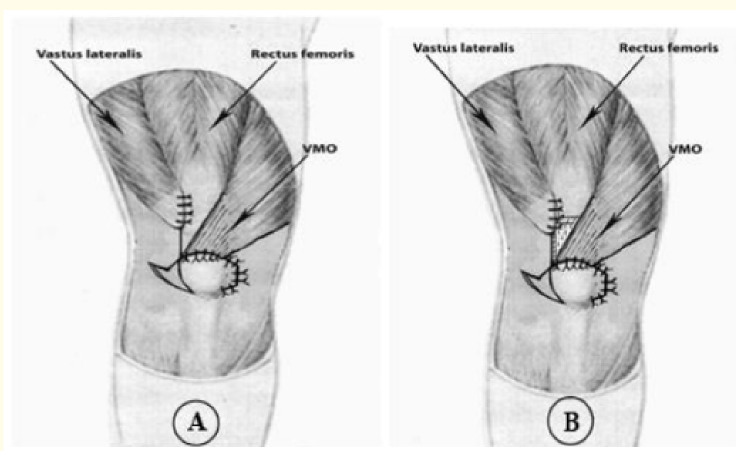

Figure 28A and 28B: *A: VMO transferred to superior border of Patella and vastus lateralis is sutured to the lateral side of the rectus femoris in variant 1; B: VMO transferred to superior border of Patella and joint tendons (rectus femoris and vastus intermedius).*

Transplantation of the vastus medialis muscle, distal and lateral to the anterior patella, was proposed by Madigan, Wissinger and Donaldson in 1975 [3]. All authors agreed that in children with immature bones, intervention should be performed only on soft tissues.

In 1975, Madigan., *et al*. [3] (followed by others) transferred the VMO laterally and distally and sutured it directly to the anterior aspect of the patella or to the medial rectus femoris tendon, according to West [6], in the hope of stabilizing the patella and directing patellar forces medially, thereby hopefully preventing lateral displacement.

The vastus medialis muscle can be functionally divided into long and oblique components. Lieb and Perry [65] reported that in the larger and more proximal long component, the fibers are more vertically oriented, medially oriented 15 to 18 degrees from the longitudinal axis of the femur, whereas the fibers in the distal quadrant of the muscle, the oblique component, are medially oriented 50 to 55 degrees from the longitudinal axis of the femur.

Furthermore, the traction vector of the oblique component is similar to that of the long component.

The sole unique function of the vastus medialis oblique muscle is to generate an inward force to the superior border of the patella and thus prevent lateral displacement. This impression is supported by the findings of Lieb and Perry [65]. Clinical practice shows an excellent and good rate of 94.7%, without poor results or re-dislocations, demonstrating the superiority of these techniques.

In our study, the VM was often severely atrophied and had a more vertical than oblique orientation, as reported by Andrish [66]. To accomplish our first goal, which was to restore the original directional force of the VMO, we freed it from the medial intermuscular septum and transferred it to the superior border of the patella. Unlike Madigan's technique, in which they transferred the insertion site of the vastus medialis laterally and distally to the patella, in our patients the vastus medialis was tight and shortened, so that it could not be transferred distally and laterally to the anterior border of the patella. In these cases, if the VMO were sutured anteriorly to the patella, it would slip the sutures if the knee was flexed or severely limited postoperatively in knee flexion.

The author's second goal was to restore normal alignment and function of the extensor mechanism. The author freed the insertion site of the vastus lateralis and patella, so that the lateral forces on the patella were reduced. The vastus intermedius and rectus femoris also limit knee motion. We lengthened the vastus intermedius and rectus femoris muscles with the aim of improving the knee flexion and extension.

The quadriceps femoris, vastus lateralis particularis, and vastus intermedius muscles may affect the distal femoral nutrient artery and the local metabolism in the distal femur. Furthermore, mechanical damage from frequent patellar dislocations has produced bone changes around the distal femur.

Compare two techniques and other techniques [67]

If we compare Madigan's results, fifty-eight percent had good or excellent results and 42 percent had poor results after a mean follow-up of twenty-nine months; Our results were ninety-four percent had good or excellent results and no poor results or recurrent patellar dislocation; our results were better than Madigan's results with P.valuate 0.000001. If we compare the two surgical techniques for patellar dislocation in this study, it shows that before and after surgery, there are similar clinical and imaging signs (P.valuate > 0.05); and similar postoperative results (P.valuate > 0.05); postoperative complications are also similar. However, we show that VM time (mean 55 minutes) is shorter than ITT (mean 65 minutes).

Comparing ITT and VM in the two age groups under and over 14 years in this study, the postoperative results were similar (P.value > 0.05). Comparing the good and excellent results of the Baker technique and the two techniques in this study, our technique was superior to the Baker technique (P.value > 0.0085). Bensahel., *et al*. [54] performed patellar instability surgery in children with a good postoperative result of 85%. Several studies have shown good results when using the semitendinotomy technique to treat patellar dislocation. Baker., *et al*. [7] published a series of 53 knees and demonstrated that this technique provided good to excellent results in the majority of young and older children and 79% and 89% good to excellent results in young and older children, respectively. The recurrence rate was < 5%. Hall., *et* *al*. [68] described a series of 26 knees with good to excellent results > 60%. However, Hall., *et al*. [68] also performed the Roux-Goldthwait procedure on the knee in their study, making their data more difficult to interpret. More recently, Letts., *et al*. [11] presented a series of 26 knees with 82% good to excellent results with a recurrence rate of 8%.

This study is similar to the previous results reported by Baker., *et al*. [7].

Complications

Reported post-operative complications include persistent medial or lateral patellofemoral dislocation, persistent extensor lag, stiffness, and peroneal nerve palsy.

Conclusion

Diagnosis of patellar dislocation requires a combination of clinical and imaging findings. Currently, various surgical techniques for DP exist, but, tibial tuberosity transfer or osteotomy is contraindicated in children because of the risk of premature closure of the anterior aspect of the proximal tibial epiphysis with the development of a retroflexion deformity.

And all follow the same principles:

1. Extensive lateral release to allow for patellar and quadriceps centralization:

a. Iliotibial ligament release
b. Lateral capsule resection
c. Biceps femoris lengthening is performed if there is a valgus subluxation.

2. Posterior capsule release may be considered if there is significant knee flexion contracture Hung NN. Pediatric Orthopedics in Guidelines 432.
3. V-Y quadriceps femoris reconstruction, z-bone lengthening, or femoral shortening may be required if the extensor mechanism is truncated and cannot be centralized.
4. Medial capsule grafting to stabilize the patella.

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