



Infrapatellar fat pad syndrome : a review of anatomy, function, treatment and dynamics

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The infrapatellar (Hoffa's) fatpad is an important structure within the knee, whose function and role are both poorly understood. This review explores the anatomy, neural innervation, vascularity, role in biomechanics, pathology, imaging (stressing the importance of dynamic ultrasound assessment) and treatment of disorders presenting within this structure.

Keywords : infrapatellar fatpad ; Hoffa's fatpad ; ultrasound ; fatpad impingement.

INTRODUCTION

In 1904, Albert Hoffa first attributed impingement of the Infrapatellar fat pad to symptoms of knee pain. He described inflammatory fibrous hyperplasia of the infrapatellar fat pad at excision, a procedure resulting in symptomatic relief in his series of 21 patients (29). In the last 109 years there still remains an apparent paucity of understanding in the role of the Infrapatellar fat pad in health and disease (10,11,21,24). Hoffa disease has been defined as, "an impingement of the hypertrophic fat pad between the articular surfaces of the knee (femeroapatellar and femerotibial)" it has also been differentiated from Hoffa syndrome, the former being fat pad oedema and fibrosis in a normal joint, with the latter occurring in conjunction with concomitant pathology (43). Reviewing current literature yields at best subjective clinical observations and somewhat

small case series (3,14,15,17,19-21,26,29,35,38,39,43,46,47). We present an overview of information pertaining to the Infrapatellar fat pad and the role it may play in pathology and pain around the knee.

Anatomy

The infrapatellar fat pad is one of three fat pads located in the anterior aspect of the knee (24). Its macroscopic, arthroscopic and radiographic anatomic boundaries are all well described (7,11,21,24,32,37,43,49,50). Gallagher *et al* detail cadaveric anatomy, finding it to be a constant structure bounded superiorly by the inferior pole of the patella, inferiorly by the anterior tibia, intermeniscal ligament, meniscal horns and infrapatellar bursa, anteriorly by the patellar tendon and posteriorly by the femoral

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condyles and intercondylar notch. Its attachments are not only to the intercondylar notch via the ligamentum mucosum, but also into the anterior horns of the menisci, the proximal end of the patella tendon and the inferior pole of the patella (24). Lying intra-articular but extra synovial and occupying the whole anterior part of the knee joint in all joint positions (10) it consists of a central body with medial and lateral extensions, along with a superior tag, the latter of which is not always an anatomical constant (24). There are 2 clefts identified macroscopically and, especially in the presence of an effusion, via MR imaging ; one vertical cleft in the superior aspect of the fat pad and horizontal cleft in the posteroinferior aspect of the fat pad (1,12,24,32,50,53). During knee trauma the fatpad may fragment mimicking loose bodies on MRI imaging, or hide true loose bodies within its clefts at arthroscopy (2).

Vascularity

For a structure to share morphological similarities with subcutaneous fat (50), yet only be metabolised in severe malnutrition, and not expand with increasing BMI (16,24,29,50), implies a degree of biological significance (10). It possesses an abundant peripheral anastomotic blood supply, the supromedial and suprolateral geniculate arteries provide 2 vertical arteries and are linked horizontally with 2-3 arteries running distally (37). In addition, there are rich local anastomotic links to the menisci anteriorly, the tibial periosteum inferiorly and to the patellar tendon anteriorly. Anatomic studies by Pang *et al* (48) revealed a large, mostly transversely orientated, contribution of vessels to the posterior aspect and central third of the patellar tendon. The central area is relatively avascular (37), having connotations for central arthroscopy portals and in the harvest of the fatpad for reconstructive measures.

Three *in vivo* studies using Laser Doppler Flowmetry (LDF) have sought to monitor blood flow to the patellar during surgical approach for total knee arthroplasty. Although recently no significant correlation between LDF and post operative anterior knee pain has been found (36), these studies still reveal useful information pertaining to the shared vascular supply of the infrapatellar fat pad in clinical practice and the contribution of the anastomotic vas-

cular supply to surrounding structures. Hughes *et al* demonstrated a reduction in patellar blood flow of 10% following resection of the infrapatellar fat pad (31). Nicholls *et al* did not resect the fat pad in their study, believing it to be an important contributor to vascularity of the patellar tendon (45), they found no significant difference in decline in flow to the patellar from a medial or lateral arthrotomy, supporting the notion of a rich functional anastomosis. Hempfing *et al* found an increase in patellar blood flow after excision of the fatpad, with the knee in extension (27), they postulated the excision of the fatpad decompressed the patellar in extension allowing for an increased blood flow. The varying techniques and differing equipment used throughout these 3 studies makes direct comparisons impossible, but do suggest the infrapatellar fatpad makes some contribution to patellar blood flow.

Neural innervation

The nerve supply to the knee has been historically well documented firstly by Garder and latterly Kennedy *et al* (25,34), with the knee taking innervations from the femoral, obturator and sciatic nerves. The predominant nerve supply to the fatpad is the posterior tibial nerve (34), which provides the majority of fibres to the popliteal plexus ; fibres course from this innervating the posterior capsule, cruciates and anteriorly up to the fatpad.

A superfluity of Type VIa free nerve endings has been identified within the fatpad (6,10,24,50) ; these Type VIa free nerve endings can be activated by mechanical deformation or specific immunoreactive chemical agents. They initiate afferent signals of pain, pressure and thermal changes to the central nervous system and are active via both fast sharp pain pathways and slow chronic pain pathways (6). Dye *et al* (22) describe the conscious neurosensory perception of pain on arthroscopically probing structures of the knee without anaesthetic ; finding the infrapatellar fat pad capable of triggering both severe and localising pain.

Substance-P has been implicated in the genesis of both pain and the induction of a pro-inflammatory response ; several authors have found substance-P nerve fibres in abundance within the fat pad (9,40,55). Furthermore in patients experiencing anterior knee

pain the number of substance P nerve fibres within the fatpad increases significantly (40,54). The presence and number of nociceptive afferent immunoreactive nerve fibres and ability to release pro-inflammatory cytokines has led to some question of the fat pad being a potential causative structure in patellar tendinopathy (6,16). There are suggestions it performs a modulatory role in the inflammatory pathways active in osteoarthritis (13) and in an *in vitro* bovine cartilage model the infrapatellar fat pad tissue has been demonstrated to reduce catabolic mediators (3).

The role of infrapatellar fat pad in biomechanics

In 1950 MacConail surmised the role of fat pads in joints is in occupying anatomical dead space to allow synovial fluid to both circulate around a joint and provide efficient lubrication (41), the rich neurovascular supply of the infrapatellar fatpad suggests this may be a simplistic view. A correlation between weight of the infrapatellar fat pad and body height has been demonstrated, perhaps inferring a biomechanical role within the knee joint (18). Given the rich nociceptive content of the infrapatellar fat pad it comes as no surprise that a form of anterior knee pain can be reliably evoked in normal patients through injection of hypertonic saline into the fatpad (4,5,28). Patients suffering with anterior knee pain have been demonstrated to show a diminished coordination of motor units between medial and lateral vastus muscle units (44). Hodges *et al* (28) concluded that after a painful hypertonic saline injection to the infrapatellar fat pad there was a significantly later activation and reduced amplitude of contraction of quadriceps during stair stepping. This deactivation of the quadriceps has been assumed to increase patellofemoral loading leading to an increased incidence of patellofemoral cartilage degeneration in some reports of Hoffa's disease (39).

Bohnsack *et al* (10), using a cadaveric model, found resection of the infrapatellar fat pad reduced patellofemoral contact pressures, produced a significant medialisation of the patella and decreased tibial rotation during flexion. Using the same model in a later study Bohnsack *et al* (8) sought to simulate oedema and mass effect of the fat pad using an in-

flatable fluid cell implanted within the fat pad. An increase in simulated oedema again decreased patellofemoral contact area and pressure, whilst increasing measured pressure within the fatpad. However, although these cadaveric studies confer a role of the fatpad in biomechanics of the knee, they may not represent true *in vivo* findings.

Two studies have sought to link the presence of oedema within the fat pad to patellofemoral maltracking (33,52), both support the view that oedema within the suprolateral portion of the infrapatellar fat pad is linked with radiological features suggestive of patella maltracking, especially an increase in the prevalence of patella alta, the exact cause of this remains unclear.

Diagnosis (inc. prevalence) of fat pad pathology

The diagnosis of infrapatellar fat pad impingement is often considered a rare diagnosis of exclusion, or misdiagnosed in cases of recalcitrant anterior knee pain (39,43). The population prevalence is not known; Smillie, in 1962, latterly supported by subsequent authors, separated the rarer primary hypertrophy and impingement from the more common secondary disease, occurring in the presence of other pathology such as meniscal tears and ligamentous injuries (39,43,51). Kumar *et al* report a series of 2623 patients undergoing knee arthroscopy, finding its presence as an isolated (primary) lesion in 34 (1.3%) of patients and coexisting with other pathologies as secondary disease in 178 cases (6.8%). Ogilvie-Haris and Giddens report incidence of 1% of patients over a ten year period containing around 1200 patients undergoing surgery.

Several authors have outlined clinical features and diagnostic aids. Anterior knee pain, typically felt in the retropatellar and infrapatellar regions can be considered a universal presentation. There may be an association with patellofemoral crepitus, with some reporting loading the knee and in particular ascending and descending stairs precipitating pain (11,21,38,39,43,47,53).

The importance of excluding causes of other pathology by careful examination in particular of the hip and spine should be accepted. Detailed examination of the knee should be performed to locate

maximal area of discomfort. The infrapatellar fat pad may appear enlarged and firm in consistency to palpation (21,29). Compression of the patellofemoral joint may also cause pain (39,43) Hoffa's test can be performed and is well described. It is performed with the hips and knees both flexed to 90 degrees ; pressure is then applied to the medial and lateral joint lines. The test is positive for impingement if pain is produced during the last 10 degrees of extension (39,43,47). Kumar *et al* suggested a modification of this test ; stating the fat pad should not be palpated during the test as pressure around the joint could trigger pain in surrounding structures giving rise to false positive results. Instead they recommend a passive forced hyperextension by lifting the heel keeping pressure on the anterior tibia, believing if the fat pad is pathological pain will be reproduced without any direct pressure (39). No figures for the sensitivity or specificity of Hoffa's test, or its modifications could be found in the construction of this review.

Synovial inflammatory and proliferative causes should be actively excluded through history and the relevant haematological, biochemical and immunological investigations (39,47). Radiographic assessment of the knee should be undertaken as needed, the acquisition of basic roentgenographic examination is well supported (23,39,43,47). The role for further imaging remains controversial. Most authors recommend further imaging, usually in the form of MRI (although CT and radio nucleotide scans have also been advocated (43)). Von Engelhardt *et al* undertook preoperative MRI scans in 62 patients with clinically suspected and arthroscopically confirmed secondary infrapatellar fatpad impingement. Whilst there was no single pathognomonic features they found some associations present statistically significantly more often than in case controlled MRI scans in patients without impingement. These changes included oedema of the superior/posterior fat pad (present in up to 48% vs 27% of non impingement control) and the presence of an inflamed infrapatellar bursa (present in up to 66% of patients with impingement and up to 43% of patients without) (53). Kumar *et al* refute the need for routine MRI scans unless additional pathology is suspected, stating that impingement will not be visible as an



Fig. 1. — T2 weighted MRI scan in coronal plane illustrating typical appearances of suprolateral fatpad oedema from impingement

awake patient will not fully extend their knees to the point impingement occurs (39). During a pilot study for ultrasound guided alcohol ablation of the infrapatellar fat pad House and Connell (30) noted pronounced sonographic anomalies in consisting of hypoechogenicity and neovascularity on colour Doppler. The widespread validity in the use of ultrasound in diagnosis is still unknown. We have tended to note an relationship of suprolateral fatpad impingement in association with a perceived tight Iliotibial band and through dynamic sonographic assessment have noted a correlation with lateral patella compression syndrome ; but stress that any part of the fatpad can be responsible for impingement. In addition we have observed cases of pre-femoral fatpad impingement presenting with similar symptoms (Fig. 2).

Diagnostic and therapeutic injections of local anaesthetic and steroid into the fat pad have been recommended by some (21). In these cases it is reported



Fig. 2. — T2 weighted MRI scan in coronal plane illustrating typical appearances of prefemoral fatpad oedema from impingement.

an immediate relief of pain and restoration of movement. There is a strong consensus of recommending a course of physiotherapy prior to proceeding to operative intervention (39,43,47).

Fat pad Dynamics

We believe fat pad impingement to be a dynamic phenomenon that is difficult to fully appreciate through the medium of static imaging. Using high resolution ultrasound we have begun to explore fatpad kinesiology and have noted varying sites of impingement in both knee flexion and extension.

The superolateral portion of the fatpad can be noted to adopt a relaxed, freely expansive and relatively fluid state in flexion. On moving to extension the morphology changes as the fatpad becomes incarcerated by the lateral patella facet and quadriceps tendon ; in symptomatic patients the onset of symptoms follows this process ; which we have observed to be the most common site of impingement. There may follow an association with the lateral patellar compression syndrome.

The less commonly seen infrapatella fatpad impingement follows an opposite pattern, becoming

incarcerated between the patella tendon and anterior femur, often creating deformation of the tendon in doing so, and again associated with the onset of pain.

Findings at arthroscopy

Aside from Hoffa's original report in 1902, most authors recommend arthroscopically treating impingement of the infrapatellar fat pad. Due to the more common occurrence in conjunction with other pathology (21,39) a careful and thorough arthroscopic evaluation of the whole joint should be performed. Portals described for adequate visualisation and access are : antrolateral, antromedial, high antrolateral, high antromedial, midpatella lateral and high antromedial (39,43,47). The typical arthroscopic findings are of a hypertrophic, firm, inflamed fatpad. Often signs of fibrosis will be present with a whitish covering instead of the normal yellow appearances. There may be adhesions present and villous changes to the overlying synovium (39,43,47,53). The impingement lesion should be visualised with the knee fully extended and the knee deflated (39). Arthroscopic shavers are the instrument of choice for resection, which is carried out cautiously to protect integrity of the patellar tendon. Kumar *et al* (39) describe three separate arthroscopic morphologies, the first pertained to acute injury related disease with duration of less than 4 months (defined as type 1) ; the fatpad appeared inflamed with contused villous extensions encroaching over the anterior horns of the menisci, engorged blood vessels and signs of recent haemorrhage may be visible. In the more chronic cases (with and without fibrosis – classified as type 2 and 3 respectively) changes included less contusion, but indentation of hyaline cartilage over the femoral condyle at the point of abutment in full extension (type 2), progressing to a firmer fibrotic fatpad with occasional areas of calcification or cartilaginous degeneration (type 3).

Histology in infrapatellar fat pad impingement

Biopsy at time of surgery is recommended to confirm diagnosis and if there is any doubt of the presence of impingement normal histology should prevent excision (47). Histology will tend to show

Table I. — Summary of clinical studies reporting treatment of infrapatellar fatpad impingement

| Author | Number of patients | Average age | Treatment | Mean duration of symptoms | Functional score pre Treatment | Functional score post Treatment |
|-----------------------------------|--------------------|--|--|--|--|--|
| Magi <i>et al</i> (43) | 63 | Not recorded | Failed conservative, proceeding to arthroscopic excision | Not recorded | Nil | Nil |
| Ogilvie-Harris and J Giddens (47) | 12 | 29 (25-50) | Arthroscopic resection | 20 (7-60) | Cincinnati rating system Symptoms 32/50 Function 31/50 | Cincinnati rating system Symptoms 46/50* Function 46/50* |
| Duri <i>et al</i> (21) | 53 | Total average not recorded, subgroups analysed | Local anesthetic and steroid injection | Total average not recorded, subgroups analysed | Nil recorded | Nil recorded |
| Kumar <i>et al</i> (39) | 34 | 38 | Arthroscopic resection | 10 months | Lysholm knee score 37.44 | Lysholm knee score 93.02 (1 year post op)* |
| House and Connell (30) | 12 | 30.8 | Ultrasound guided alcohol ablation | Not recorded | VAS 7.75 | VAS 2.92* |

* = Reached statistical significance ($P < 0/05$).

dilated vessels, synovial hypertrophy, fibrosis or chronic hypertrophy occasionally progressing to calcification or transformation to fibrocartilaginous tissue (21,39,43). Fibrosis and vascular neoformation has been shown to be present in the majority of patients undergoing total knee replacement, with chronic inflammatory changes present in over a third (42), perhaps reflecting the complex involvement of the fatpad in the presence of intra articular pathology (13).

RESULTS OF CURRENT STUDIES

There are currently five observational studies without control (level 4 evidence) describing treatment of infrapatellar impingement in the recent literature, the findings of these are summarised in table I.

The disparity of data presented from the above studies renders aggregation difficult, however Magi *et al* (43) and Duri *et al* (21) both reported objective improvement in their cohorts post treatment.

Maculé *et al* (42) carried out a randomised prospective trial to investigate the effect of fatpad re-

section on pain after total knee replacement. They found statistically significantly more patients were pain free at 6 months if the fat pad was removed opposed to retained.

CONCLUSION

Despite being a 110 year old condition the mechanisms involved in pathogenesis of pain from the infrapatellar fat pad are still largely misunderstood. What is clear from current literature is the complexity of the structure and its assessment. If infrapatellar impingement is diagnosed correctly, treatment from resection will see improvement in symptoms of pain and function. Further prospective, controlled trials are warranted in the assessment of the roles of surgery, chemical ablation and steroid injections. The indications and value for ultrasonographic dynamic assessment is still in the infancy of widespread clinical adoption, but is a technique we have found to be of great assistance and continue to develop.

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