Operative Techniques in
Orthopaedics

Femoroacetabular Impingement

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Femoroacetabular impingement (FAI) syndrome is a dynamic clinical disorder in which morphologic abnormalities of the acetabulum and/or femur combined with vigorous hip motion cause repetitive abutment between the femoral head-neck junction and acetabular rim. FAI is classified as cam (loss of femoral head-neck offset and asphericity), pincer (acetabular overcoverage) or combined type and has been increasingly recognized as a common cause of hip pain and functional limitation. Symptomatic FAI occurs predominantly in young, white, nonobese patients without prior hip disease. The etiology of FAI is controversial and likely multifactorial, with both genetic and acquired mechanisms proposed. There is growing evidence that participation in aggressive athletic activities during adolescence increases the risk of cam development due to repetitive injury to the proximal femoral physis. Sports at risk include football, hockey, soccer, and dance. Up to 50% of asymptomatic hips demonstrate radiographic FAI, particularly in athletes, highlighting the importance of considering symptoms and examination findings when making the diagnosis of FAI syndrome. Patients often report a gradual onset of motion- or position-related groin pain. Examination typically reveals limited hip motion, usually restricted internal rotation in flexion, and pain with provocative impingement tests. Numerous imaging parameters have been described to identify cam (α angle, head-neck offset) and pincer (center edge angle, acetabular index, crossover sign, and posterior wall sign) morphology on radiographs. While the natural history of FAI syndrome is thought to involve progressive chondrolabral damage leading to early osteoarthritis, currently there is no evidence that surgical treatment prevents or delays onset of osteoarthritis.
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KEYWORDS Hip, impingement, cam, pincer

Introduction

The concept of hip impingement was originally described in 1936 by Smith-Petersen et al who proposed that abnormal contact between the femoral neck and the anterior acetabular margin in a patient with protrusion of the acetabulum was leading to painful synovitis and periosteal irritation.[1] Decades later, the presence of anatomic abnormalities of the proximal femur, termed a *tilt deformity*[2] or *pistol grip deformity*,[3] were identified in association with hip osteoarthritis. However, it was not until 2003 that Ganz et al proposed femoroacetabular impingement (FAI) as the primary mechanism of early-onset osteoarthritis in the nondysplastic hip.[4] Ganz et al[4] further subdivided FAI into 2 distinct types based on the pattern and various stages of chondral and labral lesions: cam impingement and pincer impingement. In cam impingement, an abnormally-shaped femoral head-neck junction abuts the anterosuperior aspect of the acetabular rim during hip flexion, leading to chondral delamination and labral detachment. In pincer impingement, an abnormal area of acetabular overcoverage impinges on the femoral head-neck junction during hip motion leading to anterosuperior labral tearing and posteroinferior chondral injury ("contrecoup injury"). Over the past 15 years, the number of patients identified with FAI has risen dramatically, with a corresponding surge in volume of publications on the topic.[5,6] In 2016, the Warwick Agreement on FAI syndrome was convened and an international consensus statement was

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released in an effort to clarify the appropriate diagnosis and management of patients with FAI.[7]

Epidemiology

The true prevalence of FAI is challenging to define, as many epidemiology investigations use radiographic criteria alone without consideration of clinical symptomatology. Gosvig et al[8] performed a cross-sectional study of 3620 asymptomatic adults (mean age 60 years, 63.2% female) without a history of childhood hip disease and reported a prevalence of 17.8% for pincer morphology, 10.5% for cam morphology, and 1.6% for combined cam and pincer morphology. While the prevalence of pincer morphology was equal between the sexes, cam morphology was far more common in males, with a prevalence of 19.6% compared to 5.2% in females. Similar results were found by Hack et al,[9] who examined 400 hips from 200 asymptomatic adults (mean age 29 years, 79% white, 55.5% female) and reported a prevalence of 14% for cam morphology, of which 79% were male. In a prospective population-based study of 2081 asymptomatic young adults, both cam and pincer were found to be significantly more prevalent in males (35% and 34.3%, respectively) compared to females (10.2% and 16.6%, respectively).[10] In addition to male sex, participation in athletics has also been associated with a higher prevalence of radiographic FAI. Frank et al[11] performed a systematic review of 26 studies evaluating 2114 hips in asymptomatic individuals and found the prevalence of cam morphology in the athletic population to be 54.8% compared to 23.1% in the general population.

In clinically symptomatic hips, the prevalence of FAI morphology is much higher. Ninety percent of patients with symptomatic labral tears demonstrate structural femoral and/or acetabular abnormalities.[12] Nepple et al[13] found that 94% of National Football League Combine athletes with prior documented hip or groin pain had radiographic evidence of FAI. A systematic review of 60 studies involving 11,451 hips found that pincer morphology was most prevalent in asymptomatic hips (57%) while cam morphology was most prevalent in symptomatic hips (49%), followed closely by combined cam and pincer morphology (40%). A male predominance for FAI findings was noted in both asymptomatic and symptomatic patients. The most commonly represented sport was American football, followed by soccer and ice hockey.[14] Clohisy et al[15] performed a multicenter prospective longitudinal cohort study of 1130 symptomatic hips undergoing surgical treatment and found FAI occurred predominantly in young (mean age 28.4 years), white (87.8%), nonobese (53.8%) patients without history of childhood hip disease (89%). Cam morphology was identified in 48% of hips, combined cam and pincer morphology in 45% and isolated pincer morphology in only 8%. Contrary to previous reports, the authors found a higher percentage of female patients with symptomatic FAI (55%) compare to males and attributed this to the marked increase in female sports participation over the recent years, particularly in the preadolescent and adolescent age groups.[15]

Etiology

The precise etiology of FAI remains controversial, though both genetic and acquired causes have been described. A number of conditions may result in abnormal contact between the proximal femur and the acetabulum, including slipped capital femoral epiphysis (SCFE), Legg-Calve-Perthes disease, acetabular retroversion, coxa profunda, protrusio acetabuli, prior periacetabular osteotomy or femoral osteotomy, and prior femoral neck fracture malunion with decreased head-neck offset or widened femoral neck. Although these conditions may predispose to the development of FAI, in the vast majority of cases there is no history of prior hip pathology.[4,15]

Numerous studies have investigated a possible genetic or geographic link to the development of idiopathic FAI. Pollard et al[16] performed a sibling study comparing radiographs from the siblings of 64 FAI patients to a spousal control group. The sibling cohort was found to have relative risks of 2.8 and 2.0 of having similar cam or pincer morphology, respectively, and demonstrated a higher prevalence of clinical hip symptoms and osteoarthritis compared to the control group. Several investigations have explored the potential role of various single nucleotide polymorphisms (SNPs) in the development of abnormal hip morphology and osteoarthritis.[17-19] Baker-Lepain et al[18] studied SNPs associated with different proximal femur shapes and found that the presence of a specific variant allele, rs288326, increased the likelihood of developing hip osteoarthritis. As for pincer morphology, Sekimoto et al[19] identified five HOX9 SNPs that had significant association with the development of acetabular overcoverage. While recent studies suggest that genetic factors may have a role in the development of FAI, a systematic review by Packer and Safran[20] concluded that there is no definitive evidence that FAI is transmitted genetically.

There is mounting evidence, however, that participation in aggressive, high-impact athletic activities, particularly during adolescence, plays an important role in development of cam morphology. Many cross-sectional studies of both asymptomatic and symptomatic athletes have shown a high prevalence of cam morphology in this population.[21-25] This correlation is particularly worrisome given the increasing trend toward early single-sport specialization and year-round athletic training in youth sports. Nepple et al[26] performed a systematic review of studies evaluating the association between sports participation during adolescence and cam development and concluded that high-level athletes are 1.9-8 times more likely to develop cam morphology compared to nonathletes. The risk of cam development was strongest for hockey (10 times the risk) and basketball (4 times the risk). Several theories have been proposed regarding the development of cam morphology in young high-level athletes. It has been postulated that recurrent high stresses at the femoral head-neck junction lead to reactive bone formation independent of the physis.[20] Another explanation is that the cam morphology arises from a subclinical SCFE that was missed in adolescence; however, the majority of cam deformities do not exhibit the typical posterior-inferior migration of the epiphysis seen in post-SCFE hips.[27] The most widely accepted hypothesis is that repetitive axial loading and torsional force across the skeletally immature hip during high-impact and/or extreme motion sports injures the proximal femoral physis and results in abnormal lateral physeal extension along the anterosuperior femoral neck.[28,29] As the physis closes, the abnormal physeal extension results in bony overgrowth at the femoral head-neck junction and subsequent development of cam morphology.

Pathoanatomy

The normal hip joint is a ball-in-socket synovial articulation between the femoral head and pelvic acetabulum. Normal hip motion requires a spherical femoral head with an appropriate head-neck offset and proper containment by a congruent acetabular socket. The labrum is a fibrocartilaginous structure that expands the articular surface of the acetabulum, increases joint stability and fully seals the joint space to maintain pressurized fluid between the femoral head and acetabular chondral surfaces, enhancing joint lubrication and minimizing articular cartilage wear.[30] The labrum blends with the hyaline cartilage of the acetabulum at a 2 mm transition zone termed the chondrolabral junction.[30,31]

In the setting of FAI, aberrant morphology of the proximal femur and/or the acetabulum leads to abnormal contact between the 2 structures, particularly at extreme ranges of hip motion. With repetitive motion, altered femoroacetabular contact causes recurrent impingement with subsequent chondral and labral injury. The pathoanatomy of FAI is categorized into cam and pincer morphology, based on the anatomic location of the deformity and pattern of chondrolabral injury.

Cam impingement is defined by loss of femoral head-neck offset and asphericity of the femoral head in the setting of a normal acetabulum. During hip flexion and internal rotation, entry of the abnormally shaped femoral head into the acetabular socket generates shear forces at the chondrolabral junction and adjacent articular cartilage, displacing the labrum toward the capsule and the articular cartilage into the joint. Repetitive abutment of the cam lesion against the acetabulum results in a characteristic inside-out injury with eventual labral detachment and chondral delamination, most commonly involving the anterosuperior rim.[32] Prior to frank delamination, softening of the articular cartilage can be observed intraoperatively as a *wave sign* when arthroscopically probed.[33] Frequently, the acetabular cartilage is separated from the labrum and subchondral bone, while the substance of the labral tissue is relatively spared from direct injury and remains spared until advanced cartilage damage is established. Compared to intrasubstance labral tears, chondrolabral detachment injuries have superior healing rates because of the improved tissue quality and preserved vascular supply from the capsular margin.[30,34]

Pincer impingement is characterized by focal or global osseous acetabular overcoverage in the setting of a normally-shaped femoral head. Acetabular overcoverage can occur in a focal manner, arising from distinct lesions of the anterior or posterior rim and acetabular retroversion. Global overcoverage can develop in the setting of coxa profunda or acetabular protrusio deformities.[35] With hip flexion and internal rotation, repetitive contact between the normal femoral head-neck junction and the abnormal acetabular rim results in compression of the labrum trapped between the 2 structures and development of intrasubstance labral injury.[32] In contrast to the labral detachment injuries seen in cam impingement, these intrasubstance labral tears are less reparable due to diminished vascular supply and poor-quality tissue.[36] Continued impingement can progress to the development of heterotopic bone formation at the adjacent acetabular rim and mineralization of the anterosuperior labrum.[37] While the abnormal contact forces may transmit to the nearby anterosuperior acetabular cartilage, the chondral damage tends to be relatively limited compared to the deep delamination injuries characteristic of cam impingement. With continued hip flexion, levering of the femoral head against the prominent anterior acetabular rim causes transient posterior hip subluxation; impaction of the posteromedial femoral head against the posteroinferior acetabulum results in a classic contrecoup pattern of chondral injury to the femoral head and acetabulum.[32,38]

Combined impingement with both femoral and acetabular deformities is the most common type of FAI and typically demonstrates a combination of labral and chondral injury patterns of both types.[32,35] Philippon and Schenker reported that hip pain and loss of motion impairing athletic performance was predominantly related to combined cam and pincer impingement.[39] FAI syndrome can also occur concurrently with other deformities of the proximal femur and acetabulum independent of the typical cam and pincer morphology. Abnormal femoral version influences baseline hip motion and can impact the presentation of FAI syndrome. Femoral retroversion reduces hip internal rotation and flexion and can exacerbate underlying FAI; relative or absolute retroversion of the proximal femur worsens the already restricted hip motion associated with FAI and results in mechanical impingement at lesser degrees with hip rotation. Conversely, a cam and/or pincer lesion in a patient with femoral anteversion may remain asymptomatic due to increase baseline internal rotation minimizing mechanical impingement and subjective loss of motion.[12,38] Combined FAI and paradoxical acetabular dysplasia has also been described.[40,41] Abnormal loading of the hip joint in the setting of acetabular malformation is thought to generate protrusions at the acetabular rim or femoral head-neck junction, subsequently predisposing to impingement.[41]

Compensatory Extra-articular Injury

In addition to intra-articular injury, alteration of normal functional hip motion in patients with FAI results in increased mechanical stress across the entire hemipelvis and can lead to compensatory injury involving osseous and soft tissue structures around the hip.[42]

Restricted hip internal rotation leads to increased shear stress across the bony hemipelvis, which can manifest as pain within the pubic symphysis joint (osteitis pubis), sacroiliac joint, and lumbosacral spine. Osteitis pubis is characterized by tenderness over the pubic symphysis and pain with resisted hip adduction. There is a significant association between the development of osteitis pubis and loss of hip rotation.[43] Similarly, dysfunction of the sacroiliac joint has also been linked to limited internal rotation of the hip.[44] Sacroiliac joint dysfunction manifests as pain over the posterior pelvis and lower back that can be precipitated by hip internal rotation on examination.

The musculature of the hemipelvis is responsible for dynamic stability of the hip and is also susceptible to injury in the setting of altered hip biomechanics. In patients with FAI, the abnormal mechanical stress transmitted to the osseous hemipelvis subsequently imparts increased strain to the muscles that attach to the bony pelvis, leading to a variety of soft tissue injuries including enthesopathies, tendinopathies, and muscular strains. Injury to the periarticular soft tissue structures can be categorized by location relative to the hip joint into anterior, posterior, lateral, and medial. Anterior injuries include hip flexor strains involving the rectus femoris muscle and iliopsoas strains and/or impingement.[45] Posteriorly, proximal hamstring tendinopathies and strains are most common; injury to the short external rotators of the hip and piriformis muscle (*deep gluteal syndrome*), as well as irritation or compression of the sciatic nerve are other posterior injuries.[42] Lateral injuries include iliotibial band syndrome and injury to the gluteus medius and/or minimus tendons in the peritrochanteric space. Medially, injury to the core musculature (*athletic pubalgia*) encompasses injuries to the adductor, rectus abdominis, internal oblique and/or external oblique musculature, with adductor tendinopathy, and rectus tendinopathy being most common.[46]

It is critical to recognize both intra-articular and extra-articular etiologies of mechanical hip pain. Failure to identify and appropriately manage concomitant extra-articular injuries can result in persistent symptoms and hip dysfunction.

Clinical Presentation

Clinical evaluation of a patient with suspected FAI syndrome should begin with a thorough history. Any history of childhood hip disease, including developmental dysplasia of the hip, SCFE and Legg Calvé Perthes disease, trauma to the hip or prior surgery should be noted. FAI syndrome primarily affects active young adults and therefore detailed information regarding the patient's athletic activities should be obtained.

Patients with symptomatic FAI may present with a wide variety of clinical symptoms. Motion-related or position-related groin or hip pain is the predominant symptom and often has an insidious onset following a minor trauma or, in most cases, no identifiable event. The pain is typically intermittent and incited by activities that require loading of the hip joint and hip flexion or rotation. While the majority of patients describe pain in the groin, pain may also be reported in the lateral hip, anterior thigh, buttock, posterior and lateral thigh, lower back, and may be referred to the knee.[47] Patients often use the *C sign* to demonstrate the location of the pain, by cupping the hand around the lateral hip between the thumb and fingers just above the level of the greater trochanter.[48] The pain is typically described as deep, aching, and occasionally sharp with provocative motions. Positions of hip flexion, including prolonged sitting in a car or rising from a seated position, often reproduce the pain and patients may alter their posture to avoid excessive hip flexion beyond 90°. Subjective hip stiffness and mechanical symptoms may also be reported, including clicking, catching, locking, or giving way. It is imperative to note the presence of painful snapping or popping and clarify the location, reproducibility, and temporal association with pain.[49]

Physical Examination

A comprehensive examination of a patient with suspected FAI syndrome includes assessment of gait, hip range of motion, hip musculature strength, and symptoms with provocative hip maneuvers. Evaluation of gait may reveal an antalgic gait with a shortened stance phase. In stance, the patient may hold the irritated hip in slight flexion. A Trendelenburg gait, or lateral lurch, may be present and is characterized by lateral deviation of the trunk toward the affected side. This can indicate hip abductor weakness or can be compensatory as the patient attempts to shift the center gravity over the hip, thereby reducing the forces on the hip joint. Single-leg stance and single-leg squat tests are useful to assess for pain with unipedal weightbearing, as well as muscle strength and pelvic stability. Observation of the patient in a seated position may reveal a slouched posture toward the uninvolved side to minimize flexion of the hip. The patient may also hold the hip in slight external rotation and abduction, which relaxes the hip joint capsule and lessens discomfort.

A thorough evaluation of the lumbar spine, pelvic girdle, and lower extremities is necessary to identify any coexisting disorders or compensatory injuries. A standard lower extremity neurologic examination, including assessment of strength, sensation and reflexes, evaluation of lumbar spine motion and tenderness, and a straight-leg test should all be performed to rule out a neurogenic source of pain. Tenderness over the pubic symphysis, sacroiliac joint, greater trochanter or over the origin of the rectus femoris, hamstring, and adductor muscles should be noted. Muscle strength testing may reveal weakness and/or pain suggestive of various compensatory strains or tendinopathies including hip flexor strain, proximal hamstring tendinopathy, and iliopsoas impingement. Pain with resisted hip adduction or a resisted sit up is indicative of core muscle pathology.[46]

Passive hip range of motion is measured in multiple planes with the patient in a supine position and typically reveals limited hip motion, particularly in internal rotation and adduction. Range of motion is compared to the contralateral extremity but may be fairly symmetric as FAI deformities are often bilateral.[50] Less than 20° of internal rotation with the hip positioned in 90° of flexion is suggestive of FAI morphology.[49] The *flexion abduction external rotation (FABER) distance test (FDT)* can also be used to assess restricted hip motion. With the patient in a supine position, the leg is flexed and the heel of the affected leg is placed on the contralateral leg, just above the patella. With the pelvis stabilized and the patient relaxed, the distance between the lateral femoral epicondyle of the knee and the examination table is measured. A positive test is defined as greater than 4 cm difference compared to the contralateral hip and has been shown to correlate with cam impingement and larger alpha angles.[51]

Numerous provocative hip tests have been described for diagnosis of FAI syndrome and are detailed in Table.[52-54] The most common examination maneuvers are the anterior impingement test, FABER or Patrick test, posterior impingement test, Stinchfield test, and log-roll test. These clinical tests are performed with the patient in a supine position and are deemed positive if the maneuver recreates the patient's familiar pain in the appropriate location. It is imperative to note the distribution of the pain during provocative testing as one maneuver can aggravate multiple injury patterns. Pain in the anterior hip or groin area during the FABER test is suggestive of symptomatic FAI, while pain localized to the posterior pelvis is more indicative of sacroiliac joint dysfunction. Though the majority of these provocative examination maneuvers demonstrate good sensitivity, none are specific for the diagnosis of FAI syndrome.[52-54] Therefore, it is necessary to consider the patient's clinical history, including location of pain and triggering activities, in addition to positive findings on physical examination and diagnostic imaging.

Imaging Studies

Imaging studies play a vital role in confirming the diagnosis of FAI syndrome in concert with suggestive clinical symptomatology and physical examination findings. Suspected FAI is typically evaluated with a combination of radiography, magnetic resonance imaging, and less commonly computed tomography.

Plain radiographs are the initial diagnostic modality of choice and can reveal cam and pincer morphologies as well as provide information on the location and extent of the cam lesion, acetabular coverage and version, and identify the presence and severity of osteoarthritis.[7] An anteroposterior (AP) radiograph of the pelvis and a lateral femoral neck view of the affected hip should be obtained. The AP pelvis radiograph should be performed with the patient supine, in neutral pelvic tilt and rotation, and the x-ray beam centered midline between the pubic symphysis and the anterior superior iliac spine.[31] A true AP radiograph has 1-3 cm between the tip of the coccyx and the pubic symphysis (neutral tilt), with relative symmetry of the 2 hemipelvises and the coccyx centered on the pubic symphysis (neutral rotation).[55] Alterations in pelvic tilt and rotation impact the radiographic measurements, most significantly acetabular version.[56,57] Multiple lateral hip views are commonly used and choice depends on clinician preference, including frog-lateral, 45° and 90° Dunn, and cross-table lateral in 15° internal rotation. The 45° Dunn view, obtained with the hip in 45° flexion, 20° abduction and neutral rotation, gives the best view of the anterior head-neck junction and reveals the maximum cam morphology.[58]

A variety of quantitative measurements have been described to identify cam and pincer morphology. Radiographic measurements to detect cam morphology include the α angle,[59] femoral head-neck offset, and the head-neck offset ratio (Fig. 1).[60] The *α angle* is the most common parameter used to quantify cam morphology and is measured on the lateral hip radiograph. It is determined by fitting a circle to the femoral head, drawing one line from the center of that circle to the center of the femoral neck at its narrowest point, connecting a second line from the center of the circle to the first point of the head-neck junction that lies outside the margin of the circle, and then measuring the angle included between those 2 lines (Fig. 1A).[59] Higher α angles represent larger and more extensive cam lesions; however, there continues to be no standard agreed-upon threshold value differentiating normal and abnormal α angles. Recommendations ranging from 42° to 63° with an α angle greater than 55° being the most widely accepted as indicative of abnormal cam morphology.[7,31] The *femoral head-neck offset (HNO)* is assessed on the lateral hip view and is measured as the distance between 2 lines drawn parallel to the femoral neck axis – one placed along the anterior femoral neck and a second drawn tangential to the anterior margin of the femoral head (Fig. 1B).[60] The *femoral HNO ratio* allows standardization by femoral head size and controls for radiographic magnification. The HNO ratio is calculated by dividing the HNO by the diameter of the femoral head. Threshold values of HNO less than 8 mm and HNO ratios less than 0.17 have been proposed.[55] Other radiographic parameters that have been reported include the triangular index and the anterior femoral distance.[61] Radiographs may also reveal abnormal wasting and synovial herniation pits at the femoral head-neck junction.[31]

Radiographic evaluation for pincer morphology focuses predominantly on differentiating global overcoverage, acetabular retroversion, and focal rim lesions using a combination of quantitative measurements and radiographic signs (Fig. 2). Global acetabular overcoverage is quantified by measuring the center edge angle[62] and acetabular index or Tonnis angle[63] on the AP pelvis radiograph. The lateral center edge angle or simply *center edge angle (CEA)* is measured by fitting a circle to the femoral head, drawing one line from the center of the circle perpendicular to the transverse axis of the pelvis, connecting a second line from the center of the circle to the lateral edge of the acetabular sourcil, and then measuring the angle included between those 2 lines (Fig. 2A).[62] A normal CEA is 25°-39°, with angles less than 25° indicating acetabular dysplasia and angles greater than 39° indicating global acetabular overcoverage.[61] The *acetabular index (AI)* or *Tonnis angle* is measured between one line parallel to the transverse axis of the pelvis and a second line drawn between the medial and lateral margins of the acetabular sourcil.[63] A normal AI is 0-10°; global acetabular overcoverage is

Table Femoroacetabular Impingement Examination Tests

Test	Description	Pertinent Findings	Sensitivity	Specificity
Anterior impingement or **flexion adduction internal rotation (FADIR)**	■ Patient is supine. ■ Examiner moves hip into 90° flexion, 20° adduction and maximum IR	■ Anterior hip pain predominating in flexion/IR ■ ↓ pain-free flexion arc in maximum IR	0.88 – 1.0	0.67 – 0.86
Scour or **hip quadrant**	■ Patient is supine with the hip in maximum flexion and adduction. ■ Examiner applies a downward axial force to the hip joint and moves the hip through a circular arc of motion.	■ Reproduction of pain ■ Restricted motion	0.50 – 0.62	0.29 – 0.38
Internal rotation over pressure (IROP)	■ Patient is supine with the hip in 90° flexion and maximum IR. ■ Examiner exerts an IR overpressure, further internally rotating the leg, while stabilizing the contralateral pelvis to minimize pelvic rotation.	■ Anterior hip pain with IR overpressure	0.88 – 0.91	0.17 – 0.18
Flexion abduction external rotation (FABER) or **patrick**	■ Patient is supine with the hip in flexion, abduction and ER so the lateral ankle rests on the contralateral leg just proximal to the knee. ■ Examiner applies a downward force to the knee, exerting an ER force to the hip.	■ Anterior hip pain with downward force applied ■ Posterior pelvic pain with this maneuver is suggestive of sacroiliac joint dysfunction.	0.69 – 0.97	0.24 – 0.25
Dynamic external rotatory impingement test (DEXRIT)	■ Patient is supine and holds the contralateral leg is maximum hip flexion. ■ Examiner moves the hip into 90° flexion, then passively moves the hip through a wide arc of abduction and ER.	■ Reproduction of pain	NA	NA
Dynamic internal rotatory impingement test (DIRIT)	■ Patient is supine and holds the contralateral leg is maximum hip flexion. ■ Examiner moves the hip into 90° flexion, then passively moves the hip through a wide arc of adduction and IR.	■ Reproduction of pain	NA	NA
McCarthy	■ Similar to Scour test. ■ Patient is supine. ■ Examiner moves the hip through a wide arc of IR and ER from flexion to extension.	■ Reproduction of pain	NA	NA

Table (*Continued*)

Test	Description	Pertinent Findings	Sensitivity	Specificity
Lateral rim impingement	▪ Patient in lateral position with the hip abducted. ▪ Examiner moves the hip from flexion to extension while externally rotating the hip	▪ Reproduction of pain	NA	NA
Resisted straight leg raise (RSLR) or **Stinchfield**	▪ Patient is supine and asked to raise the leg to 30° of hip flexion. ▪ Examiner exerts downward force on the leg while patient resists.	▪ Reproduction of pain ▪ Hip flexion weakness compared to the contralateral leg	0.56 – 0.75	0.29 – 0.38
Log-roll or **passive supine rotation**	▪ Patient is supine. ▪ Examiner places both hands on the thigh then internally and externally rotates the hip.	▪ Reproduction of pain ▪ Restricted motion	0.30	NA
Posterior impingement	▪ Patient is supine at the edge of the table with both legs drawn up to the chest. ▪ Examiner lowers the affected leg off the table, extending, abducting and externally rotating the hip.	▪ Reproduction of pain	0.21	NA
Foveal distraction	▪ Patient is supine with the hip in 30° abduction. ▪ Examiner applies axial traction to the leg.	▪ Relief of pain	NA	NA

ER, external rotation; IR, internal rotation; NA, not available.

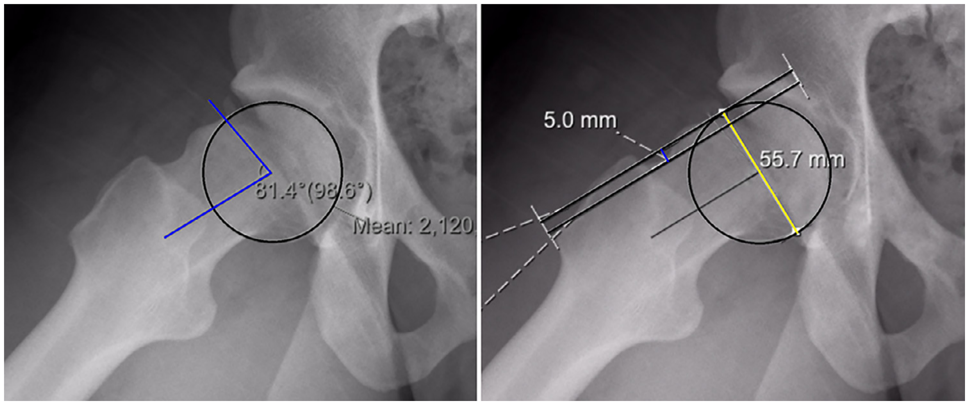

Figure 1 Radiographic Assessment of Cam Morphology. (1A) A 45° Dunn lateral radiograph of a right hip demonstrating an abnormal α angle measuring 81.4°. To determine the α angle, a best-fit circle is first drawn around the femoral head. The α angle is then measured between one line drawn from the center of the femoral head to the center of the femoral neck and a second line drawn from the center of the femoral head to the first point of head-neck junction that lies outside the margin of the circle. (B) A 45° Dunn lateral radiograph of a right hip demonstrating an abnormal femoral head-neck offset measuring 5 mm and abnormal head-neck offset ratio measuring 0.09. The head-neck offset (blue line) is measured as the distance between one line drawn tangential to the anterior margin of the femoral head and a second line drawn along the anterior femoral neck, both parallel to the femoral neck. The head-neck offset ratio is calculated by dividing the head-neck offset (blue line) by the diameter of the femoral head (yellow line) to control for radiographic magnification. (Color version of figure is available online.)

indicated by a down-sloping sourcil with an AI angle less than 0°.[61] Though less common, acetabular protrusio can result in global overcoverage and is identified radiographically as excessive medialization of the hip joint such that the medial femoral head contacts or lies medial to the ilioischial line on the AP pelvis view.[55] Presence of the posterior wall sign, crossover sign, and ischial spine sign on the AP pelvis radiograph all suggest focal acetabular overcoverage. The posterior wall is normally located lateral to the center of the femoral head and remains lateral to the anterior acetabular rim. The *posterior wall sign* is present when the posterior wall lies medial to the center of the femoral head, signifying deficiency of the posterior wall. The *crossover sign* is present when the anterior rim extends lateral to the posterior rim before converging at the lateral acetabular sourcil (Fig. 2B).[55] Existence of both the posterior wall sign and the crossover sign indicates overcoverage related to acetabular retroversion.[61] The *ischial spine sign* is present when any portion of the ischial spine projects within the pelvic brim and suggests acetabular retroversion related to deformity of the entire hemipelvis.[64] Focal overcoverage from a distinct rim lesion and normal acetabular version is indicated by presence of the

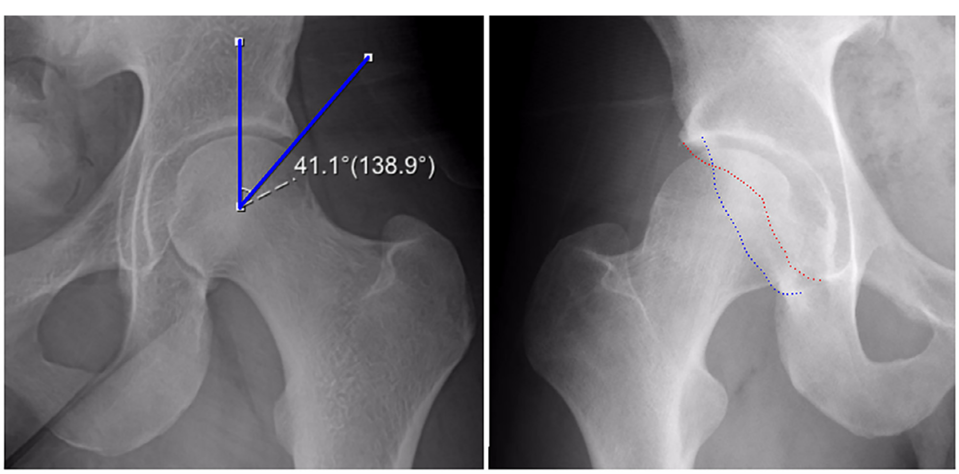

Figure 2 Radiographic Assessment of Pincer Morphology. (2A) An anteroposterior radiograph of a left hip demonstrating an abnormal center edge angle measuring 41.1°. The center edge angle is measured between one line drawn vertically from the center of the femoral head perpendicular to the transverse axis of the pelvis and a second line drawn from the center of the femoral head to the lateral edge of the acetabular sourcil. (B) An anteroposterior radiograph of a right hip demonstrating a crossover sign. Normally, the posterior wall remains lateral to the anterior wall. In this projection, the posterior wall (blue dotted line) can be seen crossing over medial to the anterior wall (red dotted line). (Color version of figure is available online.)

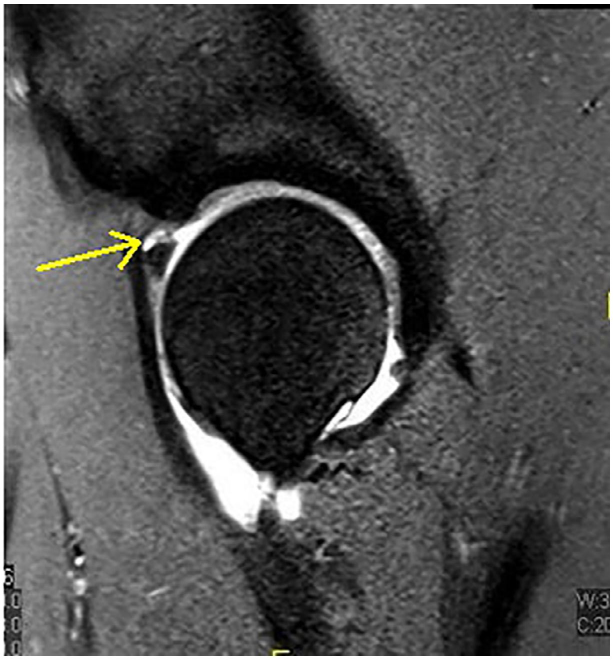

Figure 3 Magnetic resonance arthrogram T2 sagittal image of a right hip demonstrating a detached labral tear (arrow).

crossover sign with a negative posterior wall sign. Position of the crossover point relative to the femoral head center can further clarify the location of the overcoverage. Most commonly, the anterior and posterior walls cross near the center of the femoral head and indicate focal overcoverage by a prominent anterior wall. Crossing of the anterior and posterior walls lateral to the femoral head center suggest focal posterior overcoverage.[31]

Three-dimensional imaging provides multiplanar visualization of the femoral and acetabular osseous anatomy and a thorough evaluation of intra-articular pathology. Magnetic resonance (MR) imaging allows precise measurement of the acetabular version, acetabular depth, labral size, and mineralization. An MR arthrogram with injection of intra-articular gadolinium contrast provides better visualization of labral tears and detachment, as well as cartilage delamination (Fig. 3).[65] Early chondral injury, however, is poorly detected on standard MR and MR arthrogram imaging studies. Use of advanced cartilage MR (cMR) imaging techniques has been proposed to better identify and characterize the histochemical changes in cartilage composition that occur early in the disease process.[66]

Though osseous structures can be visualized on MR imaging, computed tomography (CT) is the best imaging modality to accurately delineate the bony architecture of the femoral and acetabular deformities. CT is particularly useful for characterizing severe osseous deformities and better defining the morphology in borderline cases.[61] The primary drawback of CT use is the increased radiation exposure compared to other imaging modalities. Newer protocols with low-dose CT scanning have been developed and have a radiation dosage approximately equivalent to 6 pelvic radiographs; however, these advanced protocols are not consistently available at different medical centers and therefore, radiation exposure remains a significant concern.[67] While the routine use of preoperative CT is controversial, 3-dimensional surface renderings of the bony architecture can be generated from CT images and used in preoperative planning, patient education, and intraoperative localization of the deformity.

Natural History

Morphologic variations of the proximal femur and acetabulum have long been associated with development of hip osteoarthritis (OA).[4,8,35] Ganz et al[4] originally proposed a link between FAI and early-onset OA of the hip, suggesting that repetitive abnormal contact between the femoral head-neck junction and acetabular margin results in progressive chondrolabral damage that ultimately deteriorates and progresses to arthritis. Though numerous cross-sectional studies have investigated the connection between FAI and hip OA, it is difficult to distinguish primary FAI morphology from secondary osseous changes related to arthritis progression in those analyses.[68]

More recent prospective longitudinal studies of younger patients have revealed a high prevalence of asymptomatic FAI morphology and associated increased risk of developing hip OA over time; though, it should be noted that many hips with FAI morphology will never progress to OA.[69] The presence of cam morphology and higher α angles has specifically been linked to an elevated risk of hip OA.[70,71] A 20-year longitudinal cohort study of 1003 female patients found that for every 1° increase in α angle, risk of OA development increased by 5% and need for total hip arthroplasty increased by 4%.[70] Agricola et al[71] performed a prospective cohort study of 723 patients between 45 and 65 years of age and reported that an α angle greater than 60° had an odds ratio of 3.67 for development of end-stage hip OA. The OR increased to 9.66 with an α angle greater than 83°. The combination of an α angle greater than 83° and restricted hip internal rotation less than 20° had a positive predictive value of 52.6% for development of hip OA.[71] In a separate analysis of the same cohort, the authors did not find pincer morphology, defined as CEA greater than 40°, to be associated with hip OA.[72] At the present time, there is insufficient evidence to conclude that the presence of pincer morphology leads to the development of hip OA.

Although presence of cam morphology is linked to an increased risk of end-stage hip OA, there is no evidence to support prophylactic surgical intervention in asymptomatic individuals. Surgical management should be reserved for the treatment of symptomatic FAI that has failed conservative treatment and is indicated to relieve pain and improve function, not to prevent degenerative changes of the hip. The long-term impact of surgery on the natural history of FAI and risk of hip OA has not been established and requires future investigation.

Conclusion

FAI syndrome is a motion-related disorder of the hip arising from anatomic abnormalities of the proximal femur (cam morphology) and/or acetabulum (pincer morphology) with repetitive abutment leading to progressive chondrolabral damage. The prevalence of FAI morphology is much higher than previously thought and is particularly high in young athletes who participated in high-level aggressive athletics at an early age. Diagnosis of FAI syndrome does not hinge on a single clinical sign; rather, a combination of patient history, clinical symptomatology, examination findings, and imaging results are required for proper diagnosis. The presence of FAI, specifically cam morphology, is associated with an increased risk of end-stage hip OA and need for total hip arthroplasty. However, the impact of surgical intervention on the natural history of FAI remains unclear. At the present time, surgery should be reserved for symptomatic patients; there is no evidence to support surgical intervention solely for the purpose of preventing or delaying development of hip OA in asymptomatic individuals.

Competing Interest

No conflict of interest.

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