Hip Osteoarthritis: Definition and Etiology

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Contents

Abstract

Hip osteoarthritis (OA) is a degenerative process where continued cartilage breakdown results from mechanical overload, causing secondary bony and synovial changes and characteristic clinical and radiographic findings. Evidence is accumulating that primary hip OA is actually secondary to a subtle mechanical problem like mild dysplasia or femoroacetabular impingement (FAI). Dysplasia causes increased cartilage stress at the lateral acetabular rim, with labral hypertrophy and cartilage breakdown. FAI causes damage when the hip is flexed. Cam-type FAI causes cartilage delamination and separation of the labral-chondral junction, while pincer-type FAI causes a crushing injury to the labrum and a linear pattern of cartilage damage. Family history is a known risk factor for hip OA, and both FAI and dysplasia can be inherited. In addition, certain genotypes appear to make the cartilage more vulnerable to mechanical overloading. Nonetheless, not all radiographic hip OA is symptomatic, and not everyone with FAI or dysplasia ultimately develops hip OA. Thus, it appears that end-stage hip OA is a multifactorial process, caused by a combination of a structural deformity, wear due to activity, the inherent "robustness" of the cartilage, and the amount of inflammation that the individual experiences. The understanding of the structural factors that contribute to hip OA is advancing rapidly. It also appears that

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identification and treatment of FAI and dysplasia help symptoms that result from early chondrolabral damage.

Introduction

Both patients and physicians often use the terms arthritis, arthrosis, and osteoarthritis interchangeably. Arthritis is, however, a nonspecific term that denotes inflammation of a joint, whereas arthrosis is defined as a degenerative affliction of a joint [1]. In contrast, osteoarthritis is a distinct pathologic process: "arthritis characterized by erosion of articular cartilage, either primary or secondary to trauma or other conditions, which becomes soft, frayed, and thinned with eburnation of subchondral bone and outgrowths of marginal osteophytes; pain and loss of function results" [1]. In this vein, the terms degenerative joint arthritis, degenerative joint disease, and osteoarthrosis are true synonyms for osteoarthritis.

Although there is debate about the factors that initiate osteoarthritis, the pathologic process is characterized by progressive loss of the articular surface (Fig. 1) [2]. Initially there is cartilage fissuring, chondrocyte clustering, and some attempt at repair [2]. In this early state when the cartilage damage is confined to the articular surface and there is no associated subchondral reaction, this could also be considered "arthritis" as there is no "osteo" component. As the degenerative process progresses, the subchondral bone remodels and appears sclerotic on radiographs. Among osteoarthritis researchers, there are competing theories about the cause of these subchondral bone changes and whether they occur in response to the cartilage damage or if they occur in response to increased load even before the cartilage has been macroscopically damaged. Nonetheless, as the joint degenerates further, the process is consistent. The synovium and capsule thicken, marginal osteophytes form, and the subchondral bone may develop cysts.

Although osteoarthritis is not an inflammatory process in the same sense as the rheumatologic diseases that cause joint destruction, inflammation is clearly part of what causes radiographic osteoarthritis to become painful. When osteoarthritis becomes symptomatic, patients complain of joint pain, decreased motion, effusions, and crepitation and, in more advanced cases, may notice deformity due to ongoing bony destruction. Thus, to gather all of these concepts into a broad definition, osteoarthritis should be defined as a degenerative process where continued cartilage breakdown results from mechanical overload, causing secondary bony and synovial changes and characteristic clinical and radiographic findings.

In the hip, there are many new ideas about anatomic and biomechanical factors that may ultimately cause osteoarthritis (OA), and the basic science in this area is evolving rapidly. When evaluating a potential risk factor or cause of a disease, the Bradford-Hill criteria are helpful for determining if an association between a risk factor and a disease is actually a cause-and-effect relationship [3]. These criteria consist of the following:

- Strength of association: This refers to the relationship between the possible cause and effect. If there is a stronger relative risk of developing a disease for a patient with a particular risk factor, the risk factor is more likely to be a causal factor. Occasionally, however, the observed association is slight, and the risk factor is nonetheless proven to be a cause of a disease.
- Consistency: This means that the same association is observed repeatedly, in studies that occur in different populations, with different study designs, and by different observers.
- Specificity: This describes how precisely a potential risk factor can predict that the disease will occur. It is important, however, to keep in mind that diseases may have more than one cause and that one-to-one relationships between a risk factor and a disease are infrequent.
- Temporality: This means that the proposed risk factor for the disease always precedes the disease.
- Dose–response effect: This means that the frequency of the disease increases with the dose

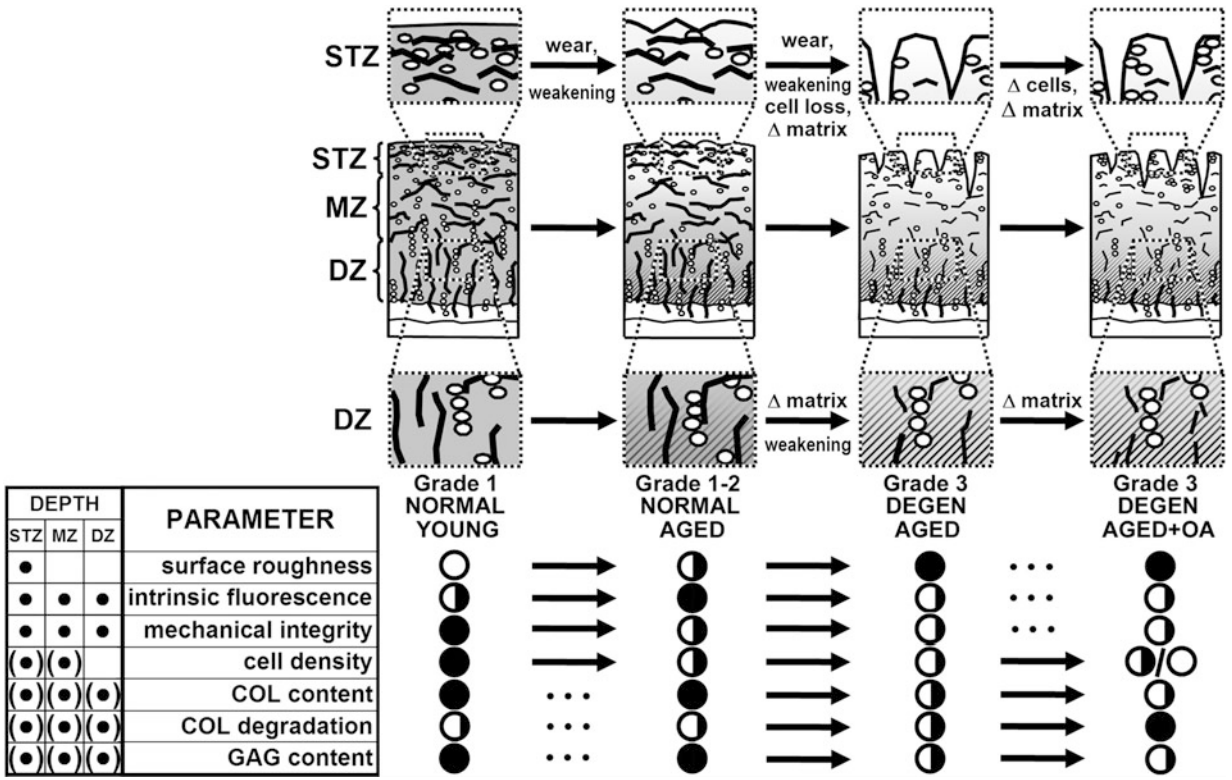

Fig. 1 Normal aged cartilage is biomechanically different from normal young cartilage. Over time, the surface (STZ: superficial tangential zone) becomes rougher and there are fewer chondrocytes. Degenerative aged and/or arthritic cartilage has deep surface clefts and fibrillation with chondrocyte clustering. In this diagram, the degree of *gray shading* indicates biomechanical integrity and loss of glycosaminoglycan (GAG). The table shows the location of changes in the STZ, middle zone (MZ), and deep zone (DZ). (•) indicates the full presence of changes, (o) the complete absence, with the *arrow* indicating a change between cartilage states (Reprinted from Osteoarthr Cartilage, Vol. 17, Temple-Wong MM, Bae WC, Chen MQ, Bugbee WD, Amiel D, Coutts RD, Lotz M, Sah RL. Biomechanical, structural, and biochemical indices of degenerative and osteoarthritic deterioration of adult human articular cartilage of the femoral condyle, 1469–1476, Copyright (2009), with permission from Elsevier)

or level of exposure. In orthopedics the dose or level exposure can also be the magnitude of a deformity.

- Biological plausibility: This means that, with what is currently known about biology or biomechanics, the proposed risk factor could reasonably cause the disease. Nonetheless, it is important to keep in mind that sometimes the basic science also needs to advance to elucidate the relationship between the cause and effect.
- Coherence: This means that the proposed association should not contradict current knowledge about the natural history and biology of the disease.
- Experimental evidence: This means that an experiment validates the cause-and-effect relationship in the expected manner. For example, modifying a risk factor decreases the frequency of a disease, or addressing the proposed cause of the disease brings about a cure.
- Analogy: This is the process of thinking about a proposed risk factor by comparing other similar and known cause-and-effect relationships for a particular disease. Reasoning by analogy can help to ascertain a cause-and-effect relationship when the observed association is slighter but similar to a known effect.

Hill himself cautioned, however, that these criteria are not necessary or sufficient for making a causal judgment and should be used more as guidelines for considering whether an observed risk factor truly causes a disease [3]. He also reminded the reader that "the 'cause' of illness may be immediate and direct, it may be remote and indirect underlying the observed association." Thus, returning to the question of the etiology of hip OA, it is more likely that the "cause" is multifactorial and different for different individuals. Furthermore, the Bradford-Hill criteria provide a useful framework for evaluating current hypotheses and evidence about the etiology of hip OA.

Although global prevalence of radiographic hip OA varies considerably, it is a common condition in the United States and Europe. The lifetime risk of developing symptomatic hip OA has been estimated to be as high as 25 % after adjusting for race, body mass index, sex, and prior injury [4]. However, not everyone who has radiographic evidence of hip OA becomes symptomatic. In one recent study, only 20 % of people with radiographic hip OA eventually became symptomatic enough to require total hip arthroplasty [5]. The natural history of asymptomatic radiographic hip OA is, however, difficult to elucidate because it requires long-term prospective cohort studies of large populations. Furthermore, the number of patients who progress to arthroplasty is likely to increase because many middle-aged and elderly patients expect to remain active indefinitely and would rather undergo arthroplasty than modify their activities. Age is one of the known risk factors for developing hip OA, and the incidence of hip OA increases with age. Not only does cartilage accumulate damage over time, but older mesenchymal stem cells also have less repair capacity and a decreased ability to protect cartilage from biomechanical stress [6]. Thus, as the expected human lifespan increases, the amount of hip OA and rates of hip arthroplasty are also projected to increase [7].

Other risk factors for hip OA include physical activity like long-term frequent lifting and standing [8] as well as intense or impact sports in young adulthood [9, 10]. There is an association between higher body mass index (BMI) and hip OA, although this association is much weaker than the association between BMI and knee OA [11]. Sex also appears to be a risk factor, with women having higher rates of hip OA than men [4]. Finally, family history and known congenital deformities have also been categorized as risk factors for hip OA.

Historically, hip OA was categorized as primary or idiopathic and secondary, meaning that the hip became arthritic as a result of a prior traumatic injury, pediatric deformity, or following infection. Primary or idiopathic hip OA was attributed to having "bad genes" – essentially that the patient had inherited weak cartilage. However, evidence is accumulating that primary hip OA is actually secondary to a subtle mechanical problem like femoroacetabular impingement (FAI) or mild dysplasia. The concept that hip OA is a

mechanical process was proposed by a number of authors and summarized nicely by Ganz in 2008:

> Most, if not all, hip OA is secondary, often secondary to subtle but definite and commonly overlooked, ignored, or not recognized dysplasia or pistol grip deformities (FAI). [12]

The Genetics of Hip Osteoarthritis

While there is clearly an inheritance pattern to hip OA, the nature of the genetic contribution is not entirely known. Have family members with a history of hip OA all inherited a bone structure like dysplasia or FAI that causes chondral damage and subsequent OA, or have they simply inherited less rigorous cartilage that is more likely to be damaged in the setting of subtle FAI or dysplasia? Or, as seems likely, is it some combination of the two factors?

Twin studies done in Caucasian females found a genetic contribution of about 60 % for both center-edge angle (as a measure of acetabular depth) and radiographic hip OA [13]. The magnitude of the genetic contribution is not the same for other joints, meaning that the etiology of OA is likely specific to mechanical factors and anatomy at that joint. This also implicates morphology rather than poor-quality cartilage as the bigger risk factor for hip OA. Other studies have shown that femoral head shape is heritable in families with a history of arthroplasty for "idiopathic" OA. However, in one of these studies, patients with a positive family history were more symptomatic than patients with the same degree of FAI morphology but no family history of hip OA. This suggests that bony morphology may not be entirely responsible for symptom development [14]. Genes have been identified that are associated with both cartilage thickness and hip shape [15, 16]. These genes are expressed in developing limb buds and in developing cartilage as well as being expressed in response to increased biomechanical loads [15, 16]. Thus, the genes associated with hip OA could affect either the hip shape or the cartilage microstructure. Finally, genetic variability influences the association between hip OA and bony morphology, meaning that certain genotypes appear to make the cartilage more vulnerable to mechanical overloading from subtle FAI or dysplasia [15].

Acetabular Dysplasia

Acetabular dysplasia is defined as a shallow or small acetabulum that inadequately covers the femoral head. Moderate to severe acetabular dysplasia has long been recognized as a risk factor for the early development of hip OA [17]. The risk of developing hip OA due to mild or borderline dysplasia is less clear, however, and may be influenced by external factors like soft tissue laxity, femoral version, and sport or dance activities.

Although dysplasia has historically been thought of in the context of infantile hip subluxation or dislocation, there is growing recognition that adolescent- or adult-onset dysplasia may represent a developmental process distinct from infantile dysplasia [18]. Furthermore, very few younger adults undergoing hip arthroplasty for arthritis secondary to dysplasia are identified as neonates [19]. The demographics of the infant and adolescent dysplasia populations are different, with adolescent-onset dysplasia patients having more bilateral hip involvement, a stronger family history, and a higher proportion of male patients [18]. Infantile dysplasia may represent a "packaging problem," meaning that mechanical factors play a greater role in the shape of the neonatal acetabulum and containment of the femoral head. The risk factors for infantile dysplasia – breech positioning, left-sided laterality, and first-born females – implicate the intrauterine environment as a mechanical factor influencing acetabular development. Furthermore, the historical prevalence of dysplasia was substantially higher in populations that had a tradition of infant swaddling with the legs in extension. When this connection was recognized and parents were instructed not to swaddle their children, the incidence of dysplasia decreased [20].

The prevalence of acetabular dysplasia varies widely [20]. It is somewhat difficult to compare the prevalence of dysplasia between countries or

regions because some studies have evaluated adults whereas for some populations the data are only available for infants. In addition, some studies have defined dysplasia as a center-edge angle of <25° whereas others have used a center-edge angle of <20°. Nonetheless, it is well recognized that the prevalence of dysplasia is higher in Asia and is the most common cause of hip OA in Japan [21].

A family history of dysplasia is a known risk factor for dysplasia and is consistent across all studied populations. Dysplasia is even more prevalent in areas where consanguinity (e.g., marriage between first cousins) is common [20]. Twin studies have revealed that the heritability of dysplasia is likely polygenic, with a higher incidence of dysplasia in monozygotic twins as compared to dizygotic twins. These findings have led investigators to propose that the genetic mechanism involves inheritance of excessive soft tissue laxity as well as acetabular shape [20].

Biomechanics of Dysplasia

In normal hips, the peak cartilage contact pressure when standing is located near the acetabular dome. The peak contact site varies between the lateral edge and the superior dome of the acetabulum, becoming more medial if the acetabulum is deeper and more lateral if the acetabulum is shallow [22, 23]. There is a direct relationship between the degree of acetabular coverage (as measured using the center-edge angle) and the contact area of the acetabular surface. As the contact surface area decreases, the peak contact pressure increases – meaning that a small center-edge angle is a marker for higher contact pressure [22]. This translates to increased force on the acetabular rim, particularly in stance, and causes characteristic chondrolabral pathology, including labral tears, ganglia, and, in some cases, acetabular rim fractures [24]. The tissue loss predictably occurs at the superior and anterosuperior regions of the acetabulum [24], which corresponds to the area of the highest load [22]. Acetabular version also influences contact pressures. Highly anteverted dysplastic hips have higher anterior contact stresses as a result of minimal anterior femoral head coverage [25]. In contrast, patients with dysplasia and retroversion have impingement-type contact stresses at the anterior edge of the acetabulum. Correcting the version and coverage with an acetabular reorientation osteotomy has been shown to decrease contact pressure by up to 50 % [23].

Natural History of Dysplasia

Radiographic dysplasia, variably defined as a center-edge angle of <20° or <25°, is clearly associated with an increased risk of hip OA [17]. The risk of developing hip OA is also clearly related to the grade of dysplasia, indicating that hips with worse biomechanics and higher contact pressures have a higher likelihood of sustaining joint damage and ultimately becoming arthritic (Fig. 2) [17]. If hip pain in a young person (<40) is considered to be a precursor of hip OA, it is notable that 25–35 % of young patients with hip pain have dysplasia [26]. Version may also play a role in the natural history of dysplasia. Patients who have retroversion and dysplasia experience an earlier onset of hip pain as compared to patients with normal anteversion [27].

If the loading biomechanics of a dysplastic hip are changed as a result of a femoral or acetabular osteotomy, the natural history of that hip appears to improve. The results are better for periacetabular or rotational acetabular osteotomy than for femoral osteotomy however. The long-term (20-year) survival rate of the native hip after a periacetabular osteotomy is about 60 % [28]. Even with the improvement in hip biomechanics, most patients have some progression of osteoarthritis and, on average, advance one radiographic Tönnis grade after 10 years [28]. Because dysplasia is largely a problem related to static loading across the hip, one might expect that weight loss in an overweight patient with dysplasia could improve hip pain and natural history because it decreases the overall static load. Although weight loss is known to improve pain and function in patients with knee OA, this has not been studied for patients with dysplasia. The

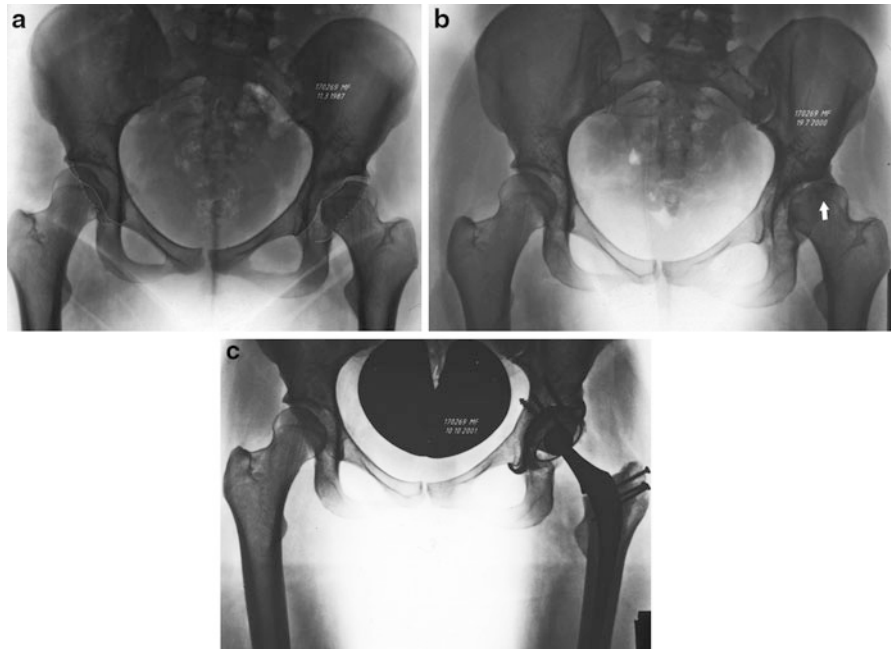

Fig. 2 The natural history of a patient with severe *left* anterior dysplasia (outline). At the time of presentation, the hip had already subluxed laterally, but the joint space was relatively preserved (**a**). Thirteen years later, there was advanced and severe cartilage degeneration on the femur, with a subchondral cyst and sclerosis (*arrow*) in the femoral head, as well as on the acetabulum (**b**). She underwent a total hip arthroplasty 1 year later (**c**)

potential for an osteotomy to improve hip function does have some limits. The success rates of osteotomy are poor for patients older than 35 with Tönnis grade 2 or more radiographic hip OA [28]. Thus, there appears to be a "tipping point" of cartilage damage, after which an osteotomy is unlikely to improve the natural history of a dysplastic hip.

Femoroacetabular Impingement (FAI)

Broadly speaking, FAI is defined as the abnormal contact between the femur and the acetabulum during hip motion that occurs as a result of a subtle deformity at the femoral head-neck junction or acetabular rim and that causes progressive chondrolabral damage [29]. Although Reinhold Ganz generally receives credit for describing FAI [29], the earliest description of impingement appears to have been in 1899 in the French literature, with the author noting "empreinte iliaque," or an impression on the head-neck junction produced by the ilium at the area of the anterior-inferior iliac spine with the hip in flexion [30]. Subsequent authors correctly described impingement in the context of coxa vara, severe protrusio deformities, and slipped capital femoral epiphysis [31–33]. Depending on the site of the deformity, these authors also recommended femoral neck osteoplasty and/or acetabular rim trimming to restore range of motion and provide pain relief. In contrast to all of the previous authors however, Ganz substantiated his ideas about FAI with observations of chondrolabral damage at the site of the impinging lesions and with the results of treatment [29, 34], both of which were made possible with the development of the technique for a safe surgical dislocation of the hip. The description of FAI also coincided with technical improvements in hip arthroscopy that resulted in an increase in hip arthroscopy for labral tears. As a result, arthroscopists began to recognize and describe early chondrolabral damage, which

ultimately helped to substantiate the association between hip pain, impingement anatomy, and eventual hip OA [35].

FAI is broadly grouped into cam and pincer types of impingement, which have different mechanisms of damage and different prognoses for the cartilage. Considering the mechanical type of impingement injury is a useful way to think about FAI because it allows for the realization that different anatomic abnormalities can cause the same type of impingement. Although cam impingement can result from many distinct anatomic abnormalities, it ultimately causes an inclusion type of injury where a bony deformity at the head-neck junction enters the joint with hip flexion. Most commonly, the abnormality is a "cam deformity" which occurs as a result of an extension of the physis onto the femoral neck [36] causing either decreased head-neck offset or a prominence at the head-neck junction. However, the femoral head deformities that occur after Legg-Calve-Perthes' disease and mild or moderate slipped capital femoral epiphysis also cause cam-type impingement and can be considered extreme examples of cam impingement [33, 37]. In cam impingement, the deformity at the head-neck junction causes shear stress and delamination of the acetabular cartilage with separation at the chondrolabral junction [29, 34]. This type of impingement has a worse prognosis for the cartilage and can cause end-stage arthrosis in a relatively young (40–50-year-old) adult. Although the cartilage over the non-spherical portion of the femoral head is abnormal, with histologic changes like cell clustering and surface fibrillation that are consistent with early arthritis [38], the macroscopic chondral damage occurs initially on the acetabulum. The weight-bearing cartilage on the femoral head remains relatively preserved until the acetabular chondral defect advances to the point that the femoral head migrates into the defect. At this time, the chondral damage becomes radiographically apparent, with visible joint space narrowing on x-rays. Pincer impingement, in contrast, causes an impaction type of injury with hip flexion, with the acetabular rim contacting the femoral head, neck, or metaphysis. Global acetabular overcoverage and focal acetabular overcoverage from acetabular retroversion are the two more classic causes of pincer impingement. However, a prominent anterior-inferior iliac spine can also cause rim impingement [39], as can acetabular protrusio [40] and a severe SCFE deformity [33]. The rim impaction causes a crushing injury to the labrum and a linear wear pattern of cartilage damage and, over time, causes rim ossification [29, 34, 41]. In addition, a "pincer groove" is often visible on the femoral neck. Although pincer impingement may not cause chondral damage as rapidly as cam impingement, the crushing injury to the labrum appears to be quite painful for the patient. A smaller number of patients with pincer impingement have femoral levering on the acetabular rim, causing contrecoup injury to the cartilage in the posterior acetabulum [29, 34]. Patients with true acetabular protrusio also develop medial cartilage thinning [40], which may be a result of increased medial contact pressure. While the distinction between cam and pincer FAI helps to explain the nature of the observed cartilage injuries, in practicality most patients with FAI have mixed cam and pincer morphotypes [29].

Biomechanics of FAI

Impingement can be observed directly during a surgical hip dislocation. Nonetheless, these observations have also been validated with finite element analysis of the cartilage contact forces during hip flexion. When the deformity at the head-neck junction is increased (by increasing the alpha angle), the non-spherical portion of the head intrudes into the acetabulum, causing increased cartilage stress on the anterior acetabulum at the site of the cam deformity. In a similar manner, increasing the amount of acetabular coverage (by increasing the center-edge angle) causes higher contact stresses at the acetabular rim and contact with the femoral neck.

Natural History of FAI

There is clearly some heritability for impingement-type anatomy, although the genetic influence may not be as strong for FAI as it is for dysplasia. Interestingly, cam morphology seems to be more heritable than pincer morphology. One sibling study observed a relative risk of 2.8 for inheriting cam-type anatomy and a relative risk of 2.0 for inheriting pincer morphology [14].

Although the evidence that FAI ultimately causes hip OA seems convincing, it is indirect (Fig. 3). Labral tears and FAI morphology are frequent in asymptomatic volunteers [42, 43]. What remains unknown about these populations is whether the subjects are asymptomatic because they are in an early stage of the disease process and have minimal chondrolabral damage or if not all FAI ultimately progresses to become symptomatic. All of the currently available natural history studies are level III or IV prognostic studies based on pelvic radiographs [44, 45]. The rates of radiographic progression for patients with FAI are quite variable, ranging from 18 % to 73 % over 10 years [44, 45]. However, FAI morphology was found in nearly all (96–99 %) hip arthroplasty patients <55 years old who were previously diagnosed with primary or idiopathic hip OA [45].

Studies of hip OA in athletes provide evidence that OA may result from a combination of FAI and abnormal loading or motion requirements. Compared to the general population, both male and female athletes have higher rates of hip OA. Contact sports and higher exposure to sports increase the risk of hip OA [9, 10]. A few studies have looked at the prevalence of hip OA in former professional dancers. Here the effect is less clear; one study showed an increased incidence of hip OA in former dancers [46], whereas a later study found no difference in rates of hip OA between dancers and the general population [47]. One potential reason for this may be that the range of motion and the amount of trained soft tissue laxity

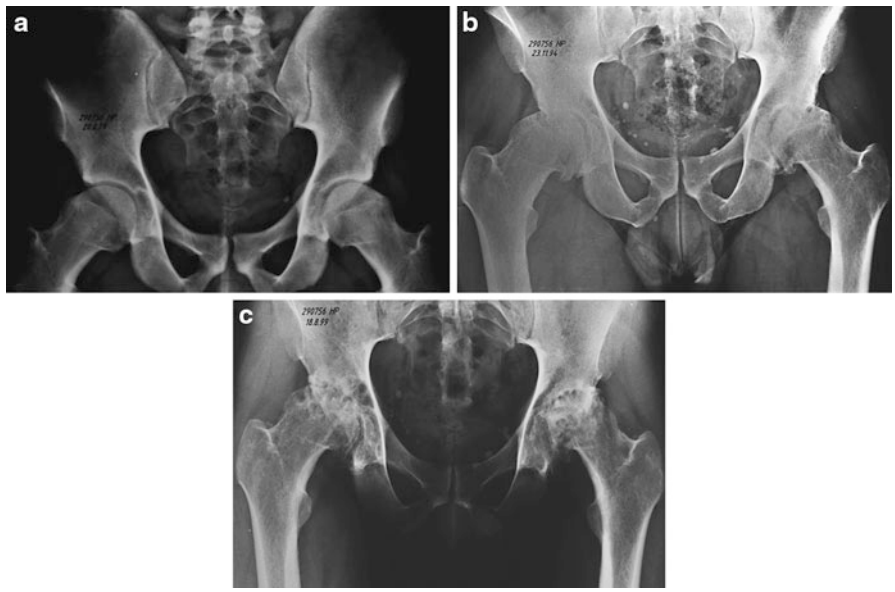

Fig. 3 This patient presented with bilateral FAI from cam deformities and acetabular retroversion on the *right*. At the initial presentation, there was acetabular subchondral sclerosis, but no joint space narrowing (**a**). Fifteen years later he had complete joint space destruction in both hips (**b**). Twenty years after his initial presentation, there are extensive cystic changes and femoral head collapse in both hips (**c**)

required for dancers weed out patients with impingement morphology before they reach elite or professional levels.

In addition to higher rates of hip OA, high-level athletes also have a higher prevalence of FAI morphology compared to the general population. This was actually first observed in the 1970s but was described as a "tilt deformity" and attributed to a mild subclinical SCFE [48]. More recently, cam deformities were found in 78 % of US collegiate football players, and a radiographic crossover sign was observed in 61 % of these same athletes [49]. Both professional and adolescent soccer players had high rates of FAI-type anatomy, with 72 % of the male professional players and 50 % of the females having some radiographic finding consistent with FAI [50]. Among asymptomatic professional and collegiate hockey players, 39 % had an elevated alpha angle but 77 % had hip and groin abnormalities on MRI [51]. Finally, a study of elite-level basketball players found that 89 % had an elevated alpha angle [52]. Cam deformities appear to occur from an extension of the femoral physis onto the femoral neck. Thus, one cause of high rates of FAI and cam deformities specifically in athletes may be the frequent high-intensity sporting activity itself. High-intensity sports have been shown to affect the proximal humeral physis and glenoid version in the young thrower as well as the distal radial physis in the gymnast. Similarly, repetitive rotational stress across the hip and proximal femoral physis as it is closing may cause the high rates of cam deformity seen in these athletes.

Summary and Conclusions

End-stage hip OA is caused by a combination of a structural deformity (either dysplasia or FAI), wear caused by the motion or activity required from an individual's hip, the inherent "robustness" of the individual's cartilage, and the amount of inflammation that the individual experiences. It is clear that not all radiographic hip OA is equally symptomatic and that many, but not all, people with FAI or dysplasia ultimately develop hip OA. The understanding of the structural factors that contribute to hip OA is advancing rapidly. It also appears that identification and treatment of FAI and dysplasia appears to help symptoms that result from early chondrolabral damage. There is good evidence that changing the biomechanics of the dysplastic joint with an acetabular osteotomy changes the natural history of the disease. If this occurs before the cartilage damage has advanced, acetabular reorientation may prevent end-stage OA or at least considerably delay an eventual arthroplasty. For FAI, there is evidence that correcting a cam or pincer deformity improves the symptoms from early OA. Although it seems likely, it is not yet known if surgical treatment can change the natural history of FAI and prevent progression of hip OA. One caveat, however, is that causing further chondral damage with surgery or incomplete treatment of these structural factors, e.g., not recognizing dysplasia in a patient with a cam deformity, might not be helpful and may incite the cascade of OA. Thus, the correct diagnosis and meticulous care of the cartilage are important when treating these patients in an attempt to prevent or delay the onset of hip OA.

References

1. Steadman's medical dictionary. 26th ed. Baltimore: Williams &Wilkins; 1995. Arthritis; p. 149, arthrosis; p. 151; osteoarthritis; p. 1267.
2. Temple-Wong MM, Bae WC, Chen MQ, Bugbee WD, Amiel D, Coutts RD, et al. Biomechanical, structural, and biochemical indices of degenerative and osteoarthritic deterioration of adult human articular cartilage of the femoral condyle. Osteoarthr Cartil. 2009;17:1469–76.
3. Hill AB. The environment and disease: Association or causation? Proc R Soc Med. 1965;58:295–300.
4. Murphy LB, Helmick CG, Schwartz TA, et al. One in four people may develop symptomatic hip osteoarthritis in his or her lifetime. Osteoarthritis Cartilage. 2010;18:1372–9.
5. Franklin J, Ingvarsson T, Englund M, Ingimarsson O, Robertsson O, Lohmander LS. Natural history of radiographic hip osteoarthritis: a retrospective cohort with 11–28 years of followup. Arthritis Care Res. 2011;63:689–95.
6. Payne KA, Didiano DM, Chu CR. Donor sex and age influence the chondrogenic potential of human femoral bone marrow stem cells. Osteoarthritis Cartilage. 2010;18:705–13.

7. Kurtz S, Ong K, Lau E, Mowat F, Halpern M. Projections of primary and revision hip and knee arthroplasty in the United States from 2005 to 2030. J Bone Joint Surg Am. 2007;89-A:780–5.
8. Sulsky SI, Carlton L, Bochmann F, et al. Epidemiological evidence for work load as a risk factor for osteoarthritis of the hip: a systematic review. PLoS ONE. 2012;7:e31521.
9. Vingard E, Alfredsson L, Goldie I, Hogstedt C. Sports and osteoarthritis of the hip. An epidemiologic study. Am J Sports Med. 1993;21:195–200.
10. Vingard E, Alfredsson L, Malchau H. Osteoarthrosis of the hip in women and its relationship to physical load from sports activities. Am J Sports Med. 1998;26:78–82.
11. Wang Y, Wluka AE, Simpson JA, et al. Body weight at early and middle adulthood, weight gain and persistent overweight from early adulthood are predictors of the risk of total knee and hip replacement for osteoarthritis. Rheumatology. 2013;52:1033–41.
12. Ganz R, Leunig M, Leunig-Ganz K, Harris WH. The etiology of osteoarthritis of the hip: an integrated mechanical concept. Clin Orthop Relat Res. 2008; 466:264–72.
13. MacGregor AJ, Antoniades L, Matson M, Andrew T, Spector TD. The genetic contribution to radiographic hip osteoarthritis in women. Results of a classic twin study. Arthritis Rheum. 2000;43:2410–6.
14. Pollard TCB, Batra RN, Judge A, et al. The hereditary predisposition to hip osteoarthritis and its association with abnormal joint morphology. Osteoarthritis Cartilage. 2013;21:314–421.
15. Baker-LePain JC, Lynch JA, Parimi N, et al. Variant alleles of the Wnt antagonist FRZB are determinants of hip shape and modify the relationship between hip shape and osteoarthritis. Arthritis Rheum. 2012;64:1457–65.
16. Castaño Betancourt MC, Cailotto F, Kerkhof HJ, et al. Genome-wide association and functional studies identify the DOT1L gene to be involved in cartilage thickness and hip osteoarthritis. Proc Natl Acad Sci USA. 2012;109:8218–23.
17. Murphy SB, Ganz R, Müller ME. The prognosis in untreated dysplasia of the hip. A study of radiographic factors that predict outcome. J Bone Joint Surg Am. 1995;77-A:985–9.
18. Lee CB, Mata-Fink A, Millis MB, Kim YJ. Demographic differences in adolescent-diagnosed and adult-diagnosed acetabular dysplasia compared with infantile developmental dysplasia of the hip. J Pediatr Orthop. 2013;33:107–11.
19. Engsæter IO, Lie SA, Lehmann TG, Furnes O, Vollset SE, Engesæter LB. Neonatal hip instability and risk of total hip replacement in young adulthood. Acta Orthop. 2008;79:321–6.
20. Loder RT, Skopelja EN. Epidemiology and demographics of hip dysplasia. ISRN Orthop. 2011; Article ID 238607, p. 47.
21. Jingushi S, Ohfuji S, Sofue M, et al. Multiinstitutional epidemiological study regarding osteoarthritis of the hip in Japan. J Orthop Sci. 2010;15:626–31.
22. Chegini S, Beck M, Ferguson SJ. The effects of impingement and dysplasia on stress distributions in the hip joint during sitting and walking: a finite element analysis. J Orthop Res. 2009;27:195–201.
23. Zhao X, Chosa E, Totoribe K, Deng G. Effect of periacetabular osteotomy for acetabular dysplasia by three-dimensional finite element analysis. J Orthop Sci. 2010;15:632–40.
24. Klaue K, Durnin CW, Ganz R. The acetabular rim syndrome: a clinical presentation of dysplasia of the hip. J Bone Joint Surg Br. 1991;73-B:423–9.
25. Matej D, Iglic A, Kral-Iglic V. Hip contact stress during normal and staircase walking: the influence of acetabular anteversion angle and lateral coverage of the acetabulum. J Appl Biomech. 2008;24:88–93.
26. Nogier A, Bonin N, May O, et al. Descriptive epidemiology of mechanical hip pathology in adults under 50 years of age. Prospective series of 292 cases: clinical and radiological aspects and physiopathological review. Orthop Traumatol Surg Res. 2010;96S:S53–8.
27. Fujii M, Nakashima Y, Yamamoto T, et al. Acetabular retroversion in developmental dysplasia of the hip. J Bone Joint Surg Am. 2010;92:895–903.
28. Steppacher SD, Tannast M, Ganz R, Siebenrock KA. Mean 20-year follow-up of Bernese periacetabular osteotomy. Clin Orthop Relat Res. 2008;466: 1633–44.
29. Ganz R, Parvizi J, Beck M, Leunig M, Nötzli H, Siebenrock KA. Femoroacetabular impingement: a cause for osteoarthritis of the hip. Clin Orthop Relat Res. 2003;417:112–20.
30. Poirier P. Traité d'anatomie humaine, vol. 1. Embryologie (Prenant A.) Ostéologie et Arthologie [Treatise of human anatomy, vol. 1 embryology (Prenant A) osteology and arthrology] (in French) Paris: Masson; 1899. pp. 516–26.
31. von Mikulicz J. In: von Bergmann E, von Bruns P, von Mikulicz J, editors. Handbuch der praktischen Chirurgie, Bd IV, Chirurgie der Extremitäten. [Handbook of surgical practice, vol. 4, Extremity surgery.] (in German). Stuttgart: Enke; 1903. p. 600.
32. Smith-Petersen MN. Treatment of malum coxae senilis, old slipped upper femoral epiphysis, intrapelvic protrusion of the acetabulum, and coxa plana by means of acetabuloplasty. J Bone Joint Surg. 1936; 18:869–80.
33. Leunig M, Casillas MM, Hamlet M, et al. Slipped capital femoral epiphysis. Early mechanical damage to the acetabular cartilage by a prominent femoral metaphysis. Acta Orthop Scand. 2000;71:370–5.
34. Beck M, Kalhor M, Leunig M, Ganz R. Hip morphology influences the pattern of damage to the acetabular cartilage: femoroacetabular impingement as a cause of osteoarthritis of the hip. J Bone Joint Surg Br. 2005;87(B):1012–8.
35. Kelly BT, Weiland DE, Schenker ML, Philippon MJ. Arthroscopic labral repair in the hip: surgical technique and review of the literature. Arthroscopy. 2005;21:1496–504.

36. Kienle KP, Keck J, Werlen S, Kim YJ, Siebenrock KA, Mamisch TC. Femoral morphology and epiphyseal growth plate changes of the hip during maturation: MR assessments in a 1-year follow-up on a cross-sectional asymptomatic cohort in the age range of 9–17 years. Skeletal Radiol. 2012;41:1381–90.
37. Eijer H, Podeszwa DA, Ganz R, Leunig M. Evaluation and treatment of young adults with femoroacetabular impingement secondary to Perthes' disease. Hip Int. 2006;16:273–80.
38. Wagner S, Hofstetter W, Chiquet M, et al. Early osteoarthritic changes of human femoral head cartilage subsequent to femoro-acetabular impingement. Osteoarthritis Cartilage. 2003;11:508–18.
39. Hetsroni I, Larson CM, Dela Torre K, Zbeda RM, Magennis E, Kelly BT. Anterior inferior iliac spine deformity as an extra-articular source for hip impingement: a series of 10 patients treated with arthroscopic decompression. Arthroscopy. 2012;11:1644–53.
40. Leunig M, Nho SJ, Turchetto L, Ganz R. Protrusio acetabuli: new insights and experience with joint preservation. Clin Orthop Relat Res. 2009;467:2241–50.
41. Corten K, Ganz R, Chosa E, Leunig M. Bone apposition of the acetabular rim in deep hips: a distinct finding of global pincer impingement. J Bone Joint Surg Am. 2011;93 Suppl 2:10–6.
42. Reichenbach S, Jüni P, Werlen S, et al. Prevalence of cam-type deformity on hip magnetic resonance imaging in young males. A cross-sectional study. Arthritis Care Res. 2010;62:1319–27.
43. Leunig M, Jüni P, Werlen S, et al. Prevalence of cam and pincer-type deformities on hip MRI in an asymptomatic young Swiss female population: a cross-sectional study. Osteoarthritis Cartilage. 2013;21:544–50.
44. 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;91:162–9.
45. Clohisy JC, Dobson MA, Robison JF, et al. Radiographic structural abnormalities associated with premature, natural hip-joint failure. J Bone Joint Surg Am. 2011;93 Suppl 2:3–9.
46. Andersson S, Nilsson B, Hessell T, et al. Degenerative joint disease in ballet dancers. Clin Orthop Relat Res. 1989;236:233–6.
47. van Dijk CN, Lim LSL, Poortman A, Strübbe EH, Marti RK. Degenerative joint disease in female ballet dancers. Am J Sports Med. 1995;23:295–300.
48. Murray RO, Duncan C. Athletic activity in adolescence as an etiological factor in degenerative hip disease. J Bone Joint Surg Br. 1971;53-B:406–19.
49. Kapron AL, Anderson AE, Aoki SK, et al. Radiographic prevalence of femoroacetabular impingement in collegiate football players. AAOS Exhibit Selection. J Bone Joint Surg Am. 2011;93:e111.
50. Gerhardt MB, Romero AA, Silvers HJ, Harris DJ, Watanabe D, Mandelbaum B. The prevalence of radiographic hip abnormalities in elite soccer players. Am J Sports Med. 2012;40:584–8.
51. Silvis ML, Mosher TJ, Smetana BS, et al. High prevalence of pelvic and hip magnetic resonance imaging findings in asymptomatic collegiate and professional hockey players. Am J Sports Med. 2011;39:715–21.
52. Siebenrock KA, Ferner F, Noble PC, Santore RF, Werlen S, Mamisch TC. The cam-type deformity of the proximal femur arises in childhood in response to vigorous sporting activity. Clin Orthop Relat Res. 2011;469:3229–40.