Thigh Region Injuries

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This resource will cover the following 3 thigh region injuries separately:

1. Rectus Femoris Strain
2. Proximal Hamstring Tendinopathy
3. Sciatic Nerve Compression (Piriformis Syndrome)

Rectus Femoris Strain

Anatomy

Rectus Femoris (RF) is the most superficial of the quadriceps muscles.[1,2] It is situated in the middle of the front of the thigh and is fusiform in shape. It originates from two heads, the direct (also known as anterior or straight) and indirect (also known as posterior or reflected). The direct head arises from the anterior aspect of the inferior iliac spine and is superficial, whilst the indirect arises from the acetabular ridge and is a deep intramuscular tendon. Its long straight fibers conjoin with the three other vastus muscles to insert into the patella via the quadriceps tendon.[3] (Figure 1) Rectus femoris is the only muscle of the quadriceps to cross both the hip and knee joints acting as a hip flexor, knee extensor and stabiliser of the pelvis in weight-bearing.[4,5]

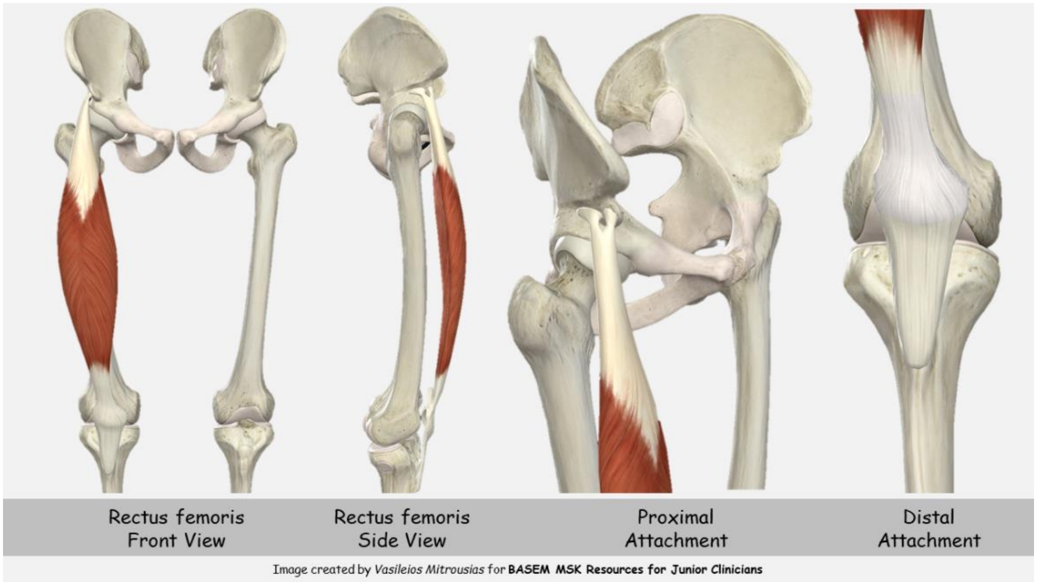

Figure 1.

Aetiology

RF strains frequently occur in sports that require repetitive kicking, sprinting and jumping. It is these actions that directly cause RF strains, although it is not known which part of the action causes injury. In general, muscle strain injuries often occur during eccentric muscle action.[5]

Previous quadriceps injury is the only proven intrinsic risk factor for RF strain, other risk factors may include leg dominance, flexibility and strength. A dry field/pitch is the only recognised extrinsic risk factor for injury.[5]

Grading

Multiple muscle strain classification systems exist. One of the oldest is the one advised by O'Donoghue in 1962[6]. This system classifies strains based on the amount of tissue damage and functional loss in 3 grades: Grade I – No appreciable tissue tear, Grade II – Moderate tissue damage and reduced strength, Grade III – Complete tear and complete loss of function.

One of the more recent and clinically relevant is the British Athletics muscle injury classification system. MRI features are used to grade injuries 0-4, grades 1–4 include a suffix 'a', 'b' or 'c' if the injury is 'myofascial', 'musculo-tendinous' or 'intra-tendinous'. Higher number and letters signify a worse prognosis.[7]

Lastly the system introduced by Valle et al. in 2016 is also widely used. This evidence-informed and expert consensus-based classification system for muscle injuries is based on a four-letter initialism system: MLG-R, respectively referring to the mechanism of injury (M), location of injury (L), grading of severity (G), and number of muscle re-injuries (R).[8]

Clinical Features

History	Sudden onset anterior thigh pain during kicking Tearing sensation
Look	Bruising Thigh asymmetry Muscle defect (in higher-grade injuries)
Feel	Tenderness at the site of injury Palpation of a muscle defect
Move	Pain on passive and active stretching Pain on resisted knee extension

RF is the most commonly injured of the quadriceps muscles and contributes to more missed games than hamstring and groin muscle injuries. Re-injury rates are high (up to 17%).[9,10] Proximal and deeper strains can be more difficult to diagnose, often not having the classical signs, imaging is therefore often useful.[4]

Investigations

Plain radiographs are useful only to exclude avulsion injuries in the skeletally immature player. MRI is used to locate and grade a tear. MRI features may include a high-intensity signal either focally or diffusely at the myotendinous junction on fluid-sensitive images, a feathery appearance of the muscle on all pulse sequences consistent with interstitial haemorrhage, a 'bull's-eye' sign indicative of intrasubstance tendon tears, a longitudinal scar, retraction, pseudocyst or hematoma.[5, 11] Ultrasound is a dynamic but operator-dependent study which may contribute to diagnosis and monitoring of rectus femoris strains. Alterations in echogenicity, discontinuation of muscle fibres, hypervascularisation at the disruption site and hematomas can be found. Imaging (MRI & USS) may help indicate return to play (RTP) time.[11-13]

Management Options

General acute management is with protection, haemorrhage control, ice, compression and elevation in the acute phase, with introduction of early mobilisation to preserve range of movement. Crutches can be used acutely for partial weight bearing in high grade injuries.[13,14] The aim is to prevent muscle atrophy and loss of range of movement.[4]

In general, conservative management is in favour as the RTP time is described to be anywhere from 3 to 10 months after surgical repair, compared to 6 to 12 weeks with conservative treatment.[15-18] Surgical management is appropriate in complete ruptures and complex proximal tears and can have lower rates of injury recurrence.[4,19] In proximal avulsion injuries primary tenodesis has been associated with reduced time for return to preinjury level of sporting function and decreased risk of injury recurrence.[20]

Take Home Message:

Longer length tears, injuries involving the central aponeurosis, more proximal injuries and tears at the musculotendinous junction have a worse prognosis and longer time to RTP. [13, 21]

Return To Play

Players should have normal range of movement, relatively symmetrical strength to the non-injured leg and have undertaken full training in a graduated way prior to RTP. Graduated return is usually done in a stepwise fashion:

1. Restoration of pain free ROM
2. Functional rehabilitation
3. Graduated return to sport[4]

Prevention

There is currently no evidence-based prevention programme for RF strains. Prevention strategies for RF muscle injuries include general flexibility of muscles of the thigh and leg, adequate balance of concentric and eccentric strength of the hip flexors and knee extensors and adequate core stability. The FIFA 11+ programme has been shown to have a small but not statistically significant effect on thigh injury prevention.[10] Exercises based on deceleration under specific sport situations should be included in prevention programmes for RF muscle injuries.[5] It is important to undertake prevention programmes due to high rate of re-injury.[4]

Proximal Hamstring Tendinopathy

Anatomy

The origin of the three hamstring muscles is complex. The hamstrings consist of the semimembranosus (SM), the semitendinosus (ST) and the biceps femoris (BF) muscles. Only the long head of the biceps femoris belongs to hamstrings, since the short head arises lower, from the lateral lip of the Linea aspera. Hamstrings arise from the ischial tuberosity, ST and long head of the BF form a conjoined tendon and SM forms a more lateral tendon (Figure 1). The ischiogluteal bursa sits between the tendon and the ischial tuberosity. The three muscles cross two joints, the hip and knee and are therefore exposed to large eccentric forces. Due to their upper posterior insertion, the hamstring tendons are folded around the ischial tuberosity during hip flexion, resulting in compression of the tendons.[1,2]

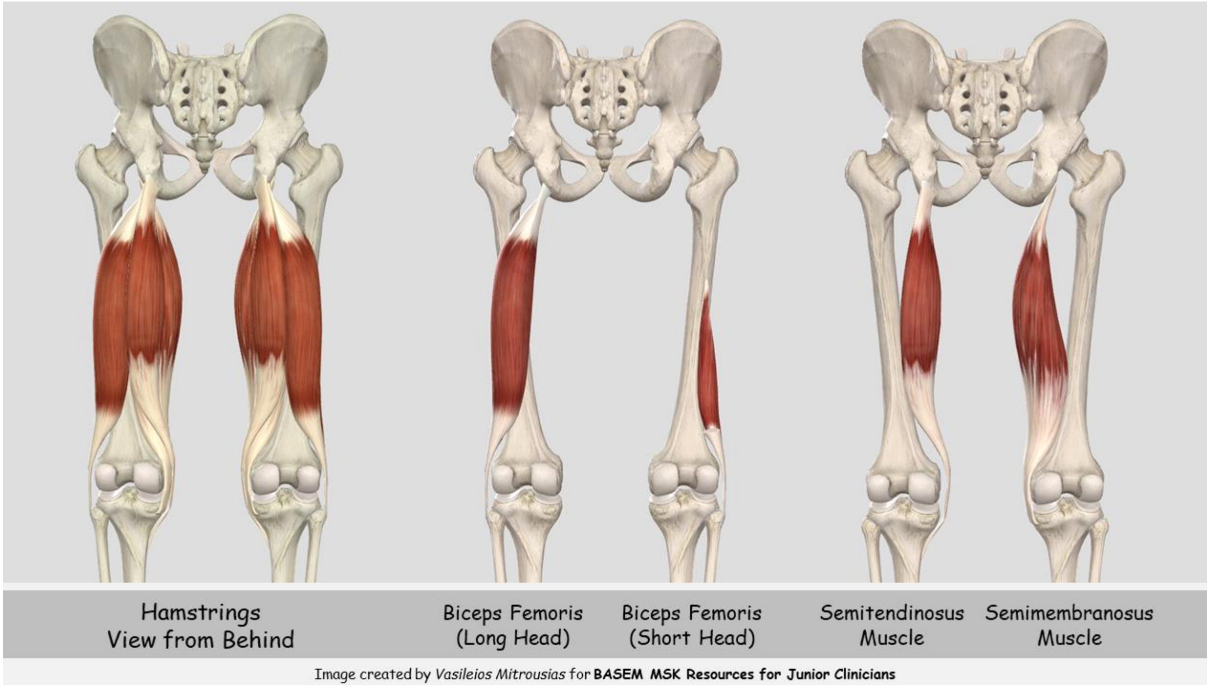

Figure 1.

Aetiology

Proximal Hamstring Tendinopathy (PHT) is a classic overuse injury, typical in endurance athletes or activities involving repeated jumping or hip flexion.[2] These actions increase tensile and compressive forces which can be contributors to the aetiology.[3] Sudden increases in training volume or intensity are common factors in aetiology, special attention should be paid to activities using greater degrees of hip flexion (e.g., hill sprints, deadlifts or yoga). PHT may affect one or a combination of the hamstring tendons.[4]

Clinical Features

The main symptom is pain in the region of ischial tuberosity, usually felt around posterior thigh and lower gluteal region which may radiate distally but rarely below the knee. History includes the usual clinical picture of tendinopathy: mild pain aggravated while starting exercise, resolved or reduced after warm-up and often returning or worsening for days, even during activities of daily living such as walking upstairs or prolonged sitting.[5]

Manual compression of the proximal hamstrings' insertion at the ischial tuberosity usually provokes symptoms. The Puranen–Orava (standing active hamstring stretch), the bent-knee stretch and modified bent-knee stretch tests have all been validated and can confirm diagnosis alongside a thorough history.[6]

Investigations

Imaging in the form of USS or MRI has limited value in proximal hamstrings tendinopathy. Ultrasound is far more challenging and less reliable than in other, superficial tendons (e.g. the Achilles tendon). Regarding MRI, the high prevalence of pathological findings in asymptomatic individuals (which increases along with age) is also discouraging for the use of this modality and its prognostic value. For these reasons, imaging should me mainly used to rule out other pathology or when symptomatology is not typical. [7-9]

Management Options

The treatment approach is almost always conservative. Research into rehabilitation is limited to case studies, mainly in runners. Rehabilitation of PHT follows management of other tendinopathies. Reduce pain by managing load and avoid provoking positions of increased hip flexion.

> ***Take Home Message:***
>
> Progressive loading is the key to rehabilitation of any tendinopathy.[10]

Progressive rehabilitation initially includes isometric contractions (e.g. single leg bridge), then eccentric exercises (e.g gym ball hamstring curls) progressed to weight bearing eccentric exercises (e.g. reverse dead lifts). Increases in hip flexion should be progressive too. Finally, prior to RTP plyometric and high-speed exercises should be performed.[2]

Prevention

Without any specific PHT prevention evidence, prevention of general hamstring injuries can be applied to PHT although not specific. The Nordic hamstring exercise protocol has been shown to have significant risk reduction and improve RTP for all hamstring injuries. Hamstring lengthening exercises may reduce re-injury risk.[11]

Sciatic Nerve Compression (Piriformis Syndrome)

Anatomy

The sciatic nerve originates from the lumbosacral plexus and specifically from the L4, L5 and S1, S2, S3 nerve roots. It is the widest and longest single nerve in the human body. The nerve travels from its origin through the greater sciatic foramen to the posterior compartment of the thigh before it branches at the popliteal fossa (Figure 1). It directly innervates the muscles of the posterior thigh (biceps femoris, semimembranosus and semitendinosus) and the hamstring portion of the adductor magnus and indirectly, via its terminal branches, all the muscles of the leg and foot. It has no direct sensory functions, however, it indirectly innervates the skin of the lateral leg, heel, and both the dorsal and plantar surfaces of the foot via its terminal branches.[1]

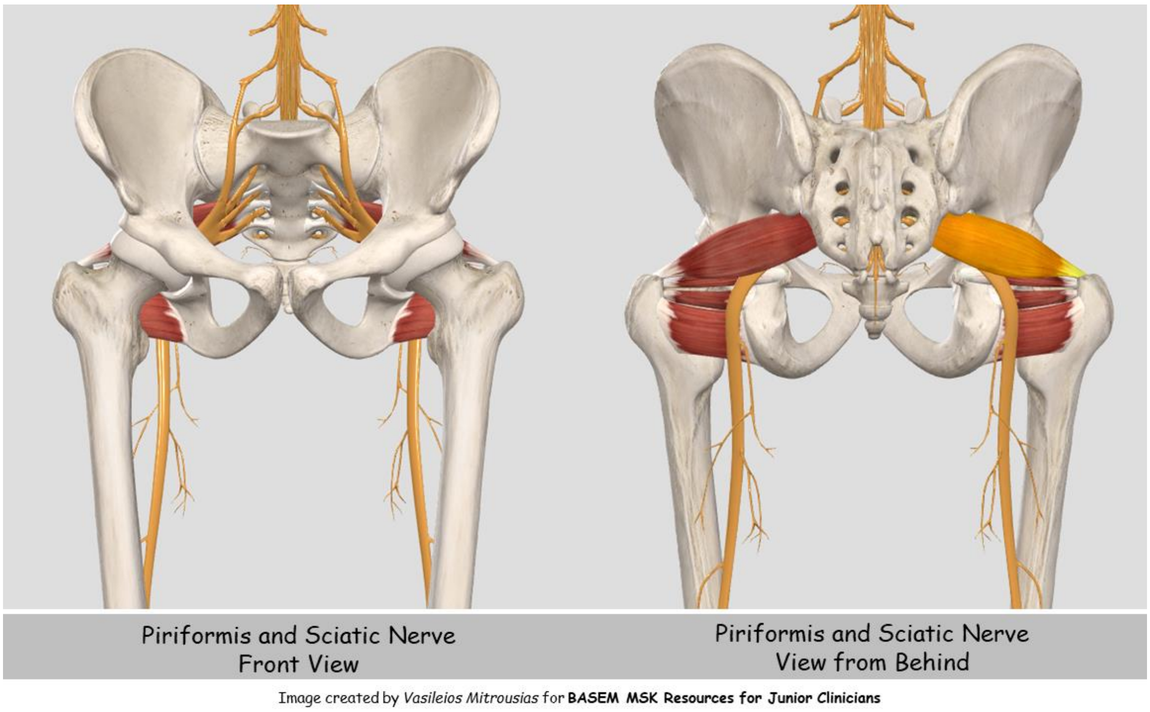

Figure 1

Piriformis, often described as a pear-shaped muscle, originates from the lateral process of the sacrum and inserts into the superior border of the greater trochanter (Figure 2). Its actions are to externally rotate the hip and abduct the hip in a position of hip flexion.[2]

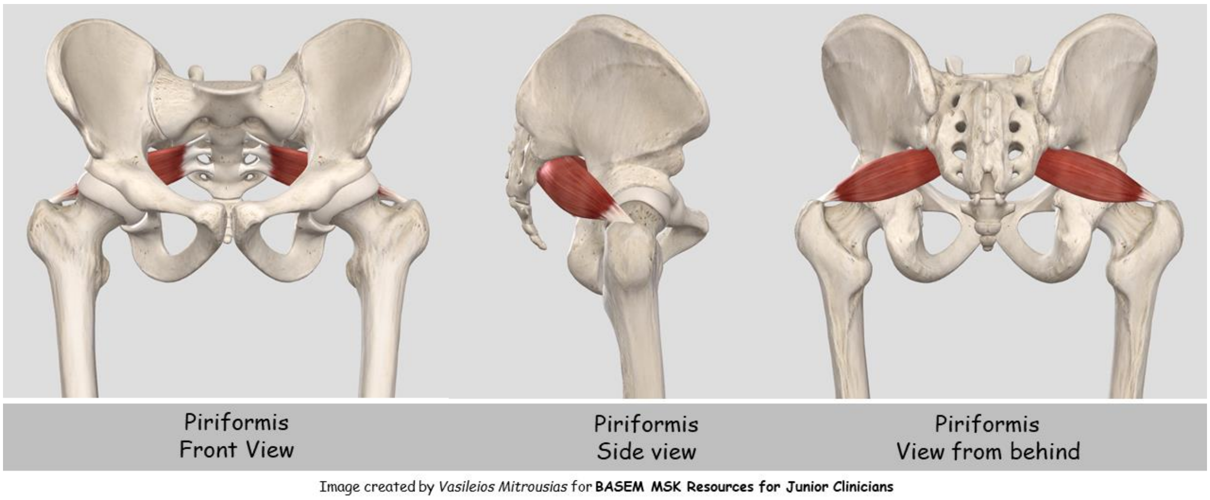

Figure 2

Most commonly (80-90% of population) the undivided sciatic nerve passes inferiorly to piriformis (Figure 1). In the remaining population, the sciatic nerve can divide more proximally and/or can pass through or superiorly to piriformis.[1] Variations of the insertion of the piriformis tendon to the greater trochanter have been also described, including fusion of the piriformis tendon with the superior gemellus, obturator internus and/or gluteus medius.[3]

Aetiology

Any compression or irritation of the sciatic nerve along its course can cause symptoms, commonly referred to as sciatica. The variations in the anatomy of the sciatic nerve in relation to piriformis or the variations of the piriformis insertions could contribute to causing piriformis syndrome, by the variant anatomy alone or hypertrophy or overuse of the muscle. Rarer causes of sciatic nerve compression include haematoma, osteophytes, or pseudo-aneurysms.[4]

Clinical Features

Regarding patients' history, deep pain felt in the buttock area or neuropathic pain along the distribution of the sciatic nerve are the most common symptoms. A positive history of trauma to the muscle during a fall or an abnormal stetch during athletic activity may be present. Pain is often exacerbated by sitting or prolonged activity. It may be more frequent in sports such as cycling, skiing and dance, due to excessive hip flexion, adduction and internal rotation, actions which stress piriformis. Female patients may also complain of dyspareunia.[5,6]

In physical examination, tenderness during palpation of the piriformis muscle and pain on palpation of the greater sciatic notch are the most common findings. A sausage-like mass or fibrous bands over the piriformis can be also felt in some patients.[5]

Many tests for piriformis syndrome, that are not specific as they also stress other structures, have been described in the literature. There are tests which passively stretch the piriformis muscles such as the Freiberg Maneuver and the FAIR test (Flexion, adduction and internal rotation of the hip) or produce active contraction of the piriformis such as the Pace test, the Beatty test or the active piriformis test.[5,7] Finally there are tests designed to stretch the irritated sciatic nerve such as the piriformis seated test or the straight leg raise test (also known as Lasègue sign). In all these tests, a positive result is indicated by the reproduction of the patient's typical gluteal pain or paraesthesia.[5]

Other causes of similar symptoms

Other causes of similar symptoms include lumbar spine and sacroiliac joint pathology, hip-joint mediated pain, greater trochanteric pain syndrome and proximal hamstring tendinopathy which should be excluded through history and examination. Since alternative compressive aetiologies other than the piriformis muscle have been described in the literature (such as a stiff obturator internus muscle) the term "deep gluteal syndrome" has been suggested to describe this muscle and nerve-mediated pain.[5]

Investigations

Imaging is often useful to exclude the rare causes of sciatic nerve compression and determine anatomy involving the muscle and nerve. Computed tomography (CT) can be used to exclude structural compression causes such as a tumor or an abscess and MRI to assess the lumbar spine, the hip joint, the size-symmetry of the piriformis and the sciatic nerve. In addition, ultrasound may be helpful for a more accurate assessment of the painful muscle using sonopalpation. Finally, electromyography, although usually normal, may help since it can reveal conduction slowing or decreased amplitude of sensory nerve action potentials, which have been shown to correlate with the duration of the syndrome.[5,8,9]

Management Options

Physical therapy is the mainstay of treatment. Initially aimed at relieving symptoms by soft tissue therapy (massage and stretching) and activity modification. Non-steroidal anti-inflammatory drugs and muscle relaxants can help. Prevention centres on strengthening of piriformis and other external rotators of the hip.[5,6]

In refractory cases local anaesthetic (with or without corticosteroid) or botulinum toxin injection into piriformis can both treat and be diagnostic.[5,8] High accuracy (up to 95%) can be achieved using ultrasound guidance.[10] Lidocaine injection has been proven to reduce symptoms, however, the additional use of the corticosteroid (such as betamethasone) should be carefully considered given the muscular atrophy and other side effects associated with intramuscular glucocorticoid injections.[11] Lastly, botulinum toxin has been found to be superior to a combination of lidocaine and steroid as well as normal saline placebo for pain relief in patients with piriformis syndrome, however its high cost should be kept in mind.[12]

References: Rectus Femoris Strain

1. Murdock CJ, Mudreac A, Agyeman K. Anatomy, Abdomen and Pelvis, Rectus Femoris Muscle. Treasure Island (FL): StatPearls Publishing, 2020.
2. Ryan JM, Harris JD, Graham WC, Virk SS, Ellis TJ. Origin of the direct and reflected head of the rectus femoris: an anatomic study. Arthroscopy. 2014;30(7):796-802.
3. Hasselman CT, Best TM, Hughes C 4th, Martinez S, Garrett WE Jr. An explanation for various rectus femoris strain injuries using previously undescribed muscle architecture. Am J Sports Med.1995;23(4):493-499.
4. Brukner P, Khan K. Brukner & Khan's clinical sports medicine: injuries (5th edition). McGraw-Hill Education (Australia), North Ryde, N.S.W, 2016.
5. Mendiguchia J, Alentorn-Geli E, Idoate F, et al. Rectus femoris muscle injuries in football: a clinically relevant review of mechanisms of injury, risk factors and preventive strategies. Br J Sports Med. 2013;47:359-366.
6. O'Donoghue DO. Treatment of injuries to athletes. Philadelphia: WB Saunders, 1962.
7. Pollock N, James SLJ, Lee JC, et al. British athletics muscle injury classification: a new grading system. Br J Sports Med. 2014;48:1347–1351.
8. Valle X, Alentorn-Geli E, Tol JL et al. Muscle injuries in sports: A new evidence-informed and expert consensus-based classification with clinical application. Sports Med. 2017;47:1241–1253.
9. Ekstrand J, Hägglund M, Walden M. Epidemiology of muscle injuries in professional football (soccer). Am J Sports Med. 2011;39:1226–32.
10. Ishøi L, Krommes K, Husted RS, et al. Diagnosis, prevention and treatment of common lower extremity muscle injuries in sport – grading the evidence: a statement paper commissioned by the Danish Society of Sports Physical Therapy (DSSF). Br J Sports Med. 2020;54:528-537.
11. Balius R, Maestro A, Pedret C, et al. Central aponeurosis tears of the rectus femoris: practical sonographic prognosis. Br J Sports Med. 2009;43:818-824.
12. Gyftopoulos S, Rosenberg ZS, Schweitzer ME, Bordalo-Rodrigues M. Normal anatomy and strains of the deep musculotendinous junction of the proximal rectus femoris: MRI features. Am J Roentgenol. 2008;190(3):W182-W186.
13. Hayashi D, Hamilton B, Guermazi A, de Villiers R, Crema MD, Roemer FW. Traumatic injuries of thigh and calf muscles in athletes: role and clinical relevance of MR imaging and ultrasound. Insights Imaging. 2012;3(6):591-601.
14. Orchard JW, Best TM, Mueller-Wohlfahrt H, et al. The early management of muscle strains in the elite athlete: best practice in a world with a limited evidence basis. Br J Sports Med. 2008;42:158-159.
15. García VV, Duhrkop DC, Seijas R, Ares O, Cugat R. Surgical treatment of proximal ruptures of the rectus femoris in professional soccer players. Arch Orthop Trauma Surg. 2012;132:329-333.
16. Bottoni CR, D'Alleyrand JC. Operative treatment of a complete rupture of the origination of the rectus femoris. Sports Health 2009;1:478-480.
17. Irmola T, Heikkilä JT, Orava S, Sarimo J. Total proximal tendon avulsion of the rectus femoris muscle. Scand J Med Sci Sports 2007;17:378-382.
18. Gamradt SC, Brophy RH, Barnes R, Warren RF, Thomas Byrd JW, Kelly BT. Nonoperative treatment for proximal avulsion of the rectus femoris in professional American football. Am J Sports Med 2009;37:1370-1374.
19. García VV, Duhrkop DC, Seijas R, et al. Surgical treatment of proximal ruptures of the rectus femoris in professional soccer players. Arch Orthop Trauma Surg. 2012;132:329–333.
20. Kayani B, Singh S, Chang JS et al. Outcomes of Surgical Repair Versus Primary Tenodesis for Proximal Rectus Femoris Avulsion Injuries in Professional Athletes. Am J Sports Med. 2021;49(1):121-129.
21. Cross TM, Gibbs N, Houang MT, Cameron M. Acute Quadriceps Muscle Strains: Magnetic Resonance Imaging Features and Prognosis. Am J Sports Med 2004;32:710-719.

References: Proximal Hamstring Tendinopathy

1. Koulouris G, Connell D. Hamstring Muscle Complex: An Imaging Review. Radiographics. 2005;25(3):571-586.
2. Brukner P, Khan K. Brukner & Khan's clinical sports medicine: injuries (5th edition). McGraw-Hill Education (Australia). North Ryde, N.S.W. 2016.Cook J, Purdam C. Is compressive load a factor in the development of tendinopathy? Br J Sports Med. 2012;46:163-168.
3. Benazzo, F, Marullo M, Zanon G. et al. Surgical management of chronic proximal hamstring tendinopathy in athletes: a 2 to 11 years of follow-up. J Orthop Traumatol. 2013;14:83–89.
4. Cacchio A, De Paulis F, Maffulli N. Development and validation of a new visa questionnaire (VISA-H) for patients with proximal hamstring tendinopathy. Br J Sports Med. 2014;48:448-452.
5. Cacchio A, Borra F, Severini G. et al. Reliability and validity of three pain provocation tests used for the diagnosis of chronic proximal hamstring tendinopathy. Br J Sports Med. 2012;46:883-887.
6. Van der Made AD, Tol JL, Reurink G, Peters RW, Kerkhoffs GM. Potential hamstring injury blind spot: we need to raise awareness of proximal hamstring tendon avulsion injuries. Br J Sports Med. 2019;53(7):390-2.
7. Thompson SM, Fung S, Wood DG. The prevalence of proximal hamstring pathology on MRI in the asymptomatic population. Knee Surg Sports Traumatol Arthrosc. 2017;25(1):108-11.
8. Bowden DJ, Byrne CA, Alkhayat A, Kavanagh EC, Eustace SJ. Differing MRI appearances of symptomatic proximal hamstring tendinopathy with ageing: a comparison of appearances in patients below and above 45 years. Clin Radiol. 2018;73(11):922-7.
9. Malliaras P, Barton CJ, Reeves ND. et al. Achilles and patellar tendinopathy loading programmes: a systematic review comparing clinical outcomes and identifying potential mechanisms for effectiveness. Sports Med. 2013;43(4):267–86.
10. Ishoi L, Krommes K, Husted RS, et al. Diagnosis, prevention and treatment of common lower extremity muscle injuries in sport – grading the evidence: a statement paper commissioned by the Danish Society of Sports Physical Therapy (DSSF). Br J Sports Med. 2020;54:528-537.

References: Sciatic Nerve Compression

1. Varenika V, Lutz AM, Beaulieu CF, et al. Detection and prevalence of variant sciatic nerve anatomy in relation to the piriformis muscle on MRI. Skeletal Radiol. 2017;46:751–757.
2. Solomon LB, Lee YC, Callary SA, Beck M, Howie DW. Anatomy of piriformis, obturator internus and obturator externus: implications for the posterior surgical approach to the hip. **J Bone Joint Surg Br**. 2010;92(9):1317-1324.
3. Windisch G, Braun EM, Anderhuber F. Piriformis muscle: clinical anatomy and consideration of the piriformis syndrome. Surg Radiol Anat. 2007;29(1):37-45.
4. Hopayian K, Song F, Riera R, Sambandan S. The clinical features of the piriformis syndrome: a systematic review. Eur Spine J. 2010;19(12):2095-2109.
5. Probst D, Stout A and Hunt D. Piriformis Syndrome: A Narrative Review of the Anatomy, Diagnosis, and Treatment. PMR. 2019;11(1):54-63.
6. Brukner P, Khan K. Brukner & Khan's clinical sports medicine: injuries (5th edition). McGraw-Hill Education (Australia). North Ryde, N.S.W. 2016.Cook J, Purdam C. Is compressive load a factor in the development of tendinopathy? Br J Sports Med. 2012;46:163-168.
7. Beatty RA. The piriformis muscle syndrome: a simple diagnostic maneuver. Neurosurgery. 1994;34(3):512-514.
8. Kirschner JS, Foye PM, Cole JL. Piriformis syndrome, diagnosis and treatment. Muscle Nerve. 2009;40: 10-18.
9. Chang CW, Shieh SF, Li CM, Wu WT, Chang KF. Measurement of motor nerve conduction velocity of the sciatic nerve in patients with piriformis syndrome: a magnetic stimulation study. Arch Phys Med Rehabil. 2006;87(10):1371-1375.
10. Finnoff JT, Hurdle MF, Smith J. Accuracy of ultrasound-guided versus fluoroscopically guided contrast-controlled piriformis injections: a cadaveric study. J Ultrasound Med. 2008;27(8):1157-1163.
11. Misirlioglu TO, Akgun K, Palamar D, Erden MG, Erbilir T. Piriformis syndrome: comparison of the effectiveness of local anesthetic and corticosteroid injections: a double-blinded, randomized controlled study. Pain Physician. 2015;18(2):163-171.
12. Fishman LM, Anderson C, Rosner B. BOTOX and physical therapy in the treatment of piriformis syndrome. Am J Phys Med Rehabil. 2002;81(12):936-942.