HIP IMPINGEMENT

WHAT DO WE KNOW AND WHAT DO WE STILL NEED TO FIND OUT?

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INTRODUCTION

Groin pain is common in athletic populations. This pain may be more prevalent in high-level athletes and in certain sports, such as hockey and soccer, which place increased stresses on the groin region. Groin pain may often be related to hip pathology, and hip pain from a number of causes may also be a common finding in high-level athletes. This confluence of hip and groin pain may challenge diagnostic and treatment algorithms in symptomatic athletes.

The clinical complaints associated with groin and hip pain after sports-related injuries are encountered across a number of medical disciplines, including orthopaedic surgery, physical therapy and radiology. According to one study, groin injuries account for 6% of athletic injuries[1]. Many athletes with chronic groin pain have multiple coexisting pathologies. In treating these patients, one must consider both musculoskeletal conditions and non-musculoskeletal conditions that can present as groin pain. A comprehensive history and physical examination should guide the clinical evaluation.

The substantial rise in the diagnosis and treatment of hip pathology is due in part to advancements in technology, coupled with an enhanced understanding of the underlying pathomechanics. The cause of primary or idiopathic osteoarthritis (OA) was, for most of the 20th century, widely thought to be unknown. Murray[2], Solomon[3] and Harris[4] each proposed a mechanical aetiology for hip arthrosis. Much of the resurgence of interest and improvement in understanding the importance of hip arthrokinematics in the development of OA is attributed to the observation of the link between osseous abnormalities, hip pain, chondrolabral damage and eventual osteoarthrosis[5]. This theory proposes that "most, if not all hip osteoarthrosis is secondary, often secondary to subtle but definite and commonly overlooked, ignored or not recognised dysplasia or pistol grip deformities"[6].

Hip deformities that may cause mechanical damage to the joint and resultant OA range from hip instability to femoroacetabular impingement (FAI). While dysplasia causes instability and static overload of the joint, FAI causes dynamic overload of the cartilage and labrum due to abnormal contact between the femur and the acetabulum during motion. At present, continued interest in the relationship between hip deformity and OA is due to advances in surgical techniques to correct the deformity and improved understanding of the role of deformity in the development of OA. While male athletes with a history of elite-level competition are at increased risk of hip OA when compared with the general population[7], the cause for this may be multifactorial.

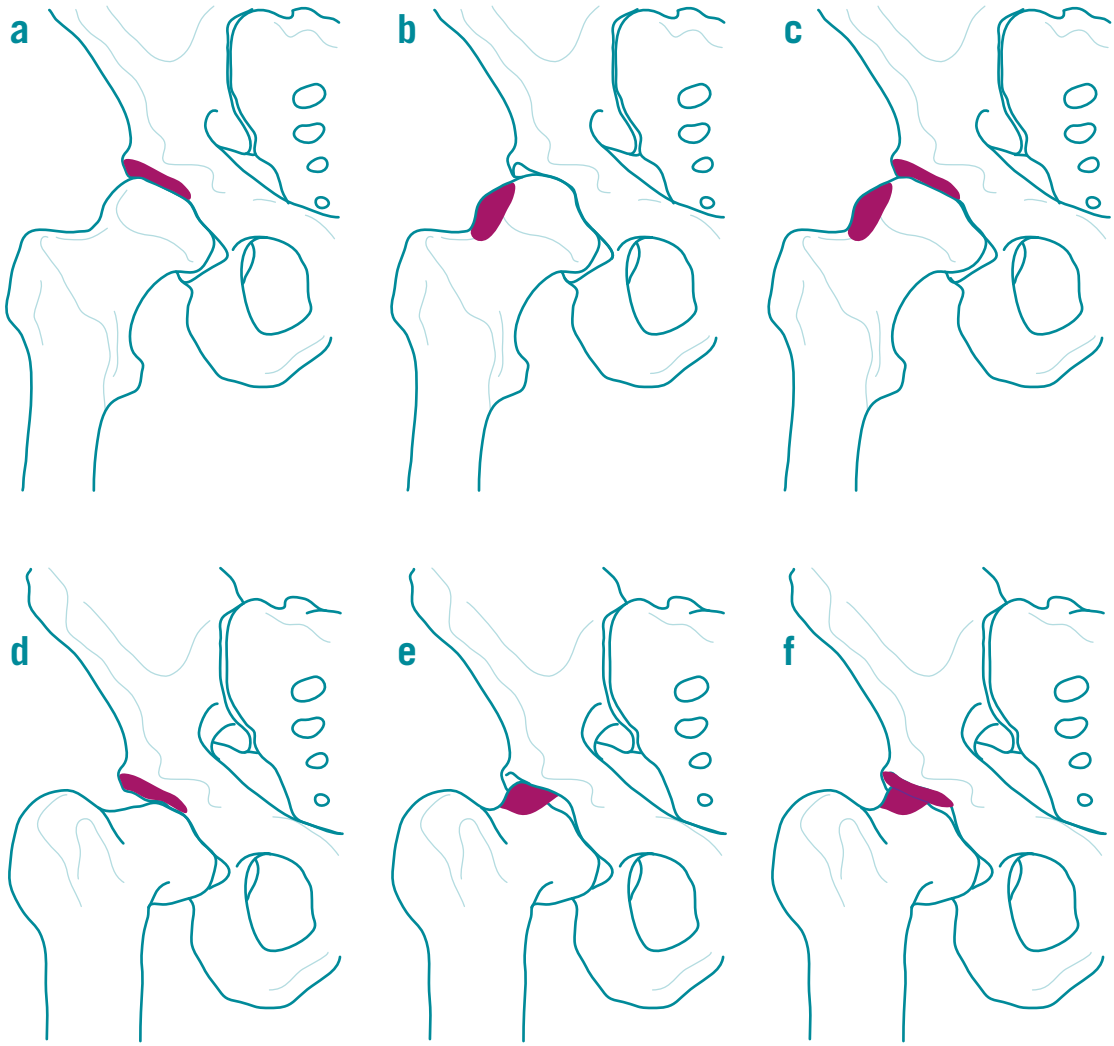

Figure 1: Pelvic model depicting the biomechanical concept of femoroacetabular impingement (FAI). Indicated in red are acetabular over-coverage (pincer FAI; a), femoral asphericity (cam FAI; b) or combinations of both deformities (c). Hip range of motion, particularly flexion and internal rotation, triggers femoroacetabular collisions. In pincer FAI this is the impact of the neck against the rim (d), in cam FAI inclusion of the asphericity into the joint (e) and in combined FAI (f), both injury mechanisms might occur.

Recent advances in the understanding of FAI have led to increasing recognition of its role as a cause of hip pain in elite athletes. Diagnoses previously attributed to muscular strains (e.g. hip flexors or adductors) or other groin pathologies may actually be caused by FAI or hip-related injuries in athletes. Conversely, there may be overlap between intra-articular diagnoses and other groin issues, with the possibility for a number of pathologies to be present simultaneously in individuals with FAI. The early detection and management of symptomatic FAI may present an opportunity to prevent further joint damage. Importantly, more advanced intra-articular disease results in worse clinical outcome and, in the athlete, may lead to diminished athletic function and long-term durability of the hip joint. This report summarises the current understanding of FAI with a focus on athletic populations (what we know), while discussing areas of future research and clinical questions (what do we need to find out).

ANATOMY RELATED TO HIP IMPINGEMENT

Proximal femur

There are a number of subtle alterations in proximal femoral anatomy that affect the biomechanics of the hip. In patients with FAI, decreased offset at the head-neck junction[8] or a cam deformity,[5] which is thought to be an extension of the epiphysis onto the femoral neck[9,10], cause cam impingement and damage at the chondrolabral junction (Figures 1a-f). The prevalence of this deformity is quite high and varies according to the population being studied and the radiographic studies used. Specifically, studies that only evaluate an anteroposterior (AP) image or only use radiographs, are likely to underestimate the prevalence and the magnitude of the deformity[11]. In a study of asymptomatic male Swiss army recruits, the prevalence of the cam deformity detected on radial MRI slices was 24% overall[12]. The maximal alpha angle is best detected with either 3D CT or radial slices through the femoral neck on either CT or MRI. Femoral version also affects the biomechanics of the hip. Femoral retroversion is thought to exacerbate the damage caused by cam impingement by limiting the amount of flexion and internal rotation that is possible before impingement occurs[13].

Acetabulum

Acetabular anatomy has historically been studied in the context of acetabular dysplasia, with the lateral center-edge angle on an AP pelvis X-ray used most commonly to measure acetabular coverage. In contrast, there are only a few studies that have evaluated the prevalence of acetabular over-coverage and acetabular retroversion, which is the pathoanatomy on the acetabular side that can contribute to FAI (Figures 1a-f). Although the most precise way to measure acetabular version is with axial MRI or CT images of the pelvis, a centred AP pelvic X-ray can also provide information about version. The crossover sign is present when

the shadow of the anterior acetabular wall crosses over that of the posterior wall. The posterior wall sign is present when the posterior wall lies medial to the centre of rotation of the femoral head. A CT study of the acetabular version in asymptomatic adults found the average version to be 26 to 27° ± 6° [14]. Regardless of the radiographic measure, acetabular retroversion is not uncommon and may be associated with other hip disorders, including OA, dysplasia and Perthes disease.

ASSOCIATION BETWEEN HIP DEFORMITY AND ARTHRITIS IN THE GENERAL POPULATION

When developing the mechanical concept of hip arthrosis, Harris, Solomon and Murray looked at the prevalence of subtle deformities in 'primary' OA[2–4] finding rates of deformity ranging from 65 to 79%. It is likely that the prevalence of subtle deformity is actually higher, however, as these early studies evaluated only AP pelvic X-rays and there was no evaluation of acetabular over-coverage or acetabular retroversion. Mild dysplasia was observed in 20 to 39% of patients with idiopathic hip OA[2–4]. All of these series found a much higher prevalence of dysplasia in females compared to males. Murray's series had a male to female ratio of 1:4.1 for mild dysplasia[2], Solomon's series had a male to female ratio of 1:10[3] and Harris observed mild dysplasia in 15% of males but 68% of females[4]. The gender ratio was reversed when looking at the 'head tilt' and 'pistol grip' deformities. Harris carefully described the spectrum of pistol grip deformities as flattening of the normally concave surface of the lateral femoral neck, development of a bump on the anterolateral surface of the femoral neck, the formation of a sharp transition or 'hook' at the junction of the articular surface of the femoral head with the femoral neck and failure of the femoral head to be centred over the femoral neck in either the AP pelvis, frog or true lateral X-rays[4]. These deformities would now be considered to be cam morphology, but at that time were considered "deformities that occurred after minimal slipping of the capital femoral epiphysis and mild cases of Legg-Perthes disease"[4]. In both Murray and Harris' series, 40% of patients with primary OA had pistol grip deformities, with a male to female ratio of 6.6:1 for Harris[4] and 5.6:1 for Murray[2]. Solomon's series included patients with secondary causes of OA, but he also observed a predominance of femoral head tilt in males, with a male to female ratio of 14:1[3].

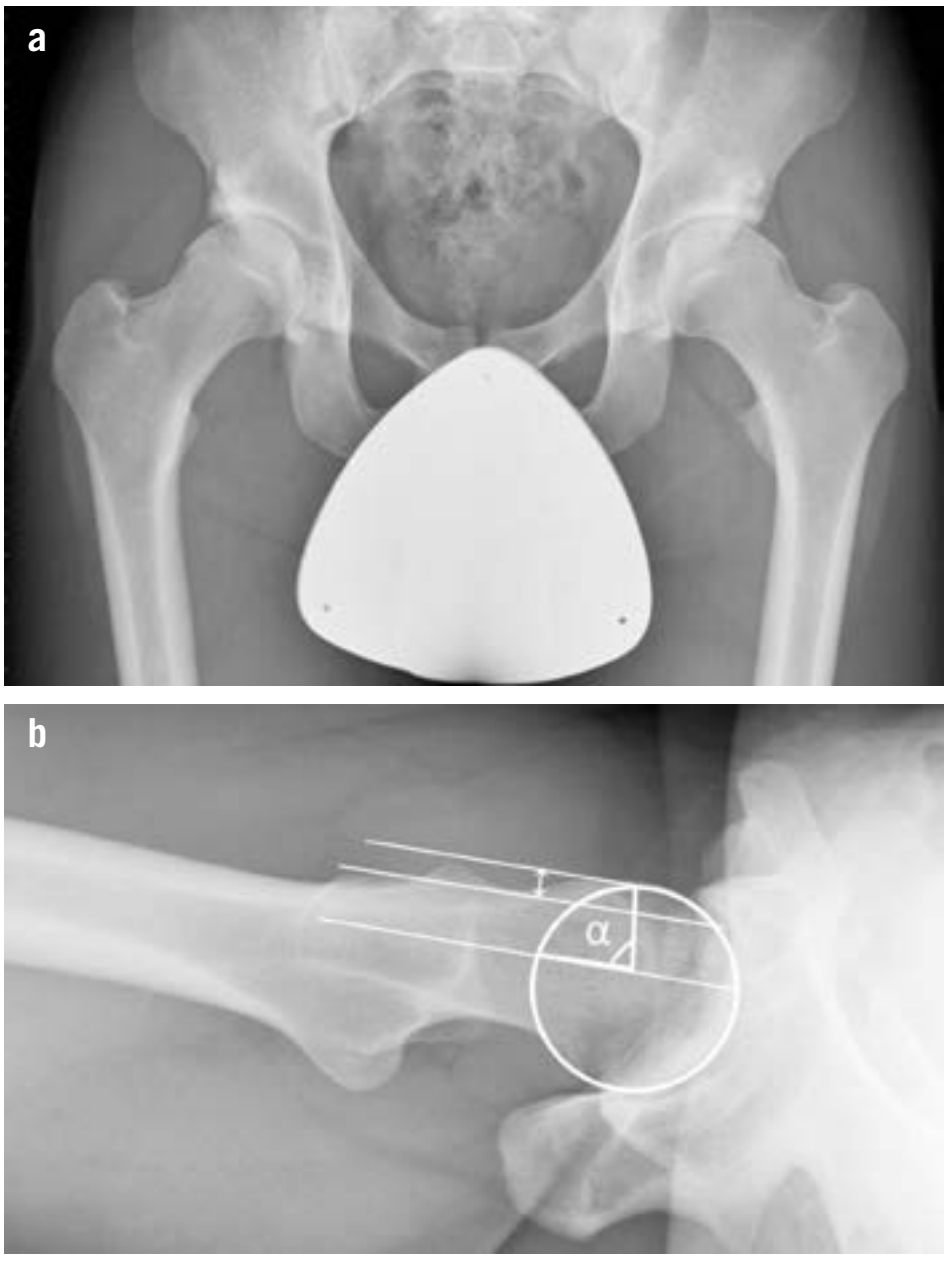

Figure 2: Anteroposterior pelvis (a) and lateral (b) radiographs showing a typical cam lesion (femoral asphericity) in an elite hockey player. The alpha angle (α) and resulting femoral offset (double-headed arrow) are presented in b.

> ***Recent advances in the understanding of FAI have led to increasing recognition of its role as a cause of hip pain in elite athletes.***

The prevalence of FAI is known to be greatly increased in patients presenting with hip pain, adductor strain, labral tears and athletic pubalgia and in some series is seen in as many as 90% of these patients[15]. In a multicentre study of French patients presenting with hip pain and who were candidates for hip arthroscopy, 63% had evidence of FAI. Of the FAI patients, 58% were found to have pure cam impingement, 19% had pure pincer impingement and 23% had evidence of mixed impingement[16]. While the cam deformity is easily and classically seen in males, the anatomy causing idiopathic arthrosis in females may be a subtler radiographic finding. Females are known to have smaller cam deformities than males and this may be a more subtle 'female equivalent' of the pistol grip deformity.

Radiographic rates of OA progression for patients with FAI are also variable. In a study of Greek patients who had radiographic evidence of FAI, after 10 years only 18% had radiographic progression of OA, regardless of whether they had cam, pincer or mixed-type FAI[17]. When compared to other European countries, however, the overall prevalence of hip OA in Greece is very low[18]. Thus, some other environmental or genetic factor may also be influencing the progression of OA in this population. In contrast, other studies have found much higher rates of progression. Of USA patients who underwent unilateral total hip arthroplasty for FAI-related OA, 73% showed either progression of at least one grade of radiographic OA or underwent total hip arthroplasty at an average of 5 years after their first arthroplasty[19]. Another study of FAI patients in the UK observed radiographic progression of OA in 65% of patients over 10 years[20]. A population-based case-control study of women in the UK obtained AP pelvis radiographs 19 years apart, allowing the investigators to evaluate risk factors for hip arthroplasty[21]. There was a higher prevalence of both acetabular dysplasia and of cam deformities in women who underwent arthroplasty[21]. There is one study that attempted to determine if the size of the cam deformity or amount of acetabular coverage correlated with the age at the time of total hip arthroplasty. With the numbers available however, no variable was predictive of the age at surgery[22].

PREVALENCE OF HIP OA AND IMPINGEMENT IN ATHLETIC POPULATIONS

Numerous studies have observed an increased rate of hip OA and, in some cases, arthroplasty for men and women who participated in sports. When the exposure to sport was examined, men and women with high exposures to sports had a significantly increased likelihood of developing hip OA compared to people with moderate and low exposure to sport. A study of former national or professional level male athletes found that athletes who played impact sports e.g. soccer, handball and hockey, had a higher risk of hip OA and total hip arthroplasty than control patients, with age-adjusted odds ratios ranging from 2.01 to 3.13, depending on the sport[7]. Athletes who played non-impact sports also had a higher risk of arthrosis, but the difference was not statistically significant (OR 1.35; CI 0.63 to 2.92)[7]. Other studies have also noted the increased incidence of OA in former elite athletes compared with controls, with former handball players having a 60% incidence of radiographic OA compared to 13% of controls[23] and former elite soccer players having an odds ratio of 2.1 [CI 1.0 to 4.2] for hip OA compared to controls[24].

As early as 1971, Murray and Duncan noted that the 'tilt deformity' was more common in young males with higher rates of activity. Nowadays this deformity would be interpreted as a cam deformity[25]. Contemporary studies of asymptomatic collegiate, national level and professional athletes have observed high rates of morphology that could cause FAI, which

may ultimately be responsible for the high rates of OA observed in former elite athletes. A study of American collegiate football players found that 61% had a crossover sign and 91% had decreased femoral head-neck offset or a cam deformity[26]. Asymptomatic professional and collegiate hockey players were found to have high rates of soft tissue abnormalities on MRI, with 77% having evidence of labral tears, osteochondral lesions or common adductor-rectus tendon dysfunction. In addition, 39% of these players had an abnormally high alpha angle[27]. A study of professional football players revealed that 72% of men and 50% of women had at least one radiographic abnormality that could cause FAI. Although these athletes were asymptomatic at the time of the X-ray, 50% of men and 25% of women reported a past groin or hip injury, indicating that these athletes may have intermittently symptomatic FAI or compensatory pathology[28].

There is some evidence that increased stress on the proximal femoral physis around the time of closure is responsible for the cam deformity[9]. Accordingly, in elite adolescent football players (ages 12 to 19), 26% had an alpha angle >60°, 13% had a prominence at the head-neck junction and 53% had flattening, compared to rates of 17%, 0% and 18% for these measures in control patients who did not play sports[29]. Elite club basketball players in Germany who had played basketball year-round, since the age of 8, were found to have greater mean alpha angles than normal controls. They also had larger mean alpha angles after physeal closure, with 89% having an alpha angle >55° in at least one site. In the control patients, there was no increase in the mean alpha angle after physeal closure. Clinical examination of these athletes revealed that 48% had pain on impingement testing (flexion, adduction and internal rotation) and 19% of athletes reported hip or groin pain in the preceding 6 months[30]. In comparison, 1% of the controls had a positive impingement test. In a case-control study of elite basketball players and age-matched non-athletes, Siebenrock et al presented evidence for growth plate alterations, rather than simply reactive bone formation, contributing to cam-type deformity in the athletic subgroup[31].

CONCLUSIONS AND FUTURE DIRECTIONS

Groin pain is common in athletes. The overlap between hip pathology and groin pain is a common clinical scenario with sports injuries. The prevalence of FAI in the younger asymptomatic population is high. In addition, FAI is probably responsible for the majority of what was formerly considered idiopathic hip joint OA. The prevalence rates of cam FAI in younger patients are higher, with 25% for males and 5% for females. However, when compared to the prevalence rates for hip arthrosis (3 to 6%) in Caucasians, this indicates that additional factors also play a role. Improvements in the biomechanical understanding of the anatomy and motion or static stress required to cause chondrolabral damage will help to explain which patients ultimately develop pain or OA. Additional studies using high-quality axial CT or MR images and incorporating long-term follow-up are needed for determining the true prevalence of FAI as well as monitoring disease progression.

Although our understanding of FAI continues to improve, we still have much to learn as the natural history of FAI is unknown. Radiographic identification of subtle alterations in bony morphology consistent with FAI may aid in identifying a hip 'at risk' of FAI, but these findings are not necessarily diagnostic. Several studies have helped to establish normal values for radiographic parameters of FAI in asymptomatic subjects. Our understanding of the radiographic findings of FAI in athletic populations remains even more limited. Athletes may be at a greater risk of FAI, particularly those involved in repetitive hip flexion and/or internal rotation[5]. Some authors have suggested a link between aggressive adolescent sport training and the development of bony changes of FAI[30]. There is evidence suggesting that genetic factors may influence the development of FAI, although it appears the evidence is stronger for dysplasia than for FAI.

Although large-scale studies of radiographic findings of FAI in elite athletes may not be feasible, investigations into clinically available information can be helpful. While it is clear that structural hip deformities (FAI) are frequent and can lead to hip damage, the association between FAI and OA must be more closely elucidated. The impact of treatment options on the natural history of disease must be evaluated, along with the role of surgical and non-surgical measures in slowing disease progression. These studies should examine the potential for prevention, especially in young athletes.

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