Patellofemoral Pathologies

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Introduction

Patellofemoral joint pain is one of the most common conditions presenting to a musculoskeletal physician's office. Pathology of this joint is common in athletes and other young active individuals. Due to its anatomical location, most pathology related to the patellofemoral joint is on the anterior aspect of the knee.

The posterior surface of the patella articulates with the femur and is divided into seven facets. Each facet is convex, allowing for articulation with the concave femoral surface. The distal femur will form an inverted U-shaped intercondylar groove to articulate with the patella. The lateral facet of the patella and femur are larger and extend proximally to improve patellar stability. The angle between the lateral and medial femoral condyles is the sulcus angle. Patellar subluxation is associated with a greater sulcus angle and can contribute to patellofemoral pain syndromes.

The orientation of the patella can affect biomechanics of the knee joint. When the patella's orientation is superior or inferior to the normal alignment, this is known as patella alta or patella baja, respectively. The patella can also have an inferior, superior, lat-

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eral, or medial tilt. It is also possible to have lateral and medial rotation of the patella. These different malalignments predispose patients to patellofemoral pathologies [1].

Biomechanics of the patellofemoral joint are dependent on the components' static and dynamic stability due to the shallow fit between the patella and femur. Static stability is supplied by the patellar tendon, joint capsule, medial ligaments (MPFL, medial patellofemoral ligament), lateral ligaments (IT band, lateral patellofemoral joint ligament, lateral retinaculum), and the joint capsule. The Q-angle is important for static and dynamic stability. To conserve patellar alignment in motion, the dynamic stabilizers such as the quadriceps, pes anserine muscles, and biceps femoris are necessary.

These components of stability enable the patella to act as a mechanical pulley for the quadriceps as it changes the direction of the extension force in the knee throughout range of motion. Contribution from the patella increases with progressive extension and is critical in the last 30 degrees of extension [2].

Patellar tracking describes the dynamic movement of the patella and depends on active contraction of the quadriceps and alignment of the patella in the trochlear groove. With open chain movements, the patella will glide superiorly with knee extension, as previously mentioned, and inferiorly with knee flexion. The contact surface area of the patella on the femur increases with increased knee flexion to disperse forces over a greater joint area. In addition to superior and inferior motion, the patella moves slightly laterally with extension and medially with flexion [3]. The patella will be restrained by the quadriceps in closed chain exercises, and therefore the femoral surface will glide posteriorly to the patella [1]. Maltracking of the patella may create narrow contact pressure points between the patella and the femoral groove, associated with anterior knee pain.

Anatomic and biomechanics are important to consider in any patient with patellofemoral pathology. Identification of malalignment or poor biomechanics may be crucial in determining appropriate treatment.

Patellofemoral Pain Syndrome

Patellofemoral pain syndrome (PFPS) is characterized by anterior knee pain around or behind the patella that cannot be attributed to another intra-articular or peripatellar pathology. Although no true consensus exists about the etiology of PFPS, the concept of overloading the knee's extensor mechanism is fundamental to all theories of the underlying pathophysiology. PFPS appears to be multifactorial with causative factors including overuse, malalignment, and trauma. Of these factors, overuse is the most common. PFPS is associated with periods of increased physical activity, high mileage in runners, and activities that overload the knee's extensor mechanism. The overload leads to subchondral bone degeneration, retinacular strain, and damage to small nerves [4–6].

Although the majority of patients with PFPS do not demonstrate malalignment during physical or radiographic examination, multiple authors suggest patellar malalignment, and therefore abnormal patellar tracking, is the primary etiology of PFPS. Static biomechanical risk factors for malalignment include leg length discrepancy, hamstring and hip muscle tightness, abnormal patellar mobility, abnormal trochlear morphology, angular and rotational deformities of the lower extremity, abnormal foot morphology, and hallux valgus. Dynamic biomechanical risk factors include muscle weakness or imbalance (ex. vastus medialis and hip abductors), knee abduction impulses, and excessive or insufficient foot pronation [7, 8]. The dynamic Q-angle is an important anatomical factor in PFPS. Females tend to exhibit a greater dynamic Q-angle than males when performing tasks associated with patellofemoral pain [9, 10].

Prior research has also indicated the subtalar joint of the foot may influence the positioning of the patella, potentially leading to PFPS. Tiberio suggests excessive pronation leads to tibia and femoral internal rotation, causing a relatively lateral patella, which may lead to PFPS [11]. Even though subtalar joint pronation may be a factor leading to PFPS, not all individuals with PFPS will have subtalar pronation during ambulation [12].

PFPS can also be caused by direct or indirect trauma to structures around the patellofemoral joint. For example, injury can occur secondary to falls, contact sports, or motor vehicle collisions [13].

PFPS is most common in active individuals accounting for nearly 25% of all identified knee injuries. The pathology occurs more often in patients participating in competitive sports than age matched controls. The ratio of women to men is nearly 2:1 and it occurs disproportionately in the second and third decades of life [13, 14].

Clinical Presentation

PFPS often presents as anterior knee pain poorly localized under or around the patella. Onset can be acute or gradual. Presentation can be unilateral or bilateral. Pain is often characterized as achy or dull but can be sharp. It is typically aggravated by loading the patellofemoral joint during weight bearing on a flexed knee, which occurs with activities such as squatting, running, prolonged sitting, or ascending and descending steps. History of overload or trauma should be obtained upon presentation [5].

Physical Exam

Physical exam should begin with observation of risk factors such as obesity, vastus medialis atrophy, or angular and rotational deformities. Assess for localized erythema, warmth, or effusion. Assess active and passive range of motion and observe any maltracking (J-sign). Patellar tendon tenderness may be present, though tenderness at the inferior border of the patella is more associated with tendinopathy. Test for risk factors such as quadriceps weakness, hip abductor and external rotator weakness, or strength discrepancies between affected and unaffected lower extremities. Patients usually have full range of motion of the knee. Observe gait for excessive varus or valgus knee movement and observe foot strike for excess supination or pronation. Of note, 80% of patients with PFPS experience pain with squatting [5, 6, 15].

Diagnostic Studies

PFPS is a clinical diagnosis and therefore imaging studies are unnecessary for initial management. Indications for imaging include history of trauma, overt instability, prior surgery, pain at rest, or failure to improve after a comprehensive rehabilitation program. When indicated, plain films of the knee (weight bearing PA, weight bearing lateral, sunrise view) are useful to rule out other sources of anterior knee pain such as bipartite patella, osteoarthritis, loose bodies, and occult fractures. Similarly, advanced imaging modalities (MRI, CT, US, radionuclide scanning) are not indicated in initial evaluation but can aid in diagnosis of pathology other than PFPS [16].

Treatment

In the acute phase (weeks 1–2), activity modification should be encouraged. Patients should avoid activities that cause pain such as running, jumping, squatting, and ascending or descending stairs. NSAIDs should be used acutely and then tapered off as pain decreases. Although evidence is lacking, applying ice to the anterior knee is reasonable for pain relief.

In the recovery phase (weeks 3–6), combined physical therapy and adjunctive therapy is recommended. Physical therapy should begin with a physical therapist or comparable professional and exercises should not cause pain. Exercise programs should include: quadriceps stretching, hamstring stretching, iliotibial band stretching, hip strengthening, quadriceps strengthening, core stability, and proprioceptive exercises. The lateral forces on the patellar are often stronger so a program that addresses muscle imbalance (ex: vastus medialis, hip abductors) would be appropriate. Adjunctive therapy can include foot orthoses to control excessive foot pronation or supination, patellar bracing, and patellar taping to allow pain-reduced range of motion and balance contractile forces [6, 15, 17].

Evidence is lacking for surgical intervention in PFPS and is considered a treatment of the last resort. Operative treatment should only be considered after 24 months of failed conservative management or when an obvious surgical lesion exists [16, 18].

Patellar alignment, patellar resurfacing, and patellar arthroplasty are the three principal categories of operative intervention for PFPS.

Currently there is no high-quality evidence supporting the treatment of PFPS with injectable therapies such intra-articular glucocorticoid, hyaluronic acid, platelet-rich plasma, glycosaminoglycan polysulfate, and botulinum toxin [19].

Return to Activities

Before returning to full activity, the patient should demonstrate equal range of motion and at least 80% strength to that of the uninvolved extremity. Mild pain that diminishes with activity is generally not concerning but severe or increasing pain should prompt termination of the activity and reassessment by a clinician. While recovering and awaiting returning to sport, athletes can maintain aerobic fitness with activities such as a stationary bicycle, upper body cycle, swimming, water running, or other painless exercises.

Idiopathic Chondromalacia Patellae

Chondromalacia patellae is often used synonymously with patellofemoral pain syndrome. However, there is consensus that patellofemoral pain syndrome applies only to individuals without cartilage damage. Chondromalacia patellae is defined as idiopathic articulate changes of the patella and may lead to anterior knee pain. The primary pain generator in this syndrome is not well understood and is thought to be multifactorial. The leading theory is the articulate changes along with roughening or damaging of the undersurface of the cartilage of patella lead to pain. Subchondral bone has weak potential to generate a pain signal. However, the anterior fat pad and joint capsule are sensitive areas with high potential for creating pain signals [20, 21].

Involved factors in chondromalacia patellae may include limb malalignment, muscle weakness, chondral lesions, and patellar maltracking. This pathology is more common in adolescent females.

Clinical Presentation

The patient will likely present with vague and diffuse knee pain in the peripatellar or retropatellar aspect of the knee, worsened with squatting, prolonged sitting, or ascending stairs. There may be pain on patellar compression when the knee is extended. Movie theater sign (pain with knee in flexed position for extended period of time) is also positive in these patients.

Anatomic characteristics to evaluate for in patients with suspected chondromalacia patellae include increased Q-angle, genu valgum, external tibial torsion, pronated feet or subtalus, and femoral anteversion.

Physical Exam

Findings are the same as those with PFPS, except these patients may also have palpable crepitus. Clark's test is a special test specific for chondromalacia. The examiner will compress the patella into the femur while the patient contracts their quadriceps. This maneuver is positive if there is a grinding sensation or pain.

Diagnostic Studies

Plain radiographs may show chondrosis, shallow sulcus, patella alta or baja, or lateral patella tilt. CT is indicated if there is suspicion of patellofemoral malalignment or fracture.

T2 weighted MRI is the best method to assess articular cartilage. Abnormal cartilage will appear with a higher signal compared to normal.

The Outerbridge Classification is used to describe the stages of degeneration. The first level is simple softening of the cartilage. Level two is classified by fibrillation of the hyaline cartilage, reflecting further degeneration. The third level is fissuring of the cartilage to the subchondral bone. The last and most severe level four refers to the area of bone devoid of articular cartilage covering [21].

Treatment

Conservative methods are the mainstay of treatment and should be trialed for a minimum of 1 year. In this condition, NSAIDs are superior to steroids. The patient should be advised to modify activities for relative rest and initiate a physical therapy program with focus on VMO, core, and hip external rotator strengthening, and closed chain short arc exercises. Orthotics may be used in patients with pronation of the foot. PRP (platelet-rich plasma) and prolotherapy may be used, but is not standard of care [22].

Surgery is indicated for patients who do not improve after 1 year of conservative management with extensive physical therapy or for patients with Outerbridge grade three or four. The patient may undergo an arthroscopic debridement or patellar realignment [21].

Return to Activities

Most patients recover in months to years depending on the severity of pain. Younger patients often achieve better long-term recovery.

Patellar Tendinopathy

Patellar tendinopathy, commonly known as "jumper's knee," is an overuse injury of the patellar tendon that develops after repetitive, forceful, eccentric contraction of the extensor mechanism [23].

This pathology of the knee was previously described as "tendonitis," which was a misnomer as the histology will show degeneration and microtears of the tendon, rather than inflammation [24, 25]. Inflammation is involved but is not the primary source of this pathology [26].

Clinical Presentation

Like PFPS, the patient will present with anterior knee pain. Most commonly, this will affect adolescent jumping athletes and males more than females, which distinguishes it from quadriceps tendinopathy that is more common in older adults. The pain will have an insidious onset. In addition to pain located in the anterior knee, it may be specifically localized to the inferior border of the patella. Movie theater sign may be positive, which is when the patient reports pain with prolonged knee flexion.

Patellar tendinopathy can be classified into four stages based on the Blazina et al. criteria [27]. Stage one is pain after sports or activity only. Stage two is pain at the beginning of sports or activity that disappears after warm-up, but reappears during fatigue. Stage three is constant pain at rest and with activity. Stage four is complete rupture of the patellar tendon [27].

Physical Exam

On inspection, there may or may not be swelling over the patellar tendon of the knee. The inferior border of the patella will be tender to palpation. Basset sign is a two-part provocative sign that can be used to identify patellar tendinopathy. The patient will be asked to flex and extend their knee. Basset sign is positive if the patient has no tenderness to palpation at the site of the patella insertion on the distal pole of the patella with full flexion, but does have tenderness when the knee is fully extended. This maneuver is relatively nonspecific [28].

Other factors identifiable on exam are intrinsic biomechanics of the knee joint. Individuals with patellar tendinopathy may also

have tight quadriceps, quadricep atrophy, hamstring tightness, weak VMO, increased Q-angle, patella alta, and genu varum or valgus [23].

Physical exam alone is unable to clearly diagnose patellar tendinopathy.

Diagnostic Studies

Plain radiographs will not usually reveal patellar tendinopathy but can rule out other potential causes of anterior knee pain. Recommended views are AP, lateral, and sunrise or Merchant. Merchant view can identify or rule out patellar subluxation [23]. In chronic cases, an enthesophyte, or inferior traction spur, can be present. Ultrasound will demonstrate thickening of the patellar tendon and hypoechoic areas within the tendon. However, ultrasound is user dependent [29]. MRI is indicated if the pain is chronic and without relief from conservative treatment, and for surgical planning. Findings on MRI include tendon thickening, increased signal on T1 images, and loss of the posterior border fat pad [30].

Treatment

Conservative treatment is first line to treat patellar tendinopathy, with the goal of returning the individual to their usual activity level. This includes a short period of relative rest and activity modification to reduce the repetitive stress on the patellar tendon, a short course of NSAIDs (no more than 14 days), and modalities, such as ice. In addition, the patient should participate in a formal physical therapy program with focus on quadriceps, hamstring, and core strengthening and stretching, in addition to eccentric exercises [23, 31]. Taping or a chopat's strap may be useful to reduce tension across the patellar tendon [32].

Sclerotherapy (also known as prolotherapy), iontophoresis, and extracorpeal shock wave therapy have few studies supporting their use. However, more research should be performed before recommending use of these modalities [33–35]. Steroid injections are contraindicated due to risk of possible patella tendon rupture [36].

Operative treatment is indicated in patients with symptoms refractory to conservative management or patients with Blazina stage III, stage IV, and partial tears. Open or minimally invasive surgery may be performed with the goal to resect the angiofibroblastic and mucoid degenerative area. Coleman et al. observed sympathetic benefit to a large percentage of patients regardless of approach type [37].

Return to Activities

Individuals who underwent conservative treatment may return to their sport once completing a physical therapy program, as described above, and when pain has become mild.

For patients who underwent operative treatment, there is a period of initial immobilization of the knee in extension, followed by progressive range of motion and mobilization exercises with weight bearing as tolerated. 80–90% of athletes will return to their sport. However, activity related aching can last 4–6 months postoperatively.

Patellofemoral Osteoarthritis

Knee osteoarthritis (OA) is a commonly diagnosed condition which can affect all three compartments of the knee. Up to 25% of cases involve the patellofemoral (PF) compartment and up to 40% of these cases are considered isolated PFOA [38]. PFOA typically results from the loss of articular cartilage of the patella and the trochlear groove from one or more of the following: overload, trauma, or instability. The lateral patellar facet is more often overloaded than the central or medial aspect of the patella so wear and tear of the lateral facet is most common. Incidence is higher in women and increases with age, primarily affecting those over 45 years of age. Additional risk factors include increased Q-angle, excessive hip anteversion, patellofemoral dysplasia, and history of patellar subluxation or dislocation [39].

Clinical Presentation

Patellofemoral osteoarthritis typically presents with anterior knee pain. Pain can be localized to the medial knee but this more commonly suggests medial-compartment tibiofemoral OA [40]. PFOA is usually bilateral but can be unilateral. The pain is aggravated by walking on inclined terrain, ascending/descending stairs, kneeling, squatting, and rising from a sitting position. Some patients report associated stiffness, locking, clicking, crackling, or crepitus from friction between exposed bones. Morning stiffness typically lasts less than 30 min. Widespread knee pain and distal radiation suggest moderate to severe knee OA. Persistent pain at rest or night pain interrupting sleep suggests more advanced OA [39, 41].

Physical Exam

Knee effusion is common though usually mild and correlates with severity of disease. Pain on palpation is usually localized to the patellar facets. Patellar grind test is often positive for crepitus [39].

Diagnostic Studies

Osteoarthritis in general may be diagnosed on clinical grounds alone with the presence of typical symptoms and risk factors. Additionally, it is important to note that patients with robust OA symptoms may have normal radiographs and vice versa. Diagnostic imaging should be considered, though, in the presentation of young patients, atypical pain location, severe pain at rest or night, severe locking or catching, weight loss, or constitutional symptoms. X-ray will show asymmetric narrowing of the joint space, osteophyte formation, subchondral sclerosis, and subchondral cysts. Isolated PFOA is diagnosed when characteristic findings are seen in the patellofemoral compartment on imaging and no other compartment of the knee is affected [39].

Treatment

Conservative treatment is the first line option for management of patellofemoral osteoarthritis. Activity modification should include avoiding stairs, squats, lunges, jumping, and impact sports. Physical therapy should focus on stretching and strengthening surrounding muscles such as quadriceps femoris. In obese patients, weight loss is helpful in reducing the load on the anterior knee. Evidence is lacking to promote the use of patella unload sleeves, braces, and taping. Pharmacotherapy with acetaminophen or non-steroidal anti-inflammatory drugs (NSAIDs) can be used for pain relief. If experiencing an acute flare or refractory PFOA, intra-articular injection of steroids or hyaluronic acid is recommended. Surgical intervention should only be considered after 3–6 months of failed conservative treatment [42].

For isolated PFOA, surgeons may recommend unicompartmental patellofemoral joint replacement, leaving the healthy medial and lateral compartments intact. Additional options include soft tissue realignment of the extensor mechanism (e.g., lateral retinaculum release), tibial tuberosity osteotomy to improve the Q-angle, and total knee replacement for severe or multicompartmental OA [39].

Return to Activities

While ceasing certain activities such as impact sports can be beneficial to patellofemoral osteoarthritis, patients should remain active during the recovery period. In addition to or following a physical therapy program, patients can gradually increase activity level provided it does not make symptoms worse. Patients can start with low-impact activities that do not stress the joint such as walking, bicycling, and water aerobics and increase from there to their desired activity level.

Prepatellar and Superficial Infrapatellar Bursitis

Bursitis refers to inflammation of a bursa, a synovial-lined fluid-filled sac which reduces friction between tissues of the body. The prepatellar and superficial infrapatellar bursae are commonly affected anatomical structures in the knee. Given their superficial location, these bursae are susceptible to microtrauma as well blunt and penetrating trauma. Septic bursitis can also occur secondary to local inoculation from trauma and accounts for nearly 1/3 of cases. After injury, synovial bursa cells thicken, granulation tissue and fibrous tissue develop, and the bursa fills with fluid [43, 44].

Clinical Presentation

Acute bursitis presents with a short history of localized pain, redness, warmth, and swelling at the anatomical location of the bursa. Chronic bursitis presents as a more indolent course, with pain and swelling elicited by prolonged periods of kneeling. Obtaining an accurate history upon presentation is critical to the diagnosis of prepatellar and superficial infrapatellar bursitis. Jobs requiring prolonged kneeling such as clergy, plumbing, gardening, etc. and sports with frequent direct blows to the knee such as wrestling are known as risk factors [45]. Nearly 80% of all cases occur in men aged 40–60 years. Chronic bursitis can also be due to gout, rheumatoid arthritis, or indolent infection [43, 44].

Physical Exam

Examination of acute bursitis may reveal localized tenderness, erythema, and edema. There may be evidence of skin abrasion or a puncture wound overlying the bursa. Knee effusion is observed in approximately 30% of cases. Chronic bursitis, however, presents as a soft, fluid-filled globular mass in front of the patella or the patellar tendon with typically little to no tenderness. Mild skin inflammation may be present. Strength and range of motion are often unaffected by bursitis but may be limited secondary to pain, particularly in the acute setting [43].

Diagnostic Studies

Prepatellar and superficial infrapatellar bursitis can be diagnosed with clinical history and physical examination alone but diagnostic testing can be helpful in determining the etiology. Often a blood cell count with differential, serum glucose, and serum uric acid are obtained. If there are signs of infection, blood cultures are also obtained. This underscores a key component of evaluation which is the exclusion of septic bursitis. Patients with severe knee pain, limited range of motion, or inability to beat weight should undergo aspiration of the inflamed bursa for evaluation of septic bursitis. Patients with symptoms such as fever, chills, night sweats, tachycardia, or tachypnea should undergo a full workup for sepsis [43]. Imaging studies of the knee are rarely necessary for cases of prepatellar and superficial infrapatellar bursitis, though the soft tissue edema can be detected on MRI [43, 44].

Treatment

Excluding gout and septic bursitis, most cases are self-limiting. Chronic microtraumatic is treated conservatively with addressing the underlying cause [43]. In cases where the damaging activity such as kneeling cannot be avoided, protective knee braces or pads are encouraged [46]. Oral or topical NSAIDs can be used for pain relief as needed. Glucocorticoid injections are not recommended for chronic bursitis due to increased risk of infection [43, 44]. Septic bursitis is managed with antibiotics, knee splint immobilization, and repeated aspiration if fluid reaccumulates [47]. Patients with bursa aspiration identifying monosodium urate crystals should be treated for gout. If conservative measures fail to resolve prepatellar and superficial infrapatellar bursitis, bursectomy is an effective treatment [44, 48].

Return to Activities

Patients should avoid activities that increase pain or inflammation but can re-incorporate activities of daily living provided they do

not cause trauma to the bursa. As mentioned above, patients in high risk occupations and those who cannot avoid activities such as kneeling can return to work/sport with the use of protective knee braces or pads. Traumatic effusions in general tend to resolve slowly over weeks to months. In cases requiring surgical bursectomy, 80% of patients have complete resolution of symptoms and return to pre-injury activity level.

Lateral Patellar Compression Syndrome

Lateral patellar compression syndrome (LPCS) is a disorder of overload and increased pressure on the lateral facet of the patella due to pathologic lateral soft tissue restraints and improper tracking of the patella in the trochlear groove. LPCS is usually caused by adaptive tightening and shortening of the lateral retinaculum. This leads to lateral tilt of the patella and chronic stress imbalance between the medial and lateral articular surfaces. The increased lateral stress on the patella leads to patellar maltracking, patellar cartilage injury, patellofemoral joint pain, and in the long-term osteoarthritis. Because pressure over the lateral patellar facet is increased with knee flexion, LPSC is sometimes referred to as lateral patellar pressure in flexion (LPIF) [49–51].

Clinical Presentation

LPCS typically presents with localized dull anterior knee pain. The pain is aggravated by activities that stress the patellofemoral joint such as climbing stairs, squatting, and prolonged sitting with flexion of the knee. Patients deny instability or crepitus [51, 52].

Physical Exam

Physical exam findings associated with LPCS include pain with compression of the patella, tenderness to palpation of the lateral facet, and inability to evert the lateral edge of the patella [51, 52].

Diagnostic Studies

The primary imaging modality to diagnose LPCS is X-ray. Sunrise knee radiographs often show patellar tilt in the lateral direction. However, the detection rate of patellar tilt by measuring the congruence angle (CA) and patellar tilting angle (PTA) is approximately 70–80% [53].

Treatment

Initial management of LPCS is conservative with NSAIDs, activity modification, and physical therapy focusing on quadriceps stretching and strengthening. Physical therapy should emphasize vastus medialis strengthening and closed chain short arc quadriceps exercises.

Operative intervention is indicated in cases refractory to a comprehensive rehabilitation program. No consensus exists on the gold standard surgical treatment for LPCS but arthroscopic lateral patellar retinaculum release (LPRR) is the most widely used in clinical practice. Other options include patellar realignment, extension of lateral patellar retinaculum or lateral patelloplasty [49, 54].

Intra-articular injection of hyaluronic acid can result in improvement of knee pain and functional outcomes after LPRR in patients with degenerative cartilage changes secondary to LPCS [52].

Return to Activities

Most patients successfully return to activity with conservative management. After arthroscopic lateral patellar retinaculum release though, patients are placed on knee immobilizer, educated on active range of motion and quadriceps strengthening exercises, and allowed to bear weight as tolerated with crutches for 2–4 weeks. The knee immobilizer is then removed when the patient demonstrates sufficient quadriceps control [51].

Patellar Instability

Patellar instability is defined as patellar subluxation or dislocation episodes because of injury, ligamentous laxity, or increased Q-angle. This condition is most common in young patients with patellar malalignment, Ehlers Danlos, or other ligamentous laxity. Osseous factors involved are patella alta, trochlear dysplasia, excessive lateral patellar tilt, and lateral femoral condyle hypoplasia [55].

Clinical Presentation

These patients will present with complaints of anterior knee pain associated with feelings of instability. The classification is based on whether the etiology was acute, chronic, or habitual. Traumatic or acute etiology is equal in male and female gender, and will occur from a direct blow, such as a helmet to knee collision [55, 56]. Chronic cases of patellar instability are more common in women with malalignment of the patella. These patients tend to have recurrent subluxation episodes.

Physical Exam

On inspection, a large hemarthrosis will be present if the etiology of the patellar instability is an acute process. The patient will have medial sided tenderness and increase in passive patellar translation. Lateral translation of the medial border to the lateral edge of the trochlear groove is considered an abnormal amount.

Patellar apprehension test, or passive lateral translation, is positive when it results in guarding. The patient may also have a positive J sign, which is excessive lateral translation in extension, popping the patella into the groove as the patella engages the trochlea early in flexion [57].

Diagnostic Studies

With chronic patellar instability, diagnosis is made with passive patellar translation and a positive J sign. Plain radiographs can be used to rule out fractures or loose bodies. The medial patellar facet is the most common site of fracture. Lateral X-ray can assess for patellar alta or baja and for signs of trochlear dysplasia, including flattened trochlear groove, hypoplastic medial condyle, and supratrochlear spur. Sunrise or merchant X-ray views can assess for lateral patellar tilt and sulcus angle. CT would be useful to assess tibial tubercle–trochlear groove (TT–TG) distance and MRI may rule out suspected loose bodies [57].

Treatment

Patients with first time dislocation without bony avulsion or presence of loose articular bodies should be treated with bracing, NSAIDs, activity modification, and physical therapy. These patients should undergo a short-term immobilization with a patellar stabilizing sleeve or J brace for comfort, then 6 weeks of controlled motion with emphasis on strengthening. Physical therapy should include closed chain short arc exercises, and quadriceps, core, and hamstring strengthening.

Operative treatment is indicated in patients with recurrent patellar instability and includes arthroscopic debridement with removal of loose bodies. Recurrent dislocation with nonoperative treatment is approximately 15–50% at 2–5 years, and recurrence rate is highest for those with a primary dislocation who are less than 20 years old [57].

Return to Activities

Patients undergoing nonoperative treatment may return to activities when asymptomatic or with mild symptoms after completion of a physical therapy program. After surgery, a patient must be cleared by their surgeon prior to returning to activities.

Patellar Tendon Rupture

Patellar tendon rupture is rare and caused by tension overload during activity in a patient at risk. It is more common in the quadriceps tendon than the patellar tendon. The mechanism is overload of the extensor mechanism of the knee through sudden quadriceps contractions with the knee in a flexed position, such as missing a step on the stairs, or jumping. Most ruptures occur with the knee in the flexed position, as the greatest forces on the patellar tendon are with knee flexion greater than 60°. The most common type of rupture is avulsion from the proximal insertion or inferior pole of the patella. The rupture can also occur midsubstance or from a distal avulsion on the tibial tubercle [58, 59].

Clinical Presentation

This pathology is most common in males in their third and fourth decade of life. Usually the injury occurs with a jumping event involving sudden quadriceps contraction in the knee flexed position. Patients will usually report feeling or hearing a pop and then immediate swelling, difficulty weight bearing, and infrapatellar pain. There are many risk factors for patellar tendon rupture. The collagen structure of the tendon can be weakened due to systemic disease, such as diabetes, lupus, rheumatoid arthritis, chronic kidney disease, or local injury from patellar degeneration, previous injury, or patellar tendinopathy [59, 60]. Corticosteroid injections in the area can also place individuals at risk for rupture.

Physical Exam

On inspection, patients with patellar rupture will have patellar elevation with large ecchymosis and/or hemarthrosis, localized tenderness to palpation below the patella, and a palpable gap between the inferior pole of the patella and the insertion site on the tibia. There will be reduced range of motion and difficulty

weight bearing of the affected knee. If only the patellar tendon is ruptured but the retinaculum is intact, then active extension is possible, but with extensor lag of a few degrees [61].

Diagnostic Studies

Physical exam and plain radiographs can confirm a full thickness tear. On X-ray, the patella will be elevated. MRI is the most sensitive modality and is required to diagnose partial tears or differentiate a partial from complete tendon rupture. Ultrasound can also be used to identify partial and full thickness ruptures (Fig. 5.1) [58, 61].

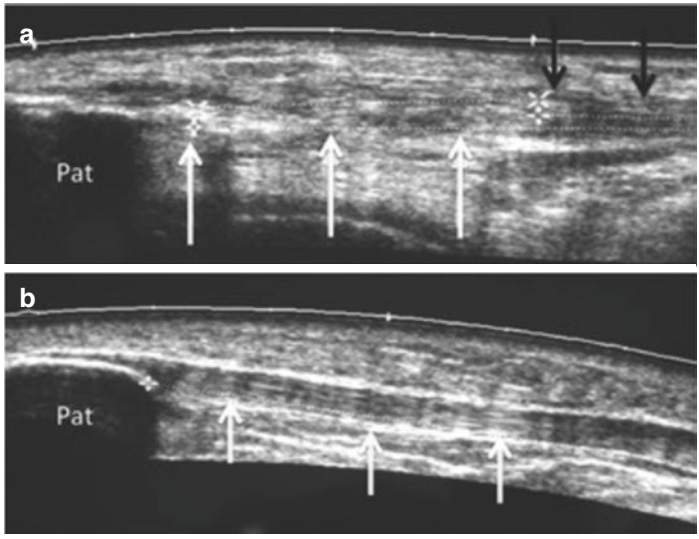

Fig. 5.1 Ultrasound of a normal patellar tendon and a chronic patellar rupture. (**a**) The proximal patella (Pat) is visualized on the left side of the image. The hypoechoic region (white arrows) indicate development of scar tissue. The remaining distal tendon is visible (black arrows). (**b**) Ultrasound image of a normal patellar tendon. (Source: Magnussen R.A., Demey G., Archbold P., Neyret P. (2014) Patellar Tendon Rupture. In: Bentley G. (eds) European Surgical Orthopaedics and Traumatology. Springer, Berlin, Heidelberg. https://doi-org.eresources.mssm.edu/10.1007/978-3-642-34746-7_137)

Treatment/Return to Activities

In patients with an intact extensor mechanism and partial tear on MRI, treatment begins with immobilization in full extension for 6 weeks with weight bearing in a hinged knee brace. After this 6 week period, rehab is a progressive active flexion and passive extension protocol [61].

Complete tears require timely operative repair. The most important prognosticating factor is timing of the repair of the tendon. Post-operative rehabilitation will begin after 6 weeks with a locked extension brace. Patients can immediately weight bear and then may undergo early motion protocols at 7–10 days post-op with a focus on passive extension and active flexion [58].

Patella Fracture

The patella is a trabecular bone with articular cartilage on the posterior aspect and is susceptible to fracture as it is superficial and unprotected by overlying soft tissue. As discussed, the patella is an important part of the extensor mechanism of the knee. The mechanism of fracture is usually direct trauma onto the anterior aspect of a flexed knee or a direct impact of an extended knee. When the knee is flexed on impact, the result is usually a complex comminuted fracture from impact and compression against the femoral condyles and trochlea. Impacts on the patella while extended usually cause nondisplaced fractures. Rarely, the extensor mechanism can result in an avulsed portion of the patella, more common in pediatric or athletic cohorts.

Clinical Presentation

The patient will usually present after a fall or direct trauma to the knee. The extensor mechanism may still be intact depending on the severity and location of the fracture. Often, there will be painful swelling of the knee and fractures may be palpable on exam.

Diagnostic Studies

Plain radiographs can confirm the diagnosis and analyze the fracture pattern. Fractures visualized may be classified in one of seven patterns—nondisplaced transverse fractures, displaced transverse fractures, longitudinal, or vertical, fracture, comminuted nondisplaced fracture, comminuted displaced fracture, osteochondral fracture, avulsion (sleeve) fracture (Fig. 5.2). These fractures were further classified by Duparc et al. into three types. Type I is

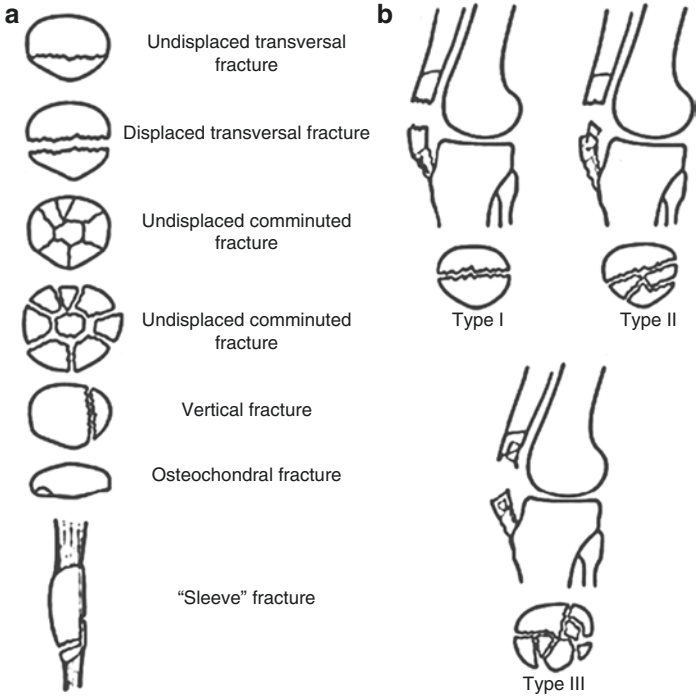

Fig. 5.2 (**a**) Morphologic classification of patellar fracture. (**b**) Duparc classification of patellar fractures. (Source: Weppe F., Demey G., Fary C., Neyret P. (2014) Fractures of the Patella. In: Bentley G. (eds) European Surgical Orthopaedics and Traumatology. Springer, Berlin, Heidelberg. https://doi-org.eresources.mssm.edu/10.1007/978-3-642-34746-7_230)

a transverse fracture located between the proximal two thirds and the distal one third. The etiology is usually pure knee flexion with a strong quadriceps muscle contraction. Type II is a transverse fracture with a comminuted distal fragment. The trauma etiology is a fall on a flexed knee causing sagittal compression of the distal pole of the patella, which compresses the patella against the condyles. Type III is the "stellate fracture," in which the patella fragment is comminuted. The mechanism is violent anterior top posterior compression, commonly seen when a knee undergoes a trauma against a solid object, just as a car dashboard (Fig. 5.2).

Treatment/Return to Activities

Nonoperative treatment, including physical therapy, is indicated if the extensor mechanism is preserved, the articular step off is less than 1 mm, and the fracture is stable. Mobilization should be initiated as early as tolerated. During the first 3 weeks post-injury, the patient should range the knee between 0° and 60°. After 6 weeks, the patient can begin to exceed 90° of flexion. Between physical therapy sessions, the patient should have the knee splinted at 30° to avoid patella baja. Weight bearing is allowed if the knee is in a full extension splint. Monitoring by plain radiograph should be performed at 1 week, 2 weeks, and 6 weeks to ensure no secondary displacement. Operative management is indicated if the extensor mechanism is not intact [62].

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