

REFLECTIONS ON THE CAUSE OF OSTEOARTHRITIS OF THE HIP

REINHOLD GANZ, MD, MICHAEL LEUNIG, MD, KATHARINE LEUNIG GANZ, MD, AND WILLIAM H. HARRIS, MD, DSC
MASSACHUSETTS GENERAL HOSPITAL

ABSTRACT

Osteoarthritis of the hip has long been separated into so-called secondary osteoarthritis (due to severe congenital or developmental deformities) or “primary osteoarthritis,” with the latter category presumed to be caused by an unidentified basic abnormality of articular cartilage. New information from Ganz and his colleagues now strongly supports the hypothesis that many cases of so-called “primary osteoarthritis” are mechanical in origin, rather than biologic. In many of these cases the developmental abnormalities are more subtle and frequently overlooked. The newly defined mechanism of the etiology of the osteoarthritis in these cases is the previously unrecognized femoro-acetabular impingement (FAI). These cases have often been described in the past to be “primary.”

Relatively minor abnormalities in the orientation and morphology of the hip have now been clearly identified to compromise the clearance for motion sufficiently to cause impingement. In such cases, osteoarthritis of these hips is initiated by chronic impingement.

Two types of FAI have been identified namely Cam-FAI and Pincer FAI. Clearly, surgical correction of the impingement can effectively relieve symptoms. Moreover, increasing data suggest that the correction of impingement can retard the progression of osteoarthritic process.

INTRODUCTION

Murray initially suggested a relationship between a rather subtle deformity of the proximal femur as seen on AP radiographs which he called the “tilt deformity” to the subsequent development of osteoarthritis of the hip. (1) The deformity he identified had the general characteristics of a mild degree of slipped capital femoral epiphysis (SCFE).

Solomon and co-workers in South Africa (2-6) and Harris and co-workers (7-13) in the United States extended Murray’s suggestion, leading to their controversial hypothesis on the

etiology of the osteoarthritis of the hip that most if not all cases of true osteoarthritis of the hip were mechanical in origin and were the result of developmental abnormalities.

For clarity about this theory of the etiology of osteoarthritis of the hip, other conditions that lead to joint degeneration must be excluded, including inflammatory arthritis, calcium pyrophosphate disease, infection, DISH, gout, hemochromatosis, osteonecrosis and fractures involving the joint. Unless these other cases are excluded from consideration, it is not possible to access accurately those cases that truly represent osteoarthritis:

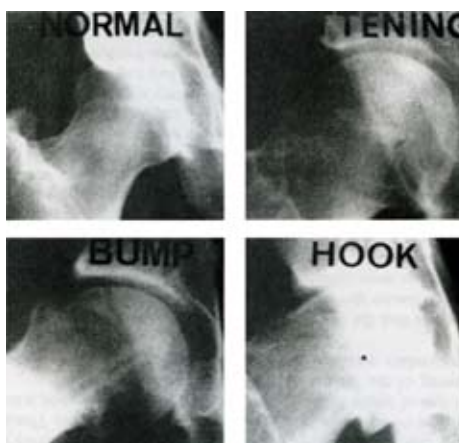


Fig. 1A: AP radiographs of a normal hip (upper left) and three forms of abnormalities associated with mild slipped capital femoral epiphyses, called “flattening”, “the bump” and the “hook.” Reproduced with permission from Clinical Orthopedics and Related Research.

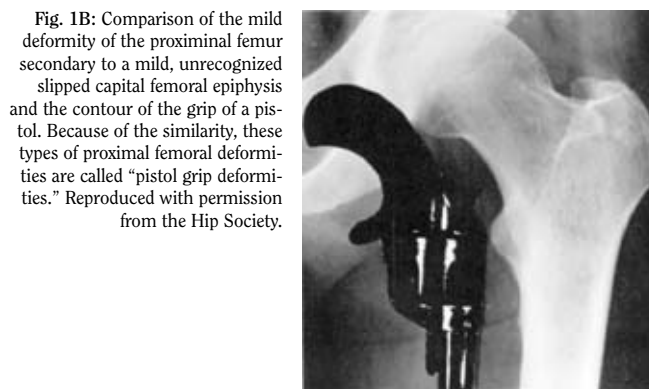


Fig. 1B: Comparison of the mild deformity of the proximal femur secondary to a mild, unrecognized slipped capital femoral epiphysis and the contour of the grip of a pistol. Because of the similarity, these types of proximal femoral deformities are called “pistol grip deformities.” Reproduced with permission from the Hip Society.

This concept of the mechanical origins of the etiology of osteoarthritis of the hip, obviously incorporates those cases secondary to gross developmental abnormalities. These cases are the well-recognized “secondary osteoarthritis” cases. However the current focus of this concept addresses cases of osteoarthritis of the hip in which the deformity was considered to be mild, slight, or even in the eyes of some to be normal hips. In the past, such cases have been commonly considered to be “primary” i.e. without known etiology. (Figures 1A and B)

Reinhold Ganz, MD, Michael Leunig, MD, Katharine Leunig Ganz, MD,
Department of Orthopedic Surgery, University of Zurich, Balgrist, Switzerland.

Address correspondence to:

Dr. Harris
55 Fruit St
Boston MA 02114



Fig. 2A



Fig. 2B

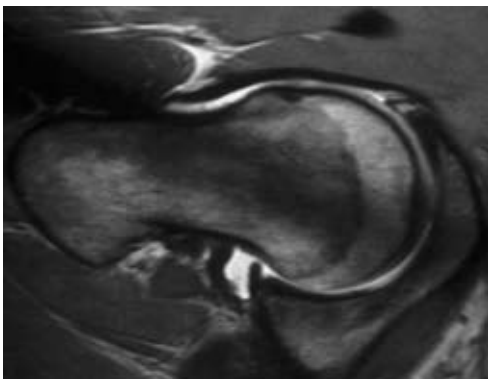


Fig. 2C

Harris and co-workers and Solomon and co-workers presented longitudinal data on documented cases of mild developmental hip disease which led, years later, to osteoarthritis of the hip. The leading conditions contributing to these subtle deformities were developmental hip dysplasia, SCFE, Legg-Perthe's disease, multiple epiphyseal dysplasia and spondyloepiphyseal dysplasia. (Figures 2A-C)

Then, retrospective analyses of patients presenting in adult life with osteoarthritis of the hip without any of the excluded conditions, specifically for whom prior radiographs were available for analysis after adulthood had been reached but before any radiographic signs of arthritis had begun to be visible, revealed that 79% of these cases had antecedent subtle but definite developmental configurational abnormalities. Among women, unrecognized and untreated developmental dysplasia predominated. Among men, abnormalities on the femoral side,

Fig. 2A-C: A) Femoroacetabular impingement (FAI) is shown in a 34-year-old man with an apparently normal AP radiograph. (B) The nonspherical femoral head leading to reduced offset at the neck and predisposition to Cam-FAI is visible on the lateral radiograph. (C) The MRI scan confirmed the labral tear and chondral injury resulting from FAI. Reproduced with permission from Clinical Orthopedics and Related Research.



Fig. 3A

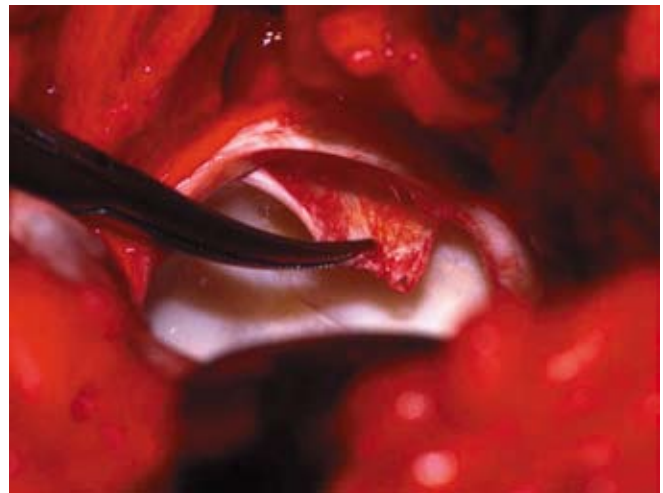


Fig. 3B

Fig. 3A-B: Intraoperative photographs of acetabular cartilage damage during the treatment of young adult patients suffering from FAI. (A) In Pincer FAI, the linear impact leads to significant labral damage, while in early phases the adjacent cartilage remains intact. (B) In contrast, Cam FAI frequently reveals already early during the disease deep reaching flap-like cleavage lesion of cartilage from the subchondral bone.

representative of the deformities resulting from slipped capital femoral epiphysis or Legg-Perthe's disease predominated. These observations challenged the traditional concept of a basic cartilaginous abnormality underlying primary osteolysis. While the data supporting this hypothesis appeared to be strong, two key factors were still missing. First, no mechanism was apparent by which some of these subtle abnormalities produced osteoarthritis. Second, there was no "low risk" way to halt or delay the progression of the osteoarthritic process.

Two current developments have dramatically strengthened the hypothesis. The two key developments are 1) the identification of the mechanism by which subtle developmental configurational abnormalities lead to osteoarthritis (Figure 3A and B) and 2) evidence that correction of that mechanism can provide major symptomatic relief and, more importantly, possibly retard the progression of development of the osteoarthritis.

Ganz and co-workers (14-38), and subsequently others (39-71) described the mechanism to be "femero-acetabu-

lar impingement” (FAI). The two distinct types of FAI exist, namely, 1) the Pincer-FAI and 2) Cam-FAI (Figure 4A and B). The Pincer-type is more common in females and develops more slowly. The Cam-type is more common in males, particularly in athletic males. This concept of the FAI mechanism leading to osteoarthritis of the hip appears to counter the existing theory of so-called primary osteoarthritis.

In conclusion, there is an increasing body of evidence that, many if not most, hips which fail from osteoarthritis without severe developmental deformities of the joint (those so-called cases of “primary osteoarthritis”) do have subtle developmental configurational abnormalities and that these abnormalities can lead to femoral acetabular impingement. It is this FAI that initiates major joint disruption in many such cases. Moreover, it is clear that surgical correction of the impinging deformities alleviates the symptoms in many cases and may retard the progression the pathologic process.

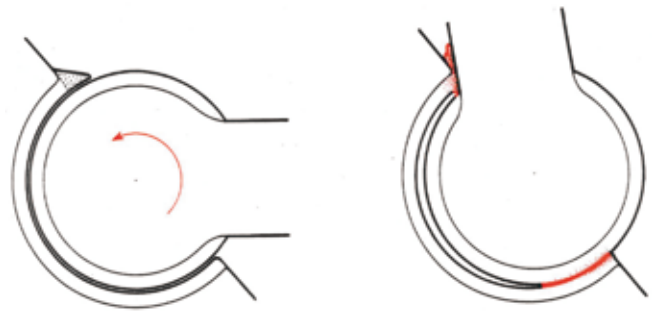


Fig. 4A

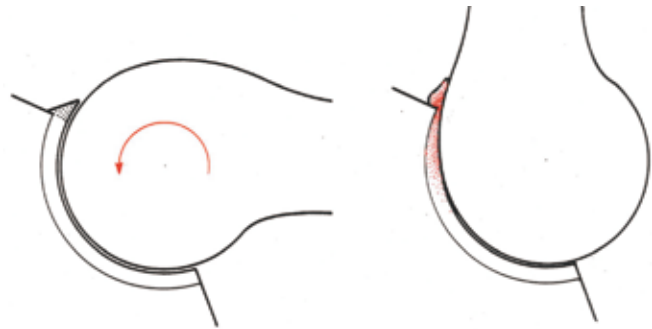


Fig. 4B

Fig. 4A-B: The proposed mechanisms of joint damage in FAI. (A) In Pincer FAI linear impact due to the acetabular overcoverage occurs anteriorly. The persistent anterior abutment with chronic leverage of the head in the acetabulum sometimes results in chondral injury in the “contre-coup” region of the posterior-inferior acetabulum (red). (B) In Cam FAI the prominent femoral head/neck junction is jammed into the acetabulum causing damage to the peripheral cartilage (red). Reproduced with permission from Clinical Orthopedics and Related Research.

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