Fat Pads as a Cause of Adolescent Anterior Knee Pain

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Abstract:
Anterior knee pain is one of the most frequently encountered symptoms in pediatric sports medicine. The fat pad is a structure with mounting evidence supporting its dynamic involvement in many pathological states in the anterior knee. There are three peripatellar fat pads that occupy much of the extrasynovial space of the knee. This review explores the anatomy, innervation, vasculature, function, imaging, and pathology of these fat pads. Fat pad pathology is likely underestimated given the limited literature on such disease in the pediatric population. In particular, the prefemoral fat pad is the least described of the fat pads with only a few reports detailing chronic pathological processes. To highlight the relevance of the fat pad, particularly in the pediatric population, we describe an atypical case of a self-limiting acute prefemoral fat pad impingement due to a hyperextension injury in a young athlete.

Key Concepts:
• The three peripatellar fat pads of the anterior knee, although often overlooked, are important nociceptive structures with robust vasculature that undergo dynamic changes in many pathological states.
• Fat pad impingement is often described as a chronic process predominantly involving the infrapatellar fat pad; however, acute impingement is also clinically significant and all three of the fat pads may be implicated in disease.
• Acute fat pad impingement injury and chronic fat pad pathology should be included in the differential diagnosis in adult and pediatric patients with anterior knee pain.

The Anterior Knee Fat Pads
Structure and Anatomy
Articular fat pads are intracapsular, synovial lined adipose structures that make up much of the extrasynovial space of the knee by occupying irregularities within the joint cavity. Histologically, the fat pads are ultrastructurally distinct from other types of adipose tissue by their small adipocytes ensheathed in a dense network of collagenous fibers which provide resistance to mechanical stress.1,2,42 The fat pad is also composed of vasculature, nociceptive nerve fibers, immune cells, and multipotent stem cells capable of differentiating into chondrocytes, osteoblasts, or adipocytes.3,4 Embryologically, adipocytes are first present in the joint by the 17th week of gestation and lobules begin to form in the infrapatellar region by the 18th week with increasing size and collagenous density thereafter.4,5 In children, the fat pad volume increases linearly with age until maturity, while maintaining a constant ratio to body weight.8,6 The exact developmental process in the genesis of the fat pad is not yet understood.
Three peripatellar fat pads exist within the anterior knee capsule, the infrapatellar fat pad (IFP) or Hoffa’s fat pad, the suprapatellar fat pad (SFP) or quadriceps fat pad, and the prefemoral fat pad (PFP) or posterior suprapatellar fat pad (Figure 1). The IFP is the most well described and often studied of the fat pads. Its attachments are to the anterior meniscus, the patellar tendon, and the patella. Structurally, there is a central body with two protrusions from the central body termed the superior tag superiorly and the ligamentum mucosum inferiorly which is also known as the infrapatellar plica. Two additional anatomical variants of note are the synovial recesses, a vertically oriented superior recess and a horizontally oriented inferior recess, which occasionally collect fluid that may be mistakenly interpreted on imaging as a pathological area of effusion (Figure 2). These recesses can also harbor loose bodies during arthroscopy making it difficult for identification and retrieval.

The IFP is vascularized by a rich anastomotic network supplied peripherally by vertical contributions from the superomedial and superolateral genicular arteries anastomosing with the inferior genicular arteries. Two to three horizontal anastomoses connect these vertical arteries. The central body of the fat pad remains the least vascularized of the fat pad and is the ideal access site to reduce hemarthrosis and fibrosis should the fat pad be incised. The IFP shares anastomoses with local structures in the knee including the patellar tendon, menisci, and tibial periosteum. Small branches have also been found to supply the cruciate ligaments from the synovial network supplying the fat pad. In less common variants where the middle genicular artery persists in its vestigial association with the ligamentum mucosum, a large portion of the blood supply to the ACL is shared with the IFP. This rich vascular supply with local connections supports the hypothesis that the fat pad aids with healing of local structures but also that it may be a secondary locus of pain and pathology in the knee. However, multiple studies have attempted to elucidate the direct correlation between IFP blood supply and local pathology with inconclusive findings.

There is also rich innervation of the IFP originating predominately from the posterior articular nerve, a branch of the posterior tibial nerve that also courses through the menisci, synovium, and cruciate ligaments. Dye et al. describe conscious pain perception through arthroscopic probing of intraarticular structures without anesthesia; this procedure identified the IFP to be one of the most sensitive structures in the knee, along with the capsule and synovium, in triggering and localizing pain. In addition, healthy subjects with hypertonic saline injected into the...
IFP experienced significant knee pain equivalent to the quality and location of anterior knee pain. Similar to the synovium, there is evidence of a high density of Type IVa free nerve endings and substance-P immunoreactive nerve fibers in the fat pad which increase in density in patients with chronic knee pain, indicating substantial nociceptive potential that is hypothesized be involved in anterior knee pain and inflammation in the setting of local mechanical or chemical irritation.

There is limited literature regarding the SFP and PFP, with the PFP being the least studied structure of the anterior knee fat pads. The SFP is a triangular structure found on the base of the superior pole of the patella between the quadriceps tendon insertion anteriorly and the suprapatellar recess posteriorly. The suprapatellar recess is continuous with the joint space and as such the SFP is lined with synovium posteriorly. The PFP is found directly posterior to the suprapatellar recess with synovium lining its anterior surface. Directly posterior to the PFP is the cortex of the distal femoral metaphysis, extending just superior to the trochlea.

**Function**

In the appendicular skeleton, fat serves a mechanical role beyond its widely recognized biochemical properties. In fact, the fat pads of the knee are only metabolized in severe malnutrition and do not expand with increasing BMI. Although the exact evolutionary purpose of these structures has not been elucidated, many studies, primarily focusing on the IFP, have hypothesized on the normal function of the fat pad in the knee. From a mechanical perspective, the fat pad may serve to absorb compressive stress by accommodating the dynamic shape and volume of the joint space in flexion-extension, thus also allowing expansion of the synovial membrane. This pressure modulating activity has also been attributed to the PFP and SFP. During flexion-extension of the knee, bone movement results in deformation and compression of the PFP between the distal femoral cortex and deep aspect of the quadriceps tendon and patella, thereby protecting the surfaces from bone and tendon friction.

The SFP is thought to provide a similar fortification and congruency of the extensor mechanism, while enhancing gliding of the quadriceps tendon and trochlear articulation during flexion. Cadaveric and animal model studies suggest an increased joint stability and decreased contact pressure of articular structures with the fat pads in place, while significant changes in kinematics result following resection (Figure 3). These mechanical properties are of particular relevance in pathological states, such as osteoarthritis, as several features, including composition and stiffness, may be altered.

Furthermore, there is increased inflammatory cells, edema, fat necrosis, and fibrosis in the IFP in subjects with osteoarthritis, rheumatoid arthritis, and induced arthritides. In particular, the IFP has been found to be a source of adipokines (i.e., leptin, adiponectin, chemerin), interleukins (IL-6 and IL-8), TNF-α, as well as growth factors (fibroblast growth factor, vascular endothelial growth factor). These molecules infiltrate the synovial fluid and alter joint homeostasis which is thought to play a role in the development and progression of knee osteoarthritis.

**Pathology**

There are multiple pathologic processes that can affect the anterior knee peripatellar fat pads. The first description of pathology of the fat pad was in 1904 by Albert.
Hoffa who found inflammatory fibrous hyperplasia of the IFP as a cause of knee pain and impairment of function.\(^5\) Eponymously termed Hoffa’s disease (HD), we now know that acute injury or repetitive microtrauma leads to hemorrhage, inflammation, hypertrophy, and edema of the IFP (Figure 4). Consequent enlargement of the inflamed IFP beyond the margins of the patellar tendon leads to repetitive intraarticular impingement that culminates in fat necrosis and fibrosis.\(^5\) Primary hyperplasia of the IFP is uncommon and the vast majority of cases are secondary to an initial insult from blunt impact or from shearing forces due to impingement, patellar dislocation, cruciate ligament tearing, iatrogenic trauma, and arthroscopic port insertion.\(^7\) Other less common pathologies may too be an etiology for HD impingement syndrome. Several case reports in the literature have identified more rare and likely underdiagnosed pathological processes of the fat pad, including ossifying chondroma, pigmented villonodular synovitis, synovial hemangioma, ganglion cyst, fibroma, and other tumorous process.\(^58\)–\(^63\),\(^69\)–\(^72\) (Figure 5).

Patients with IFP impingement syndrome typically present with anterior knee pain exacerbated by movement, swelling near the patellar tendon, reduced range of motion, and a positive Hoffa’s test (supine with the knee and hip flexed at 90 degrees, pain provocation with palpation along the patellar tendon as the patient extends the leg).\(^5\)–\(^6\),\(^64\)–\(^66\) (Figure 6). Ultrasound has been used for diagnostic evaluation with limited success, thus MRI is the reference modality for definitive diagnosis.\(^1\)–\(^6\),\(^65\) In the acute phase, edema will manifest as high signal intensity on T2 and there may be gadolinium enhancement with active inflammation. Later in the course, once fibrosis has occurred indicating chronic phase, there will be hypointense signals on all sequences with an underlying hypertrophied IFP.\(^6\),\(^67\) Active impingement is difficult to identify on MRI as the knee will not be fully extended during imaging.\(^6\) The initial treatment is conservative with nonsteroidal anti-inflammatory drugs, ice, and rest. Physical therapy can be utilized in an attempt to restore biomechanics through active intervention (ie., muscle training, gait training) and passive intervention (ie.}
taping, stretching).\textsuperscript{66,72} Injections of the fat pad with local anesthetic and corticosteroids have been used with limited success.\textsuperscript{57,73} Surgery may be considered in recalcitrant disease or disease with evidence of metaplastic intra-pad chondral or osseous nodules, a finding suggestive of end-stage HD.\textsuperscript{65} Surgical options include fat pad excision, partial resection, debridement of fibrosis, anterior interval release of the IFP from the tibia, synovectomy, infrapatellar plica release, and denervation, all of which have some evidence of acute symptom improvement.\textsuperscript{66-68,74,76}

Nearly all cases of fat pad impingement syndrome in the literature involve the IFP with very few cases reported involving the PFP or SFP. Literature regarding the SFP predominantly concerns radiologic findings. Studies have evaluated MRI appearance of edema in the SFP which appears analogous to HD. This is theorized to be secondary to repetitive microtrauma with ensuing inflammation and impingement similar to HD although true association to anterior knee pain is indeterminate.\textsuperscript{81-83} Two reports showed symptomatic relief following steroid and anesthetic injection into the SFP in the setting of anterior knee pain and SFP edema.\textsuperscript{84-85} The similarity of this phenomenon to HD on imaging and histology suggests that this is a similar process to HD, but further research is necessary to understand the clinical impact of this disorder.\textsuperscript{81-83}

With maximum extension of the knee, the PFP may be compressed between the posterior patella and anterior femur resulting in impingement and anterior knee pain. This is rarely described in the literature and often overlooked, but in the limited reports available, it is a chronic process with pathophysiology similar to HD.\textsuperscript{87,90-91} There have been two case reports and one retrospective imaging study discussing this entity. The first case describes a chronic impingement of the PFP by the patella that caused fibrous changes, enlargement of the PFP, and a fatty soft tissue mass extending from the PFP to the femoral condyle which was excised arthroscopically with resolution of symptoms.\textsuperscript{90} The second reported case involved hyperplasia of the SFP that was felt to be causing chronic compression of the PFP; symptom resolution was achieved with SFP excision.\textsuperscript{91} In addition, PFP edema and alterations were identified in 68% of patients diagnosed with HD suggesting that it may be an unrecognized component of that pathology.\textsuperscript{95}

**Epidemiology**

In all age groups, the limited epidemiologic data on fat pad pathology makes it difficult to estimate the prevalence of disease. It is thought that fat pad impingement and inflammation syndromes of the knee as a whole are underdiagnosed, both as a primary and secondary entity. Concerning the IFP, in a series of 2623 patients...
undergoing knee arthroscopy, 1.3% had an isolated primary IFP lesion while 6.8% had a secondary lesion co-existing with other pathology. Biopsy specimens in patients undergoing primary total knee arthroplasty have shown fat pad pathology in about 33% of the cases. Further surgical studies describe hypertrophy of the fat pad in 28% of the cases with meniscal lesions and in 39% of the patients with chondropathy of the retropatellar cartilage. Regarding the SFP, two studies of consecutive knee MR examinations found evidence of SFP edema in 4.2% and 13.8% of patients. Finally, a single retrospective imaging study on the PFP identified edema in 6.9% of patients. These data provide insight into the pervasiveness of fat pad abnormality, yet little is known concerning the incidence of clinically relevant disease and the rates of misdiagnosis.

Furthermore, there is a paucity of data on fat pad pathology in the pediatric literature. Three reports detailed ganglion cysts and pigmented villonodular synovitis, while no studies were identified with a primary focus on fat pad impingement syndrome in this population, with the exception of one case report on a child with HD. Two limited epidemiological studies on sports injuries in adolescents reported a prevalence of 0.4-4.5% for fat pad injury as a cause of anterior knee pain. One study also detailed herniation of the IFP through the patellar retinaculum as a rare cause of an asymptomatic anterior knee mass in children. The true incidence of fat pad pathology in the pediatric population is unknown. With the limited amount of literature on pediatric fat pad disease and prefemoral fat pad abnormalities, there is likely underestimation, both clinically and radiologically, of such pathology. To the best of our knowledge, there have been no reports of an acute impingement of the PFP as a cause of knee pain. Here, to highlight the relevance of the fat pad, particularly in the pediatric population, we describe an atypical case of a self-limiting acute PFP impingement due to a hyperextension injury in a young athlete.

Statement of Informed Consent
The parents were informed that data concerning the case would be submitted for publication and they provided consent.

Case Report
The patient is a healthy 10-year-old male with no significant past medical history, past knee pathology, known arthropathy, or prior injuries. He was playing in a youth recreational soccer game when he attempted to kick the ball with his right foot but missed the ball and forcefully hyperextended his leg. The momentum of his kick caused him to lose his balance and he fell onto his back. He immediately experienced pain in the knee as well as swelling. There was no direct knee collision, instability, locking, dislocation, neurovascular symptoms, or subjective “popping” of the knee. This injury prevented him from running and continuing play.

The patient was seen at our orthopaedic clinic the next day with continued symptoms of anterior knee pain and swelling. On exam he had tenderness to palpation over the medial and lateral femoral condyles as well as the medial patella. A large suprapatellar effusion was noted. His passive flexion was limited to 90 degrees compared to 150 degrees on the contralateral knee and his extension was also limited by 10 degrees. The patient had no meniscal signs, and he had a normal ligamentous exam. Plain radiographs of the knee demonstrated a large knee effusion as well as an anterior fat pad sign (Figure 7).
Given his traumatic knee effusion, an MRI was obtained which revealed the following abnormalities: 1) a moderate to large joint effusion; 2) elevation of the prefemoral fat pad with underlying hemorrhage and edema; 3) an impaction injury involving the apex of the patella (Figure 8a and 8b).

Based on the patient’s injury pattern and imaging findings, he was diagnosed with an acute prefemoral fat pad compressive injury. Following this diagnosis, the patient’s symptoms were managed conservatively with a short course of nonsteroidal anti-inflammatory medication and activity modification. After a week, the patient’s effusion had resolved, and his activities were slowly progressed. At four weeks, the patient had returned to full sport with no pain and no recurrence of symptoms.

Discussion

Given that the few previous reports of PFP impingement are chronic in nature, this case is unique in that it is an acute, transient process that has not been previously described in the literature. Typically, hyperextension injuries involve primary tensile resistance on the posterior cruciate ligament and secondary resistance from the anterior cruciate ligament and the posterior capsule.96 These typically are the structures of concern in a patient presenting with knee pain and swelling following traumatic hyperextension. However, MR imaging of our patient showed no internal derangement of these structures, rather a PFP contusion was identified with increased signal intensity to the posterior patella. We suspect that the hyperextension caused the patella to compress the PFP against anterior femur leading to acute inflammation and hemorrhage of the PFP (Figure 9). Unlike the chronic fat pad impingement cases discussed in this review, our patient had a brief impingement and was able to recover over the succeeding weeks. Given that this is the first case describing acute fat pad impingement in a child, it is not known whether there are irreversible changes to the PFP that may predispose the patient to future PFP impingement or other pathology. We expect that given the brevity of the injury the patient will continue to have full resolution of symptoms. However, it is of consideration that repeated trauma of this nature may contribute to development of chronic intraarticular pathology.

With our case description and analysis, we conclude that this novel case is a model for an acute prefemoral fat pad impingement injury. This case report is of value because it demonstrates that impingement of the PFP can be clinically significant, that it can be an acute process, and that it can occur in the pediatric population. Even if fat pad impingement occurs with remarkable frequency as a secondary pathology of the knee joint, this pathology is not given due attention, not only in the literature, but also in common clinical practice of MRI diagnostics.65 Furthermore, anterior knee pain still remains one of the less understood conditions although it is one of the most
commonly encountered problems in a pediatric sports medicine practice. The fat pad is a structure with mounting evidence supporting its dynamic involvement in many pathological states in the anterior knee. More needs to be done to establish the clinical importance of these entities to ensure that patients with pain originating from the fat pad, consequence of any of the multitude of etiologies, are diagnosed and treated appropriately to prevent chronic pain and impairment of function.\textsuperscript{57,65,67}

The fat pads have been described as windows to the disorders of the knee given their nociceptive potential and dynamic changes in articular, synovial, and intrinsic disease. Acute impingement injury following hyperextension and chronic fat pad pathology should be included in the differential diagnosis in those with anterior knee pain. The fat pads, particularly the SFP and PFP, should be subject to further investigation.

References

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