

Is Posterior Hip Instability Associated with Cam and Pincer Deformity?

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Abstract

Background Posterior hip instability is an increasingly recognized injury in athletes; however, the function of patients after these injuries and an understanding of the pathoanatomy and underlying mechanism are currently unclear.

Questions/Purposes We determined (1) the function of patients after these hip injuries using validated, self-reported outcome instruments and (2) the specific

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Each author certifies that his or her institution approved the human protocol for this investigation, that all investigations were conducted in conformity with ethical principles of research, and that informed consent for participation in the study was obtained.

This work was performed at the Hospital for Special Surgery, New York, NY, USA.

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pathoanatomy sustained in these events to better understand the mechanism of posterior hip instability.

Methods We reviewed the records of all 22 athletes presenting to our clinics with a posterior acetabular rim fracture confirming a posterior hip instability episode. Radiograph, CT, and MRI findings were documented in all patients. Intraoperative findings were recorded in patients undergoing surgery. There were 19 males and three females with an average age of 22 years (range, 13–31 years). Minimum followup was 2 years (average, 4 years; range, 2–16 years).

Results The mean modified Harris hip score was 94, Hip Outcome Scores for Activities of Daily Living and Sport were 99 and 87, respectively, and 20 of 22 athletes returned to sport. The most common constellation of pathoanatomy was a posterior labral tear with rim fracture, anterior labral tear, capsular tear, ligamentum teres avulsion, and chondral injury of the femoral head with loose bodies. Sixteen of the 18 patients with femoroacetabular impingement (FAI) had a twisting or noncontact mechanism of injury.

Conclusions When posterior hip subluxation is recognized and avascular necrosis avoided, these athletes generally have high functional outcome scores and high rates of return to sport. There is an apparent association between the occurrence of posterior hip instability and the presence of structural abnormalities often associated with FAI, which may contribute to a mechanism of FAI-induced posterior subluxation.

Level of Evidence Level IV, therapeutic study. See the Instructions for Authors for a complete description of levels of evidence

Introduction

The spectrum of posterior hip instability ranges from subluxation to frank dislocation. While the diagnosis of

dislocation is obvious in severe cases, a low-energy hip subluxation instability episode can be surprisingly subtle in its clinical presentation and has been initially misdiagnosed as a simple hip sprain or strain [16]. However, traumatic posterior subluxation of the hip is a potentially severe injury, resulting in osteonecrosis in 7% [14] to 25% [16] of patients. Normally the hip has a deep acetabulum and strong capsulolabral complex that can withstand high joint reaction forces during athletic activity [2, 8]. In the general population, the most common mechanism for hip dislocations has been a high-energy dashboard injury in a motor vehicle accident [9], which overpowers the strong osseous and soft tissue stabilizers of the hip. In athletic competition, however, lower mechanisms of hip instability have been reported [3, 16]. Posterior hip subluxations reportedly occur in athletes of various sports, including football, skiing, rugby, gymnastics, basketball, jogging, soccer, and biking, even with noncontact mechanisms of injury [20].

The increasing case reports of posterior hip subluxation [3, 6, 16, 17] suggest these injuries may manifest by low-energy mechanisms and present subtly as a hip sprain. However, these reports have not generally explained how these injuries occur or how they subsequently affect hip function in these patients. Additionally, there have been no absolute indications for nonoperative versus operative management, making it difficult for the clinician to follow a treatment algorithm. Moorman et al. [16] showed, on the basis of the posterior acetabular lip fracture and the disruption of the iliofemoral ligament, all athletes presenting with this constellation of pathoanatomy can be assumed to have had a transient episode of posterior subluxation. However, the specific injury patterns are unclear, resulting in a lack of understanding of the underlying mechanism in these seemingly low-energy injuries.

Femoroacetabular impingement (FAI) is a clinical syndrome associated with structural abnormalities of the hip causing abnormal contact stresses in the hip that can lead to pain, dysfunction, and early osteoarthritis [7, 11]. FAI can be secondary to structural abnormalities, including cam and/or pincer lesions, both of which restrict hip flexion and internal rotation [5], movements necessary for many athletic maneuvers [7]. It has been our clinical experience that many athletes sustaining a posterior hip subluxation also have preexisting FAI, which may be implicated in the mechanism of these hip injuries. In certain cases, a posterior hip subluxation or dislocation event may be the first manifestation of an asymptomatic cam or pincer structural deformity in competitive athletes [21].

Our purposes were therefore to (1) investigate the function of a cohort of athletes sustaining a posterior hip instability episode and (2) define the constellation of pathoanatomy associated with a posterior hip instability episode in an athletic population to better understand the

underlying mechanism of instability in these seemingly low-energy injuries.

Patients and Methods

We retrospectively reviewed all 25 athletes who presented at our clinics with an acetabular rim or lip fracture on imaging between 1998 and 2010. Based on the evidence of Moorman et al. [16], we presumed all of these occurred during posterior instability episodes. We excluded three patients from the study because their injury occurred during alpine skiing and could potentially be a higher-energy mechanism. This left 19 males and three females with an average age of 22 years (range, 13–31 years). The athletes consisted of 15 football players, two baseball players, two lacrosse players, one basketball player, one soccer player, and one wrestler. Eight of the athletes participated at the professional level, six at the collegiate level, five at the high school level, and three were recreational athletes. The mechanism of injury was a twisting, noncontact injury in eight athletes; fall on a flexed knee with the hip adducted and flexed in six athletes; hyperflexion in five athletes; twisting, contact mechanism in two athletes; and a flexion abduction mechanism in one athlete. All patients presented with groin and/or buttock pain. Additionally, all patients reported aggravation of hip pain with stairs and pivoting maneuvers. Interval from injury to presentation in our clinics was an average of 83 days (range, 2–384 days). Eight of the 22 patients with a frankly dislocated hip required a closed reduction on the field or at the emergency department. They later followed up to one of three authors' clinics (CML, JWTB, BTK). Postinjury MRI was obtained within 4 weeks after injury in all patients but varied based on the interval of presentation to the authors' clinics. The minimum followup was 2 years (average, 4 years; range, 2–16 years).

From the medical records, we extracted demographics, reported mechanism of injury, imaging characteristics, and intraoperative findings (if surgery was performed). We also recorded their return to sport at their preinjury level.

Eleven athletes were treated nonoperatively and 11 athletes were treated operatively using hip arthroscopy. No patients underwent acute (within 6 weeks) hip arthroscopy. Hips that had a closed reduction did not ultimately undergo surgery more frequently (four of eight hips, 50%) than hips that did not require a closed reduction (seven of 14 hips, 50%). Nonoperative treatment included 6 weeks of protected weightbearing with a protective brace and was the same regardless of whether a closed reduction was performed. We instituted posterior hip precautions to avoid deep flexion and internal rotation. Surgical treatment most commonly included treatment of the posterior and/or

Table 1. Patient demographics, structural abnormality, mechanism of injury, associated MRI findings, and intraoperative correlation

Age (years)	Sex	Sport	Structural abnormality	Mechanism	Closed reduction	MRI finding	Intraoperative pathology
18	Male	Lacrosse	Cam Pincer	Noncontact twisting	No	AVN, PLT, ALT, FHCI, LB, LTA, CT	PLT, ALT, FHCI, LB, LTA
27	Male	Football	Cam Pincer	Noncontact twisting	Yes	PLT, ALT, FHCI, LB, LTA, CT	PLT, ALT, FHCI, LB, LTA, CT
17	Male	Lacrosse	Cam Pincer	Noncontact twisting	No	PLT, ALT, FHCI, LB, LTA, CT	PLT, ALT, FHCI, LB, LTA, CT
18	Female	Soccer	Cam	Contact twisting	Yes	PLT, ALT, FHCI, LB, LTA, CT	PLT, ALT, FHCI, LB, LTA
27	Male	Baseball	Cam	Contact twisting	No	PLT, ALT, LTA, CT	No surgery
25	Female	Football	Cam	Noncontact twisting	No	PLT, ALT, CT	No surgery
21	Female	Basketball	Cam	Noncontact twisting	No	PLT, ALT, FHCI, LB, LTA, CT	PLT, ALT, FHCI, LB, LTA, CT
18	Male	Football	Cam	Noncontact twisting	Yes	PLT, ALT, FHCI, LB, LTA, CT	No surgery
16	Male	Football	Cam Pincer	Hyperflexion	Yes	PLT, ALT, FHCI, LB, LTA, CT	No surgery
13	Male	Football	Cam Pincer	Hyperflexion	No	PLT, CT	PLT
13	Male	Football	Cam Pincer	Contact twisting	Yes	PLT, ALT, FHCI, LB, LTA, CT	No surgery
19	Male	Wrestling	Cam Pincer	Extreme flexion abduction	Yes	PLT, ALT, FHCI, LTA, CT	No surgery
13	Male	Football	Cam Pincer	Noncontact twisting	No	PLT, ALT, LTA, CT	No surgery
20	Male	Football	None	Fell on knee with flexed adducted hip	No	PLT, CT	No surgery
26	Male	Football	Cam	Hyperflexion	No	PLT, ALT, LTA, CT	No surgery
25	Male	Football	None	Fell on knee with flexed adducted hip	Yes	PLT, ALT, FHCI, ACI, LB, LTA, CT	PLT, ALT, FHCI, ACI, LB, LTA
31	Male	Football	Cam Pincer	Noncontact twisting	No	PLT, ACI, LB, CT	PLT, ACI, LB
25	Male	Football	Cam Pincer	Noncontact twisting	No	PLT, ALT, FHCI, LB, LTA, CT	PLT, ALT, FHCI, LB, LTA
31	Male	Football	Cam	Hyperflexion	No	PLT, ALT, CT	No surgery
24	Male	Football	Cam	Hyperflexion	No	PLT, ALT, LTA, CT	No surgery
20	Male	Baseball	None	Fell on knee with flexed adducted hip	No	PLT, ALT, LTA, CT	PLT, ALT, LTA
31	Male	Football	None	Fell on knee with flexed adducted hip	Yes	PLT, ALT, FHCI, LB, LTA, CT	PLT, ALT, FHCI, LB, LTA

AVN = avascular necrosis; PLT = posterior labral tear with bony avulsion; ALT = anterior labral tear/injury; FHCI = femoral head chondral injury; LB = loose body; LTA = ligamentum teres avulsion; CT = capsular tear; ACI = acetabular chondral injury.

anterior labral tear with débridement or repair, removal of loose bodies, and treatment of the underlying FAI with resection of the cam and/or pincer lesions (Table 1).

At 6 weeks, we introduced progressive weightbearing, ROM, and a hip rehabilitation program and allowed running at 3 months if there was no evidence of osteonecrosis. Eleven patients underwent arthroscopy at 6 to 52 weeks after initial treatment for a symptomatic loose body

(n = 6), failure of nonoperative management defined as continued pain (n = 4), or sensation of hip instability (n = 1). Postoperatively, patients were evaluated at 10 to 14 days for suture removal, at 6 weeks for ROM check and with repeat radiographs to assess for heterotopic ossification and possible osteonecrosis, at 3 months to advance therapy, at 6 and 12 months to repeat radiographs to assess for any heterotopic ossification and osteonecrosis, and then

yearly thereafter. MRI scans were obtained at 6 to 12 months of followup. Modified Harris hip score (HHS) and Hip Outcome Scores (HOS) for Activities of Daily Living (ADL) and Sport were calculated routinely for all patients evaluated in our clinics [13].

Two of the authors (AJK, MT, nontreating surgeons) evaluated all radiographs and MRIs for FAI and/or osteonecrosis. The diagnosis of osteonecrosis was based on the system of Ficat and Arlet [15], although this scheme is reportedly associated with poor interobserver variability [19]. We defined a cam lesion for FAI as an alpha angle of greater than 45° [12] and a pincer lesion as acetabular retroversion as defined by the crossover, or figure-of-eight, sign on AP pelvic radiographs [12]. Followup MRI at times ranging from 6 to 12 months was available in all patients.

Results

The overall function in athletes with posterior hip instability was generally good, with a mean modified HHS of 94 (range, 90–96), a mean HOS ADL of 99 (range, 98–99), and a mean HOS Sport of 87 (range, 81–100) at an average 4-year followup. Twenty of 22 athletes returned to sport at their previous level. One hip had focal osteonecrosis of 12 by 12 mm. This hip did not require closed reduction at

the time of injury. Three months after injury, this hip was treated surgically with core decompression and also had concomitant arthroscopic anterior labral débridement, posterior lip fracture and labral repair, loose body removal, and ligamentum teres débridement. We advised this athlete not to return to sport, and this athlete had no evidence of secondary arthrosis or femoral head collapse at a 2-year followup. Nine of the 11 operatively treated athletes returned to sport at their previous level. All of the 11 athletes treated nonoperatively returned to sport at their previous level.

Based on the preoperative MRI scans, the most common constellation of pathoanatomy was a posterior labral tear with bony Bankart lesion ($n = 22$), anterior labral tear ($n = 19$), capsular tear ($n = 22$), ligamentum teres avulsion ($n = 17$), and chondral injury of the femoral head ($n = 12$) with loose bodies (Fig. 1). The most common arthroscopic findings included bony Bankart lesion of the posterior labrum, anterior labral tear and/or injury, avulsion of the ligamentum teres, and chondral injury to the femoral head with loose bodies (Fig. 2). Review of plain radiographs demonstrated underlying combined cam- and pincer-type FAI in 10 athletes, cam-type FAI in eight athletes, and no underlying FAI in four athletes (Fig. 3) [4]. All four athletes without underlying FAI had a mechanism of injury consistent with a fall on a flexed knee with the hip

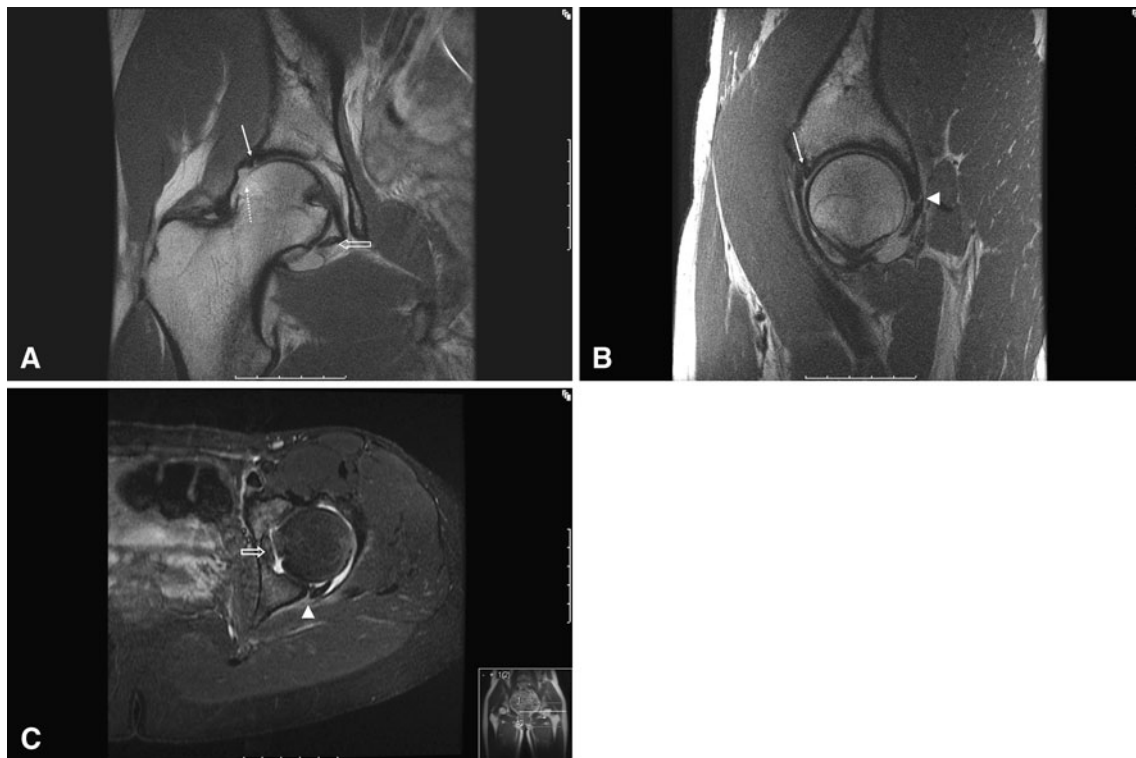
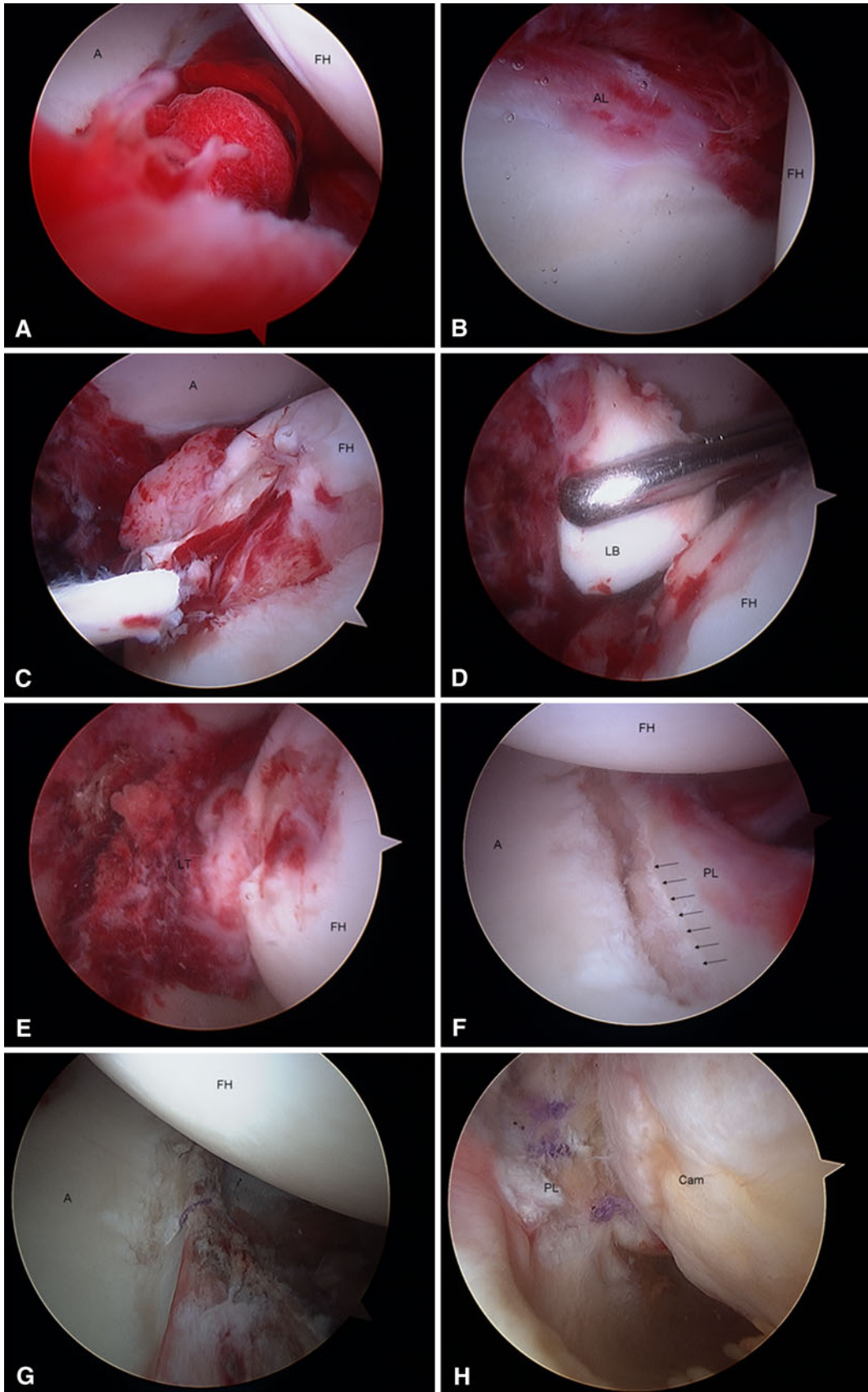


Fig. 1A–C (A) Coronal, (B) sagittal, and (C) axial MR images demonstrate typical findings, including anterior labral injury (arrow), posterior bony Bankart lesion (arrowhead), ligamentum teres avulsion (open arrow), and chondral injury to the femoral head (dotted arrow).



◀ **Fig. 2A–H** Intraoperative arthroscopy photographs through a lateral viewing portal demonstrate (A) synovitis of the hip, (B) anterior labral crush injury, (C) femoral head chondral injury, (D) loose chondral body, (E) ligamentum teres avulsion, (F) posterior labral tear with bony Bankart component (arrows), and (G) posterior labral status postrepair. (H) A view of the peripheral compartment demonstrates chronic cam lesion of the femoral head-neck junction with a view of posterior labral repair. FH = femoral head; A = acetabulum; LT = ligamentum teres; PL = posterior labrum; AL = anterior labrum.

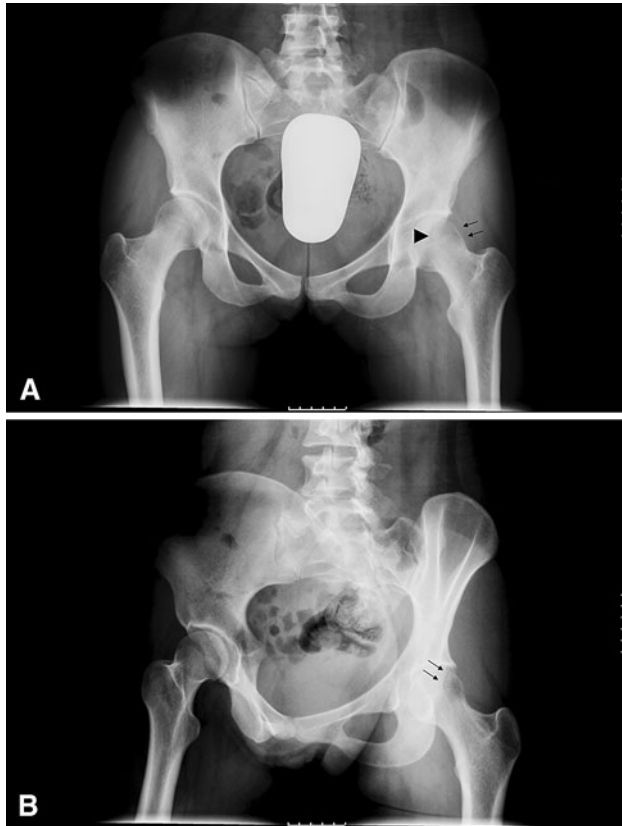


Fig. 3A–B (A) An AP pelvic radiograph demonstrates a subtle posterior wall rim fracture (arrowhead) and underlying cam-type FAI (arrows). (B) An obturator oblique radiograph of the left hip better demonstrates the posterior wall rim fracture (arrows).

in an adducted and flexed position, while 16 of the 18 patients with underlying FAI had a mechanism of twisting or hyperflexion. In athletes with underlying FAI, the average alpha angle was 64° (range, 54° – 80°) [4]. The femoral head-neck offset ratio was an average of 0.12 (range, 0.10–0.20; < 0.17 suggested cam lesion [4]). The average lateral center-edge angle was 26° (range, 22° – 32°). Ten hips had a crossover sign, three hips had coxa profunda, and no hips had evidence of protrusio. CT scans were available for review in 10 patients, and all demonstrated posterior acetabular rim fracture (Fig. 4). We were able to calculate femoral version from the CT scans in 10 hips, with an average of 11° anteversion (range, 6° – 16°).



Fig. 4 A three-dimensional CT scan demonstrates a typical posterior acetabular wall rim fracture (arrows).

Discussion

Posterior hip subluxations have become increasingly recognized in athletes in a variety of sporting activities, even with noncontact mechanisms of injury. Despite these accounts, a lack of functional outcome data, indications for operative and nonoperative treatment, and an understanding of the underlying mechanism in these low-energy injuries remain elusive. In this retrospective study, we (1) described the function of a cohort of athletes sustaining a posterior hip instability episode and (2) defined the constellation of pathoanatomy associated with these injuries.

There were several limitations to this retrospective study. First, the 22 athletes included in this series presented to three different surgeons' clinics. Somewhat different treatment algorithms were utilized and surgical indications varied among surgeons. Nevertheless, these were relatively uncommon injuries, and treatment recommendations were continuing to evolve. Second, there was relatively short-term followup of these athletes. These hips may develop posttraumatic osteoarthritis at further imaging followup. However, our main purpose was to determine patient function, including return to sport, and all patients included in this series had a mandatory 2-year followup. Third, while we demonstrated a clear association between FAI and posterior subluxation of the hip in an athletic population, this study did not definitively prove this mechanism of FAI-induced posterior hip subluxation. Further biomechanical work needs to be performed in this regard.

In our series, 20 of 22 athletes returned to sport. In a study by Moorman et al. [16], eight American football players were treated nonoperatively with 6 weeks of protected weightbearing and rehabilitation program. Six of

these eight returned to full, unrestricted activity at an average of 13 weeks. Unfortunately, two of the hips developed osteonecrosis and subsequently had THA for the treatment of severe hip arthrosis [16]. In a study by Philippon et al. [18], all 14 patients who were treated operatively returned to professional sport at their previous level. None of the patients in their series developed osteonecrosis. In our series, one athlete developed focal osteonecrosis and underwent core decompression and was advised against returning to competition. At a 2-year followup, his radiographs did not demonstrate any secondary arthrosis or femoral head collapse. It has also been our experience that early signal changes (1–3 months after injury) in the femoral head (impaction injuries) are common. In an MRI study of hip dislocation, six of the 14 patients with posterior dislocation had isolated femoral head contusions (trabecular microfractures) [5]. It is important to distinguish these signal changes from osteonecrosis, although the long-term prognosis of trabecular microfractures is unknown. Overall, athletes seem most likely to return to sport if osteonecrosis and subsequent chondrolysis are avoided [6].

The most common pathologic findings in our series included a posterior bony Bankart lesion, anterior labral injury, synovitis, chondral injury to the femoral head with loose bodies, and ligamentum teres avulsion. The triad of findings in eight American football players with posterior hip subluxation has been described as hemarthrosis/effusion, iliofemoral ligament disruption, and posterior acetabular lip fracture [16]. In the series of 14 athletes treated with arthroscopic surgery, Philippon et al. [18] found labral tears, ligamentum teres avulsion, and chondral defects to be common. In their series, five of 14 hips sustained acetabular rim fractures, although none were repaired arthroscopically. Laorr et al. [9] described MRI findings in 18 consecutive posterior hip dislocations, with six of 18 having acetabular rim fractures. The variation of pathoanatomy in the different series of patients may have been attributed to the differing mechanisms of injury, presence or absence of underlying FAI, and the difference in MRI and arthroscopy findings.

We found 18 athletes with structural cam and/or pincer deformities of the hip. This was similar to a study of intraarticular hip pathology in professional athletes who sustained hip dislocation during competition and were treated with arthroscopy. In that series including 12 posteriorly dislocated hips, Philippon et al. [18] found preexisting FAI in nine hips. This may suggest some correlation in that cohort of professional athletes as well, although no mention of this specific mechanism was made. In chronic FAI, a contrecoup lesion in the posterior capsular-labral junction develops by a distraction force due to the femoral head levering out of the hip socket with

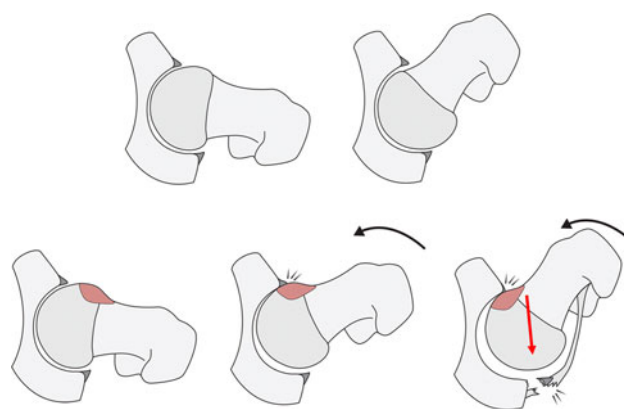


Fig. 5 A diagram illustrates a theorized mechanism of injury for patients with FAI and posterior hip instability. Top row: With normal osseous hip anatomy, internal rotation is not impeded. Bottom row: In patients with cam-type FAI, internal rotation is limited. Attempts at achieving internal rotation cause anterior impingement and levering, which drives the femoral head posteriorly (red arrow).

continued hip flexion [1]. This microlevering of the femoral head caused chronic changes to the posterior acetabular cartilage and labrum [22]. It is logical to deduce, during competition, attempts to achieve increased flexion and internal rotation may cause intrusion of the cam lesion into the hip, creating abnormal anterior contact between the cam lesion and the anterior acetabulum, which could result in levering of the femoral head posteriorly (Fig. 5). This could lead to failure of the soft tissue and osseous structures, with a subsequent posterior acetabular rim fracture and posterior capsulolabral tear. Moorman et al. [16] proposed athletic posterior traumatic hip instability results from more of a classic mechanism of injury similar to that of posterior dislocation in motor vehicle accident, with a posteriorly directed force to an adducted, flexed hip. Six patients in our series had a similar pathomechanism with the hip in an adducted and flexed position. Four of these patients had no preexisting FAI, and we submit that a hip with normal osseous anatomy may subluxate posteriorly with this pathomechanism, agreeing with the original analysis of force vectors of Letournel and Judet [10]. This mechanism is distinctly different from the torsion and hyperflexion mechanism of posterior hip subluxation in the setting of cam and pincer structural abnormalities. Letournel and Judet [10] also stated the type of hip injury sustained depends on the amount and direction of applied force, as well as the position of the hip. They demonstrated through vector analysis the relationship of the position of the leg and pelvis to the injury sustained, accounting for the difference in an anterior dislocation, posterior dislocation, or fracture-dislocation of the hip [10]. Letournel and Judet [10] reported the degree of internal or external rotation of the hip dramatically affects the position of the head within

the acetabulum and therefore the resulting injury complex. Expanding this concept, ultrasonographic measurements performed by Upadhyay et al. [23] on a series of patients who had sustained a posterior hip dislocation demonstrated substantially less anteversion on both the injured and the uninjured sides compared with a control group. They proposed relative femoral retroversion might cause relative medial rotation of the hip, predisposing to posterior hip dislocations. It may be that femoral retroversion exacerbates the abnormal contact between the femur and acetabulum with a cam and/or pincer lesion, making the hip more susceptible to a subluxation event.

There is an increasing recognition of athletic hip subluxation, and clinicians need to be aware of this injury pattern and mechanism. If promptly recognized and osteonecrosis is avoided, athletes generally function well. There is an apparent association between the occurrence of posterior hip instability and the presence of structural abnormalities often associated with FAI, which may contribute to a mechanism of FAI-induced posterior subluxation.

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