The management of symptomatic femoroacetabular impingement: what is the rationale for non-surgical treatment?

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INTRODUCTION

Femoroacetabular impingement (FAI) is a pathomechanical process often associated with bony deformities at the proximal femur and/or acetabulum in conjunction with repetitive, rigorous or supraphysiological hip motion.¹ It may cause acetabular labral injuries and hip pain, and can lead to developing early hip osteoarthritis.¹ Since FAI is thought to be caused primarily by structural hip abnormalities, surgery -whether open or arthroscopic-is considered to be the main treatment.¹ Nevertheless, dynamic hip joint instability, defined as excessive femoral head translation relative to the acetabulum during daily and sports activities,² may also contribute to the FAI pathomechanism. Indeed, this may lead to mechanical overloading of the hip joint structures and pain.³ Managing dynamic hip joint instability has often been overlooked in terms of treatment options for FAI. This editorial proposes a rationale, discussing the potential effectiveness, for nonsurgical intervention to improve dynamic hip joint stability and reduce hip pain in patients with symptomatic FAI.

FAI-RELATED OSSEOUS AND INERT **HIP ABNORMALITIES**

Hip joint structures can be systematically evaluated for their involvement in the FAI pathomechanism.4 Osseous structures include the femur, pelvis and acetabulum, and provide hip joint congruence and structural guidance.⁴ The inert structures consist of the acetabular labrum, joint capsule and ligamentous complex, and provide static hip joint stability.⁴ Symptomatic patients with FAI consistently present with bony deformities at the

proximal femur and/or acetabulum, and most demonstrate resultant acetabular labral and capsule-ligament complex injuries.¹ Nevertheless, recent studies suggest that FAI-related bony deformities as well as acetabular labrum alterations are also found in a significant number of asymptomatic subjects.⁵ This indicates that these abnormalities in the osseous and inert hip structures, which are commonly treated with hip surgery, might not be the only determinants of hip pain in patients with FAI.

FAI-RELATED MUSCULAR AND SENSORIAL HIP ABNORMALITIES

Skeletal muscles support, control and create movement about the hip joint.4 They ensure dynamic stability of the hip, pelvis and trunk.⁴ Neuromechanical structures include nociceptors and mechanoreceptors, which provide hip joint pain sensation and proprioception, respectively.⁴ They are found in several hip joint structures including the acetabular labrum. Hip muscle weakness is common in patients with symptomatic FAI⁶ and deep hip muscles could potentially be most affected because of their proximity to the painful, damaged and/or inflamed inert hip structures. Besides being primary movers for hip flexion, external rotation and abduction, the deep hip muscles are also key muscles for ensuring dynamic stability of the femoroacetabular joint.² In order to compensate for such deep hip muscle weakness and the resultant joint instability during functional activities, secondary movers may thus be overactivated.3 This may lead to anterior gliding of the femoral head into the acetabulum and exaggerated anterior mechanical hip joint loading.³ Accordingly, repeated mechanical loading, potentially enhanced by the presence of FAI-related bony deformities, may result in structural acetabular labrum alterations over time. Based on the biochemical pain model proposed for tendinopathies,⁷ we believe that pain experienced by patients with FAI may already result from repeated mechanical

loading of the acetabular labrum before structural damage occurs. Indeed, repeated mechanical loading of the labrum may lead to the upregulation of its nociceptive receptors through the production of nociceptive neurotransmitters (eg, substance P).

DYNAMIC HIP JOINT STABILITY THROUGH NEUROMUSCULAR TRAINING

We suggest that the management of muscular, proprioceptive and nociceptive hip dysfunctions could consist of active physical therapy aimed at improving hip neuromuscular function. The protocol should include hip-specific and functional lower limb strengthening, as well as core stability and postural balance exercises. In particular, strengthening the deep hip external rotator, flexor and abductor muscles has the potential to improve dynamic stability of the femoroacetabular joint in the transverse, sagittal and frontal planes, respectively, and to minimise anterior gliding of the femoral head into the acetabulum.² This would reduce the anterior mechanical loading of the acetabular labrum 3 and downregulate nociceptor activity. 7 This mechanism may also be effective if the labrum already has structural damage.7 Moreover, progressive strength training might reduce hip joint inflammation, which is common in patients with FAI.

PERSPECTIVES

We anticipate that the proposed nonsurgical intervention will probably not be successful in reducing hip pain in all patients with FAI. Symptomatic FAI is the result of abnormalities at multiple and interconnected hip structures. Some patients with FAI might indeed be able to cope with FAI-related bony deformities and hip pathologies through improved dynamic hip joint stability, while others may not. Future research should propose standardised neuromuscular treatment protocols, and evaluate their feasibility and effectiveness in reducing symptoms in prospective cohort studies. Afterwards, randomised controlled trials comparing neuromuscular treatment protocols with other non-surgical treatment options, hip surgery and sham treatments should be conducted.

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Editorial

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