Running Injuries

The Infrapatellar Fat Pad and Plica Injuries

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KEYWORDS

• Hoffa pad • Nociceptive • OA pain • Knee biomechanics • Quadriceps inhibition

KEY POINTS

- The infrapatellar fat pad (IFP) is highly innervated and when inflamed is responsible for inferior, medial, retropatellar, and in some cases posterior knee pain.
- The IFP stabilizes the patella in extremes of knee motion (<20° and >100°).
- The IFP can be injured with rapid extension or hyperextension of the knee, such as overextending the knee when running downhill.
- Inflammation of the IFP causes quadriceps atrophy.
- Inflammatory changes in the IFP seen on MRI are the consequence of trauma and degeneration, the commonest trauma being arthroscopy.

INTRODUCTION

Various intraarticular structures of the knee generate neurosensory signals that result in conscious pain perception. Pain is defined as an unpleasant sensory or emotional experience associated with actual or potential tissue damage (nociception).[1] Pain involves an individual's reaction to nociception, so it is very much a personal experience with a learned component. Pain can become memorized because pain mechanisms are not fixed (hard wired) but are plastic (soft wired).[1] Through neuroplasticity, hyperalgesia can be learned and unlearned, from both tissue-based and environmental afferent inputs.[2]

The tissue-based structures that can be the potential source of knee pain are the synovium, lateral retinaculum, subchondral bone, and the infrapatellar fat pad (IFP), with the articular cartilage because it is aneural, providing only an indirect source, perhaps either through synovial irritation or increasing subchondral bone stress.

There is, however, no correlation between the amount of articular cartilage degeneration and pain experienced by individuals with knee osteoarthritis (OA), for example.

Disclosures: None.

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Phys Med Rehabil Clin N Am 27 (2016) 79–89
http://dx.doi.org/10.1016/j.pmr.2015.08.009 **pmr.theclinics.com**
1047-9651/16/$ – see front matter

The severity of OA knee pain is associated with bone marrow lesions (edema) with subarticular bone attrition,[3–5] synovitis/effusion, and degenerative meniscal tears, but it is not associated with presence of osteophytes or reduction in joint space.[6] Hill and colleagues[7] followed 270 subjects with tibiofemoral and patellofemoral (PF) OA for 30 months finding no correlation between baseline synovitis and baseline pain score but a decrease in synovitis at follow-up was correlated with a reduction in pain. These investigators found synovitis in 3 locations, namely, the superior, medial, and inferior patella, with infrapatellar synovitis being most strongly correlated with pain severity. The synovitis was not associated with cartilage loss in either the tibiofemoral or PF compartments.[7]

Free nerve endings (IVa) are present in the synovium,[8] so peripatellar synovitis is a possible source of knee pain. Despite the evidence supporting the synovium as a potential pain source, histologic changes in the synovium of patients with PF pain are only moderate.[9] However, there is evidence of histologic changes in the lateral retinaculum with increased numbers of myelinated and unmyelinated nerve fibers, neuroma formation, and nerve fibrosis being found in some patients with PF pain.[10–12] Additionally, increased intraosseous pressure of the patella has been found in patients with PF pain who complain of pain when sitting with a bent knee ("movie goers knee"), possibly secondary to a transient venous outflow obstruction.[10,13] However, the structure that until recently has largely been ignored by the orthopedic community, even though it was first identified as a potent source of pain by Hoffa in 1904, is the (IFP). Hoffa described the symptoms originating from the IFP as being "pain felt quite suddenly on the medial side of the joint; with the patient having difficulty bending and straightening the knee and the presence of knee joint swelling on both sides of the patella."[14] Consequently, the IFP is often referred to as Hoffa's fat pad, with most clinicians thinking of Hoffa's syndrome as a result of a direct blow to the knee. Superolateral fat pad edema is a frequent finding with patellar maltracking and may precede clinically significant chondrosis.[15] In a recent study by Matcuk and Cen,[15] a patient with patellar maltracking was placed in the Hoffa group (superolateral fat pad edema), if 1 of 3 conditions was met: lateral patellar displacement greater than -3.6 mm and Insall–Salvati ratio (ratio of patellar tendon length and patellar length) greater than 0.99; lateral patellar displacement of -3.6 mm or less and Insall–Salvati ratio greater than 1.23; or lateral patellar displacement of -3.6 mm or less, Insall–Salvati ratio of 1.23 or less, and lateral trochlear inclination of 16.5° or less. These findings had 91.6% sensitivity and 88.9% specificity for identifying the Hoffa group.

ANATOMY

The IFP is a highly vascular, richly innervated, intracapsular, extrasynovial structure, lined by synovium, filling the anterior knee compartment with between 21 to 39 mL of adipose tissue, although there is considerable individual volumetric variation.[16–18] The IFP covers the extraarticular part of the posterior patellar surface and merges superiorly with the peripatellar fold. Posteriorly, the IFP extends into ligamentum mucosum, which in many individuals is continuous with the anterior cruciate ligament (ACL), finally connecting to the intercondylar notch of the femur.[16,17] Inflammation of the pericruciate portion of the IFP causes posterior knee pain in athletes.[19]

The IFP also attaches to the proximal patellar tendon, inferior pole of the patella, transverse meniscal ligament, medial and lateral meniscal horns, and the retinaculum, as well as the periosteum of the tibia. The medial and lateral patellomeniscal ligaments