Relationship between iliopsoas muscle surface pressure and anterior cup protrusion length after total hip arthroplasty: pathological evaluation of the iliopsoas impingement.

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INTRODUCTION:

Iliopsoas impingement (IPI) is a well-known cause of groin pain after total hip arthroplasty (THA). Symptomatic IPI generally occurs during active flexion or passive extension of the hip and causes anterior groin pain during activities of daily living such as climbing stairs or riding in a vehicle. Symptomatic IPI is a major concern after THA because of its negative impact on postoperative ADL and risk of reoperation.

It was well known that mechanical irritation of the iliopsoas muscle by the protruding part of the anterior cup is the main cause of the symptomatic IPI. It has been speculated that changes in implant alignment may cause changes in the surface pressure of the iliopsoas muscle, which may lead to pain. However, to our knowledge, changes in the surface pressure of the iliopsoas muscle at the site of impingement after THA have not yet been quantitatively evaluated.

The purpose of this cadaveric study was to directly measure changes in the surface pressure of the iliopsoas muscle using a seat-type pressure sensor when the anterior cup protrusion length (ACPL) were varied during THA.

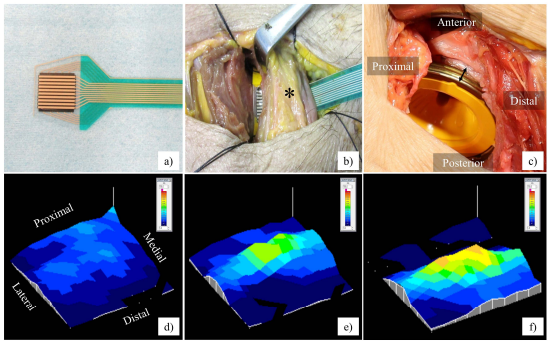
METHODS: Ten hips of 8 fresh-frozen cadavers were used in this study (4 male, 4 female). Mean age at the time of death was 79.2 (range, 65-96) years. In all the specimens, evaluation of osseous morphology on computed tomography (CT) showed no evidence of osteoarthritic changes, such as joint space narrowing, osteophytes, femoral head deformity, or previous hip surgery. We performed THA using antero-lateral supine approach with CT-based navigation system. After THA, we placed a seat-type pressure sensor (I-SCAN, Nitta, Osaka, Japan. Figure 1-A) for real-time monitoring between the iliopsoas muscle and anterior pelvic wall or anterior cup edge using an ilioinguinal approach (Figure1-B). Then, we measured the surface pressure of the iliopsoas muscle when the ACPL was 0 mm, 3 mm, and 6 mm (Figure 1-D,E,F). Each evaluation was performed with the hip in 20° and 10° of extension, the neutral position, and 10° of flexion. The maximum and mean surface pressure of the iliopsoas muscle were measured twice in each setting and average score was recorded and analyzed.

RESULTS:

The maximum surface pressure with the hip in 20° of extension was highest when the ACPL was 6 mm, showing a significant difference between 0 mm and 6 mm ($p=0.014$), but not between 0 mm and 3 mm ($p=0.576$) or between 3 mm and 6 mm ($p=0.057$). The maximum surface pressure with the hip in 10° of extension was significantly higher when the ACPL was 6 mm compared with 0 mm and 3 mm ($p=0.010$ and 0.002, respectively). With the hip in the neutral position and 10° of the flexion, the maximum surface pressure was extremely low, with no significant differences between the ACPL values. Mean surface pressure showed no statistically significant differences according to ACPL or hip flexion angle.

DISCUSSION AND CONCLUSION:

The reported ACPL for symptomatic IPI ranges from 2 mm to 27 mm in the axial plane on CT scans. However, the mean ACPL has been reported to be 0 mm to 17 mm in the axial plane even in patients who are asymptomatic after THA. Thus, the overlap of ACPL between symptomatic and asymptomatic IPI is wide, ranging from 2 mm to 17 mm. Despite general agreement that large ACPL causes symptomatic IPI, the association between small ACPL and symptomatic IPI remains unclear. We consider the minimum ACPL value associated with symptoms to be the most clinically relevant. Therefore, the present study focused on smaller ACPL values within the range of overlap between symptomatic and asymptomatic patients. We compared ACPL of 0 mm and 6 mm with the hip in 20° and 10° of extension, finding that the maximum surface pressure significantly increased when ACPL was 6 mm even though the mean surface pressure of the iliopsoas muscle tended to remain the same or decrease. When the ACPL was 6 mm, higher pressure was recorded at the protruding part of the cup, while relatively low pressure was recorded elsewhere, resulting in a lower average pressure. Similar changes occurred for comparison between ACPL of 0 mm and 3 mm, but the amount of change was relatively small. These findings suggest that symptomatic IPI is caused by strong localized stimulation of the iliopsoas muscle at the protruding part of the cup, rather than pressure applied over the entire iliopsoas muscle. Based on the results of this study, the potentially unsafe range of ACPL was considered to be 3 mm to 6 mm, suggesting that ACPL greater than 6 mm should be avoided in the clinical setting.


a)
b)
Anterior
Proximal
Distal
Posterior
c)
Proximal
Medial
Lateral
Distal
d)
e)
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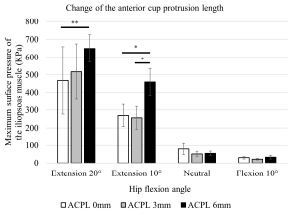
Change of the anterior cup protrusion length
Maximum surface pressure of the iliopsoas muscle (kPa)
800
700
600
500
400
300
200
100
0
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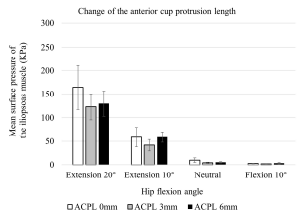
*
*
Extension 20°
Extension 10°
Neutral
Flexion 10°
Hip flexion angle
ACPL 0mm
ACPL 3mm
ACPL 6mm


Change of the anterior cup protrusion length
Mean surface pressure of the iliopsoas muscle (kPa)
300
250
200
150
100
50
0
Extension 20°
Extension 10°
Neutral
Flexion 10°
Hip flexion angle
ACPL 0mm
ACPL 3mm
ACPL 6mm