

Cam versus pincer femoroacetabular impingement. Which type is associated with more hip structural damage? An exploratory cross-sectional study

Ashraf Anbar, MD, MRCS^a, Yasser Ragab, MD, PhD^b, Fatma Zeinoh, MD^b, Nashwa El-Shaarawy, MD^c, Yasser Emad, PhD, MD^d, Ihab Abo-Elyoun, MD^d, Hanan Hussein, MD^d and Johannes J. Rasker, MD, PhD^e

^aOrthopedic Department, Faculty of Medicine, Cairo University, Cairo Egypt & Orthopedic Department, Dr. Erfan and Bagedo General Hospital Jeddah, Saudi Arabia

^bRadiology Department, Faculty of Medicine, Cairo University, Cairo, Egypt & Radiology Department Dr. Erfan and Bagedo General Hospital, Jeddah, Saudi Arabia

^cRheumatology and Rehabilitation Department, Faculty of Medicine, Suez Canal University, Ismailia, Egypt

^dRheumatology and Rehabilitation Department Faculty of Medicine, Cairo University, Cairo, Egypt

^eRheumatology Department, University of Twente, The Netherlands

ABSTRACT

Background:

Femoroacetabular impingement (FAI) occurs as a conflict between the proximal femur and the acetabular rim. The purpose of this study was to evaluate MRI findings and look for correlations with pain intensity and duration in each type of FAI separately in an attempt to identify which type is associated with more structural damage.

Methods:

Forty-four patients (78 hips) diagnosed with either cam or pincer FAI were consecutively recruited in a prospective cohort study. None of our patients had evidence of osteoarthritis (OA) on the initial plain radiography. All patients had contrast-enhanced MRI and CT scans of the hips. All patients filled in a visual analogue scale (VAS) for pain.

Results:

The frequency of bone marrow edema (BME) was 37% in cam FAI and 20.8% in pincer FAI. In cam FAI, BME positively correlated with pain severity as measured by VAS ($P < 0.0001$), cartilage degradation ($P = 0.001$), pseudocysts ($P < 0.0001$), hip effusion ($P = 0.013$) and reactive synovitis ($P < 0.0001$). However, in pincer FAI, BME only correlated with pain severity ($P = 0.004$) and duration ($P = 0.011$) and did not correlate with other MRI signs of structural hip damage.

Conclusions:

In cam FAI, BME of the femoral head and neck on MRI positively correlated with chondral damage and synovitis, but not in pincer FAI. This correlation suggests that cam FAI might be

associated with a worse long-term prognosis. This finding might have an impact on clinical practice and decision making as it would encourage surgeons to intervene early in cases of cam FAI, thus preventing the possible development of irreversible, established hip OA.

Key Words

hip impingement, FAI, hip osteoarthritis (OA), bone marrow edema (BME), magnetic resonance imaging (MRI)

INTRODUCTION

Femoroacetabular impingement (FAI) occurs when there is a conflict between the proximal femur and the acetabular rim.¹⁻⁴ It has been suggested that repetitive microtrauma between the femur and the acetabular rim causes tearing of the labral-chondral transitional zone, especially in the anterosuperior region. This may then predispose to degeneration of the adjacent articular cartilage in the form of softening, fraying, and separation. Eventually, articular cartilage detachment or fragmentation may occur, leading to exposure of bone and subsequent development of osteoarthritis (OA).^{5,6}

FAI is a clinical condition that usually presents in physically active adults as intermittent hip or groin pain that is exacerbated by exertion. In recent years, FAI has become increasingly recognized as a potential cause of early-onset OA in nondysplastic hips.^{3,4} It has been classified into three patterns, cam, pincer, or mixed. Cam and pincer FAI can be differentiated on the basis of the site of pathology being either femoral or acetabular, respectively. Cam deformity is an abbreviation for camshaft in which the nonspherical shape of the femoral head at the femoral head-neck junction and reduced depth of the femoral waist lead to abutment of the femoral head-neck junction against the acetabular rim. The name "pincer" deformity describes excess of growth of the acetabular margin, pinching the femoral head. The

Financial Disclosure: The authors report no conflicts of interest.

Correspondence to Ashraf Anbar, MD, MRCS, Cairo University, Faculty of Medicine, Kasr Al-Aini, Manial Elroda, Cairo 11562, Egypt

Tel: +201005520908; fax: +202005520908;

e-mail: ashraf.anbar@gmail.com.

1940-7041 Copyright © 2017 Wolters Kluwer Health, Inc. All rights reserved.

hallmark of pincer FAI is acetabular overcoverage limiting range of motion and leading to a conflict between the acetabulum and the femur. This overcoverage may be diffuse in patients with coxa profunda and protrusio acetabuli or localized in patients with acetabular retroversion.^{1,3,7-9} It is important to identify the type of FAI because surgical treatment differs for each type.¹⁰

Bone marrow edema (BME) is a general term describing an area of low-signal intensity on T1-weighted (w) and high-signal intensity on T2-weighted and short T1 inversion-recovery (STIR) MRI. The term bone marrow edema was first used by Wilson *et al.*¹¹ in 1989 who found ill-defined bone marrow hyperintensities on T2-weighted MRI in patients with knee and hip pain.^{11,12} Within the OA research community the term “bone marrow lesions” is used to describe the MRI findings.¹³ BME affecting the hip joint is neither a specific MRI finding nor a specific diagnosis. It may occur in transient osteoporosis of the hip, avascular necrosis (AVN), trauma, occult stress fracture, infection, myeloproliferative disorders, and infiltrative neoplasms;¹⁴ it may also be primary BME syndrome in the hip, knee, ankle, or foot.¹⁵

The aim of this work was to investigate the pattern and magnitude of hip structural damage associated with each type of FAI before the development of established hip OA. This was achieved by studying pain intensity and duration in association with BME and other MRI signs of inflammation and hip structural damage. To the best of the authors’ knowledge, there has been no similar previous work reported in the literature.

MATERIALS AND METHODS

The design of the study was approved by the local ethics committee and all patients gave informed written consent to be enrolled into the study according to the Declaration of Helsinki. From April 2010 to December 2013, 44 patients, with a total of 78 hips affected with FAI, were consecutively recruited in a multicenter, prospective, cohort study; 31 patients with cam pattern and 13 with pincer pattern (Table 1). The study sample was collected from the authors’ respective institutions after standardization of the imaging protocols. All patients had a history of chronic unilateral or bilateral hip pain with no history of trauma and demonstrated a positive impingement sign. Cases with either cam or pincer pattern were only recruited and mixed cases of FAI were excluded to examine the pathological changes of the hip associated with each pattern of FAI separately. Exclusion criteria were established OA Kellgren and Lawrence grade 2, 3, and 4 on plain radiographs,¹⁶ transient osteoporosis, patients with inflammatory arthropathy involving the hip joint or any arthritis or enthesitis, avascular necrosis, infection, sickle cell disease, previous hip trauma or fracture, neoplasms, infectious disease, regional pain syndrome, previous surgery, radiotherapy, drugs that might affect the blood supply of the femoral head such as corticosteroids and cancer chemotherapy, metabolic diseases like chronic kidney disease and neurological diseases like Charcot’s joints. Pain assessment was performed using 100mm visual analogue scale (VAS).

TABLE 1. Demographic data and MRI signs in patients with femoroacetabular impingement

| Variable | Cam FAI (n = 31) | Pincer FAI (n = 13) | Significance |
|------------------------------|------------------|---------------------|--------------|
| Age (yr) | 44.09 ± 7.23 | 36.38 ± 2.84 | 0.00061** |
| Sex: | | | |
| Male | 23 (74.2) | 11 (84.6) | 0.295 |
| Female | 8 (25.8) | 2 (15.4) | |
| Duration of hip(s) pain (mo) | 31.097 ± 17.87 | 28.38 ± 11.46 | 0.23 |
| FAI Pattern: | | | |
| Unilateral | 8 (25.8) | 2 (15.4) | 0.581 |
| Bilateral | 23 (74.2) | 11 (84.6) | |
| VAS (0-10) | 6.484 ± 1.95 | 5.77 ± 1.83 | 0.26518 |
| BME: | | | 0.5 |
| Unilateral | 12 (38.7) | 5 (38.5) | |
| Bilateral | 4 (12.9) | 0 (0) | |
| Cartilage denudation: | 22 (71) | 3 (23.1) | 0.002** |
| Unilateral | 10 (32.3) | 3 (23.1) | |
| Bilateral | 12 (38.7) | 0 (0) | |
| Labral degeneration | 22 (71) | 8 (61.5) | 0.05 |
| Unilateral | 15 (48.4) | 7 (53.8) | |
| Bilateral | 7 (22.6) | 1 (7.7) | |
| Pseudocysts | 6 (19.4) | 2 (15.4) | 0.65 |
| Unilateral | 7 (22.6) | 1 (7.7) | |
| Bilateral | 4 (12.9) | 1 (7.7) | |
| Osteophytes | 11 (35.5) | 9 (69.2) | 0.18 |
| Unilateral | 7 (22.6) | 4 (30.8) | |
| Bilateral | 4 (12.9) | 5 (38.5) | |
| Hip(s) effusion | 23 (74.2) | 5 (38.5) | 0.002** |
| Unilateral | 16 (51.6) | 3 (23.1) | |
| Bilateral | 7 (22.6) | 2 (15.4) | |
| Reactive synovitis | 15 (48.4) | 5 (38.5) | 0.001** |
| Unilateral | 10 (32.3) | 3 (23.1) | |
| Bilateral | 5 (16.1) | 2 (15.4) | |

** , statistically highly significant.
FAI, Femoroacetabular impingement; VAS, Visual Analogue Scale; BME, bone marrow edema.

Imaging Protocols

All patients had plain radiography, intravenous gadolinium enhanced MRI scan and CT scan.

Plain Radiography Protocol

Imaging included standing anteroposterior and lateral hip cross-table radiographs. The anteroposterior pelvic radiographs were adjusted so that the tip of the coccyx and the center of symphysis pubis were aligned and in the midline, with a distance of 1-1.5 cm between them.¹⁷ Frog lateral radiographs were not used as they lack reliability.¹⁸

CT Protocol

Axial CT at 2mm intervals from just above the articular surface to the distal end of the socket was obtained.¹⁹ An axial oblique plane parallel to the axis of the femoral neck and running through the center of the head was used as a reference plane for measuring alpha angle (AA),² anterior femoral distance (AFD),²⁰ anterior femoral head-neck offset (AFHNO),^{1,21} and acetabular depth (AD). The value of AD was considered positive if the center of the femoral head was

lateral to the line connecting the anterior and posterior acetabular margins.⁸ The acetabular version (AV) angle was measured using the method described by Reynolds *et al.*¹⁹

MRI Protocol

A 1.5 TESLA MR Unit was used in all MRI studies. After acquisition of sets of transverse T1-weighted scout MRI and comparative coronal T1-weighted MRI of both hips, coronal T1-weighted images of the symptomatic hip or hips were systematically obtained by a surface coil wrapped around the symptomatic hip or hips. Sagittal T1-weighted images were obtained in a plane with approximately 10 degrees of internal rotation with respect to the plane perpendicular to the femoral neck. T1-fast spin-echo (FSE)-weighted images were obtained with the following parameters (repetition time ms 350–550/echo time ms 20–25, 14-cm to 20-cm field of view, 3.5-mm to 5.0-mm section thickness, 0.5-mm intersection gap, two to four signals acquired, and a 230 × 256 matrix). Coronal or sagittal images were obtained as necessary, depending on the topography of the marrow changes on the T1-WI. The sagittal plane was favored when femoral head changes predominated anteriorly or posteriorly, and the coronal plane was selected when the predominant area of involvement was the upper aspect of the femoral head. T1-weighted images were obtained again after administration of 0.1 mmol/kg gadolinium in all orthogonal planes, adding fat saturation technique. Other sequences included T2-weighted gradient echo, T2-weighted FSE, fat-saturated fast spin-echo, proton density sequences, and T2-weighted imaging sequences (TR/TE 2,500–3,000/20–25 and 40–60 ms, respectively).^{1,14,22}

Image Analysis and Interpretation

The diagnostic criteria for cam FAI included one or more of the following: a flattened head-neck junction or pistol-grip deformity of the proximal femur,^{4,17} AA greater than 55 degrees,² AFD greater than 3.6 mm,²⁰ and AFHNO less than 8 mm.^{1,21}

The diagnostic criteria for pincer FAI included one or more of the following: central edge (CE) angle greater than 39 degrees,^{21,23} acetabular retroversion; diagnosis by the cross-over sign, the posterior wall sign, AV angle more than 15 degrees of retroversion,^{17,19,24,25} coxa profunda; floor of the acetabulum touched or overlapped the ilioischial line,¹⁷ protrusio acetabuli; contour of the femoral head touched or overlapped the ilioischial line,¹⁷ os acetabulare or ossification of the acetabular rim,⁴ and AD of -5 mm or deeper.⁸

All MRI scans were analyzed with respect to the following pathological features: FAI pattern, diffuse extended BME of the femoral head with or without involvement of the neck, cartilage denudation, subchondral bone marrow lesions (BML), subchondral cysts, osteophytes, acetabular labral degeneration, synovitis (diagnosed on contrast enhanced images) and joint effusion (Figures 1 and 2). All MRI scans were interpreted in accordance with the method described in details by Roemer *et al.*²² To eliminate single-observer bias, images were interpreted independently by two senior

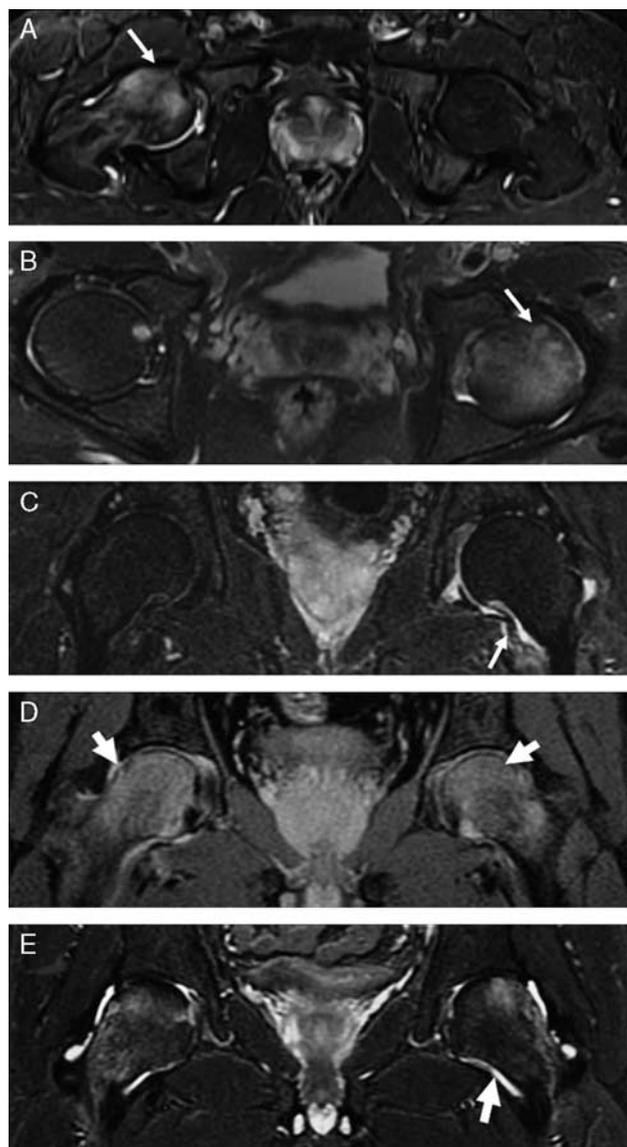


FIGURE 1. Cam FAI. (A) Axial T2-WI FAT SAT showing right hip cam FAI, with femoral head bone marrow edema (arrow). (B) Axial PD FAT SAT showing left hip cam FAI with subchondral pseudocysts (arrow), bone marrow edema and anterior cartilaginous denudation. (C) Coronal T2-WI FAT SAT showing bilateral cam FAI with left hip effusion (arrow). (D) Coronal PD FAT SAT showing bilateral cam FAI with bilateral femoral head-neck bone marrow edema (arrows) and superior cartilaginous denudation on the right side. (E) Coronal T2-WI FAT SAT showing bilateral cam FAI with bilateral femoral head-neck bone marrow edema and effusion (arrow).

radiologists (YR and FZ) and the interobserver agreement was measured.

We are not aware of a grading system to quantify BME but only clearly visible BME was taken into account.

Statistical Analysis

Data were coded and summarized using SPSS version 12.0 for Windows (Chicago, IL). Quantitative variables were described using mean ± standard deviation (SD) and categorical variables using absolute values and percentages.

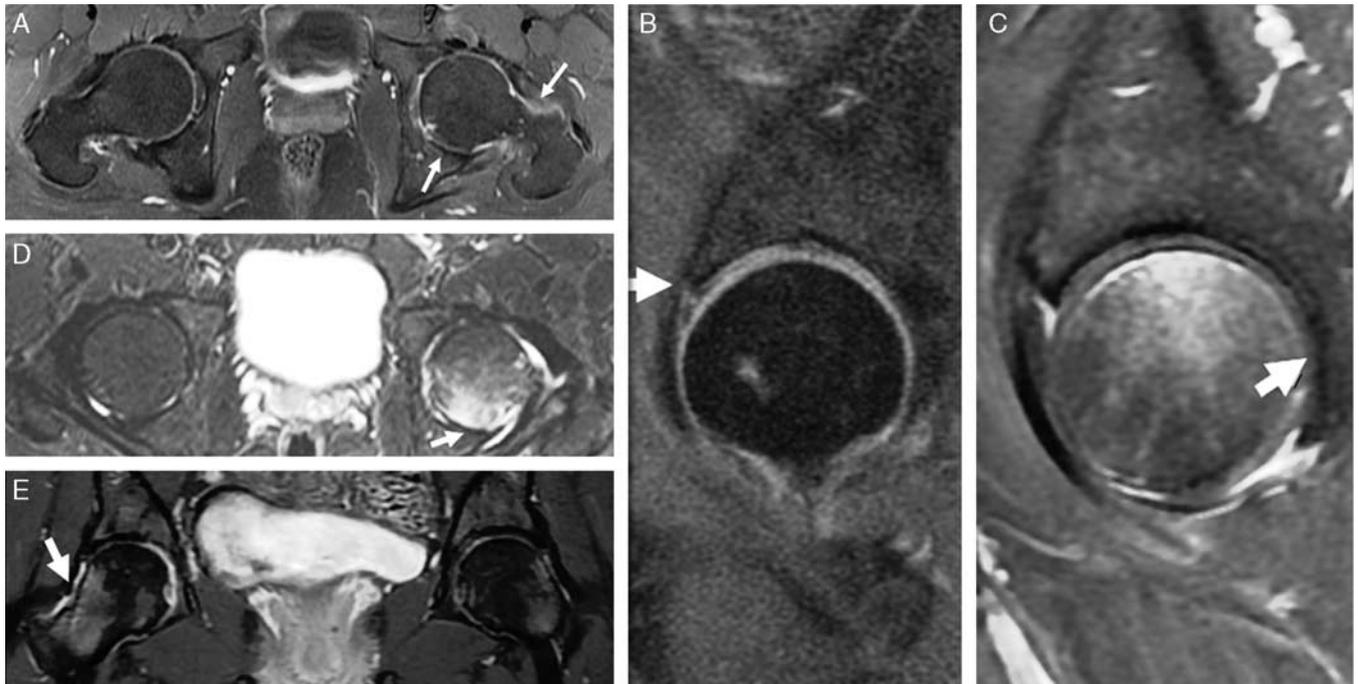


FIGURE 2. Pincer FAI. (A) Axial post contrast T1-WI FAT SAT showing pincer FAI with head overcoverage resulting in posterior cartilaginous denudation (lower arrow) and synovial enhancement denoting reactive synovitis (upper arrow). (B) Sagittal T2 FAT SAT showing acetabular labrum signal alteration denoting degeneration (arrows). (C) Sagittal PD FAT SAT, showing posterior cartilage denudation (arrow) and bone marrow edema. (D) Axial PD FAT SAT showing left pincer FAI as denoted by long abutting posterior acetabular rim (arrow), with adjacent bone marrow edema of the femoral head and hip effusion. (E) Coronal T2-WI FAT SAT showing right hip pincer FAI with minimal hip effusion and bone marrow edema of the head-neck junction.

Associations between categorical groups were tested using the chi square test with Fisher's exact test as appropriate. Spearman's rank correlation test was used as a measure of association of quantitative variables. In all tests P values < 0.05 were inferred as statistically significant. Interobserver agreement between the two radiologists was measured by Kappa coefficient. The interobserver agreement was assessed using Landis and Koch scale who characterized values less than 0 as indicating no agreement, 0–0.20 as slight, 0.21–0.40 as fair, 0.41–0.60 as moderate, 0.61–0.80 as substantial, and 0.81–1 as almost perfect agreement. This is an exploratory study, so we had no idea what to expect and no power analysis could be done.

RESULTS

Seventy eight hips with FAI in 44 consecutive patients were prospectively studied, 31 patients (54 hips) with cam FAI (23 men 74.2% and eight women 25.8%) with mean age 44 yr ($SD \pm 7.23$), and 13 patients (24 hips) with pincer FAI (11 men 84.6% and two women 15.4%), with mean age 36.4 yr ($SD \pm 2.84$). The mean duration of hip pain was 31 mo ($SD \pm 17.87$) and 28.38 mo ($SD \pm 11.46$) in cam and pincer FAI patterns, respectively. In cam FAI, eight (25.8%) were unilateral and 23 (74.2%) were bilateral, whereas in pincer FAI, two (15.4%) were unilateral and 11 (84.6%) were bilateral (Table 1). The frequency of BME was 37% ($n = 20$) in hips with cam FAI and 20.8% ($n = 5$) in hips with pincer FAI.

The frequencies of labral degeneration and pseudocysts were comparable in both FAI types, whereas osteophytes were more frequent in pincer FAI albeit a nonsignificant difference. Cartilage denudation, hip effusion and reactive synovitis were observed significantly more in cam than in pincer FAI (Table 1).

Out of the 54 hips affected with cam FAI, the classic "pistol grip" deformity caused by a moderate to large lump at the femoral head/neck junction was found in 18 hips only. The remaining 36 hips showed a flattened head-neck junction, with an AA greater than 55 degrees, AFD greater than 3.6 mm and AFHNO less than 8 mm. The most frequent anatomical abnormality associated with pincer FAI was acetabular retroversion (14 hips, 58%; Table 2). Coxa profunda was found bilaterally in two male patients and was associated with CE angle greater than 39 degrees (Table 2). In addition, one of those two showed elongated superior acetabular lips and the other showed BME of the posterior part of left femoral head and neck on MRI.

Cartilaginous lesions in cam FAI were predominant in the anterosuperior part of the hip (Figure 1) whereas in pincer FAI they were predominant in the posterior part of the joint (Figure 2). In cam FAI, BME positively correlated with pain severity as assessed by VAS ($r = 0.659$, $P < 0.0001$), cartilage denudation ($r = 0.569$, $P = 0.001$), pseudocystic bone erosions ($r = 0.679$, $P < 0.0001$), hip effusion ($r = 0.442$, $P = 0.013$) and reactive synovitis ($r = 0.613$, $P < 0.0001$). However, in pincer pattern BME only correlated with pain severity ($r = 0.732$, $P < 0.004$) and pain duration ($r = 0.676$,

TABLE 2. The breakdown of radiographic criteria for pincer femoroacetabular lesions

| Cause of overcoverage | No. | Sex | Affected side |
|----------------------------------|-----|-----|---------------|
| Acetabular retroversion | 7 | M | Bilateral |
| Coxa profunda* | 2 | M | Bilateral |
| Protrusio acetabuli | 2 | F | Bilateral |
| Over growth of acetabular rim | 1 | M | Unilateral |
| Ossification of acetabular rim** | 1 | M | Unilateral |

*Associated with CE angle > 39 degrees.

**On the right side of a case of bilateral acetabular retroversion with asymptomatic left side.

$P=0.011$) (Table 3). Interobserver agreement between the two radiologists was found to be almost perfect ($K=0.84$).

DISCUSSION

FAI can cause hip pain in young adults and predispose to hip structural damage and OA.^{3,26,27} Morphological changes associated with FAI have been identified in asymptomatic children as early as the age of 10yr,²⁸ as well as asymptomatic adults^{27,29} and may be genetically influenced.³⁰ The current study was conducted on a series of patients with no apparent structural damage on the initial plain radiography. The aim of this work was to study the pattern and magnitude of hip structural damage associated with each type of FAI separately, before the development of established hip OA. The parameters used to determine disease aggression were pain severity and duration, diffuse BME of the femoral head-neck region and other MRI signs indicative of inflammation and hip structural damage.

The results showed that BME sign in cam FAI positively correlated with cartilage denudation, pseudocystic bone erosions, hip effusion, and reactive synovitis, as opposed to pincer pattern. To the best of the authors' knowledge no previous studies found these associations.

BME is a common finding when patients with knee pain are evaluated by MRI. The typical MRI signal patterns for

BME are nonspecific, however, and occur in several diseases of the knee. Painful BME of the knee joint can be categorized into three distinct etiological groups: ischemic BME (bone marrow edema syndrome, osteochondritis dissecans, complex regional pain syndrome), mechanical BME (bone bruise, microfracture, stress-related BME, and stress fracture), and reactive BME (inflammatory gonarthrosis, degenerative gonarthrosis, postoperative BME, and tumor-related BME).³¹

The pathophysiology of pain in BME syndromes is poorly understood and thought to be multifactorial. Thus, increased intraosseous pressure, with irritation or disruption of sensory nerves within the bone marrow, venous hypertension, raised focal bone turnover with or without microfractures and irritation of the periosteum and periarticular structures could all be possible mechanisms.^{32,33}

In the current study, the frequencies of labral degeneration and pseudocysts were comparable in both FAI types, whereas osteophytes were more frequent in pincer FAI albeit a nonsignificant difference. Cartilage denudation, hip effusion, and reactive synovitis were significantly more in cam than in pincer FAI (Table 1).

The current study also showed that cartilaginous lesions in cam FAI were predominant in the anterosuperior part of the hip (Figure 1B and D), whereas in pincer FAI they were predominant in the posterior part of the joint (Figure 2A and C). This matches well with the findings of Pfirrmann *et al.*,⁸ who compared characteristic MR arthrographic findings of cam and pincer FAI.⁸ They found that cartilage lesions at the anterosuperior and superior positions were significantly larger in patients with cam FAI than in patients with pincer type, while cartilage lesions at the posteroinferior position were significantly larger and labral lesions at the posterior and posteroinferior positions were more pronounced in patients with pincer FAI than in patients with cam FAI.

According to Schmid *et al.*,³⁴ cartilage lesions seen during surgery (42 hip joints in 40 patients) were located most often in the anterosuperior part of the acetabulum ($n=37$), followed by the posterosuperior ($n=23$), anteroinferior ($n=12$), and posteroinferior ($n=10$) parts of the acetabulum. However, the authors did not specify the locations of

TABLE 3. Correlation of bone marrow edema in both femoroacetabular impingement patterns with age, pain, and structural abnormalities by MRI

| Variable | BME in cam FAI (n=20) | | BME in pincer FAI (n=5) | |
|--------------------------|-----------------------|-----------|-------------------------|---------|
| | r | P | r | P |
| Age | 0.339 | 0.062 | -0.053 | 0.862 |
| Pain severity: VAS(0-10) | 0.659 | <0.0001** | 0.732 | 0.004** |
| Pain duration (mo) | 0.266 | 0.149 | 0.676 | 0.011* |
| Cartilage denudation | 0.569 | 0.001** | -0.217 | 0.477 |
| Labrum degeneration | 0.148 | 0.427 | 0.402 | 0.174 |
| Pseudocysts | 0.679 | <0.0001** | -0.337 | 0.260 |
| Osteophytes | 0.280 | 0.127 | 0.499 | 0.082 |
| Hip effusion | 0.442 | 0.013* | 0.065 | 0.832 |
| Reactive synovitis | 0.613 | <0.0001** | -0.138 | 0.654 |

*, statistically significant.

** , statistically highly significant.

BME, bone marrow edema; FAI, Femoroacetabular impingement; VAS, Visual Analogue Scale.

these lesions in relation to the pattern of FAI. Histopathologically, Wagner *et al.*²⁶ found that cartilaginous pathological changes in patients with FAI were similar to those found in OA. They concluded that an impingement conflict is the possible mechanism of peripheral degeneration of the hip joint.

MRI is an indispensable diagnostic tool that allows a detailed view of joint structural damage occurring in FAI and hip OA.^{8,22,34,35} In the current study, BME was observed in 20 (37%) hips with cam FAI and in five (20.8%) hips with pincer FAI. Importantly, BME in cam FAI positively correlated with the severity of pain assessed by VAS ($r=0.65$, $P<0.001$) and important signs indicative of structural damage observed by MRI as cartilage denudation ($r=0.569$, $P=0.001$) and pseudocystic bone erosions ($r=0.679$, $P<0.0001$). Furthermore, BME in cam FAI was found to correlate with MRI signs suggestive of irritative synovial reaction in terms of reactive synovitis and effusion. Although the incidences of BME in both FAI patterns were comparable ($P=0.5$), BME in pincer FAI was found to correlate positively with pain severity ($P=0.004$) and disease duration ($P=0.011$) but not with signs of structural damage or synovial irritation. From these data, it can be suggested that cam FAI seems to predispose to a more aggressive course of hip structural damage, whereas pincer FAI seems to be a slower disease process. However, a longitudinal study is needed to confirm those suggestions. BME positively correlated with pain severity in both disease patterns in this study. This matches well with the strong evidence in the literature that BME is a painful lesion.^{13,31–33,36} James *et al.*³⁷ studied BME adjacent to areas of fibrocystic changes at the femoral head-neck junction in six patients with FAI. They found that MRI identified fibrocystic changes in all patients, surrounded by variable grades of BME, with five patients demonstrating chondral loss. They concluded that BME is rarely identified around areas of fibrocystic changes in FAI. In the current study, the higher prevalence of BME in both disease patterns observed may be explained by the larger number of cases recruited.

In contrast to the previously published incidence of pincer FAI being more common in women and cam FAI more common in men,^{8,17} in the current study, male sex predominated in both FAI patterns. Nevertheless, the number of pincer FAI cases is relatively small and may not truly reflect sex predominance. It is worth mentioning that the pattern of BME being evaluated is the diffuse edema involving the femoral head and that may extend to the neck region. Unlike BME, subchondral bone marrow lesions (BML) are localized small spots of bone marrow edema-like lesions located always in the subchondral region and indicate, most of the time, the presence of cartilage delamination and deterioration.^{36,38,39} Although BML was included in the initial list of pathologies to be looked at by the radiologists, in the current study in the presence of diffuse BME, it was difficult to be sure about the presence of BML because both lesions have the same signal intensity and the diffuse pattern would hide the localized pattern if present. That is why it was not possible to study the correlation between those two pathological entities.

There is evidence in the literature that coxa profunda alone is unrelated to acetabular overcoverage. Anderson

*et al.*⁴⁰ reviewed hip radiographs of a large series and found that coxa profunda existed in all patterns of acetabular coverage whether normal or under or overcovered. They concluded that coxa profunda alone should not be considered a diagnostic parameter for pincer FAI. In the current study, bilateral coxa profunda was found in two male patients who presented with chronic bilateral hip pain and demonstrated positive impingement sign on examination. In both cases, the CE angle was greater than 39 degrees, confirming overcoverage. In addition, one patient had elongated superior acetabular lips and the other case had BME of the posterior part of left femoral head and neck on MRI.

A limitation of the current study is that it was an exploratory study. Thus, a power analysis could not be performed. However, the study has some strengths. A large series of patients with either types of FAI were diagnosed. There were no refusals to cooperate, and all patients underwent a complete investigation according to a standardized protocol.

A valuable future study would be to reassess the same group of patients after having surgical decompression for FAI and see how this would affect the course of hip structural damage and the associated BME.

In conclusion, we evaluated 78 hips with FAI but no radiographic evidence of OA. In cam FAI, BME of the femoral head and neck on MRI positively correlated with chondral damage and synovitis, but not in pincer FAI. This correlation in cam FAI suggests that this type might be associated with a worse long-term prognosis. However, this needs confirmation by further long-term follow-up studies. This finding might have an impact on clinical practice and decision making as it would encourage surgeons to intervene early in cases of cam FAI thus preventing the possible development of irreversible, established hip OA.

REFERENCES

1. Ito K, Minka MA2nd, Leunig M, *et al.* Femoroacetabular impingement and the cam-effect: a MRI-based quantitative anatomical study of the femoral head-neck offset. *J Bone Joint Surg [Br]*. 2001; 83:171–176.
2. Nötzli HP, Wyss TF, Stoecklin CH, *et al.* The contour of the femoral head-neck junction as a predictor for the risk of anterior impingement. *J Bone Joint Surg [Br]*. 2002; 84:556–560.
3. Ganz R, Parvizi J, Beck M, *et al.* Femoroacetabular impingement: a cause for osteoarthritis of the hip. *Clin Orthop Relat Res*. 2003; 417:112–120.
4. Crawford JR, Villar RN. Current concepts in the management of femoroacetabular impingement. *J Bone Joint Surg [Br]*. 2005; 87:1459–1462.
5. Santori N, Villar R. Arthroscopic findings in the initial stages of hip osteoarthritis. *Orthopedics*. 1999; 22:405–409.
6. McCarthy JC, Noble PC, Schuck MR, *et al.* The Otto E. Aufranc award. The role of labral lesions to development of early degenerative hip disease. *Clin Orthop Relat Res*. 2001; 393: 25–37.
7. Ganz R, Gill TJ, Gautier E, *et al.* Surgical dislocation of the adult hip a technique with full access to the femoral head and acetabulum without the risk of avascular necrosis. *J Bone Joint Surg [Br]*. 2001; 83:1119–1124.
8. Pfirrmann CW, Mengiardi B, Dora C, *et al.* Cam and pincer femoroacetabular impingement: characteristic MR arthrographic findings in 50 patients. *Radiology*. 2006; 240:778–785.

9. Giori NJ, Trousdale RT. Acetabular retroversion is associated with osteoarthritis of the hip. *Clin Orthop Relat Res*. 2003; 417:263–269.
10. Tibor LM, Leunig M. The pathoanatomy and arthroscopic management of femoroacetabular impingement. *Bone Joint Res*. 2012; 1:245–457.
11. Wilson AJ, Murphy WA, Hardy DC, et al. Transient osteoporosis: transient bone marrow edema? *Radiology*. 1988; 167:757–760.
12. Patel S. Primary bone marrow oedema syndromes. *Rheumatology (Oxford)*. 2014; 53:785–792.
13. Felson DT, Chaisson CE, Hill CL, et al. The association of bone marrow lesions with pain in knee osteoarthritis. *Ann Intern Med*. 2001; 134:541–549.
14. Ragab Y, Emad Y, Abou-Zeid A. Bone marrow edema syndromes of the hip: MRI features in different hip disorders. *Clin Rheumatol*. 2008; 27:475–482.
15. Grøvre L, Hasvik E, Rashid HU, et al. Primary bone marrow oedema syndrome: proposed outcome measures for pain and physical functioning. *Rheumatology (Oxford)*. 2014; 53:1910–1911.
16. Kellgren JH, Lawrence JS. Radiological assessment of osteoarthritis. *Ann Rheum Dis*. 1957; 16:494–502.
17. Beck M, Kalhor M, Leunig M, et al. Hip morphology influences the pattern of damage to the acetabular cartilage: femoroacetabular impingement as cause of early osteoarthritis of the hip. *J Bone Joint Surg [Br]*. 2005; 87:1012–1018.
18. Konan S, Rayan F, Haddad FS. Is the frog lateral plain radiograph a reliable predictor of the alpha angle in femoroacetabular impingement? *J Bone Joint Surg [Br]*. 2010; 92:47–50.
19. Reynolds D, Lucas J, Klaue K. Retroversion of the acetabulum. A cause of hip pain. *J Bone Joint Surg [Br]*. 1999; 81:281–288.
20. Lohan DG, Seeger LL, Motamedi K, et al. Cam-type femoroacetabular impingement: is the alpha angle the best MR arthrography has to offer? *Skeletal Radiol*. 2009; 38:855–862.
21. Wenger DE, Kendall KR, Miner MR, et al. Acetabular labral tears rarely occur in the absence of bony abnormalities. *Clin Orthop Relat Res*. 2004; 426:145–150.
22. Roemer FW, Hunter DJ, Winterstein A, et al. Hip Osteoarthritis MRI Scoring System (HOAMS): reliability and associations with radiographic and clinical findings. *Osteoarthritis Cartilage*. 2011; 19:946–962.
23. Kim JA, Park JS, Jin W, et al. Herniation pits in the femoral neck: a radiographic indicator of femoroacetabular impingement? *Skeletal Radiol*. 2011; 40:167–172.
24. Bardakos NV, Villar RN. Predictors of progression of osteoarthritis in femoroacetabular impingement: a radiological study with a minimum of ten years follow-up. *J Bone Joint Surg [Br]*. 2009; 91:162–169.
25. Diaz-Ledezma C, Novack T, Marin-Peña O, et al. The relevance of the radiological signs of acetabular retroversion among patients with femoroacetabular impingement. *Bone Joint J*. 2013; 95-B:893–899.
26. Wagner S, Hofstetter W, Chiquet M, et al. Early osteoarthritic changes of human femoral head cartilage subsequent to femoroacetabular impingement. *Osteoarthritis Cartilage*. 2003; 11:508–518.
27. Hartofilakidis G, Bardakos NV, Babis GC, et al. An examination of the association between different morphotypes of femoroacetabular impingement in asymptomatic subjects and the development of osteoarthritis of the hip. *J Bone Joint Surg [Br]*. 2011; 93:580–586.
28. Monazzam S, Bomar JD, Dwek JR, et al. Development and prevalence of femoroacetabular impingement-associated morphology in a paediatric and adolescent population: a CT study of 225 patients. *Bone Joint J*. 2013; 95-B:598–604.
29. Jung KA, Restrepo C, Hellman M, et al. The prevalence of cam-type femoroacetabular deformity in asymptomatic adults. *J Bone Joint Surg [Br]*. 2011; 93:1303–1307.
30. Pollard TC, Villar RN, Norton MR, et al. Genetic influences in the aetiology of femoroacetabular impingement: a sibling study. *J Bone Joint Surg [Br]*. 2010; 92:209–216.
31. Hofmann S, Kramer J, Vakil-Adli A, et al. Painful bone marrow edema of the knee: differential diagnosis and therapeutic concepts. *Orthop Clin North Am*. 2004; 35:321–333.
32. Korompilias AV, Karantanas AH, Lykissas MG, et al. Bone marrow edema syndrome. *Skeletal Radiol*. 2009; 38:425–436.
33. Starr AM, Wessely MA, Albastaki U, et al. Bone marrow edema: pathophysiology, differential diagnosis, and imaging. *Acta Radiol*. 2008; 49:771–786.
34. Schmid MR, Notzli HP, Zanetti M, et al. Cartilage lesions in the hip: diagnostic effectiveness of MR arthrography. *Radiology*. 2003; 226:382–386.
35. Rakhra KS, Lattanzio PJ, Cárdenas-Blanco A, et al. Can T1-rho MRI detect acetabular cartilage degeneration in femoroacetabular impingement?: a pilot study. *J Bone Joint Surg [Br]*. 2012; 94:1187–1192.
36. Manara M, Varenna M. A clinical overview of bone marrow edema. *Reumatismo*. 2014; 66:184–196.
37. James SL, Connell DA, O'Donnell P, et al. Femoroacetabular impingement: bone marrow oedema associated with fibrocystic change of the femoral head and neck junction. *Clin Radiol*. 2007; 62:472–478.
38. Sharkey PF, Cohen SB, Leinberry CF, et al. Subchondral bone marrow lesions associated with knee osteoarthritis. *Am J Orthop (Belle Mead NJ)*. 2012; 41:413–417.
39. Sowers MF, Hayes C, Jamadar D, et al. Magnetic resonance-detected subchondral bone marrow and cartilage defect characteristics associated with pain and X-ray-defined knee osteoarthritis. *Osteoarthritis Cartilage*. 2003; 11:387–393.
40. Anderson LA, Kapron AL, Aoki SK, et al. Coxa profunda: is the deep acetabulum overcovered? *Clin Orthop Relat Res*. 2012; 470:3375–3382.