[Orthopaedic Surgery]

The Etiology of Femoroacetabular Impingement: What We Know and What We Don't

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Context: Several risk factors may cause femoroacetabular impingement (FAI). Knowledge of causation would identify patients for early intervention, prior to the development of painful intra-articular damage.

Data Sources: PubMed, MEDLINE, EMBASE, and related article reference lists were screened for relevant studies published between January 2000 and December 2013.

Study Selection: Inclusion criteria were (1) etiology of FAI, (2) original FAI clinical data, and (3) English language. Case reports of fewer than 3 patients were excluded.

Study Design: Systematic review.

Level of Evidence: Level 4.

Results: In all, 754 studies were screened, with 18 meeting the eligibility criteria. There were 13 comparative observational studies and 5 case series. The studies pertained to intrinsic patient factors (n = 2), activity/developmental factors (n = 8), hip disease (n = 5), postsurgical changes (n = 2), and malunion after hip fracture (n = 1).

Conclusion: A combination of intrinsic patient and developmental factors, activities involving repetitive hip motion, pediatric hip disease, and hip-related surgical procedures may contribute to the development of FAI.

Keywords: femoroacetabular impingement; systematic review; hip; etiology

Femoroacetabular impingement (FAI) is abnormal contact between the femoral head-neck junction and the acetabulum rim, causing hip pain and dysfunction.[3,9] Over time, the collision between the acetabulum and femoral head-neck may lead to degenerative changes and, eventually, osteoarthritis of the hip.[9]

Two types of FAI are classically described. Pincer impingement is focal or generalized overcoverage of the femoral head by the acetabulum, more common in women.[3,15] Cam impingement is an aspherical femoral head-neck junction, more common in men.[3,15] The large majority (approximately 85%) of patients with FAI have mixed pathology of both cam and pincer impingement.[8,9]

Although certain risk factors may cause FAI, none have been definitively evaluated. Present theories of FAI etiology focus on genetics and associated congenital anatomy,[25] developmental adaptations following activities that involve repetitive supraphysiologic hip motion,[16] or subclinical and overt developmental pathology (such as slipped capital femoral epiphysis).[17,25]

METHODS

A comprehensive search of 3 medical databases (PubMed, MEDLINE, and EMBASE) identified studies on the etiology of FAI published between January 2000 and December 2013. The following broad search terms were used to ensure all relevant articles were included: (femoroacetabular impingement OR FAI) AND (cause OR etiology), using both the appropriate keywords and MeSH/EMTREE index headings. Inclusion criteria were (1)

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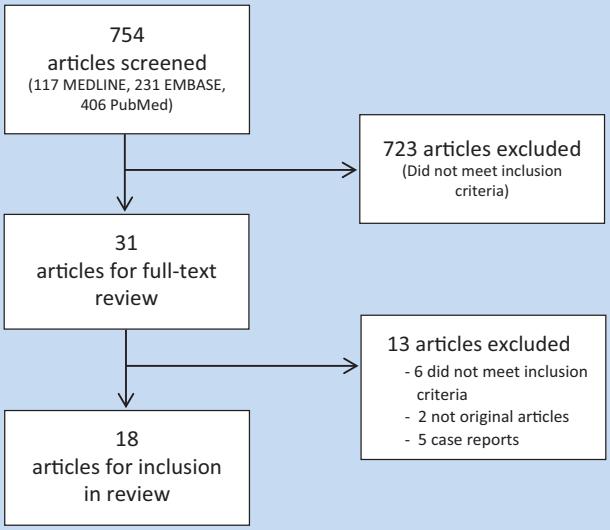

Figure 1. Flow diagram outlining the process through which articles were screened and selected for eligibility.

etiology of FAI, (2) original clinical data, and (3) English language.

RESULTS

A total of 754 titles and abstracts were screened; 31 were included for full-text review. Eighteen articles met the inclusion criteria and were included in this systematic review (Figure 1). Further screening of reference lists of these articles yielded no further articles for inclusion.

Our search yielded no randomized controlled trials or prospective observational cohort studies. There were 13 comparative studies (case-control or cross-sectional studies) and 5 case series.

Intrinsic Factors

Pollard et al[21] recruited 64 patients with a mean age of 38 years with evidence of FAI on supine anteroposterior (AP) pelvis radiographs as well as clinical confirmation with positive anterior impingement. These patients were compared with their siblings and spouses (controls). Siblings were significantly more likely to have both radiographic cam (relative risk, 2.8) and pincer (relative risk, 2.0) deformities compared with controls, as well as clinical signs and symptoms of FAI pathology. The term "genetic" was used by the authors, but the methodology lacked the strength to delineate whether the higher prevalence of FAI among siblings was attributable to genetics, common developmental activities, or some other shared factor.

Men may also have greater inherited risk of cam pathology.[11,12] Hack et al[11] examined 400 hips using magnetic resonance imaging (MRI) and delineated risk factors for the development of cam FAI. Using alpha angles on MRI, men had a far higher frequency of cam deformity (75% vs 25%, $P < 0.001$). Age, body mass index, and ethnicity had no relationship with cam deformities.

A higher prevalence of FAI among those with affected siblings as well as a predilection for cam FAI in the male gender suggests certain intrinsic characteristics to the development of FAI. Further population-based studies are needed to delineate the absolute and relative contributions of inherited/genetic versus acquired characteristics to the development of symptomatic FAI.

Developmental (Activity-Related) Factors

Repetitive physical activity, especially during the developmental years, may predispose to the development of FAI.[16] Most published studies exploring this hypothesis have focused on elite athletes because of the high prevalence of FAI in this population.[10,18]

In a small case-control, cross-sectional study, a significant difference was not found in radiographic (AP and frog-leg laterals) signs of cam FAI between 15 high-level youth soccer players and 14 controls.[12] However, there was a significant difference in prevalence of radiographic cam deformity between male and female players (29 hips vs 17 hips, $P = 0.016$).

In 89 preprofessional soccer players and 92 controls, AP and frog-leg lateral hip radiographs were evaluated for alpha angles exceeding 60° and a flattened or prominent head-neck junction.[1] There were significantly more flattened and prominent head-neck junctions in the athletic group. However, there were no significant differences in radiographic alpha angles for cam FAI.

In a case series of 76 collegiate football players with asymptomatic hips, AP and frog-leg lateral radiographs were examined for cam or pincer FAI.[13] Cam FAI was defined as an alpha angle greater than 50° (on either radiographic view) or a head-neck offset less than 8 mm; pincer FAI was defined as a crossover sign, a later center-edge angle greater than 40°, or an acetabular index less than 0°. Ninety-five percent of hips demonstrated at least 1 radiograph sign on either the AP or frog-leg lateral films of cam or pincer FAI.

In a comparison of 72 elite male basketball players and 76 controls, significantly greater alpha angles in the anterosuperior quadrant of the femoral head on MRI were seen in the athlete group (50.1°-60.5° vs 37.6°-47.5°, $P < 0.001$).[24] There was decreased internal rotation of the hip on clinical examination in elite athletes compared with asymptomatic control hips (18.9° vs 30.1°, $P < 0.001$). After stratifying physeal closure (open vs closed), alpha angle was only significantly greater in the closed physis group. Although the study was not longitudinal in design, these findings suggest that cam FAI may be developmental.

In a subsequent study of 76 elite male basketball players' hips and 76 control hips, the hypothesis was that lateral extension of the femoral head physeal line was responsible for the characteristic "pistol grip" deformity in cam hips.[23] Significantly greater extension of the epiphysis laterally was found in athlete

hips in all positions when physes were open (0.64-0.84 vs 0.49-0.69, $P < 0.001$) but only found in the anterosuperior quadrant when physes were closed (0.70-0.80 vs 0.60-0.75, $P < 0.007$).

Sixty-one asymptomatic youth hockey player hips (player age range, 10-18 years) were compared with 27 control asymptomatic youth skier hips in a comparative cross-sectional study.[20] All hips were evaluated with clinical examination and MRI for signs of FAI. Overall, youth hockey players had significantly greater alpha angles on MRI (55.2° vs 60.1°, $P = 0.05$). A significantly greater number of youth hockey players between the ages of 16 and 18 years had an MRI-measured alpha angle greater than 55° compared with age-matched skiers (93% vs 25%, $P = 0.001$). A significant difference was not detected in the younger age groups (10-15 years). A regression analysis revealed a significant positive correlation between age and alpha angle in youth hockey player hips only ($r = 0.421$, $P < 0.001$), suggesting that a higher prevalence of cam FAI exists in older youth hockey players.

In bilateral hip MRIs of 44 pediatric volunteers (23 with open physes, 21 closed), a cam deformity was only detectable in patients with closed physes.[4] This fact supports the hypothesis that developmental changes during physeal closure lead to the development of the deformity. Furthermore, using the Habitual Activity Estimation Scale, the investigators found that the 3 participants who developed cam deformity had significantly higher daily physical activity (7.1 vs 2.9 hours, $P = 0.02$). Activity level during physeal closure may promote the development of cam deformity.

Based on this limited evidence, patients that are exposed to "at-risk" activities such as basketball, football, or hockey during childhood and adolescence may undergo developmental changes in hip architecture. These sports, which invariably involve repetitive and often supraphysiologic hip rotation and flexion movements, may alter the anatomy of the growing hip. The hip joint may remodel according to the varied stresses applied to it during phases of development. An adaptive response in bony and soft tissues to repetitive activity has been seen in other sports such as baseball.[19,22] These subtle changes may lead to the characteristic FAI deformities and subsequent symptoms.

Sequelae of Pediatric Hip Disease

Slipped Capital Femoral Epiphysis

Childhood and adolescent hip disease may cause FAI.[17,25] Slipped capital femoral epiphysis (SCFE) can alter the shape of the femoral head, which may lead to abnormal contact with the acetabulum and eventual degenerative changes.

Fraitzl et al[7] retrospectively identified 73 patients with unilateral mild SCFE (slip angle <30°) treated with K-wire fixation. Sixteen patients with a mean slip angle of approximately 16° had long-term follow-up with an activity score, clinical examination, and AP/cross-table lateral radiographs, including the contralateral hip. The only significant findings were a reduced head-neck offset (16 hips vs 9 hips, $P < 0.02$) and increased prevalence of head-neck junction bony prominence (13 hips vs 9 hips, $P < 0.03$). Interestingly, these morphologic changes are characteristic of cam FAI.

A retrospective chart review at a mean 6.1 years of 26 male and 39 female hips with SCFE treated with a single cannulated screw found that one third had a positive impingement sign on clinical examination.[5] Almost 70% of these patients also demonstrated a pistol grip deformity characteristic of cam FAI pathology. The study did not demonstrate a correlation between the severity of the initial slip and the subsequent development of FAI.

Fifty-eight hips from 50 patients with remote SCFE (average Southwick slip angle of 32°) were compared with 22 normal hips with AP and frog-leg lateral radiographs.[27] Femoral head ratio, alpha angles, and head-neck offset were all significantly increased in those hips with previous SCFE pathology.

Slipped capital femoral epiphysis is a common pediatric hip pathology that results in bony changes of the femoral head and may contribute to the eventual development of cam FAI.

Legg-Calvé-Perthes Disease

Legg-Calvé-Perthes disease (LCPD) is an idiopathic hip condition, typically affecting children 4 to 12 years of age, in which there is osteonecrosis of the femoral head and progressive deformity. Although LCPD can also lead to the development of clinical FAI, this is likely a different type of impingement than classic cam or pincer impingement. Because of the complexity of the deformities associated with LCPD, the radiographic diagnosis of FAI in these hips is difficult.[14]

In a retrospective study, 13 LCPD hips were compared using computed tomography and computerized 3-dimensional motion analysis to both 27 normal hips and 22 FAI hips.[26] Zones of impingement in LCPD were more likely to be extra-articular on both the femoral and acetabular side. Intra-articular impingement was also more prevalent in LCPD hips. Overall, LCPD hips had more extra-articular and intra-articular zones of impingement than FAI hips, as determined by the virtual motion analysis algorithm.

Bony impingement is an important source of pain in LCPD; however, the nature of impingement does not mimic classic impingement mechanisms seen with FAI. As a result, the relative contribution of LCPD to the development of FAI is not completely understood.

Sequelae of Femoral Neck Fracture

Malunion following femoral neck fractures in young patients could also result in alteration in femoral head-neck contour and hip mechanics.[6]

In a case series of 9 young patients (mean age, 33 years) with previous femoral neck fracture, all suffered groin pain and demonstrated positive anterior impingement tests on physical examination.[6] Eight of 9 patients required operative intervention. All demonstrated an intraoperative flat contour at the femoral head-neck junction along with labral and acetabular cartilage damage, suggestive of FAI. Corrective osteoplasty was

performed on all 8 patients, while 2 required a supplemental intertrochanteric osteotomy to correct residual deformity. Three were pain-free postoperatively, while the other 5 had substantial improvements in pain at a mean 16-month follow-up.

Postsurgical Etiology

Structural anomalies of the acetabulum may be acquired and lead to pincer FAI.[28,29] Forty-six hips in 38 male patients underwent periacetabular osteotomies for hip dysplasia and were followed to determine any difference in pre- and postoperative signs of FAI.[28] There was no change in clinical signs or radiographic parameters that would suggest the acquisition of pincer FAI postoperatively at a minimum of 12 months follow-up.

A total of 115 hips (mean age, 34.7 years) that underwent a rotational acetabular osteotomy for hip dysplasia had a clinical follow-up and radiographic postoperative analysis at a mean 13 years.[29] Only 8 hips demonstrated a preoperative acetabular crossover sign (suggestive of pincer impingement), while 49 hips demonstrated a postoperative crossover sign. The radiographic crossover sign was significantly associated with a positive clinical impingement sign, suggesting acquired pincer FAI. Thus, overcorrection of conditions such as hip dysplasia may lead to acetabular overcoverage and the development of pincer FAI symptoms.

DISCUSSION

The available literature on the etiology of FAI suggests that FAI is likely multifactorial. Intrinsic patient factors (genetic or acquired), repetitive athletic activity, pediatric hip disease, and prior hip surgery may contribute to its development. The combination of inherited or genetic factors with certain types of physical activity—namely sports involving vigorous flexion and rotation, such as basketball, football, and hockey—during the adolescent years may lead to alteration of the anatomy of a developing hip. Childhood hip disease, such as SCFE and LCPD, as well as femoral neck malunion and overcorrection of hip dysplasia may also lead to FAI.

Strengths and Limitations

The primary limitation of this review is the quality of the included studies and the resultant conclusions. There were no prospective observational studies. The included studies have bias commonly associated with level 3 and 4 study designs.[2] Finally, the FAI-related radiographic and clinical parameters were not consistent across studies, limiting the ability to conduct a meta-analysis.

CONCLUSION

Femoroacetabular impingement is defined as abnormal mechanics between the femoral head-neck junction and the acetabulum rim, which can lead to pain and intra-articular damage. Several factors may contribute to the etiology of FAI. Longitudinal and natural history studies will be required to better elucidate the contributions of, and relationships between, genetics, developmental changes, hip disease, and hip-related surgical procedures to FAI.

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