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# The clinical presentation, diagnosis and pathogenesis of symptomatic sports-related femoroacetabular impingement (SRFAI) in a consecutive series of 1021 athletic hips

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**Abstract**

**Aim:** To examine the pathogenesis and clinical presentation of sports-related femoroacetabular impingement (SRFAI) in a large consecutive series of symptomatic athletes.

**Methods:** Between January 2009 and February 2017 prospectively collected data from competitive athletes within the Gaelic Athletic Association (GAA), and who subsequently underwent arthroscopic treatment for symptomatic FAI, were analysed. Data was collected using internationally validated health questionnaires (Harris Hip Score, UCLA, SF-36, WOMAC) and recognised clinical (ROM, symptom presentation, provocation tests) and radiological (AP pelvis, Dunn, False profile) indicators/measures of FAI.

**Results:** A total of 1021 consecutive cases (mean 26.6 $\pm$ 6.2 years) were included. In every case, conservative treatment failed to resolve symptoms with athletes attending an average of 2.4 $\pm$ 1.1 health care professionals prior to referral. Symptoms developed gradually (78%) and consisted primarily of groin pain (76.1%) and hip stiffness (76.5%) following activity.

An acetabular rim deformity (pincer) was present in all cases; a cam deformity in 72.1%. The prevalence and degree of cam deformity increased with progressing age groups ($p < 0.001$); mean lateral centre-edge angle remained static ($p = 0.456$). Increasing CEA, alpha angle and presence of rim fracture was associated with a reduction in all ranges of hip movement ($p < 0.001$).

**Conclusion:** Symptomatic SRFAI presented in this large series of GAA athletes failed to resolve with non-operative treatment. Increasing hip deformity resulted in poorer ROM. Abnormal acetabular morphology remains static with increasing athletic age while cam deformity is progressive and most likely a secondary pathology.

**Keywords**

Diagnosis, FAI, hip arthroscopy, hip impingement, sports injury

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## Introduction

Gaelic football and hurling are 2 of Ireland's fastest and most popular field sports with more than 2600 Gaelic Athletic Association (GAA) clubs located within Ireland and internationally.[1] Although not classed as 'professional' sports, the intensity and commitment with which GAA athletes train and compete is comparable to professional athletes.[2] The typical GAA culture follows that athletes become involved from a very young age, often competing at school, club and inter-county levels, simultaneously.

Hip and groin pain is a highly prevalent disabler among athletes, particularly those involved in activities requiring rapid bursts of acceleration/deceleration, twisting/turning,

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jumping and kicking movements. Femoroacetabular impingement (FAI) in particular has emerged as one of the leading contributors to progressive pain and stiffness, loss of range of motion of the hip joint, reduced function and sports activity dropout.[3,4] Characterised by a disruption to the natural mechanics of the hip joint through abnormal morphology at either the femoral head-neck junction (cam deformity) or acetabular rim (pincer deformity), the presence and consequence of FAI within the GAA has received little attention to date.

The pathogenesis of FAI is postulated to be multifactorial. A number of researchers have attempted to establish a genetic link;[5–9] childhood developmental abnormalities such as slipped capital femoral epiphysis and Perthes' disease have also been attributed to the development of a cam-like deformity capable of causing impingement.[7,10]

Pincer impingement resulting from morphological changes to the acetabulum, such as acetabular retroversion, coxa profunda and protrusio acetabuli, may also be considered the result of a developmental process, however studies examining its effect among athletes are limited.[11]

What appears a greater catalyst, however, toward the development of FAI-specific hip deformity is engagement in high impact physical activity,[7,12,13] particularly at a young age during the critical period of skeletal development.[14–16] The nature of development of cam deformities in particular, during this growth phase, has been explored in athletes with a cam becoming radiographically visible from as early as 12–14 years.[13,17]

This study aims to examine a very specific athletic population with the aim of highlighting the characteristic clinical symptoms and signs, and provide insight into the pathogenesis of sports-related FAI (SRFAI). In particular, 3 areas were considered for special analysis: (1) the relationship between age of participation and sporting intensity with the development of abnormal bony morphology; (2) the relationship between progressive abnormal bony morphology with increasing athletic age; and (3) the impact of abnormal bony morphology on range of hip motion and level of clinical symptoms and signs.

To our knowledge this is the largest study to date examining the pathogenesis and clinical presentation of symptomatic FAI, in a consecutive series of competitive athletes.

## Methods

All data presented and analysed were prospectively collected between January 2009 and February 2017 at a single institution. We included athletes competitively involved in either of the primary GAA codes who subsequently underwent arthroscopic treatment for symptomatic FAI under the care of a single experienced hip surgeon (PC), with a final study cohort of 700 athletes (1021 symptomatic hips). Patients provided written consent for the use of their collected data which received institutional board approval.

Internationally validated health questionnaires comprised the Harris Hip Score, University of California at Los Angeles (UCLA) Activity Scale, 36-item Short-Form Health Survey (SF-36) and the Western Ontario and McMaster Universities Osteoarthritis Index (WOMAC) and were completed by athletes at the initial consultation. A full clinical assessment, including a structured patient history and physical examination was undertaken and recorded. Athletes were specifically assessed for classical signs and symptoms commonly associated with chronic hip and groin pathology. Hip joint range of movement (ROM) was measured by 2 examiners using a hand-held goniometer with the patient lying supine (hip flexion, abduction) and with the knee and hip flexed to 90° (adduction and internal/external rotation).

Hip provocation tests including FADIR (90° flexion, adduction, internal rotation) and FABER (90° flexion, abduction, external rotation) were performed and considered positive if hip/groin pain was reproduced on testing.

Plain radiography (including standardised anteroposterior [AP] pelvis, 90° Dunn and False profile (FP) view)[18–20] was utilised to establish the nature and extent of bony impingement. The alpha angle, lateral centre-edge angle (LCEA), Tönnis grade and the presence/absence of a crossover sign were assessed from the standardised AP pelvic x-ray;[21,22] the alpha angle (Dunn) was measured using the technique described by Notzli et al.[18] from the 90° Dunn view. A cam deformity was considered present if the alpha angle was $>55°$ (Dunn view) or $>65°$ (AP view). A pincer deformity was considered to be present when a crossover sign was evident on the AP pelvis or a clear bony prominence or rim fracture was observed on the acetabular rim, on the FP view. The degree of acetabular coverage of the femoral head was determined by measuring the LCEA on the AP view: 25–30° (normal); $>30°$ (overcoverage); $<25°$ (dysplasia).[23]

### Statistical analysis

Statistical testing was performed using SPSS v.25 software. Differences between 2 or more groups was assessed using the t-test and ANOVA with post hoc analysis, respectively for parametric data and Mann-Whitney U-test for non-parametric data. Correlation was assessed between continuous variables with Pearson's (parametric) or Spearman's test (non-parametric). A $p$-value of $<0.05$ was considered significant.

## Results

A total of 700 GAA athletes (1021 symptomatic hips) comprised of 93.9% males were included in the study (Table 1). Gaelic football was reported as the main sport in 48% of cases and hurling in 52% of cases with an average age commencing competitive play at 7 years of

**Table 1.** Sporting demographics: including sporting frequency and ability data at the time of initial consultation.

| CATEGORY | VALUE |
|---|---|
| **Age** (years) (mean, SD) (range) | 26.6 (SD 6.2) (14.6–48.2) |
| **Age commence sports** | 7 years ± 2.3 years |
| **Symptoms at consultation (region)** | Hip (99.6%); Groin (31%); Adductor (5.6%); Knee (0.7%) |
| **Position played** | |
| ▪ Forward/Offense | 359 (36.3%) |
| ▪ Midfield | 211 (21.4%) |
| ▪ Back/Defense | 382 (38.7%) |
| ▪ Goalkeeper | 29 (2.9%) |
| ▪ Other | 7 (0.7%) |
| **Training frequency** | |
| ▪ 1–2 days/week | 140 (14.1%) |
| ▪ 3–5 days/week | 730 (73.4%) |
| ▪ >5 days/week | 124 (12.5%) |
| **Match play frequency** | |
| ▪ 1–2 matches/month | 183 (18.5%) |
| ▪ 3–5 matches/month | 579 (58.6%) |
| ▪ >5 matches/month | 226 (22.9%) |
| **Sporting ability** | |
| ▪ Able to play full match? | Yes (36.9%); No (63.1%) |
| ▪ Fully participate in training? | Yes (28.5%); No (71.5%) |
| ▪ Fully participate in recreational? | Yes (41.9%); No (58.1%) |
| ▪ Sprint? | Yes (43.9%); No (56.1%) |
| ▪ Kick a long ball? | Yes (43.5%); No (56.5%) |
| **Competitive sports played** | |
| • Hurling | 639 (62.6%) |
| • Gaelic football | 752 (73.7%) |
| • Soccer | 329 (32.2%) |
| • Rugby | 74 (7.2%) |
| • Running/Athletics | 124 (12.1%) |
| • Other | 107 (10.5%) |
| **Multiple competitive sports** | 1 sport (35.7%)<br>2 sports (38.4%)<br>3+ sports (26%) |

SD, standard deviation.

age ± 2.3 years. The average age at time of presentation overall was 26.6 years (range 14.6–48.2 years, standard deviation [SD] 6.2); with no difference between males and females ($p = 0.650$); 290 athletes (41.4%) were <25 years, 337 (48.1%) from 25–34 years and 73 (10.4%) 35 years and over. There were 379 (54.1%) unilateral and 321 (45.9%) bilateral cases; in athletes with unilateral surgery the right hip was operated on in 57.8% and left side in 42.2%.

For all athletes, conservative treatment failed to resolve their symptoms. Athletes attended at least 1 health care professional (HCP), (average 2.4 ± 1.1) for conservative treatment prior to referral to our clinic; 39% attending at least 3 different HCPs for treatment (90% physiotherapist, 57.1% general medical practitioner [GP], 24.2% sports doctor, 23.2% physical therapist, 27.9% other).

At the time of initial consultation, athletes presented with a wide range of symptoms relating to SRFAI (Figure 1). The most consistent symptoms of SRFAI were pain and stiffness in the hip joint both during and following activity. Symptoms developed gradually in 78.7% of cases; the overall duration of symptoms was <6 months in 21.3% of athletes, 6–12 months (23.4%), 1–2 years (24.0%), 2–5 years (22.8%) and >5 years (8.5%).

FADIR impingement test was positive in 69.4% of cases. FABER test was positive in 41.6% of cases. The passive log roll (internal rotation of the extended leg) elicited pain in only 9.3% of cases.

In general, a reduction in the range of hip motion ($n = 1000$) was a consistent feature of the clinical examination of the symptomatic hip; mean ROM (with standard deviation) of hip flexion was 113.3° ± 11°; internal rotation 23.6° ± 10.9°; adduction 21.0° ± 7.9°; external rotation 37.0° ± 8.2° and abduction 45.8° ± 9.3°. Females in general displayed greater ROM than males ($p = 0.025$) but this difference only reached statistical significance specifically for internal ($p = 0.000$) and external ($p = 0.048$) hip rotation (Table 2). In athletes undergoing unilateral surgery, the asymptomatic contralateral hip demonstrated better preoperative ROM ($p = 0.000$), with the exception of external rotation, which showed no significant difference ($p = 0.196$) (Table 2).

Internationally validated health questionnaires were completed by all athletes to assess the effect of SRFAI on their physical and mental well-being; median preoperative HHS score was 81 with an interquartile range (IQR) of (71–93); UCLA Activity Scale: 8 (5–10); SF-36: 73.7 (61.3–84.9); WOMAC: 16 (7–29). Females scored poorer in all preoperative outcome tests compared to males and this difference was highly significant ($p < 0.01$) (Table 3).

Radiographic findings are summarised in (Table 4). The mean alpha angle was 61.7° ± 13.5° (Dunn) and 70.3° ± 17.4° (AP) for males and 47.2° ± 8.4° and 45.8° ± 9.3° for females. Differences in mean alpha angles between genders were highly significant ($p = 0.000$).

In all cases, an acetabular rim deformity was present. The mean LCEA was 34.1° ± 6.7° for males and 33.1° ± 60° for females, demonstrating no significant difference ($p = 0.325$).

As expected, there was a highly significant correlation between the alpha angle on the AP view with the alpha angle on the Dunn view ($p = 0.000$). There was also a statistically significant correlation between increasing LCEA and Alpha angle on both the Dunn view ($p = 0.008$) and AP view ($p = 0.048$).

The prevalence of a rim fracture increased with higher LCEA ($p = 0.012$) and Tönnis grade ($p = 0.00$); mean LCEA was greater in patients with a rim fracture (36.5° SD 7.0) compared with those without a rim fracture (33.6° SD 6.5), ($p = 0.000$).

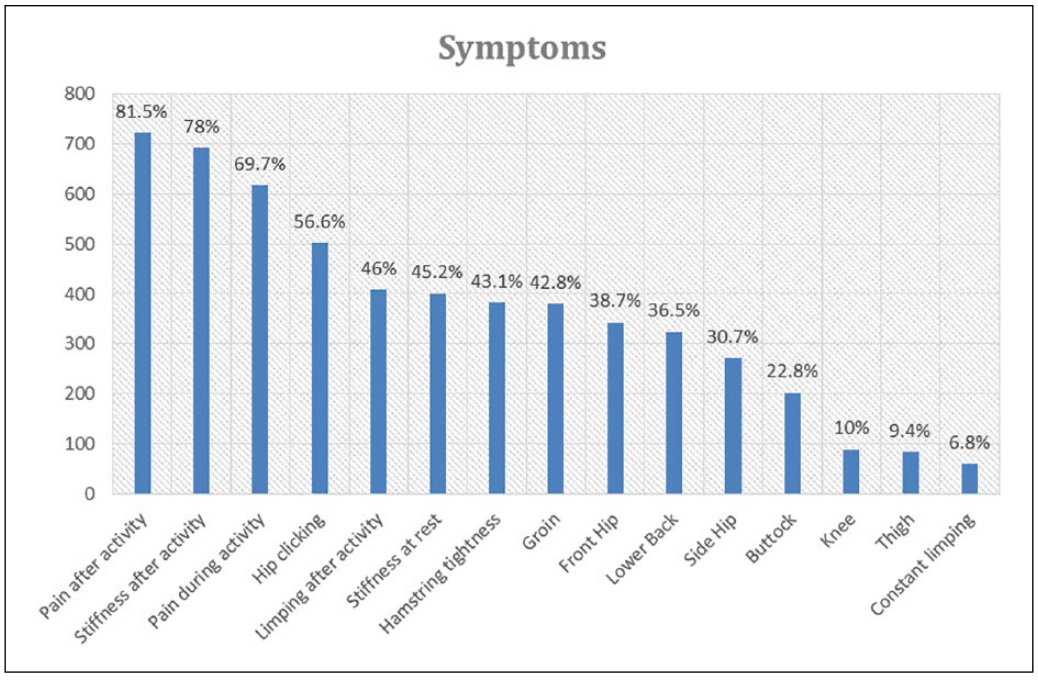


**Figure 1.** Chart above outlines the range of symptoms and regions of pain relating to sports-related FAI (SRFAI) in GAA athletes at initial presentation ($n = 891$).

**Table 2 (a–c).** Mean hip range of movement (ROM).

| | Symptomatic hip | Asymptomatic hip | *p* value | Male hip | Female hip | *p* value | Rim fracture | No Rim fracture | *p* value |
|---|---|---|---|---|---|---|---|---|---|
| | **(a)** | | | **(b)** | | | **(c)** | | |
| **Flexion** | 113.0 ± 11.4 | 118.4 ± 8.2 | *0.00* | 113.2 ± 10.8 | 115.4 ± 15.0 | *0.351* | 112.5 ± 9.9 | 113.5 ± 11.1 | *0.272* |
| **Abduction** | 46.7 ± 10.4 | 49.4 ± 10.1 | *0.00* | 45.7 ± 9.1 | 47.5 ± 11.6 | *0.316* | 45.0 ± 7.7 | 46.0 ± 9.6 | *0.128* |
| **Adduction** | 21.1 ± 8.5 | 24.8 ± 6.4 | *0.00* | 21.0 ± 7.9 | 22.5 ± 9.2 | *0.293* | 19.2 ± 8.2 | 21.5 ± 7.8 | *0.001* |
| **Ext. Rot** | 37.7 ± 8.5 | 38.3 ± 7.6 | *0.19* | 36.9 ± 8.1 | 39.4 ± 9.5 | *0.048* | 35.8 ± 9.4 | 37.2 ± 7.9 | *0.063* |
| **Int. Rot** | 24.4 ± 10.9 | 31.2 ± 8.9 | *0.00* | 23.3 ± 10.7 | 31.3 ± 12.8 | *0.000* | 18.8 ± 10.5 | 24.7 ± 10.6 | *0.000* |
| **Total ROM** | 242.7 ± 34.9 | 262.3 ± 26.8 | *0.00* | 240.0 ± 31.6 | 256.0 ± 44.8 | *0.025* | 231.3 ± 29.2 | 243.0 ± 32.5 | *0.000* |

Rot, rotation; ROM, range of hip motion.
**(a)** Preoperative mean hip ROM with standard deviation, in athletes with unilateral FAI ($n = 367$); comparing hip movements in the symptomatic versus asymptomatic hip joint (with t-test statistical *p* values);
**(b)** ROM among gender, comparing mean ROM of males ($n = 957$) and females ($n = 43$).
**(c)** Group 1 (rim fracture, $n = 175$) had a highly significant reduction in overall ROM measures ($p = 0.000$) when compared with Group 2 (no rim fracture, $n = 836$); a large effect size was observed (Cohen's d, 0.57) and adduction ($p = 0.001$) and internal rotation ($p = 0.000$) were considered clinically significant.

## *The effect of increasing athletic age on the prevalence of abnormal bony morphology*

Participants were divided into 3 groups (group 1: <25 years, group 2: 25–34 years and group 3: 35+ years). The prevalence of a cam deformity increased among the progressing age groups ($p < 0.001$) and alpha angle on AP and Dunn views increased with age ($p < 0.001$); to reduce the potential effect that advanced disease and secondary osteophyte may have on alpha angle measures, further analysis was undertaken with Tönnis 3 grade and older age group (>35 years) excluded; the relationship between increasing age, age groups and cam progression remained highly significant ($p < 0.001$).

A one-way between-groups analysis of variance was conducted to explore the impact of age levels on Alpha angle as measured on the Dunn view. There was a statistically significant difference at the $p < 0.01$ level in alpha angles for the 3 age groups; $F(2, 770) = 4.37$, $p = 0.002$.

Post-hoc analysis revealed the mean alpha angle in the <25 group was significantly different from that of the 25–34 and 35–44 years groups. No difference between

**Table 3.** Preoperative validated outcome scores.

| | *Female (n = 46)* | *Male (n = 975)* | *p value* |
|---|---|---|---|
| **HHS** | 69 (61–79) | 81 (71–93) | <0.01 |
| **UCLA** | 5 (3–7) | 8 (5–10) | <0.01 |
| **SF-36** | 62 (51.8–76.9) | 74 (62–85) | <0.01 |
| **WOMAC** | 33 (13–51) | 16 (7–29) | <0.01 |

HHS, Harris Hip Score; UCLA, University of California at Los Angeles activity score; SF-36, 36-item Short-Form Health Survey; WOMAC, Western Ontario and McMaster Osteoarthritis index.
Note: Female athletes demonstrated poorer preoperative outcome scores when compared to male athletes and this difference was highly significant for all test scores. Median score with interquartile range is displayed.

group 2 and group 3. A similar result was evident for AP view alpha angles ($p < 0.01$) but there was no significant difference between any age groups or with increasing age when analysing LCEA ($p = 0.824$) or prevalence of a rim fracture ($p = 0.264$).

### *The effect of training intensity level on bony morphology of the hip?*

A one-way between-groups analysis of variance was conducted to explore the impact of intensity levels on bony morphology (alpha and centre-edge angle). Participants were divided into 3 groups (group 1: 1–2 days, group 2: 3–5 days and group 3: >5 days). There was no statistically significant difference found between level of intensity (at time of presentation) and increased prevalence of abnormal bony morphology.

### *The effect of bony morphology on the range of hip motion and functional assessment*

The correlation between ROM and abnormal bony morphology was explored using Pearson's and Spearman's rank analysis for parametric and non-parametric data distribution. Increasing LCEA and alpha angle on either view was associated with a statistically significant restriction in all ranges of hip movement ($p < 0.001$), irrespective of Tönnis grade. LCEA and reduced hip abduction was significant at $p < 0.05$ level. Although alpha angles increased with higher Tönnis grades ($p = 0.00$), the LCEA demonstrated no relationship with advancing pathology.

T-test analysis revealed a statistically significant reduction in mean ROM tested in athletes with a rim fracture present ($p < 0.001$) (Table 2). Internal rotation and total range of motion differences were considered clinically significant and independent of Tönnis grade ($p < 0.001$).

There was no correlation with degree of abnormal bony morphology and pre-operative patient-reported outcome scores.

## Discussion

The pathogenesis of FAI in athletes is unknown. It has been proposed that stresses across the developing proximal femoral physis generated from high repetitive activities such as running and kicking may lead to asymmetric growth and a reduced head-neck offset.[24,25] This dynamic and impact-like nature of mechanical loading associated with a variety of impact sports together with the frequency and intensity of this load application could stimulate bone formation.[14,15,26–29] Capsular forces against an immature epiphysis may also induce bone growth and cam formation; decreased acetabular depth has been reported in patients with a cam deformity, potentially exposing the epiphysis to greater capsular contact than subjects with deeper coverage.[30]

Although a genetic predisposition to developing FAI has been proposed,[5–9] the majority of studies investigating the development of FAI in athletes report developmental factors as the main driver; the earlier age of sports commencement and increased intensity of training and playing have been implicated in development of cam deformity.[14,17,27] The type of physical activity may also be effective and a number of different impact sports have been explored for their causal relationship in the development of FAI.[15,26,31]

This study did not demonstrate a relationship between the intensity of regular sports and training (prior to developing symptoms) with the prevalence of cam deformity or increasing alpha angle measured on AP or Dunn views, although all athletes in this cohort were involved in regular competitive sports. The duration of sports involvement (increasing athletic age) was a more important factor in the development of cam morphology. Although much of the focus in the literature has been on cam-type FAI in athletes,[15,17,26,32] the degree of pincer impingement can too be considered developmental in nature.[33,34] An acetabular rim deformity was present in every case and it is our opinion that the rim deformity is the primary cause of progressive and symptomatic FAI in these athletes.

LCEA or the prevalence of overcoverage did not change with increasing age, indicating the coverage of the femoral head is relatively constant and non-progressive, within this cohort of athletes. Abnormal morphology of the acetabular rim was invariably associated with sclerotic, thickened or prominent bone in the subspine region at the attachment of the anterior hip capsule and Iliofemoral ligament (Figure 2). As GAA sports necessitates repeated and excessive strain on the hip capsule during running/sprinting and twisting/turning, the pull of the hip capsule and the strain placed on the anterior labrum intensify the traction forces at their bony attachments which may promote the formation of new bone.[35,36] The anterior acetabular physis (os acetabuli)[37,38] may be particularly prone to this during the rapid growth years of

**Table 4.** Quantification and prevalence of radiographic parameters among GAA athletes (Gaelic football and hurling codes), classified by age, gender and presence of rim fracture.

| | Measure of deformity (mean ± SD) | | | | | | | | | |
|---|---|---|---|---|---|---|---|---|---|---|
| | ALL | Age Groups | | | | Gender | | | Acetabular rim | | |
| | | <25 years ($n$ = 441) | 25–34 years ($n$ = 484) | 35+ years ($n$ = 96) | $p$ value | Male ($n$ = 975) | Female ($n$ = 46) | $p$ value | No rim fracture ($n$ = 836) | Rim fracture ($n$ = 175) | $p$ value |
| Alpha angle (Dunn view) | 61° ± 13.6° | 59.2° ± 13.1° | 62° ± 13.5° | 64.5° ± 15° | *0.001* | 61.7° ± 13.5° | 47.2° ± 8.4° | *0.000* | 59.0° ± 13.3° | 70.0° ± 11.0° | *0.000* |
| Alpha angle (AP view) | 69.3° ± 17.9° | 66.2° ± 17.8° | 71.8° ± 17.5° | 71.5° ± 17.4° | *0.000* | 70.3° ± 17.4° | 45.8° ± 9.3° | *0.000* | 67.4° ± 18.1° | 78.2° ± 13.7° | *0.000* |
| CEA (AP view) | 34.1° ± 6.7° | 34.1° ± 6.3° | 33.9° ± 6.9° | 34.8° ± 7.4° | *0.456* | 34.1° ± 6.7° | 33.1° ± 6.0° | *0.325* | 33.6° ± 6.5° | 36.5° ± 7.0° | *0.000* |
| Radiographic Parameter | Prevalence of radiographic parameter | | | | | | | | | | |
| | ALL | Age Groups | | | | Gender | | | Acetabular rim | | |
| | | <25 years ($n$ = 441) | 25–34 years ($n$ = 484) | 35+ years ($n$ = 96) | p value | Male ($n$ = 975) | Female ($n$ = 46) | p value | No rim fracture ($n$ = 836) | Rim fracture ($n$ = 175) | p value |
| **FEMUR** | | | | | | | | | | | |
| CAM (Dunn view) >55° | 60.7% | 54.4% | 64.4% | 70.7% | *0.000* | 62.7% | 16.2% | *0.000* | 53.9% | 89.9% | *0.000* |
| CAM (AP view) >65° | 61.2% | 53.1% | 67.4% | 67.7% | *0.000* | 63.5% | 4.9% | *0.000* | 56.2% | 84.5% | *0.000* |
| Total CAM (either view) | 72.1% | 66.9% | 75.1% | 81.7% | *0.002* | 74.3% | 22% | *0.000* | 67.2% | 96.6% | *0.000* |
| **ACETABULUM** | | | | | | | | | | | |
| Over-covered (CEA>30°) | 70.1% | 70.5% | 69.5% | 70.7% | *0.660* | 70.5% | 61.0% | *0.065* | 79.7% | 20.3% | *0.005* |
| *Normal (CEA 25–30°)* | 22.7% | 23.4% | 22.7% | 19.6% | | 22.1% | 36.6% | | 88.4% | 11.6% | |
| *Dysplasia (CEA <25°)* | 7.2% | 6.0% | 8.0% | 9.8% | | 7.4% | 2.4% | | 88.6% | 11.4% | |
| Crossover sign | 80.5% | 81.0% | 78.8% | 87.2% | *0.216* | 80.5% | 80.4% | *0.994* | 82.1% | 84.0% | *0.553* |
| Rim Fracture | 17.3% | 16.9% | 18.2% | 14.7% | *0.674* | 18.1% | 0.0% | *0.001* | n/a | n/a | *n/a* |
| **Tönnis Grade:** | | | | | | | | | | | |
| Grade 0 | 68.2% | 80.2% | 60.5% | 51.1% | *0.000* | 67.1% | 91.3% | *0.017* | 86.8% | 13.2% | *0.000* |
| Grade 1 | 20.4% | 15.9% | 22.8% | 28.7% | | 21.0% | 4.3% | | 77.1% | 22.9% | |
| Grade 2 | 9.3% | 3.6% | 13.5% | 14.9% | | 9.6% | 4.3% | | 69.1% | 30.9% | |
| Grade 3 | 2.1% | 0.2% | 3.2% | 5.3% | | 2.2% | 0% | | 61.9% | 38.1% | |

SD, standard deviation.
Note: Chi-square tests for independence is significant if $p < 0.05$.

adolescence when the intensity and frequency of training and competition reaches its greatest (Figure 3).

The study findings would indicate that pincer deformity and alterations in acetabular coverage most likely occur during adolescence and remain largely unchanged following closure of the acetabular physes with skeletal maturity.

The alpha angle in contrast demonstrated a progressive and highly significant increase in mean value and prevalence on both the Dunn ($p < 0.001$) and AP pelvic ($p < 0.001$) views with increasing age of the athlete. This would indicate that development of a cam deformity in athletes may be progressive and not restricted to the early adolescent period.[39]

The cam deformity is most likely a secondary phenomenon and not due to epiphyseal overgrowth;[18,40–42] we believe the cam deformity develops from a combination of repeated impingement from a prominent acetabular rim (pincer deformity) during flexion, adduction and internal rotation (kicking/jumping) and from pulling, rubbing and abrasion of the hip capsule, zona orbicularis and iliofemoral ligament against the femoral head and neck primarily during extension and external rotation (running/twisting).[30]

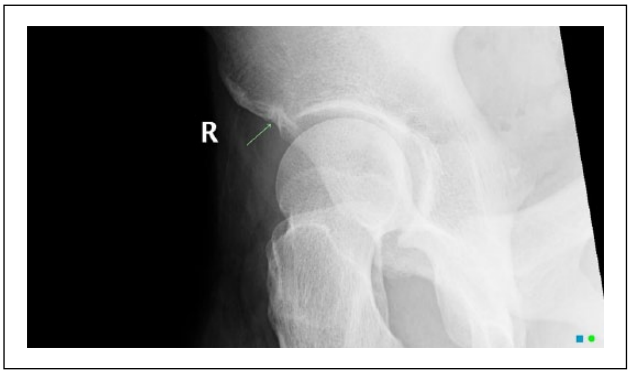


**Figure 2.** Abnormal morphology of the acetabular rim was invariably associated with sclerotic, thickened or prominent bone in the subspine region at the attachment of the anterior hip capsule and Iliofemoral ligament (arrow).

A greater LCEA, indicative of overcoverage, demonstrated a highly significant correlation with increased alpha angle (AP and Dunn) supporting the theory that a cam deformity may develop secondary to recurrent pincer impingement.

The clinical presentation of SRFAI is similar to that described in other studies with chronic hip pain and stiffness the predominant symptom.[11,43,44] Although in a recent consensus statement on the diagnosis of FAI,[45] activity related hip stiffness (1 of the 2 most important symptoms described in SRFAI) was surprisingly not considered a primary symptom. The most clinically relevant provocation test was FADIR which was positive in 69.4% of cases and a reduced range of hip motion was a consistent finding.

The restriction in ROM, although partly due to soft tissue pathology and pain, is primarily due to a mechanical block to natural motion from the abnormal bony morphology of the femoral neck and acetabular rim; increasing LCEA and alpha angle on either view demonstrating a high correlation with increased restriction of hip ROM ($p < 0.001$); the presence of a rim fracture, which may be indicative of a more chronic and severe type of pincer impingement, resulted in a more significant restriction of hip ROM.

## *Limitations*

This is not a longitudinal study so true progression of deformity cannot be assessed, however, the strength of the

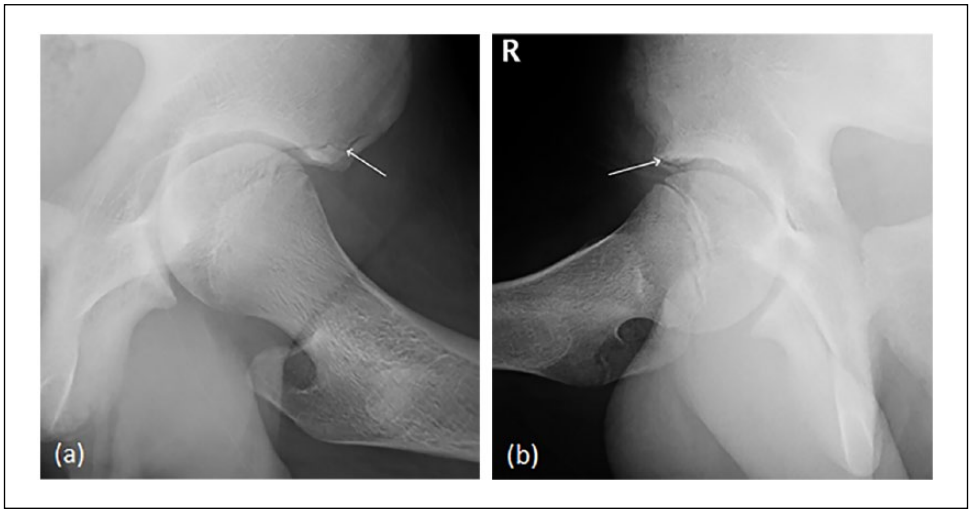


**Figure 3.** False Profile view demonstrating the secondary ossification centre of the anterior acetabular physis (os acetabuli) in (a) 15-year-old and (b) 14-year-old male athletes (arrow).

study is in the large number of competitive athletes with confirmed symptomatic FAI included; comparing prevalence and deformity measures through sequential age groups permits observations which may represent true FAI progression.

## Conclusion

The pathogenesis of sports-related FAI (SRFAI) results primarily from the development of abnormal acetabular morphology during the adolescent period when sporting intensity combined with skeletal growth induces bone formation at the acetabular epiphysis and subspine region. Changes to the LCEA (an indication of femoral head coverage) and prevalence of radiological signs of pincer morphology remain static with increasing athletic age. However, abnormal morphology of the femoral head-neck junction increases with age most likely as a secondary process from recurrent impingement against an abnormal acetabular rim during flexion, adduction and internal rotation (jumping/kicking) and from abnormal capsular abrasion at the head-neck junction with hip extension and external rotation (sprinting/turning).

Hip pain and stiffness during and following activity are the classical symptoms of SRFAI; pain on FADIR and reduced range of hip motion are the classical signs. The reduction in range of hip motion is related to the extent of the abnormal bony morphology. Differences in the intensity of sports demonstrated no significant effect on the development of bony morphology, although all athletes were actively involved in competitive sports prior to developing symptoms. Symptoms of SRFAI had a detrimental effect on many aspects of the athletes' ability to train and play; conservative treatments failed to resolve symptoms in all cases.

SRFAI results in an enormous physical and emotional toll on athletes in the GAA and further studies are needed to examine the true prevalence of SRFAI within the GAA athletic population. The comprehensive findings of this study should help provide important information to health care professionals involved in treating athletes in the GAA (and other similar field sports) regarding the clinical presentation and radiological findings of SRFAI; consideration should also be given to the introduction of hip screening protocols to assist with timely diagnosis, treatment and future prevention strategies.

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