Anterior Knee Pain in the Young Athlete
Diagnosis and Treatment

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Abstract: The underlying etiology of anterior knee pain has been extensively studied. Despite many possible causes, often times the diagnosis is elusive. The most common causes in the young athlete are osteosynchondroses, patellar peritendinitis and tendinosis, synovial impingement, malalignment, and patellar instability. Less common causes are osteochondritis dissecans and tumors. It is always important to rule out underlying hip pathology and infections. When a diagnosis cannot be established, the patient is usually labeled as having idiopathic anterior knee pain. A careful history and physical examination can point to the correct diagnosis in the majority of cases. For most of these conditions, treatment is typically nonoperative with surgery reserved for refractory pain for an established diagnosis.

Key Words: anterior knee pain, patellofemoral syndrome, idiopathic knee pain

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Anterior knee pain (AKP) is a broad clinical entity that includes all causes of pain in the anterior aspect of the knee. The differential diagnosis in the young athlete is extensive (Table 1), and every effort should be made to narrow this list by history and physical examination. Although AKP technically describes a location of knee pain, it seems to be used frequently as a diagnosis and has been used synonymously with patellofemoral syndrome or even chondromalacia patella.[1] The latter should be used less frequently in the skeletally immature athlete as chondromalacia is an infrequent occurrence in this age group.[2,3] Chondromalacia should be reserved as a term to describe a degenerative condition of articular cartilage.[4] The anatomic sources of intra-articular knee pain have been described by Dye et al,[5] who underwent arthroscopic evaluation of his own knees without general or regional anesthesia and noted that the synovium and fat pad were exquisitely sensitive in comparison to the menisci and articular cartilage. This is consistent with other studies that showed high amounts of substance-P nerve fibers in the synovium and fat pad of knees in patients with AKP, even higher than in those with osteoarthritis.[6] Other studies describe a vascular phenomenon in which tight retinacular structures about the knee cause subtle ischemia within the retinaculum leading to neural proliferation and pain.[7,8]

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Despite extensive research, the exact etiology and pathophysiology of AKP is often still unclear. This review will discuss some of the common etiologies of AKP in the young athlete with an overview of diagnosis and management of the various disorders.

SYNOVIAL IMPINGEMENT SYNDROMES

Synovial impingement is a common cause of AKP. More specifically, synovial impingement may be associated with a pathologic plica, or the result of fat pad impingement. The 3 most commonly found plicae in the knee are the superior, medial, and inferior plica.[9] An inferolateral plica[10] and an arch-type suprapatellar plica[11] have also been described.

The diagnosis of a pathologic plica is a difficult one. It is important to note that synovial plicae are a normal finding, and that identification of a plica on arthroscopic examination does not necessitate resection unless the physical examination is abnormal. Controversy exists in defining what constitutes a pathologic plica. Typically, focal pain that impairs function in which the only objective finding is a thickened hypertrophic plica is considered a pathologic plica.[12] This is an elusive diagnosis but careful history and physical examination can identify those that are pathologic.

Classically, the medial plica has been described to be pathologic.[12,13] At times, patients sustain a direct blow to the knee and have a "window" period free of symptoms. They subsequently develop pain that is most symptomatic with repetitive activities like running. The pain is characteristically aggravated by knee flexion and relieved with extension. There may be an associated thick, palpable cord.[12,13] In addition, patients may have a positive active

TABLE 1. Differential Diagnosis of Knee Pain in the Adolescent Athlete

Synovial impingement syndromes Pathologic plica Hoffa syndrome	Infection
Osteochondroses/tendinitis Osgood-Schlatter Sinding-Larsen-Johannsen Patellar tendinitis	Idiopathic anterior knee pain
Patellar instability	Psychiatric disorder (ie, stress, depression, etc)
Patellar malalignment	Meniscus tears
Osteochondritis dissecans	Fracture
Hip pathology (ie, SCFE, stress fracture, etc)	Ligamentous injury
Tumors (ie, osteosarcoma, osteoid osteoma, etc)	Iliotibial band friction syndrome

SCFE indicates slipped capital femoral epiphysis.

extension test in which pain is reproduced with quick active extension of the leg from a flexed knee position.[14] A flexion test can also be conducted by allowing the leg to flex with gravity from an extended position, and the patient is asked to stop the flexion moment. This causes eccentric contraction of the quadriceps that can cause pain if the plica is pathologic.[14] Radiographs are typically normal, and an magnetic resonance imaging (MRI) scan may identify a prominent plica though often times it will be inconclusive as to whether it is pathologic. Occasionally, this thickened cord can cause wear as it rubs or snaps over the medial femoral condyle.[3] Arthroscopically, more severe pathological changes of the plicae have been associated with more severe chondral changes.[15] With the advent of stronger magnets for MRI scans, it is theoretically possible to identify wear in the medial femoral condyle consistent with the area where a thickened medial plica may exist.

Nonoperative management is the initial treatment, and symptom resolution typically occurs with patients who have had a shorter duration of symptoms. Nonoperative treatment has been described in the literature to include a period of activity limitation, anti-inflammatory medications, corticosteroid injections into the plica and physiotherapy to work on quadriceps, hamstring, and gastrocnemius stretching.[9,13] Results of nonoperative management have had reported success rates as high as 60%.[16] When conservative treatment fails, arthroscopy can be performed and should include a complete arthroscopic evaluation to rule out other etiologies of knee pain, in addition to complete plica excision.

The anterior fat pad of the knee is a collection of fatty tissue posterior to the patellar tendon just distal to the inferior edge of the patella. Hoffa in 1904[17] described irritation of this structure leading to AKP. It has also been shown that manipulation of the anterior fat pad under conscious arthroscopy resulted in the most severe pain with high spatial localization.[5] As a result of its high-density innervation, anterior fat pad syndrome can result in significant pain.

Clinically, patients with Hoffa syndrome may present with swelling in the region of the fat pad and tenderness to palpation in this area. The Hoffa maneuver is performed by applying compression to the fat pad on each side of the patellar tendon while bringing the knee into extension. A test is considered to be positive if this maneuver produces pain or apprehension. The Hoffa syndrome is not evident on plain radiography, but MRI can detect subtle areas of high signal consistent with irritation of the fat pad.[18] Specifically, edema in the fat pad, fibrosis of fat pad, infrapatellar bursitis, and calcifications on MRI has been shown to be associated with impingement[19] (Fig. 1). The described treatment options for anterior fat pad syndrome include rest, avoidance of aggravating activities, and even modalities such as transcutaneous electrical nerve stimulation, ultrasound, and cold therapy. In addition, taping has been used in order to try to decrease compression of the fat pad.[20] Surgery, consisting of debridement of the exuberant fat pad has been used in refractory cases with good results in small series.[21,22]

OSTEOCHONDROSES/PATELLAR TENDINITIS AND TENDINOSIS

Osgood-Schlatter (OS) disease and Sinding-Larsen-Johannsen (SLJ) disease are extremely common in the young athlete with the former being more common. They were originally described in the early 1900s and represent pain at the tibial tuberosity (OS) or inferior pole of the patella (SLJ).[23,24] OS is thought to be due to a traction effect of the extensor mechanism on the tibial tuberosity causing separation of the apophysis.[25] Diagnosis is often made by physical examination with tenderness, swelling, or even a bony prominence in the area of the tibial tuberosity. Plain radiographs are obtained and typically show irregularity with separation of the apophysis in the early stages and fragmentation in the later stages.[26] These radiographic findings, although commonly found, are not diagnostic of OS disease as they may also occur without symptoms. Further imaging studies are usually not required unless patients present with atypical symptoms or to rule out other diagnoses. Treatment can consist of a long course of conservative treatment involving cessation of aggravating activities, nonsteroidal anti-inflammatory drugs (NSAIDs), ice, and physical therapy.[3,26,27] Physical therapy is initially focused on stretching of the quadriceps, hamstrings, and heel cords with progressive strengthening of the hamstrings.[26] Quadriceps strengthening is avoided in the initial stages of rehabilitation because this can increase stresses across the tibial tuberosity apophysis and thus aggravating symptoms.[26] In addition, a counter-force brace may provide some symptomatic relief. Although sport activity is typically allowed unless pain is affecting performance and quality of life and we think of OS disease as a benign,

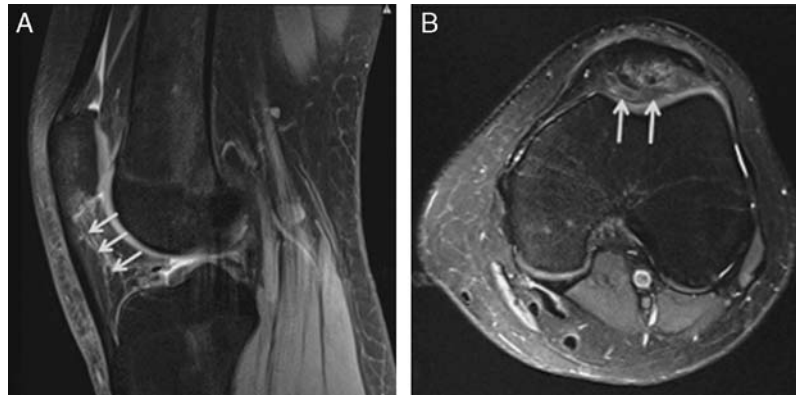

FIGURE 1. Magentic resonance imaging increased signal (arrows) in fat pad on sagittal (A) and axial (B) views characteristic of Hoffa syndrome.

self-limiting condition, it should be recognized that an increased susceptibility to epiphyseal fracture has been described.[28] Surgical treatment is reserved for patients who fail conservative treatment and is typically carried out after skeletal maturity for the removal of a tender and painful intratendon ossicle.[26]

Patients with the SLJ disease present with AKP and a physical examination that reveals tenderness to palpation near the inferior pole of the patella. Plain radiographs may be normal in the early stages with calcifications or an ossicle adjacent to the inferior pole in the later stages[23] (Fig. 2). Similar to OS disease, treatment consists of conservative treatment with activity modification, NSAIDs, ice, and physical therapy. It is invariably self-limiting with surgery being rarely indicated.[23,29]

Patellar tendinitis is another common source of AKP in the young athlete. It causes pain at the inferior pole of the patella that is exacerbated by activity. It is often related to a period of increased activity that can be debilitating during the athlete's sport. Initially, symptoms may start with activities only but as the disease progress, they can occur with activities of daily living.[30] Physical examination demonstrates tenderness to palpation at the inferior pole overlying the proximal aspect of the patellar tendon. A decline squat test can be performed by having the patient squat on a decline surface. This places increased stress on the patellar tendon and can illicit pain.[31] Similar to OS and SLJ syndromes, treatment consists of rest, NSAIDs, ice, physical therapy to work on stretching, and strengthening the muscles around the knee as this has been seen as an overload problem.[30]

If patients are refractory to these measures, an MRI study can be obtained to look for a thickened tendon or increased signal within the proximal end of the patellar tendon consistent with tendinosis. Patellar tendinosis occurs owing to rapid acceleration and deceleration forces across the tendon origin leading to microruptures.[32] Over time, this leads to chronic degenerative changes in the tendon. Ultrasound can also be used as a diagnostic modality to look for these chronic changes owing to the superficial location of the tendon. If imaging studies are consistent with significant tendinosis, surgery may be performed for patellar tendon debridement. Kaeding et al[33] in a systematic review found lower success rates when surgical treatment involved bony debridement of the inferior pole, closure of the paratenon, and immobilization after surgery. Platelet-rich plasma has been used for various tendinopathies but long-term outcomes for patellar tendinosis is nonexistent.

PATELLAR INSTABILITY

Although AKP from an acute patellar dislocation is dramatic and easily diagnosed, AKP from recurrent patellar subluxation can be insidious. The incidence of both is not exactly known. However, one study,[34] which examined a managed care database consisting of 400,000 members, noted a first time dislocation rate of 5.0 per 100,000 yearly. The majority of these individuals were younger and female. Patellar instability can result from an injury owing to a direct blow to the patella, or more commonly by an indirect mechanism during athletic participation. In most cases, the patella reduces spontaneously as the knee is extended.

In evaluating these patients, the treatment is often dependant upon differentiating between patients with normal and abnormal anatomy. Stability of the patella is conferred by both osseous and soft tissue structures. In recent times, the medial retinacular structures of the knee have been recognized for their importance. The medial patellofemoral ligament has been examined in detail and has been determined to be the primary soft tissue restraint to lateral displacement of the patella.[35–37] Numerous studies have demonstrated that in acute lateral patellar dislocation, damage to the medial patellofemoral ligament ranges from partial disruption to the more common complete disruption.[38–40]

The examiner must pay attention to any malalignment including genu valgum, external torsion of the tibia, and femoral anteversion, which may be associated with subluxation or patellofemoral malalignment. Palpation of the structures of the patellofemoral articulation should be undertaken. Patellar tilt can be assessed clinically by placing the examiner's thumb and index finger on the medial and lateral border of the patella with the knee in extension and assessing the relationship of the fingers in the sagittal plane. If the medial border of the patella is anterior to the lateral border, then the patient has lateral tilt. If the medial border is

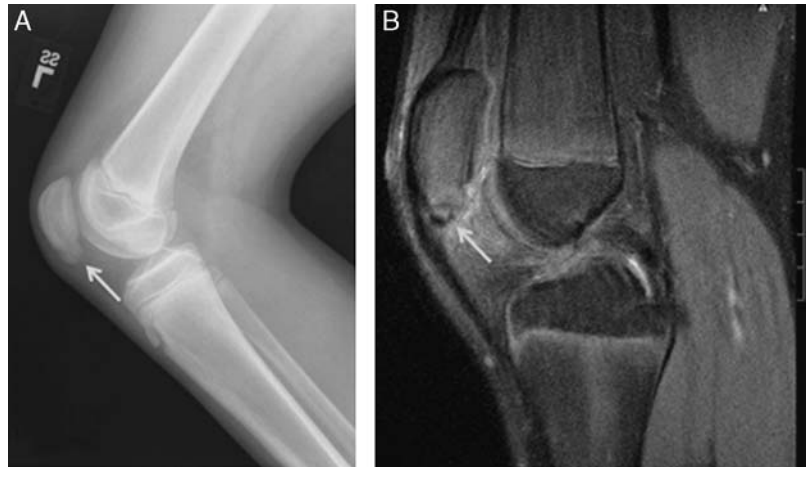

FIGURE 2. An ossicle (arrow) at the inferior pole noted on both the plain radiograph (A) and MRI (B) with increased signal around the ossicle noted on MRI. (Courtesy of Paul Saluan, MD, Cleveland Clinic Foundation). MRI indicates magentic resonance imaging.

posterior to the lateral border, then the patient has medial tilt.[41] Lateral patellar tilt can indicate a tight lateral retinaculum and increased contact forces in the lateral patella and trochlea.[42] Patellar mobility is another test that can be used to assess range of motion of the patella identifying any medial or lateral tightness.[41] The apprehension test, performed by placing a laterally directed force on the patella with the knee in approximately 30 degrees of flexion with subjective feelings of impending dislocation, is the classic examination finding of patellar instability.

Imaging of the patellofemoral instability patient should include weightbearing anteroposterior (AP), 45 degree posteroanterior flexion or tunnel view, lateral and axial views. Plain radiographs can provide information about osteochondral fracture, patellar height, morphology of the trochlear groove, patellar tilt, and the sulcus angle. The single most important view may be the lateral projection as it has been shown to be very accurate in assessing patellar malalignment.[43] Computed tomography scanning can be helpful in planning a realignment procedure once this has been determined necessary based on the patient's clinical findings. The distance between the tibial tuberosity and trochlear groove can be calculated on axial imaging to determine the necessity for a distal realignment procedure once the decision for surgery has been made.[44] A distance greater than 20 mm is considered to be sufficiently abnormal to consider medialization.[44] MRI has also gained acceptance in the imaging of patellofemoral instability, especially in the case of acute dislocation. It can provide information regarding the integrity of the medial retinacular structures, chondral damage, and possible cruciate or collateral ligament damage. In the case of an acute dislocation, Quinn et al[45] described a triad of injuries detectable on MRI consisting of focal impaction injuries of the lateral femoral condyle, osteochondral injuries of the medial patellar facet, and injury of the medial patellar retinaculum. Acute dislocation of the patella is most commonly associated with osteochondral injuries with an incidence of 24%.[46] Nomura et al[47] noted that the most common site of chondral injury is the medial patellar facet. However, one study showed that at the time of arthroscopy, only 32% of chondral injuries and 29% of loose bodies were noted on plain radiographs.[48]

The nonoperative treatment of chronic patellofemoral instability focuses on regaining strength. The role of the hip abductors (pelvic stabilizers) and quadriceps mechanism in stability of the patellofemoral articulation has been noted. Patellofemoral contact pressures have been shown to be at their lowest levels from 0 to 30 degrees of flexion thus leading to the use of short arc extension exercises in the rehabilitation program.

Historically, for an acute dislocation, the knee was immobilized in a cylinder cast to allow for healing of the medial structures. However, studies by Noyes et al[49] and Woo et al[50] have shown the negative impact of immobilization on soft tissue structures and cartilage. Others prefer early motion combined with lateral buttress bracing. The nonoperative treatment of this condition remains controversial.

The role of operative treatment in patellar instability has expanded recently. In the setting of acute patellar dislocation, surgery is indicated when there is an associated osteochondral fracture for removal or repair. Surgery may also be indicated for the repair or reconstruction of the medial retinacular structures of the knee, although this remains controversial.[51] Lateral release in the setting of recurrent lateral instability has been a commonly performed procedure. However, we should remember that biomechanical and clinical studies have shown that the lateral retinaculum may actually prevent lateral instability.[52–55] Therefore, this procedure is best confined to the treatment of AKP associated with a tight lateral retinaculum and should be used with caution for patellar instability.

The surgical treatment for chronic patellar instability involves the identification of the individual pathoanatomies that contribute to the instability and organizing the surgical procedures to correct these pathoanatomies.[56]

OSTEOCHONDRITIS DISSECANS OF THE KNEE

It is important to consider osteochondritis dissecans (OCD) lesions in the differential for AKP in an athlete as they are often missed. The exactly etiology is unclear and seems to be a polyarticular problem.[57] Repetitive microtrauma, vascular insufficiency, and genetics have all been proposed as potential explanations.[58,59] Prevalence is highest in the 10 to 15 years age group with males being more commonly affected with a 15% to 30% incidence of contralateral limb involvement.[60] Because of this, it is recommended that bilateral knee radiographs be obtained once the diagnosis of OCD has been established.

Patients often present with AKP, effusion, and if a loose body is present, variable symptoms of catching, locking, or giving way. Physical examination can demonstrate limited range of motion, an effusion, and a positive Wilson's test, which is carried out by reproducing pain with internal rotation of the tibia while passively extending the knee.[61] Plain radiographs are extremely valuable, and it is important to get a full knee series consisting of a standard weightbearing AP and lateral view in addition to a skyline or Merchant view for trochlear or patellar lesions and a "notch" view to identify posterior lesions. Classically, the majority of the lesions are found in lateral aspect of the medial femoral condyle, followed by the lateral femoral condyle and least commonly the patella.[60,62] The presence of open physes on plain radiographs has a favorable prognosis for eventual healing. However, this may be in part owing to errors in diagnosis in which normal variants of ossification in the posterior femoral condyles are interpreted as OCD lesions.[63] An MRI is also valuable in diagnosing these lesions and may be able to characterize the stability of the fragment. The presence of homogenous, high signal beneath the fragment of greater than 5 mm in diameter may represent an unstable lesion.[64] Lesions are typically classified by Berndt and Harty's system based on talar dome lesions.[65] The natural history, according to a large multicenter trial by Hefti et al,[60] showed that patients with pain and swelling indicative of unstable lesions, lesions greater than 2 cm, skeletally mature patients, and lesions in atypical locations had a worse prognosis. Peters and Mclean[66] reported their series on patellofemoral lesions and noted pain relief whether treated with or without surgery. Surgical treatment was indicated in the presence of unstable lesions or loose bodies.

Treatment of these lesions is dependant on multiple factors including the stability of the lesion as assessed by MRI and intraoperative findings. Fragmentation and the presence and quality of bone in the fragment are important intraoperative factors that determine the reparability of OCD lesions. Nonsurgical management is primarily reserved for stable lesions and consists of activity modification including cessation of running and jumping sports and

pharmacological pain management modalities. In addition, patients can be placed on crutches and kept nonweight bearing until symptom resolution.[58,67] However, if the OCD lesion is in the patellofemoral compartment, then the patient may be weightbearing as tolerated in a knee brace locked in extension. Surgical management is reserved for unstable lesions or those that have failed conservative treatment. Stable lesions that fail conservative treatment may have histological findings of instability in the deep layers of cartilage. This is evidenced by the presence of fibrous tissue or fibrocartilage indicating a delayed union or nonunion in those deep layers.[68] If arthroscopic findings show healthy cartilage and a stable lesion, then our treatment of choice is extra-articular drilling to stimulate bleeding and promote subchondral bone healing. This has shown good results in femoral condyle lesions clinically and radiographically with a healing time of approximately 4 to 8.5 months in skeletally immature patients.[69,70] Transarticular or retrograde drilling has also yielded good results with average healing time of 4.4 months for condyle lesions.[71] For unstable lesions, every effort should be made to salvage the fragment. An assortment of fixation methods, which include headless screws, K wires, bioabsorbable pins or screws, and osteochondral bone pegs, can be used to stabilize the fragment. If the fragment is not salvageable, then treatment options include simple excision, microfracture, osteochondral autograft for smaller lesions, osteochondral allograft, or autologous chondrocyte implantation for larger lesions. One recent study showed osteochondral autograft transplantation had better clinical results than microfracture for OCD lesions in skeletally immature patients, though both treatment options had improved clinical outcomes.[72] Excision is generally reserved for older patients with small lesions away from the central weightbearing portion of the knee. With autologous chondrocyte implantation, it is important to recognize that with significant bone loss, bone grafting may also be required.

HIP ETIOLOGIES

It is well know that pathologic entities of the hip can refer pain to the knee. These include slipped capital femoral epiphysis (SCFE) and femoral neck stress fracture (Fig. 3). In regard to SCFE presenting as knee pain, it has been shown that there is a delay in diagnosis in comparison to patients who present with hip pain.[73] Therefore, an examination of the hip is always included in the evaluation of the patient with knee pain. Posterolateral displacement of the femoral epiphysis relative to the metaphysis is the hallmark of SCFE. The incidence of SCFE ranges from 0.2 per 100,000 in Japan to 10 per 100,000 in the United States.[74] Mean age at diagnosis is 12 years in females and 13.5 years in males.[74] Bilateral SCFE has been reported to be as high as 63% with bilateral involvement at initial presentation in the range of 50%.[75,76] Obesity has been implicated in the etiology of SCFE owing to increasing shear forces across the femoral physis.[77] AP and frog leg lateral imaging of the hip is performed and usually leads to the diagnosis. Once diagnosed, early treatment of SCFE is indicated and is most often accomplished with in situ pinning. However, high degrees of deformity may require corrective osteotomy after initial stabilization.

TUMOR/INFECTION

Another cause of knee pain that must be kept in mind is the possibility of a tumor. Knee pain in the skeletally immature individual that continues despite a reasonable period of nonoperative treatment mandates workup with radiographs. Tumors occurring around the knee include osteosarcoma, Ewing sarcoma, enchondroma, adamantinoma, and osteoid osteoma. Problematic lesions around the knee warrant referral to an orthopedic oncologist for consultation.

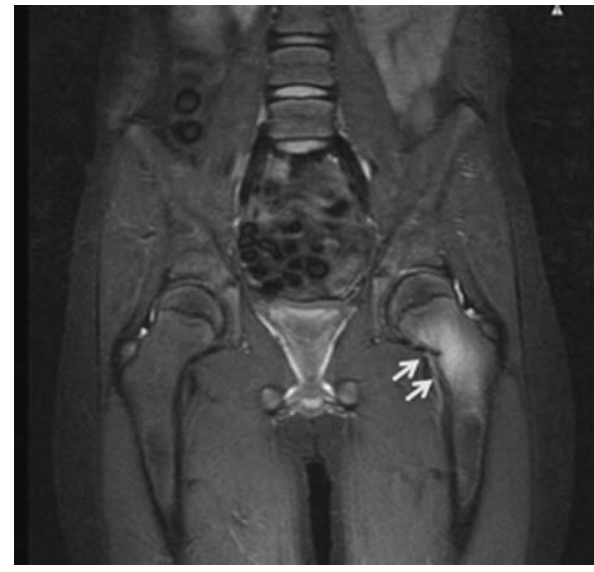

FIGURE 3. Femoral neck stress fracture (arrows) of the left hip in a patient who presented with knee pain.

Infection should also be in the differential in a skeletally immature patient who presents with AKP. Clinical features of septic arthritis include pain with range of motion, swelling, fever, and refusal to bear weight on the affected extremity. Laboratory features include an elevated white blood cell count, erythrocyte sedimentation rate, and C-reactive protein. Diagnosis is made by history and physical examination and confirmed with a knee aspiration. Typically, the bacterial isolate is *Staphylococcus aureus*.[78] Treatment includes irrigation and debridement in addition to antibiotics that the specific isolates are sensitive to.

IDIOPATHIC AKP

If the history and physical examinations as well as diagnostic modalities have not conclusively established a diagnosis, then some would say that the patient has idiopathic AKP.[3,79] An argument can be made that there is no such entity as idiopathic AKP, and most patients will at least have subtle muscle imbalances or malalignment that can subsequently alter patellofemoral contact forces causing pain.[80,81] In these patients, physical therapy is extremely important to identify imbalances or deficiencies of core and/or lower extremity strength and flexibility and then to proceed to organize a rehabilitation regimen accordingly.

Finally, interviewing the parents of the young athlete is critical. The athlete may have extraordinary pressure to succeed in their sport that originates from his or her parents.[3] In these scenarios, it is important to council the parents and the athlete on having realistic expectations and on the potential deleterious effects of overuse injuries with excessive training. In addition, a psychiatric profile to look for signs of depression, hostility, or passive attitude is useful as these findings have been associated with AKP.[82]

In our practice, patients with normal objective findings with chronic pain refractory to traditional treatment will be referred to a multidisciplinary clinic involving a pain medicine specialist and a clinical psychologist.

Fortunately, according to Nimon et al,[83] the natural history of AKP managed nonoperatively is fairly good with 22% having no pain and 71% having an improvement in pain at an average of 16-year follow-up. Though, 1 in 4 continued to have some pain, the authors recommended nonoperative management unless a surgical solution is proposed that can provide better results than in this long-term follow-up.

CONCLUSIONS

The etiology of AKP in the young athlete can be challenging to diagnose. As with most aspects of medicine, a careful history and physical examination will often lead to the correct diagnosis. Most etiologies can be successfully managed nonoperatively. Often times, these young athletes play their sport year round, and a period of rest is a necessary component of treatment. It is can be helpful to council both the child and the parents regarding the importance of relative rest to prevent overuse injuries and ultimately to increase the longevity in the child's sport.

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