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The Effect of Dynamic Femoroacetabular Impingement on Pubic Symphysis Motion

A Cadaveric Study

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Background: A link between femoroacetabular impingement and athletic pubalgia has been reported clinically. One proposed origin of athletic pubalgia is secondary to repetitive loading of the pubic symphysis, leading to instability and parasymphyseal tendon and ligament injury.

Hypothesis/Purpose: The purpose of this study was to investigate the effect of simulated femoral-based femoroacetabular impingement on rotational motion at the pubic symphysis. The authors hypothesize that the presence of a cam lesion leads to increased relative symphyseal motion.

Study Design: Controlled laboratory study.

Methods: Twelve hips from 6 fresh-frozen human cadaveric pelvises were used to simulate cam-type femoroacetabular impingement. The hips were held in a custom jig and maximally internally rotated at 90° of flexion and neutral adduction. Three-dimensional motion of the pubic symphysis was measured by a motion-tracking system for 2 states: native and simulated cam. Load-displacement plots were generated between the internal rotational torque applied to the hip and the responding motion in 3 anatomic planes of the pubic symphysis.

Results: As the hip was internally rotated, the motion at the pubic symphysis increased proportionally with the degrees of the rotation as well as the applied torque measured at the distal femur for both states. The primary rotation of the symphysis was in the transverse plane and on average accounted for more than 60% of the total rotation. This primary motion caused the anterior aspect of the symphyseal joint to open or widen, whereas the posterior aspect narrowed. At the torque level of 18.0 N·m, the mean transverse rotation in degrees was 0.89° ± 0.35° for the native state and 1.20° ± 0.41° for cam state. The difference between cam and the native groups was statistically significant ($P < .03$).

Conclusion: Dynamic femoroacetabular impingement as caused by the presence of a cam lesion causes increased rotational motion at the pubic symphysis.

Clinical Relevance: Repetitive loading of the symphysis by cam impingement is thought to lead to increased symphyseal motion, which is one possible precursor to athletic pubalgia.

Keywords: femoroacetabular impingement; athletic pubalgia; sports hernia; pubic symphysis; cam; proximal femoral retroversion

Femoroacetabular impingement (FAI) occurs in many forms. One form is dynamic impingement of the hip, which has been increasingly recognized in young patients with hip pain.[3] Dynamic impingement can be caused by the presence of a cam lesion (decreased head-neck offset), a pincer lesion (acetabular overcoverage), proximal femoral retroversion, or coxa vara.[3,14,19,23,27] As defined by Mardones et al,[19] the cam-type lesion is a bony increase in the diameter of the femoral neck at the femoral head-neck junction. This reduced femoral head-to-neck offset distance leads to contact with the acetabulum early in the arc of internal rotation of the hip. Repetitive contact through activity can lead to labral tearing and transition zone cartilage delamination, pain, and early onset osteoarthritis over time.[6,8,12,14,27,31]

Normal proximal femoral version is around 15° of anteversion, and thus any amount of version significantly less than this represents anatomic retroversion. In patients with proximal femoral retroversion, as with cam

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impingement, the femoral neck contacts the acetabular rim earlier in the arc of functional motion and causes impingement and a crush injury to the labrum, and it is associated with the development of arthritis.[8,29,31] Both of these types of FAI have been shown to decrease physiologic internal rotation of the hip, which puts the labrum and hemipelvis at risk for repetitive loading while participating in activities that require more functional rotation than the bony anatomy allows.[29]

Repetitive loading of the pubic symphysis could be a contributing factor in the origin of athletic pubalgia, or sports hernia, or osteitis pubis.[9,10] This is thought to occur as a result of the symphyseal instability, leading to injury to the parasymphyseal tendons of the rectus abdominis and adductor longus fascial sheath.[10] Other suggested causes of sports hernia include injuries to the external oblique aponeurosis, transversus abdominis, and posterior inguinal floor. Sports hernia is a common clinical presentation in athletes that typically presents as unilateral, localized groin pain without the presence of an actual hernia.[1,21,28] Ahumada et al[1] reported this as point tenderness over the pubis, rectus abdominis origin, or adductor longus tendon origin. The literature attributes this pain to micro-tears of the rectus abdominis, the adductor longus, and their continuous aponeurosis as they attach on the pubic tubercle.[1,21,28] There has been a recent report on a subset of patients who present with both femoroacetabular impingement and athletic pubalgia in whom treatment of either in isolation leads to a high rate of failure.[17] Hypermobility of the pubic symphysis has also been shown to be associated with osteitis pubis.[33] Clinically, patients with osteitis pubis and femoroacetabular impingement commonly have limited internal rotation of the hip, so this may represent another link between these 2 pathologic entities.[30]

The purpose of this study was to investigate the effect of a cam lesion on rotational motion at the pubic symphysis. Our hypothesis is that cam impingement leads to increased relative motion at the pubic symphysis.

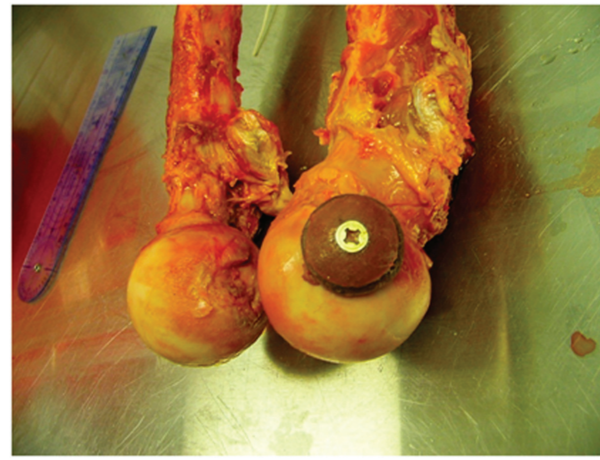

Figure 1. The cam lesion was simulated with a 5-mm dome height and base diameter of 25 mm.

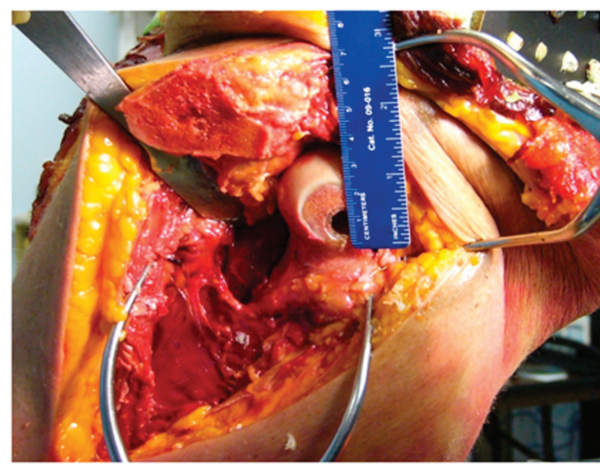

Figure 2. The cam lesion site was first prepared by planing flat the head-neck junction centered at the 1:30 position in a clock-faced representation.

METHODS

Twelve hips from 6 fresh-frozen human cadaveric pelvises were used for this study (3 male and 3 female, mean age 47.8 years, range 29-57 years). The specimens included native femurs and all related musculature and skin but without visceral organs. Donors with previous hip surgery, severe hip arthritis, or cancer were excluded. Each specimen was kept frozen at –20°C until 36 hours before testing, when it was taken out to thaw at room temperature.

Cam-type FAI was simulated by implanting a dome-shaped wood button at the femoral head-neck junction through an open surgical dislocation with trochanteric osteotomy.[7] The cam lesion was simulated with a 5-mm dome height and base diameter of 25 mm (Figure 1). The cam lesion site was first prepared by planing flat the head-neck junction centered at the 1:30 position in a clock-faced representation (Figure 2). In the clock representation used, the medial femoral head position was referenced as the 3-o'clock position for both right and left hips. The dome was fixed in place with a screw. The osteotomy of the greater trochanter was then held rigidly with 2 bicortical 3.5-mm screws, and the joint capsule was repaired with multiple 5-0 Ethibond sutures.

A custom-designed loading jig was used for the experiment. It allowed the pelvis to be suspended vertically in an upright position by anchoring an iliac wing to the jig frame with bolts and dental cement. The femoral epicondyle contralateral to the fixed iliac wing was exposed and potted with a special Plexiglas plate attachment that was used to apply internal rotation to the femur. Internal rotation was manually imposed while the leg was positioned in 90° of flexion and neutral adduction on a height-adjustable supporting stance. The 90° of flexion position was chosen because this is the position of flexion typically used to evaluate a clinical impingement sign. Adduction was positioned in the neutral position to remove it as another variable to evaluate. A 6-axis load cell (Model M3; AMTI, Watertown, Massachusetts) was mounted onto the Plexiglas plate at the distal femur to measure the moments and forces generated during the maneuver. To record the 3-dimensional motion of the pubic symphysis, we mounted marker triads from a motion-tracking system (Optotrak Certus Motion Tracking System; Northern Digital Inc, Waterloo, Ontario, Canada) onto each side of the symphysis. Additional markers were also attached to the base of the load cell to track internal rotation of the femur. A sampling rate of 30 Hz was used for the collection of load cell and marker data. Euler angles were then derived from movement of the markers based on the theory of rigid body kinematics.

Each hip was tested in 2 states: native and cam. The native state represents the hip being tested in its original state prior to trochanteric osteotomy, dislocation, and capsular repair. Load-displacement plots were generated between the internal rotational torque applied to the hip and the responding motion in 3 anatomic planes of the pubic symphysis. The mean ranges of symphysis rotation were evaluated at 10 torque levels ranging from 0.0 to 18.0 N·m and compared between the 2 groups. The maximum torque of 18 N·m was selected to represent approximately 20% of the maximum hip rotation strength reported in the literature.[18] The reported peak isokinetic internal hip rotation torque in a seated position in young healthy subjects was 62% for males and 48% for females in ft-lbs per lb when normalized with respect to body weight, which was equivalent to 110 N·m and 92 N·m in our donor group. The value of 18 N·m is also at the level of peak internal rotation torque during stair ascent for normal elderly groups.[15] It represents a moderate level of physiologic loading and low enough for the specimen to withstand repeated testing. The 10 torque levels were evenly selected within this range as points of comparison. The cadaveric model and software have been previously developed and validated in our laboratory based on previous work.[32] Load-displacement plots of the applied torque versus internal rotation of the hip were then evaluated.

Statistical Analysis

Statistical analysis was performed using software package Statview (SAS Institute, Cary, North Carolina). The effect of a cam lesion on motion across the pubic symphysis was evaluated by repeated-measure analysis of variance (ANOVA) and post hoc Fisher protected least significant difference (PLSD) tests. The rate of increase in hip rotation with respect to the applied torque was obtained from linear region(s) of the load-displacement plot using regression. The effect of a cam lesion on these slopes was compared using the same ANOVA and post hoc tests. Significance level was set at $P < .05$ for all analyses.

RESULTS

As the hip was internally rotated, the motion at the pubic symphysis increased proportionally with the degrees of the rotation as well as the applied torque measured at the distal femur. The primary rotation of the symphysis was in the transverse plane and on average accounted for more than 60% of the total rotation. The transverse rotation was in the direction of opening the joint anteriorly. The secondary rotation was in the coronal plane, which accounted for about 20% of the total rotation, and in the direction of opening the inferior portion of the joint. The mean total symphysis rotation and the transverse rotation are plotted against the applied torque and presented in Figure 3. The mean rotations of the symphysis in the transverse plane at 3 selected levels of internal rotation are listed in Table 1. Both testing states showed a change in the slope of the rotation versus torque plot around 12.0 N·m. This change in slope represents a change in the stiffness of the system, and we postulate this represents the point of bony contact.

TABLE 1
Range of Motion of Secondary Pubic Symphysis Rotation (in Degrees) in Response to Hip Internal Rotation[a]

Hip Internal Rotation	Frontal Plane		Sagittal Plane	
	Native	Cam	Native	Cam
6 N·m	0.22 ± 0.15	0.19 ± 0.12	0.21 ± 0.30	0.09 ± 0.06
12 N·m	0.37 ± 0.20	0.37 ± 0.16	0.25 ± 0.13	0.19 ± 0.10
18 N·m	0.45 ± 0.30	0.52 ± 0.15	0.39 ± 0.12	0.27 ± 0.18

[a]Values are presented as mean ± standard deviation.

In the symphysis rotation versus applied torque plots, a more rapid motion increase in the primary transverse plane was noted for the cam group once the applied torque exceeded 12.0 N·m, further supporting that this likely represents the point of contact. The mean transverse plane rotation was comparable among the groups at 12.0 N·m, valued at 0.62° ± 0.37° for the native state and 0.68° ± 0.37° for the cam state. At a torque level of 18.0 N·m, the mean transverse rotation increased to 0.89° ± 0.35° for the native state and 1.20° ± 0.41° for the cam state. The difference between the cam and native groups was statistically significant ($P < .03$) at the higher level of applied torque. Repeated ANOVA and post hoc test shows a trend toward significant difference in stiffness in the lower torque region (0-12.0 N·m), where stiffness of the native group is 52% higher than the cam group ($P < .058$), which represents the effect of the soft tissue on stiffness. The joint stiffness became comparable at the higher torque region (ANOVA, $P = .20$), which would be consistent with bone-on-bone contact.

DISCUSSION

Femoroacetabular impingement and athletic pubalgia or sports hernia have increasingly been recognized in athletes as a source of disability and inability to perform.[4,13,24] A clinical link has been reported between femoroacetabular impingement and athletic pubalgia.[17] The purpose of this

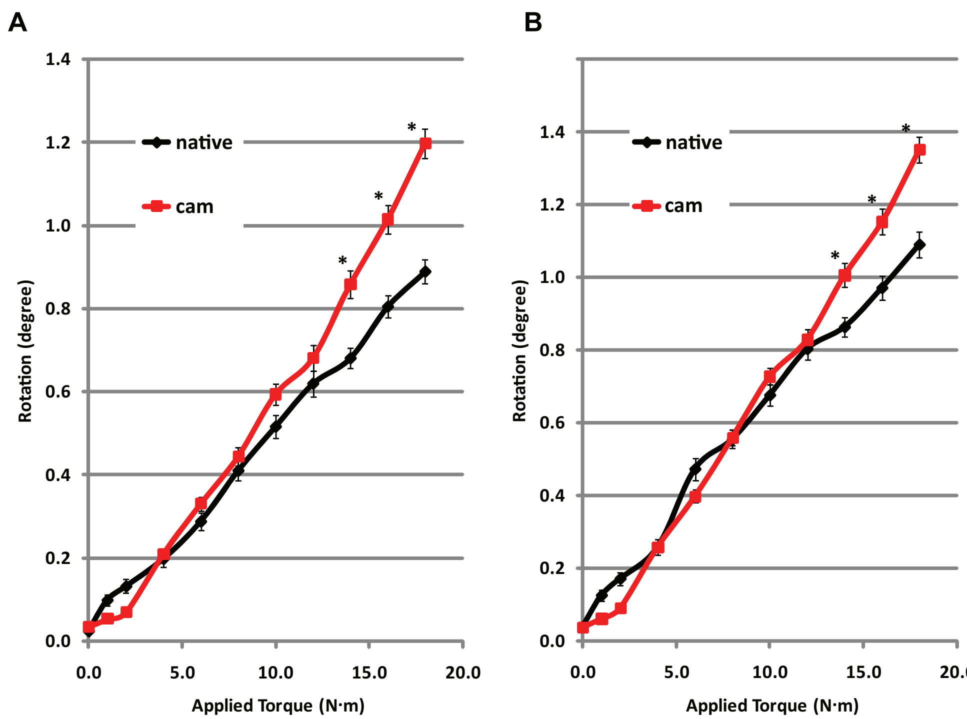

Figure 3. Mean (SE) motion of the pubic symphysis in response to the applied internal rotation torque at the hip in 2 states: native and cam. (A) Rotation in the transverse plane and (B) equivalent total rotation. *P = .05.

study was to examine the effect of simulated cam impingement on motion of the pubic symphysis.

It has been established that both proximal femoral retroversion and the presence of a cam lesion of the femoral head-neck junction cause a loss of physiologic internal rotation of the hip.[29] Mechanically, this occurs because the bone of the retroverted neck or of the cam lesion contacts the rim of the acetabulum earlier in the arc of motion and acts as a block to further rotation of the hip. The amount of functional internal rotation for sports is likely about 30°.[25] An athlete with reduced physiologic internal rotation secondary to cam or femoral retroversion to around 0° to 10° would be at risk for contacting the rim and labrum with high torque. This has been shown to cause tears of the acetabular labrum and subsequent groin pain and disability to the patient.[2,22,26] Good results with return to full activity have been reported with arthroscopic treatment of this injury.[5,11,16,24]

Loss of internal rotation of the hip has also been shown to be highly correlated with the incidence of groin injuries and osteitis pubis in a 2-year prospective study of Australian rules football.[30] Osteitis pubis in association with injuries to the low abdominal musculature has been recognized as athletic pubalgia and has been treated successfully with high return to activity with repair of these injured structures.[30]

A recent study reported on a series of patients with both femoroacetabular impingement and sports hernia. The results of treating either entity in isolation led to significant recurrence of symptoms, whereas treatment of both led to 24 of 27 patients returning to full unrestricted activity.[17] The authors suggest that the range-of-motion restrictions caused by FAI could create compensatory patterns that affect the extra-articular structures involved in athletic pubalgia. There has also been a case report linking femoroacetabular impingement to osteitis pubis.[20]

The current study demonstrates that simulated cam impingement causes rotation of the pubic symphysis after the point of bony contact, which occurred consistently between 10.0 N·m and 12.0 N·m. The point of bony contact was confirmed by correlating the amount of torque to the degrees of internal rotation on a handheld level and then repeating the same hip rotation with the capsule open and the cam lesion and acetabular rim under direct visualization. The amount of motion occurring at the pubic symphysis was in all planes but most significantly in the transverse plane (60% of the total motion). This relationship is present in the native state, as well as in the cam states. However, the cam lesion caused significantly more rotation of the symphysis (as much as 35% more) as compared with the native no-cam state at every level of torque above the point of bony contact. The rotational motion was small, on the order of about one-third of a degree, but the results were reproducible over all 12 specimens tested in triplicate. It is possible that even very small amounts of motion as a chronic repetitive loading could cause an overuse injury to the symphysis or parasymphyseal structures.

This signifies that after the femoral neck contacts the acetabular rim, motion occurs at the pubic symphysis, which can be a normal occurrence. This motion becomes pathologic when the bony anatomy causes this contact to occur earlier in the arc of internal rotation than is required for activity. Therefore, any cause of loss of internal rotation (cam, retroversion of the proximal femur, coxa vara) could lead to repetitive loading of the pubic symphysis for activities that require more motion.

This study was not without its limitations. The stiffness, as represented by the slope of the curves or degrees per unit of torque (N·m), was significantly higher for the native state, where there was less motion per unit of torque before bony contact. Therefore, the effect on stiffness of the construct by cutting the capsule is further supported by the stiffness of the native versus the cam state. Before contact, the soft tissue acts as the major restraint to motion and not the bone. The amount of motion at the symphysis recorded in our results would be underestimated given this line of reasoning.

Second, the artificial cam was not an anatomic representation of a true loss of head-neck offset. In addition, there could have been undetected motion of the cam secondary to inadequate fixation or subsidence into the bone of the neck during testing. This would lead to an underestimation of the transfer of motion to the symphysis. Finally, using a 6 degree-of-freedom robot would have produced a more reproducible hip internal rotation loading path between trials. However, despite this reproducibility, a robot model system would be more constrained than our method and would likely represent a less natural arc of motion than we reproduced manually.

In addition, the effect of a decreased head-neck offset was the only type of dynamic FAI evaluated directly. The other types of FAI not evaluated were femoral retroversion, coxa vara, and the acetabular pincer. Also, the hip was only tested in the 90° of flexion, neutral adduction position, which may not truly reproduce range-of-motion requirements during various activities. The motion transmitted through the sacroiliac joint and lumbar spine was not evaluated. The mobility or rigidity of these articulations would affect how much motion is transmitted through the pubic symphysis and would ideally have been measured. Also, the 12 hips tested were right and left hips from 6 pelvises. It is possible that the first hip tested could have altered the mechanics of the symphysis for the second hip tested.

The results of this study suggest that dynamic cam impingement causes rotational motion at the pubic symphysis after the point of bony contact. Repetitive loading of the symphysis is a known precursor to athletic pubalgia.[10] This could give one possible explanation for the clinical observation that patients with FAI also can present with athletic pubalgia or osteitis pubis.[17,20] Further studies must be carried out to support this link between pubic symphysis motion and athletic pubalgia and osteitis pubis.

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