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REVIEW ARTICLE

Current concepts in the diagnosis and management of extra-articular hip impingement syndromes

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Abstract
Purpose Extra-articular hip impingement syndromes encompass a group of conditions that have previously been an unrecognised source of pain in the hip and on occasion been associated with intra-articular hip impingement as well. As arthroscopic techniques for the hip continue to evolve, the importance of these conditions has been recognised recently and now form an important part of the differential of an individual presenting with hip pain. The aim of this article, therefore, is to provide the reader with an evidence-based and comprehensive update of these syndromes.
Methods By reviewing past literature, the anatomy, pathophysiology, clinical features and the management of the five common extra-articular hip impingement syndromes were described.
Results The common extra-articular impingement syndromes are: 1) Ischiofemoral impingement: quadratus femoris muscle becomes compressed between the lesser trochanter and the ischial tuberosity. 2) Subspine impingement: mechanical conflict occurs between an enlarged or malorientated anterior inferior iliac spine and the distal anterior femoral neck. 3) Iliopsoas impingement: mechanical conflict occurs between the iliopsoas muscle and the labrum, resulting in distinct anterior labral pathology. 4) Deep gluteal syndrome: pain occurs in the buttock due to the entrapment of the sciatic nerve in the deep gluteal space. 5) Pectineofoveal impingement: pain occurs when the medial synovial fold impinges against overlying soft tissue, primarily the zona orbicularis. Knowledge for these syndromes still remains limited for reasons mostly relating to their low prevalence and their co-existence with typical femoro-acetabular impingement.
Conclusions The knowledge of extra-articular hip impingement syndromes is essential and should form a part of the differential diagnoses alongside intra-articular pathology including femoro-acetabular impingement particularly in the younger patient with a non-arthritic hip.

Keywords Arthroscopy · Deep gluteal syndrome · Extra-articular impingement · Hip pain · Iliopsoas impingement · Ischiofemoral impingement · Subspine impingement · Pectineofoveal impingement

Introduction

Femoro-acetabular impingement (FAI) is a well-described cause of hip pain and restriction of movement in the young, non-arthritic patient [1]. Growing experience with hip arthroscopy and improved understanding of FAI has led to the recognition of many previously undiagnosed extra-articular hip impingement syndromes as well. These include ischiofemoral impingement, subspine impingement, iliopsoas impingement and pectineofoveal impingement. Although rare, these syndromes form a very important part of the differential when dealing with young adults presenting with hip pain. However, unfamiliarity with the anatomy and the rarity of these conditions makes diagnosis and differentiation amongst these pathologies more difficult.

The aim of this descriptive non-systematic review article, therefore, is to provide the reader with an evidence-based and

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comprehensive update on the anatomy, pathophysiology, clinical features and the management of each of these impingement syndromes abstracted from relevant studies.

Ischiofemoral impingement

Anatomical considerations

Ischiofemoral impingement (IFI) was first described in 1977 as a cause of hip pain [2] following total hip replacement (THR). The basic pathology is that the space between the lesser trochanter and ischial tuberosity, known as the ischiofemoral space (IFS) is reduced and this leads to compression of the quadratus femoris muscle within the space causing pain. The quadratus femoris muscle originates from the external border of the ischial tuberosity and inserts onto the upper part of the linea quadrata of the proximal femur and is at risk of compression when the IFS is reduced. With the insertion of psoas on the lesser trochanter and the hamstrings on the ischial tuberosity, bursitis of these tendons may also coexist, leading to or resulting from IFI. An increased inclination angle of the femoral neck as in coxa valga and an increased proximal hamstring area may lead to a decrease in or narrowing of the ischiofemoral space [3] leading to IFI. Bredella [4] et al have reported that patients with ischiofemoral impingement have increased ischial and femoral neck angles compared with controls by assessing MRIs. We have recently reported on the normal ischiofemoral distance (IFD) (measured as the smallest distance between the lateral cortex of ischial tuberosity and the medial cortex of the lesser trochanter) and its variations using the CT data of 298 normal hips and found that the mean IFD was 18.6 mm in females and 23 mm in males and that the IFD increased by 1.06 mm for each 1 mm of offset and dropped by 0.09 mm with each year of age [5].

Aetiology and clinical features

The aetiology for IFI is variable. A detailed description of the causes for IFI is provided in Table 1. Presentation tends to be at an older age in comparison with other types of impingement with an average age of 51-53 years. Twenty-five to 40 per cent of the patients may have bilateral impingement [3, 13]. It is more common in females, perhaps due to the pelvis being wider and shallower along with the ischial tuberosity tending to be wider as well. Commonly pain presents in the groin and/or buttock and may radiate down to the medial thigh and to the knee. Pain is usually brought on by extension, adduction and external rotation of the hip. There may also be snapping or locking associated with the pain [3, 7, 8, 13]. Symptoms mimicking sciatica may also be present as the sciatic nerve could be irritated. Clinical examination reveals that walking with a long stride causes pain and the patient may grab the hip, lateral to the ischium. The pain is then alleviated with a shorter stride. Palpation in the ischiofemoral space, just lateral to the ischium, may also reveal tenderness in the region. The ischiofemoral impingement test involves passively extending the leg with the hip in adducted or neutral position as the patient is in a contralateral decubitus position. It is positive if it reproduces the patient's symptoms. Extension with the leg abducted should not reproduce the patient's symptoms as this increases the ischiofemoral space [14].

Other differential diagnoses include iliopsoas impingement, quadratus femoris tears, chronic hamstring injuries, adductor bursitis, deep gluteal syndrome and sciatica.

Table 1 Causes for ischiofemoral impingement

Aging (muscle atrophy) [6]
Woman gender (increased width of pelvis) [7–10]
Coxa profunda [2]
Coxa valga [11]
Valgus hip due to proximal femoral osteotomy [2]
Legg-Calve-Perthes disease [12]
Total hip replacement with reduced femoral offset or medialized socket [2]
Peritrochanteric fractures with involvement of lesser trochanter [2]
Abductor muscle injury causing uncompensated hip adduction during gait [8, 10]
Multiple or isolated exostoses [6]

Management

The patient should be investigated with plain radiographs of the hip which include antero-posterior weight-bearing and cross-table lateral views. These are usually normal but may demonstrate narrowing between the lesser trochanter and the ischium along with subcortical cysts and sclerosis. MRI is considered to be the gold standard for diagnosis. Torriani [13] describes the abnormal signals around the quadratus femoris muscle and narrowed ischiofemoral space seen on MRI, which correlate well with the diagnosis. The narrowing of the ischiofemoral space and quadratus femoris space which is the smallest space between the superolateral hamstrings and posteromedial iliopsoas tendon is most predictive of ischiofemoral impingement [3]. Other causes for this pathology may also be found with trauma, burns, corticosteroid injections, immobilisation, sciatic neuropathy and spinal cord injuries, which enhances the difficulty in making a diagnosis of impingement. Compared with a quadratus femoris tear where oedema is more concentrated at the musculotendinous junction, in ischiofemoral impingement, the oedema in the quadratus femoris is more diffuse and the muscle fibres are not disrupted [3].

The mainstay of treatment in these patients is non-operative with rest, modification of activities and anti-inflammatories. CT-guided local anaesthetic and steroid injection around the quadratus femoris may also relieve pain [7, 9]. In a high-volume hip arthroscopy practice, only 5% of patients diagnosed with ischiofemoral impingement needed surgical intervention [15]. Open lesser trochanter resection to increase the IFS was originally reported in patients post THR, however, this technique could result in a significant loss of hip flexion in a younger and more active patient without prior hip surgery [2]. Also, it may lead to instability, especially if a posterior surgical approach has been used in THR [16]. A detachment of quadratus femoris has also been suggested, however, since the medial femoral circumflex arteries run over the upper border of quadratus femoris, there is a risk of avascular necrosis of the femoral head with any injury to these vessels [15]. A pre-operative angiogram is recommended to this end, especially when the local anatomy is grossly distorted, as in the presence of exostosis [17]. Little has been published on definitive treatment for this condition thus far, however, there has been reported improvement in pain and range of movement after surgical management in some patients with no complications [15, 18].

Subspine impingement

Anatomical considerations

Subspine impingement occurs when there is abnormal contact between an enlarged or malorientated anterior inferior iliac spine (AIIS) and the distal anterior femoral neck in straight flexion of the hip. The AIIS is located just above the anterosuperior portion of the acetabular rim and the upper portion of the AIIS gives origin to the straight head of the rectus femoris muscle. The rectus femoris is situated in the middle of the front of the thigh, and it arises by two tendons: one from the anterior inferior iliac spine (direct head) and the other from a groove above the rim of the acetabulum (reflected head). The average dimensions of the footprint of the direct head of the rectus femoris are 13.4 (±1.7) mm (medial-lateral) ×26.0 (±4.1) mm (cranial-caudal) [19]. The labrum, the anterior capsule, and rectus femoris can be involved in this form of impingement [20, 21]. Hetsroni [22] evaluated three-dimensional CT reconstructions hips with subspine impingement and defined three morphological AIIS variants: type I where there was a smooth ilium wall between the AIIS and the acetabular rim, type II where the AIIS extended to the level of the rim, and type III where the AIIS extended distally to the acetabular rim. They reported that type II and III variants are associated with a decrease in hip flexion and internal rotation, supporting the rationale for considering AIIS decompression for variants that extend to and below the rim.

Aetiology & clinical features

This condition may occur because of an avulsion injury of the AIIS due to the excessive muscular activity of the rectus femoris. In congenital cases, there is an association with acetabular retroversion, which makes the AIIS rotated more anteriorly and distally [21]. The AIIS may also become more prominent following pelvic osteotomy or injury to the AIIS. In adolescents, repetitive traction injuries can cause apophysitis, hypertrophy, inferior displacement of the apophysis or fracture which can lead to malunion resulting in an enlarged AIIS or a bony protrusion, thereby causing subspine impingement [23].

Patients presenting with subspine impingement are usually young, active and involved in vigorous contact sports and sometimes have a history of prior pelvic osteotomy or injury to the hip flexors [24]. Most of these patients present with anterior hip pain on flexion, internal rotation and adduction and this is sometimes also associated with a grinding sensation [24]. Clinical presentation overlaps with that of FAI [24], but examination may reveal tenderness on the AIIS and flexion may be mechanically restricted at the end-range of straight hip flexion. Otherwise, impingement test is positive and there is a limitation of internal rotation in 90 degrees of flexion. It is important to note that patients presenting with persistent symptoms following surgery for FAI should be assessed for subspine impingement.

Management

Plain radiographs may demonstrate an avulsion fracture of the AIIS but subtle findings can often be missed and should be looked for carefully [25, 26]. Over time, a displaced avulsion fracture may develop into a prominent bony deformity of the AIIS, which can be more accurately visualised at CT or MRI, and is often point shaped [23]. In some cases, calcified deposits are found within the proximal portion of the straight head of the rectus femoris tendon and impingement cysts are found on the distal femoral neck [21]. Three-dimensional CT datasets make pre-operative dynamic simulations (motion analysis) possible to detect a direct osseous impingement between the AIIS deformity and the femoral neck at maximum hip flexion. MR arthrography is also beneficial to rule out damage to the labrum and articular cartilage secondary to ongoing FAI. It is important to note that there may only be partial or no pain relief and a persistent limitation of hip flexion after intraarticular administration of a local anaesthetic [23].

Non-surgical treatment such as activity modification, physiotherapy and therapeutic anaesthetic and steroid injections may be prescribed first but their efficacy has not been closely investigated [18]. Patients who fail conservative treatment, an AIIS decompression is often performed through the standard

anterolateral and mid-anterior hip arthroscopy portals. A concomitant arthroscopic procedure is sometimes conducted to address any intra-articular pathology as well [23]. Arthroscopic resection of an impinging AIIS has also been reported by some experienced hip arthroscopists [21, 23, 25, 27], and studies have shown good short-term outcomes up to two years follow-up for a return to sporting activity, increased ROM, Harris hip score and visual analogue scale [21, 23, 27]. To date, post-surgical rehabilitation has not been objectively studied and its role in the post-operative period is poorly detailed in the literature [18], while two to four weeks of protected weight bearing with crutches and ROM exercises until basic muscle strength is regained are recommended [28]. The post-operative administration of non-steroidal anti-inflammatory agents for prophylaxis against heterotopic ossification for the first three to four weeks after surgery is also advised [23].

Iliopsoas impingement

Anatomical considerations

The iliacus muscle originates from the iliac crest and the inner table of the ilium, and the psoas muscle from the 12th thoracic and all five lumbar vertebrae. These two muscles merge to form one muscle: the iliopsoas muscle, which has a musculotendinous insertion on the lesser trochanter, though some muscle fibres of the iliacus attach directly on the lesser trochanter and proximal femur [29, 30]. The anatomical and functional relationship between the iliopsoas muscle and the hip has been highlighted in previous studies. It is postulated that the impingement is caused by a repetitive traction injury by the iliopsoas tendon that is scarred and adherent to the capsule-labrum complex of the hip or by a tight or inflamed iliopsoas tendon that causes impingement during hip extension [18, 31].

In patients with classical FAI, most labral tears occur at the anterosuperior position, and are caused by osseous abnormalities of the acetabulum and the proximal femur [32, 33]. However, in patients with iliopsoas impingement there is mechanical conflict between the iliopsoas muscle and the labrum, resulting in distinct labral lesions directly anteriorly (3 and 9-o'clock for right and left hips, respectively). It has been shown that the iliopsoas tendon directly overlies the anterior capsulolabral complex at the three-o'clock position for right hips using fresh-frozen cadavers [34]. Another study examining the findings of MR arthrograms has confirmed this finding [35]. In other words, iliopsoas impingement causes a distinct pattern of anterior labral damage that does not extend into the anterosuperior portion of the labrum (e.g., 1 to 2-o'clock position for right hips). It is an emerging diagnosis of anterior hip pain that has been linked to acetabular labral tears [28, 35], and also to impingement on the osteophytic acetabular rim in a patient with degenerative hip arthritis [36].

Aetiology & clinical features

Iliopsoas impingement is more common in young females and individuals involved in regular sporting activities [18, 28, 37]. The average age of the patients is 25–35 years (range, 15–57 years) [28, 35]. The strong female preponderance might be explained by a narrower iliopsoas tendon in females (10.2 mm) compared with females without iliopsoas impingement (11.0 mm) and males with iliopsoas impingement (11.9 mm) [35]. Iliopsoas impingement has also been reported in patients following total hip resurfacing and total hip replacement when a larger femoral head component is used which leads to the abutment of the iliopsoas tendon against the prosthetic femoral head or when the acetabular component is large or anteverted [38–41].

Patients often report of anterior hip pain with active flexion and may also report of a snapping sensation. Clinical examination may reveal non-specific focal tenderness over the iliopsoas tendon at the anterior joint line, positive hip impingement test (Flexion, Adduction, Internal Rotation test), and pain or apprehension with a resisted straight leg raise test [28, 37]. Iliopsoas snapping may be elicted by flexion, abduction and external rotation of the hip and then bringing the leg into extension [42–44].

Management

Patients with iliopsoas impingement usually do not exhibit osseous pathomorphology on their plain radiographs [28, 45]. MR arthrography, however, is often diagnostic [35]. Dynamic ultrasound is also very helpful in making the diagnosis and in some cases treating it as well. It should, however, be stressed that in the absence of established clinical or radiological criteria, the diagnosis of iliopsoas impingement can only be confirmed arthroscopically [46].

Non-surgical intervention such as rehabilitation, activity modification and therapeutic local anaesthetic with steroids may usually be prescribed first, although the efficacy of these interventions have not been investigated [18]. If conservative measures fail, then surgical intervention is indicated. Although open and arthroscopic surgical techniques for the treatment of iliopsoas impingement syndrome by release or lengthening of the iliopsoas tendon have been described [44, 47], hip arthroscopy is currently the most useful instrument in the treatment of the iliopsoas impingement, being less invasive than the open technique. Arthroscopic release of the iliopsoas tendon may be performed in a transcapsular manner in three different anatomic regions: (1) at the level of the labrum, from the central compartment, (2) via the transcapsular approach in the peripheral compartment and

(3) within the iliopsoas bursa on its insertion at the lesser trochanter. Ilizaliturri et al reported that all these three methods produced favourable results for the treatment of iliopsoas impingement syndrome and that no clinical difference was found in terms of the results with the three different approaches [48, 49]. The labral tear corresponding to the iliopsoas impingement can be resected or repaired and a tenotomy carried out for the tendon simultaneously [28, 37]. Management of the labrum (repair or resection) is dictated by the pattern of injury. Studies have reported good short-term outcomes at 1-year postoperatively for a return to sporting activity, restored ROM, decreased symptoms and better Harris hip scores [18, 28, 37, 46]. Full flexion strength at 3 months is expected when care is taken to release only the tendinous portion of the iliopsoas muscle [37, 45].

Deep gluteal syndrome

Anatomical considerations

There has been a significant amount of progress in the understanding of posterior hip anatomy and sciatic nerve kinematics and it is now well recognised that the sciatic nerve can be entrapped at many other locations (in the subgluteal space) rather than just at the piriformis alone. Therefore, the term 'deep gluteal syndrome' is now preferred to describe the presence of pain in the buttock caused from extra-pelvic entrapment of the sciatic nerve instead of the 'piriformis syndrome' [50].

The deep gluteal space is the cellular and fatty tissue located between the middle and deep gluteal aponeurotic layers [51]. Its border is defined by the following structures: superiorly, inferior margin of the sciatic notch; inferiorly, proximal origin of the hamstrings at ischial tuberosity; posteriorly, the gluteus maximus; anteriorly, posterior acetabular column, hip joint capsule and proximal femur; laterally, lateral lip of linea aspera and gluteal tuberosity; medially, sacrotuberous ligament and falciform fascia. The sciatic nerve enters the pelvis at the sciatic notch on the inferior surface, in the majority (83%) [52], to the piriformis muscle and sacrotuberous ligament. There are anatomical variations of the course of the sciatic nerve in relation to the piriformis muscle. There are six variations originally described by Beaton and Anson [53]: a) the sciatic nerve passes along the inferior surface to the piriformis, b) the sciatic nerve divides into two with the peroneal branch piercing through piriformis, c) the sciatic nerve splits with the peroneal branch running over the superior surface and the tibial branch inferior, d) the sciatic nerve pierces through piriformis, and e) the sciatic nerve passes over the superior surface of piriformis. Anomalous variation has been thought to predispose to entrapment of the sciatic nerve, but a recent review by Smoll [52] suggests this is not as significant as originally thought. However, it is important to note anatomical variation during operative management [52]. As the nerve courses distally towards the ischium and hamstring origin, it passes posterior to the gemelli-obturator internus complex and quadratrus femoris and can be entrapped at any location throughout its course.

Aetiology & clinical features

Sciatic nerve kinematics is thought to play a crucial role in understanding the pathophysiology of entrapment. It has been reported that in deep flexion, abduction and external rotation of the hip, the sciatic nerve glides across the posterior border of the greater trochanter. Moreover, in the fully flexed, abducted and externally rotated state, the semimembranosus origin and the posterior edge of the greater trochanter can come into contact. Excursion of the nerve is dependent upon the flexion-extension of the knee. When the knee is flexed, the nerve moves posterolateral and the nerve moves deeply into the tunnel when the knee is extended. During a straight leg raise with knee extension, the sciatic nerve experiences a proximal excursion of 28 mm medial towards the hip joint [54]. Additionally, it has been reported that neurapraxia can be produced at 6% strain and that a complete block can occur at 12% strain [55]. Any abnormality within the sub-gluteal space that disrupts these normal excursions may trigger deep gluteal syndrome [51].

Clinical assessment of patients with deep gluteal syndrome is difficult since the symptoms are not precise and may be confused with other intra or extra-articular hip pathology including pathology emanating from the spine. The most common symptoms include hip or buttock pain and tenderness in the gluteal and retro-trochanteric region and sciatica-like pain. They are often unilateral but sometimes bilateral, and are usually exacerbated with the rotation of the hip in flexion and knee extension. Using the ischial tuberosity as a reference, the production of pain by palpation at the sciatic notch indicates piriformis syndrome. The active piriformis test involves active abduction and external rotation against resistance and pushing the heel into the examination table, in the lateral position while the examiner palpates the piriformis. The seated piriformis stretch test involves passive flexion, adduction with internal rotation of the hip, with the knee extended, performed as the examiner palpates the deep gluteal region with the patient in the seated position. The combination of the seated piriformis stretch test with the piriformis active test has shown a sensitivity of 91% and specificity of 80% for the endoscopic finding of sciatic nerve entrapment [14]. These specific tests are especially helpful when evaluating a patient with posterior hip pain. Deep gluteal syndrome does not have any particular osseous pathomorphology on the plain radiographs. MRI is the imaging modality of choice for assessing this syndrome

and may substantially influence the management of these patients [56]. The diagnostic injection test is also a useful tool, enabling diagnosis and excluding articular pathology or other extra-articular disorders. Guided injections with the patient lying prone, utilising ultrasound, CT or open MRI, have also been reported [57, 58].

Management

In addition to the non-surgical intervention such as rehabilitation or activity modification, imaging guided local anaesthetic and steroids injections have been described to be very helpful, precise and reproducible for discriminating the pathology [59]. In rare cases that fail conservative therapy including physical therapy, analgesic or anti-inflammatory drugs and injections, open or endoscopic surgical treatment may be considered. Several studies have reported good results after open sciatic decompression in patients with deep gluteal syndrome [59–63]. Dezawa first reported the endoscopic decompression of the sciatic nerve entrapment as an effective and minimally invasive approach to six cases of deep gluteal syndrome [64]. Recently, Martin reported a case series of 35 patients presenting with deep gluteal syndrome treated endoscopically [51]. Endoscopy allows for a complete extrapelvic sciatic nerve visualisation and safe nerve decompression in the deep gluteal space, however, a surgical experience and standardised technique for diagnosis and sciatic nerve decompression is mandatory in order to identify the anatomy of the sciatic nerve, avoid iatrogenic injury and to find every potential source of sciatic nerve entrapment [65].

Pectineo-foveal impingement

Anatomical considerations

Pectine-ofoveal impingement is a rare condition, which can cause unexplained hip or groin pain in young adults, and only two articles describing this pathology have been published thus far [66, 67]. The medial synovial fold represents a fibrous band located anteromedially on the femoral neck, and it is consistently visualised during arthroscopy of the peripheral compartment of the hip. Anatomically, it originates from the head–neck junction and inserting distally onto the capsule, crossing the zona orbicularis and iliopsoas tendon. The medial synovial fold is well known for its utility as a landmark during the arthroscopic transcapsular release of the iliopsoas through the peripheral compartment [68, 69]. Pectineo-foveal impingement may be symptomatic when medial synovial fold impinges against overlying soft tissue, primarily the zona orbicularis [68].

Aetiology & clinical features

The aetiology of this impingement syndrome still remains unclear, as there has been no study focusing on the pathology. The presence of an abnormally shaped or thickened medial synovial fold may be responsible for this disorder. The authors have recently reported of a cyst in the medial synovial fold abutting against the zona orbicularis, which led to pectineofoveal impingement in a 13-year-old girl who presented with hip pain and clicking which had been affecting her for over six months. After the cyst was detected and removed arthroscopically, she was asymptomatic and remains active with no clicking or pain at the last follow-up [67].

Usually, symptoms of pectineofoveal impingement are clinically non-specific, with ill-defined hip pain aggravated by rotational movements and the occasional feeling of hip blockage, but no snapping. It can be associated with clicking. The medial synovial fold is clearly visible on MR arthrography as a band-filling defect to the extent that its dimensions can be accurately measured [71], and is also beneficial to rule out damage to the labrum and cartilage secondary to other intra-articular pathologies including FAI.

Management

Non-surgical interventions such as rehabilitation or activity modification should be considered as the first option for treatment. Refractory symptomatic pectineo-foveal impingement can be treated by arthroscopic resection of the medial synovial fold, which is usually carried out with a radiofrequency ablation device and a punch. Adding to our recent case report described above which has reported the successful outcome of treatment for pectineo-foveal impingement, Bardakos has also mentioned his experience with a female sprinter diagnosed with isolated pectineo-foveal impingement who achieved a successful outcome following arthroscopic intervention [69].

Conclusion

Although our knowledge for these extra-articular hip impingement syndromes still remains limited but evolving for reasons mostly relating to their low prevalence and their co-existence with typical FAI, they are now being recognised more often by surgeons treating young adults with hip pain. The knowledge of these syndromes is, therefore, essential and should form a part of the differential diagnoses alongside intra-articular pathology including FAI particularly in the younger patient with a non-arthritic hip. This descriptive review provides the surgeons with current concepts of each extra-articular hip impingement syndrome, however, a comprehensive review of the diagnosis and management of each of these syndromes should be performed in the future as well.

Compliance with ethical standards

Conflict of interest The authors declare that they have no conflict of interest.

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