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SYMPOSIUM: FEMOROACETABULAR IMPINGEMENT: CURRENT STATUS OF DIAGNOSIS AND TREATMENT

The Concept of Femoroacetabular Impingement

Current Status and Future Perspectives

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Abstract Femoroacetabular impingement (FAI) is a recently proposed mechanism causing abnormal contact stresses and potential joint damage around the hip. In the majority of cases, a bony deformity or spatial malorientation of the femoral head or head/neck junction, acetabulum, or both cause FAI. Supraphysiologic motion or high impact might cause FAI even with very mild bony alterations. FAI became of interest to the medical field when (1) evidence began to emerge suggesting that FAI may initiate osteoarthritis of the hip and when (2) adolescents and active adults with groin pain and imaging evidence of FAI were successfully treated addressing the causes of FAI. With an increased recognition and acceptance of FAI as a damage mechanism of the hip, defined standards of assessment and treatment need to be developed and established to provide high accuracy and precision in diagnosis. Early recognition of FAI followed by subsequent behavioral modification (profession, sports, etc) or even surgery may reduce the rate of OA due to FAI.

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Introduction

Femoroacetabular impingement (FAI) is not a disease per se but rather a pathomechanical process by which the human hip can fail. A variety of abnormalities of the bony acetabulum and/or femur combined with terminal and/or rigorous hip motion can lead to repetitive collisions that damage the soft tissue structures (labrum and/or cartilage) at the acetabular rim. Today, evidence has emerged suggesting that (1) FAI may instigate osteoarthritis (OA) of the hip and (2) adolescents and active adults with groin pain might be successfully treated by addressing FAI.

More than a century ago, hip damage due to a femoroacetabular conflict (impingement) was anecdotally reported in orthopaedic textbooks and publications as sequelae of childhood disease, mainly slipped capital femoral epiphysis (SCFE) [7, 24, 50]. The first Swiss impingement experience and the roots of the current FAI concept date back to the early 1990s with the recognition of impingement occurring after femoral neck fractures malunited in retrotorsion [17]. But it was not until the development of open surgical dislocation of the hip allowed direct observations [18] that the concept of FAI was introduced as a mechanical cause of OA. The originality and distinction of the FAI concept by Ganz et al. [20] compared to previous work by Solomon and Schnitzler [47], Harris [22], Murray [42], and Stulberg et al. [48], referring OA secondary to grossly visible deformities (acetabular dysplasia, femoral pistol grip, or head tilt), is that subtle, often unrecognized developmental alterations and spatial malorientation of the hip in the absence of overt childhood disease might instigate OA [19].

In FAI articular damage initially occurs by either point or regional loading of the femoral neck against the acetabular rim (Fig. 1A–B). The site of hip damage is similar to the acetabular rim damage in hip dysplasia; however, the failure mechanisms are almost the opposite. In hip dysplasia, the unstable femoral head migrates and subluxates in regions of the least coverage of the femoral head (Fig. 2A–B) while in FAI, the femoral head remains well-centered, but the free arc of hip motion is limited by either an acetabulum that is functionally excessive (deep or maloriented causing "pincer" FAI) or a malshaped proximal femur (insufficient head/neck offset, nonspherical head causing "cam" FAI), or a combination of the two (Fig. 2 C–D). The damage patterns of pincer and cam FAI differ substantially when one of these two types exists as an isolated deformity, with pincer FAI being more localized at the labrum and cam FAI taking place at the cartilage with outside-in abrasion and/or delamination. Extremes of hip motion [9] and variations in the tissue quality of the labral/chondral junction might contribute to an enhanced vulnerability at the anterosuperior rim [38]; however, the main damage mechanism is FAI caused by the bony abnormalities. Central parts of the joint and the femoral head are only involved in the development of more advanced hip degeneration (OA) or in pincer FAI.

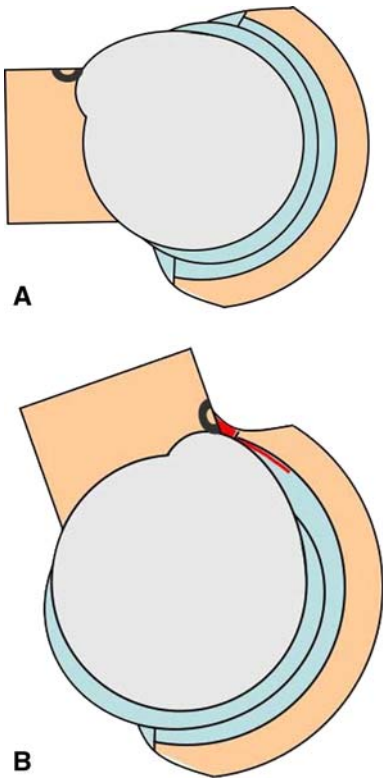

Fig. 1A–B A schematic drawing of a socket joint simulates combined FAI in (**A**) extension and (**B**) flexion. Pincer FAI (acetabular overcoverage) leads to deformation and fatiguing primarily at the labrum (red labrum) with visible indentation groove at the femoral neck. Cam FAI (femoral head asphericity) leads to shear forces primarily at the adjacent cartilage with disconnection from the subchondral bone (red line within cartilage).

This brief overview will describe the etiology of FAI, its diagnosis, and its treatment.

Etiology of FAI

Although childhood diseases such as Perthes disease, SCFE, hip dysplasia, and bladder extrophy are associated with an increased incidence in pincer- and cam-type FAI [19], the etiology of most FAI-causing abnormalities has not been identified. Acetabular protrusion leading to global pincer FAI might be caused by metabolic or inflammatory disease, although for some no such explanation can be postulated. For acetabular retroversion (focal pincer FAI), representing a posterior opening of the acetabulum [29, 30] rather than a deficiency of the posterior acetabular wall [21], the etiology is even less understood. For most cam FAI, the etiology of the proximal femoral malformation or malorientation is also unclear. While sequelae of childhood disease, in particular SCFE, were initially considered and still are etiological factors, the majority of patients with cam FAI do not show orientational growth plate abnormalities, which suggests a different developmental etiology [34, 46]. Physeal stresses (trauma, rigorous sports, etc) during development (extrinsic factors) and genetics (intrinsic factors) represent potential sources for the development of proximal femoral abnormalities. Finally, there are a substantial number of posttraumatic (acetabular dysplasia [12], femoral retrotorsion [17]) and iatrogenic deformities (femoral varus osteotomy, retroversion after pelvic osteotomy such as a Salter [11]) of the hip joint leading to FAI. Without appreciating the etiology of malformations leading to FAI, preventive measures will hardly become effective.

Diagnosis of FAI

Clinical

During the last decade, clinical tests and imaging methods facilitating the identification of morphologies at risk of causing FAI have been established. While symptoms of FAI-induced hip damage are quite variable, groin pain and a limited terminal hip motion are the clinical key symptoms and signs [4, 6, 13]. Various outcome instruments such as the nonarthritic hip score, WOMAC, or the hip outcome score have recently been either developed [8] or validated [37, 45] for their potential use in following the outcome of prearthritic hip disorders such as FAI. Owing to

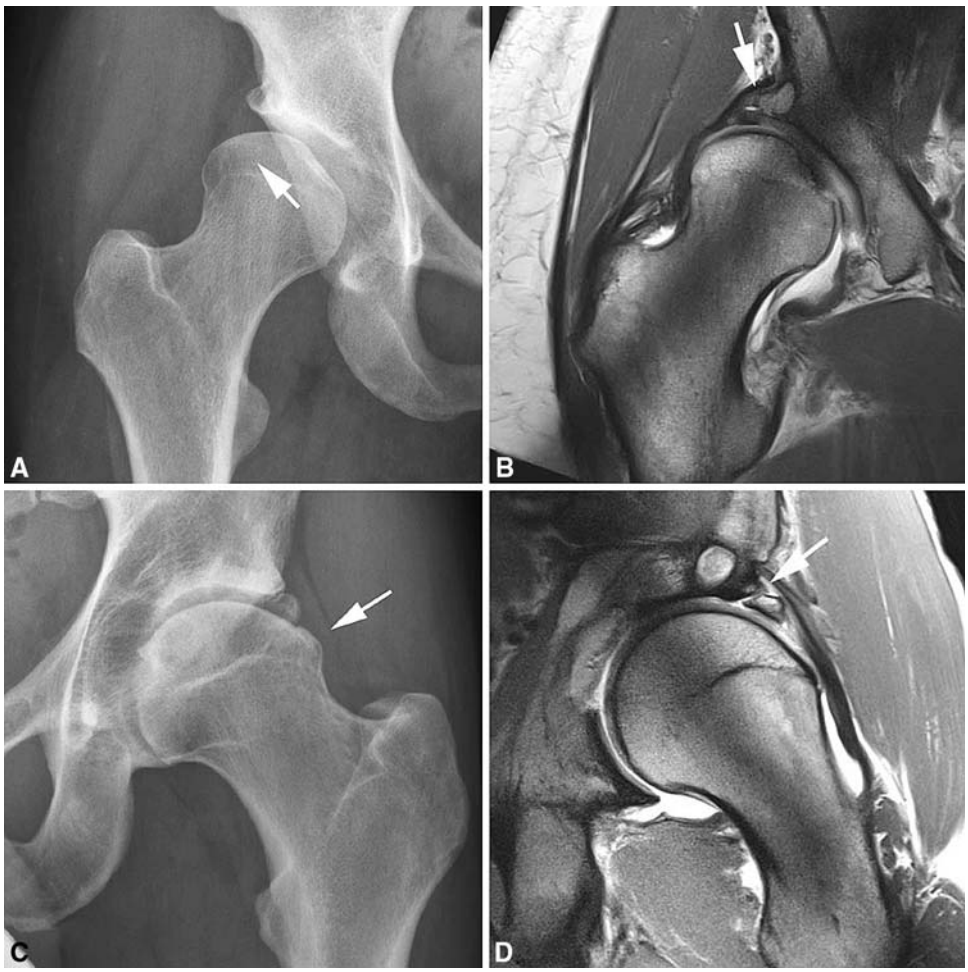

Fig. 2A–D (**A**) Acetabular dysplasia with a short and steep acetabular roof and a femoral head that has become unstable, migrating laterally (arrow) out of the acetabular socket. (**B**) The technique of MR arthrography depicts the intraarticular damage to the labrum with adjacent ganglion formation (arrow) caused by secondary joint incongruity and overload. (**C**) Femoroacetabular impingement (cam-type) (arrow) with a centered femoral head. (**D**) Shown is a cam FAI-induced acetabular rim fracture (arrow) and cartilage damage under a resulting intraosseous ganglion formation.

their ceiling effects, instruments used for assessing advanced OA such as the Merle d'Aubigne [39], Harris hip score [23], WOMAC [5], and many others (often designed for an older age group undergoing total hip replacement) do not capture the limitations of this young and active group of patients, often with high athletic levels and demands.

Radiographic/MR

Bony abnormalities causing FAI are sufficiently well-depicted on correctly imaged radiographs in two planes [40]. For evaluating acetabular version, an appropriate anteroposterior pelvic radiograph is needed since an AP of a hip alone does not allow the viewing of bony landmarks required to control for pelvic position (rotation and/or tilt). While the typical pistol grip deformity is usually visible on an AP pelvis view, the majority of nonspherical extensions of the head are located more anterior and are better visible with a cross-table lateral or a Dunn view [40]. Nonetheless, there are anterosuperiorly located sectorial abnormalities that escape detection on conventional radiographs. Although computerized tomography has a high resolution for bony abnormalities and allows 3-D reconstruction of FAI [3], we believe it should be used cautiously for diagnosis as it requires a rather high dose of radiation. That said, the treatment of FAI by computer navigation might still require CT image acquisition. In contrast, high resolution MR arthrography of one hip with radial sequences has become an important imaging tool, in particular of labral abnormalities [2, 27, 31, 36, 43]. Low-resolution pelvic MR, the absence of gadolinium, or radial sequences alone do not provide any valuable information. Future developments introducing 3-Tesla scanners and dGEMRIC [10] have been proposed to add to the imaging quality of MR, in particular for cartilage lesions or capsular adhesions.

Treatment of FAI

Identifying the appropriate timing for surgical intervention in treating hip disease secondary to FAI is still evolving

and although physiotherapy and/or antiinflammatory therapy remain the first line of treatment of musculoskeletal injuries, its benefits in FAI are questionable. More importantly, delay in the surgical correction of symptomatic patients with these bony abnormalities may lead to disease progression to the point where joint preservation is no longer indicated. Therefore we believe clinically and radiographically diagnosed FAI requires correction of the underlying bony abnormality and that is possible by surgery only. Open hip dislocation has become the primary approach to FAI, with good to excellent short- to mid-term results around 70% to 80% [1, 4, 13, 28, 41, 49]. With an improved appreciation of FAI-causing morphologies, less invasive approaches such as arthroscopy have emerged [44]. Today, complex bony abnormalities including extra-articular impingement, major deformities, and global pincer FAI still seem to be more precisely treated by open techniques. The open technique additionally allows femoral osteotomies at the level of the head/neck (Fig. 3A–B) [35], base of the neck, or intertrochanteric region when these seem appropriate. Severe acetabular retroversion with intact cartilage might even require an acetabular reorientation, a decision that needs to be based on the position of the posterior wall in relation to the center of rotation of the femoral head and on the quality of the cartilage in the superomedial area of the acetabulum. In contrast, minor structural deformities and cam FAI becomes a domain of all arthroscopic (Fig. 4A–C) or arthroscopically assisted mini open techniques. Mini open techniques without visualizing the cartilaginous surfaces are surely limited as the surgeon can obtain no information about the cartilage conditions.

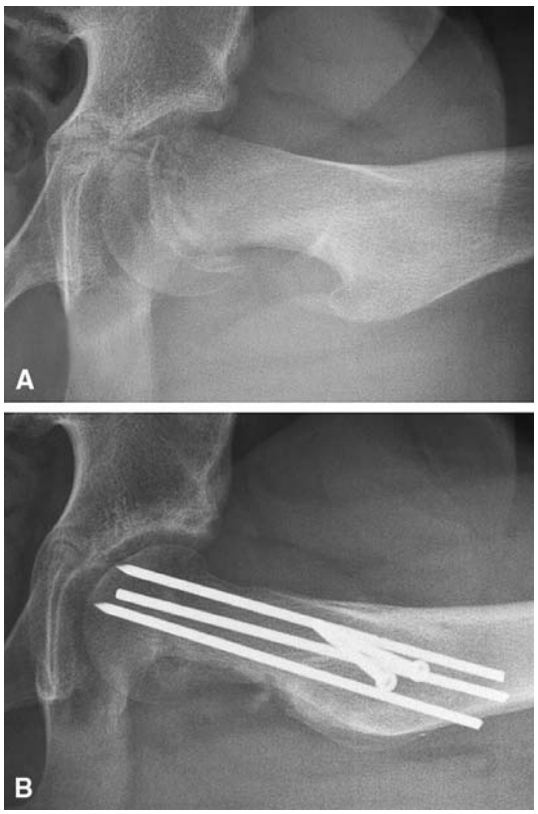

Fig. 3A–B (**A**) Preoperative lateral hip radiographs of a 13-year-old girl with a moderate acute-on-chronic SCFE of her left hip. Surgical treatment was by open hip dislocation, subcapital realignment and internal fixation (three fully threaded 3-mm K-wires for subcapital stabilization, two 3.5-mm screws for the greater trochanter). (**B**) One year later, the patient was asymptomatic with united subcapital osteotomy as evident from the lateral hip radiograph.

Several recent studies confirm that treating labral abnormalities while neglecting the underlying bony pathology (FAI) is a major cause of treatment failures [25, 32]. This is due to the fact that most, if not all, labral abnormalities occur in the presence of structural deformities [31, 51]. Currently, the indications for labral refixation are still evolving but it currently appears that resecting the labrum from its bony attachment is to be avoided whenever possible [13]. To expose the acetabular rim for trimming bony overgrowth as well as providing an intact acetabular cartilage interface, the labrum should be taken down as a part of the approach and should be reattached. Considering its physiological role [14–16], the reattachment of the intact portion of the labrum [26] appears logical since an absent labrum might, similar to meniscectomy of the knee, lead to OA. Successful refixation requires a labral substance of sufficient quality and bony procedures to correct the FAI performed with high precision [13], and presumably allowing the labrum to regain its physiological function (sealing, pressure distribution). As labral refixation can only restore labral physiology if a procedure to restore offset has been correctly performed, poor results from labral refixation might result from suboptimal impingement treatment. Today the technique of labral refixation with arthroscopy is still difficult and laborious which is likely to be overcome with technical and instrument advancements.

Discussion

This paper summarizes the current status and future perspectives of femoroacetabular impingement (FAI) as a novel pathomechanical concept. With the increased clinical interest in FAI, the number of scientific publications on FAI has increased between 1999 and 2007 almost exponentially (Fig. 5). Some of the publications addressed the etiology, clinical and radiographic assessment, and even the treatment of FAI, but a substantial number of papers are reviews on FAI. While some information exists on OA secondary to grossly visible deformities (acetabular dysplasia, femoral pistol grip, or head tilt) [22, 42, 47, 48],

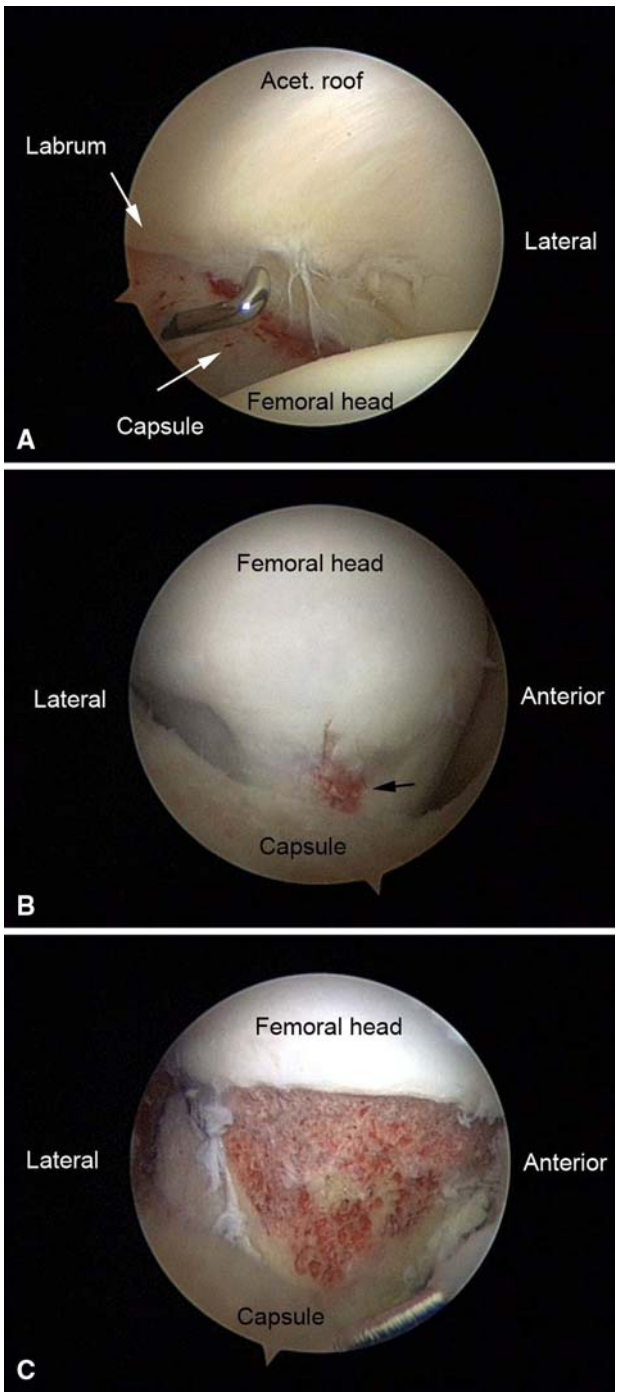

Fig. 4A–C Intraoperative photographs (hip arthroscopy) of a 34-year-old woman suffering from FAI due to mild femoral cam deformity and hypermobility. (**A**) The chondral/labral junction shows a small tear and fraying at the junction between labrum and cartilage and synovitis at the capsule and labrum as viewed from an anterior portal. (**B**) Performing peripheral hip arthroscopy, an insufficient femoral offset is present with a small area of central cartilage damage (arrow) treated by (**C**) femoral osteochondroplasty.

there is almost no information on the natural course of more subtle femoral or acetabular deformities as present in FAI disease. Currently, cross-sectional cohort studies are

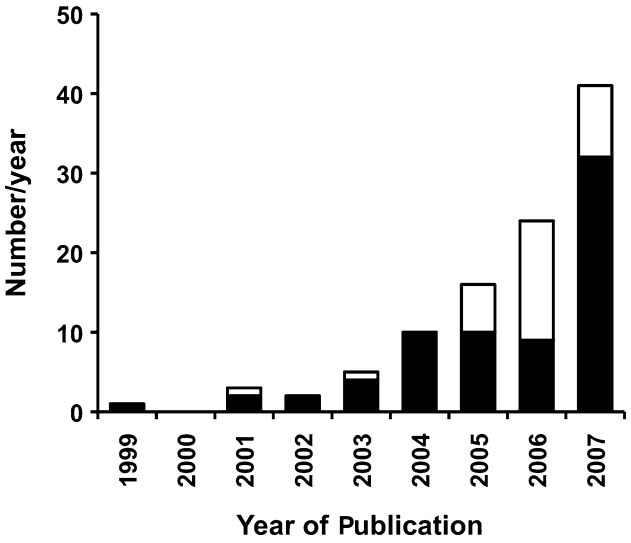

Fig. 5 The number of publications on FAI is almost exponentially increasing during the recent years. Note, however, the high number of review articles (white bars).

ongoing to determine the prevalence and familial clustering of FAI in the Swiss population (supported by the SNF National Research Program 53 grants 405340–104778 and 405340–117207). A followup study should reveal the association between the FAI morphotypes and joint degeneration and as such provide information on the natural course of FAI disease.

So far, the disease concept has been proposed mainly based on clinical findings reported from surgical hip dislocation, while mechanistic studies, in particular animal studies, supporting this disease concept are lacking. According to Koch's postulates on the pathogenesis of infectious diseases [33], experimentally created FAI morphologies (cam or pincer) should lead to similar acetabular rim damage observed in patients suffering from FAI. In addition to experimental and mechanistic research there is a need for the further effort in the development of clinical (clinical test devices, scores, etc) and radiographic (conventional radiographs, MR with dGEMRIC or 3 Tesla technology, etc) outcome instruments to allow the assessment of the natural course of FAI disease, as well as the impact of nonsurgical and surgical treatment (open and arthroscopic). Only with these investments, new information determining the significance of FAI disease and its potential treatment or even prophylaxis will become possible.

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