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HIP

Is the hip capsule thicker in diseased hips?

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Objectives

The purpose of this study was to compare the thickness of the hip capsule in patients with surgical hip disease, either with cam-femoroacetabular impingement (FAI) or non-FAI hip pathology, with that of asymptomatic control hips.

Methods

A total of 56 hips in 55 patients underwent a 3Tesla MRI of the hip. These included 40 patients with 41 hips with arthroscopically proven hip disease (16 with cam-FAI; nine men, seven women; mean age 39 years, 22 to 58) and 25 with non-FAI chondrolabral pathology (four men, 21 women; mean age 40 years, 18 to 63) as well as 15 asymptomatic volunteers, whose hips served as controls (ten men, five women; mean age 62 years, 33 to 77). The maximal capsule thickness was measured anteriorly and superiorly, and compared within and between the three groups with a gender subanalysis using student's *t*-test. The correlation between alpha angle and capsule thickness was determined using Pearson's correlation coefficient.

Results

Superiorly, the hip capsule was significantly greater in cam- ($p = 0.028$) and non-FAI ($p = 0.048$) surgical groups compared with the asymptomatic group. Within groups, the superior capsule thickness was significantly greater than the anterior in cam- ($p < 0.001$) and non-FAI ($p < 0.001$) surgical groups, but not in the control group. There was no significant correlation between the alpha angle and capsule thickness. There were no gender differences identified in the thickness of the hip capsule.

Conclusion

The thickness of the capsule does not differ between cam- and non-FAI diseased hips, and thus may not be specific for a particular aetiology of hip disease. The capsule is, however, thicker in diseased surgical hips compared with asymptomatic control hips.

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Article focus

- To determine, with the use of MRI, if the hip capsule is thickened in diseased hips, especially those with cam-femoroacetabular impingement (FAI) and non-FAI chondrolabral pathology, compared with asymptomatic control hips.
- To identify any possible gender differences in hip capsule thickness in both control and surgical diseased hips.
- To determine if there is any correlation between the alpha angle and hip capsule thickness.

Key messages

- The capsule thickness is increased in cam-FAI hips compared with asymptomatic hips.
- Hip capsule thickening is not a specific finding in surgical diseased hips, whether related to FAI or non-FAI aetiologies.
- The alpha angle does not correlate with the thickness of the hip capsule.

Strengths and limitations

- This study evaluates hip capsule thickness in cam-FAI, and has both non-FAI diseased hips and asymptomatic controls to serve as comparative references.
- MRI allows for measurement of hip capsule thickness with high inter- and intra-reader reliability.
- A future study with a larger sample size, greater gender balance, and age matching between the groups would strengthen the results of this project.

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Introduction

There has been relatively limited research on the periarticular soft-tissue structures of the normal and diseased hip.[1,2] Specifically, there has not been any study on whether or not the hip capsule is thickened in diseased hips requiring surgery compared with normal hips.

However, it has been suggested that the hip capsule is thickened in diseased hips, such as those with cam-femoroacetabular impingement (FAI), secondary to engagement of the cam deformity with the capsule.[3] FAI is a recognised risk factor of early onset hip osteoarthritis (OA).[4] Cam-FAI results from an abnormal osteochondral bulk at the femoral head–neck junction rendering the head aspherical.[5] This leads to abnormal biomechanics during normal ranges of hip movement, leading to labral tearing, cartilage damage and eventually OA.[4] The primary anatomical dysmorphisms of the hip that predispose to FAI are well established, as are the secondary derangements to the joint that can result from FAI.[4] However, to date, the focus of previous studies has been on the osseous, labral, cartilage and joint-space abnormalities. It is unknown whether the capsule thickness differs between diseased hips (cam- or non-FAI) and asymptomatic hips.

Any variation in capsule thickness might have the potential to serve as an anatomical marker specific to the aetiology of hip disease. The hip capsule thickness in adult humans *in vivo* has been evaluated in two studies, although one has only included symptomatic surgical FAI subjects pre-operatively[3] and the other only compares capsule thickness in hips with and without laxity.[6] To our knowledge, there has not been any study comparing diseased surgical diseased hips (FAI or non-FAI) with asymptomatic controls.

The purpose of this study was to determine and compare the hip capsule thickness in subjects with diseased hips with cam- or non-FAI hip pathologies against asymptomatic control hips, including a gender subanalysis. In addition, we sought to determine the effect of the cam deformity on hip capsule thickness. The correlation between the alpha angle and capsule thickness, as well as age and capsule thickness, were explored.

Patients and Methods

This study was performed in a tertiary care academic centre (The Ottawa Hospital, Division of Orthopaedic Surgery) with a hip subspecialty orthopaedic service (the Adult Hip Reconstruction and Joint Replacement Service). The data, both retrospective and prospective, were obtained from MRI scans performed between March 2010 and July 2012. Local institutional research ethics board approval was obtained (Protocol # 2010842 - 01H). Informed consent was waived for subjects whose images were reviewed retrospectively. Informed consent was obtained from volunteers enrolled prospectively.

Using an orthopaedic database, all subjects with either cam- or non-FAI chondrolabral pathology, subsequently confirmed with surgery, and who had undergone a 3 tesla(T) (Trio, Tim Syngo MRB17; Siemens, Erlangen, Germany) MRI of the hip between March 2010 and July 2012, were retrospectively identified. All patients had undergone a failed trial of non-surgical management with anti-inflammatory medications and activity modifications for a minimum of six months. Patients with congenital hip deformity, pincer-FAI (including acetabular retroversion), or hip dysplasia were excluded. Between February and May 2011, volunteers were prospectively recruited to undergo limited MRI of a unilateral hip. These subjects were scheduled for MRI of the pelvis for non-musculoskeletal pathologies, and prior to their scan, were approached during the standard pre-MRI questionnaire discussion with the technologist. Informed consent was obtained, and initial screening excluded anyone with history of hip pain, arthritis, trauma, infection or surgery.

There were 56 hips in total that met the criteria. The patients included 40 patients with 41 hips having surgically proven hip disease broken down into: 16 with cam-FAI (nine men, seven women; mean age 39 years, range 22 to 58) and 25 with non-FAI chondrolabral pathology (four men, 21 women; mean age 40 years, range 18 to 63) as well as 15 asymptomatic volunteers, whose hips served as controls (ten men, five women; mean age 62 years, range 33 to 77). The subjects were thus classified as either 'control' (n = 15) or 'hip disease' (n = 41) for initial analysis. The hip disease group was subsequently subdivided into two groups, 'cam-FAI' (n = 16) or 'non-FAI' (n = 25).

The intra-operative findings and surgical procedure performed served as the benchmark for confirmation of the diagnosis. The non-FAI group only included subjects with isolated labral tears or chondrolabral pathology. All cam-FAI patients had an osteochondroplasty and chondrolabral debridement performed – the non-FAI surgical group had chondrolabral damage (labral tear, chondral defects) and all underwent chondral or labral debridement/repair except one, who underwent a resurfacing arthroplasty. None had any reconstruction performed as a result of pincer-related deformity. Radiographs of all diseased hips were also reviewed by a fellowship-trained Musculoskeletal Radiologist (KSR), excluding any acetabular retroversion or other signs of morphological abnormality associated with pincer-type impingement. This methodology was employed because one of the aims of this study was to determine if cam-FAI (specifically due to direct mechanical abutment of the cam deformity against the hip capsule) is a cause of capsular thickening. However, it was imperative to determine if the capsular thickening was purely due to the cam deformity, or if it could be secondary to a labral tear or chondrolabral pathology, both of which are highly prevalent in cam-FAI

patients. Thus, a pure control group and a cam-FAI group would not have sufficed. For this reason a third group, termed non-FAI, with only labral tears and/or chondrolabral pathology, was included to serve as a standard with which cam-FAI hips with chondrolabral pathology could be compared. This would allow for the true isolation of the effect of the cam deformity or cam-FAI mechanism itself.

All subjects, both surgical and control, had identical MRI sequences performed on their hips. The MRI scans were performed on a 3T scanner (Trio, Tim Syngo MRB17; Siemens, Erlangen, Germany). The protocol included fast spin echo, intermediate weighted, fat suppressed sequences acquired in the oblique axial and oblique coronal planes with the following parameters: TR 2310ms, TE 30ms, NEx 2, ETL 7, FOV 180mm, matrix 320 x 256, slice thickness 3.5mm. Contrast was not administered.

Images were sent to a picture archiving and communication system (PACS) and reviewed by two fellowship-trained Musculoskeletal Radiologists (AAB, NMK) who were blinded to the diagnosis and group assignment for each subject. Two select images were used to measure the capsule thickness at the thickest point using electronic calipers. An oblique axial image through the level of the mid femoral neck, and an oblique coronal image through the level of the mid acetabulum, were used to measure the maximal capsular thickness both anteriorly and superiorly. These two images for measurement were selected, as the plane would be orthogonal to the capsule at the superior and anterior locations, and thus minimise volume averaging artifacts. On oblique axial images, measurements were not taken through the zona orbicularis if present. A clock-face nomenclature was used with superior position at 12 o'clock (12:00), and the anterior position at 3 o'clock (3:00). The first reader repeated all measurements four weeks after the initial read in order to determine the intra-reader reliability; the second reader performed measurements on ten random subjects to determine the inter-reader reliability. The alpha angle was measured at the anterior 3 o'clock position for all patients using the technique described by Nötzli et al.[5] The alpha angle is determined by first drawing a best-fit circle around the perimeter of the femoral head. The first arm of the angle is the long axis of the femoral neck, defined as the line drawn between the centre of the femoral neck at its narrowest point and the centre of the circle. The second arm of the angle is drawn from the centre of the circle anteriorly, to the point where the head extends beyond the margin of the circle. The hips of the cam-FAI and non-FAI groups were scored using the Tönnis classification.[7]

Statistical analysis. Comparison of the maximal hip capsule thickness at the anterior and superior positions between the surgical diseased hip and asymptomatic group was performed with a student's *t*-test. Subsequently, the diseased hip group was subdivided into cam- and non-FAI, and with the asymptomatic group, yielded three groups that were similarly compared with one another using analysis of variance. Gender differences in maximal capsule thickness for all groups were determined using the student's *t*-test. For gender analysis within the non-FAI group, the Wilcoxon test was used due to the disproportionately small number of male patients. The correlation between the alpha angle and capsular thickness was evaluated using Pearson correlation coefficient. The intra- and inter-reader reliabilities were determined using the intra-class correlation coefficient (ICC), and graded according to the Landis and Koch score.[8] The correlation between the age and capsule thickness was determined using Pearson's correlation coefficient. Statistical significance was defined as $p < 0.05$. All statistics were carried out using a statistical software package (SAS; version 9.2 SAS Institute Inc; Cary, North Carolina).

Results

There was substantial to almost perfect reliability for measurement of the hip capsule thickness. The intra-reader ICC was 0.948 and 0.909 at the anterior and superior positions, respectively. The inter-reader ICC was 0.935 and 0.757 at the anterior and superior positions, respectively.[8]

The mean maximal capsular thicknesses at the anterior and superior positions are presented in Table I. The hip capsule was significantly greater in the hip disease group ($p = 0.026$) (mean 6.8 mm, SD 1.6, range 2.3 to 9.0), as well as both cam- ($p = 0.028$) (mean 7.0, SD 1.4, range 4.0 to 9.5) and non-FAI ($p = 0.048$) (mean 6.7 mm, SD 1.7, range 3.2 to 10.2) surgical subgroups, when compared with the control group (mean 5.3 mm, SD 2.3, range 2.0 to 9.0), for the superior location.

Within groups, the superior capsule thickness was significantly greater than the anterior capsule thickness in the hip disease group (mean 6.8 mm *vs* 5.0 mm, $p = 0.006$), including both cam- (mean 7.0 mm *vs* 5.0 mm, $p < 0.001$) and non-FAI (mean 6.7 mm *vs* 4.9 mm, $p < 0.001$) subgroups, but there was no difference for the control group (mean 5.3 *vs* 4.4mm). Figures 1 to 3 present representative images of a single asymptomatic control, cam-FAI and non-FAI hip, respectively, demonstrating the anterior and superior capsule measurements.

There was no significant correlation between the alpha angle and the capsule thickness, at both the anterior and superior locations for all groups. The alpha angle was significantly higher in the cam-FAI group ($p < 0.001$) (mean 57.3, SD 8.9, range 47.8 to 74.0) compared with both the non-FAI (mean 46.5, SD 6.0, range 38.9 to 54.4) and control groups (mean 44.5, SD 10.0, range 32.7 to 53.60) (Table II). There were no significant gender differences in capsule thickness identified in any group (Table III).

Table I. Mean maximal capsular thickness with standard deviation at the anterior (3:00) and superior (12:00) positions for all groups

	Hip disease (cam- and non-FAI) (n = 41)	Cam-FAI (n = 16)	Non-FAI (n = 25)	Control (n = 15)
Mean capsule thickness (mm)				
Anterior (3:00)	5.0 (SD 1.4) (2.3 to 9.0)§	5.0 (SD 1.3) (3.5 to 7.5)¶	4.9 (SD 1.5) (2.3 to 9.0)**	4.4 (SD 1.1) (2.6 to 6.1)
Superior (12:00)	6.8 (SD 1.6) (3.2 to 10.2)*§	7.0 (SD 1.4) (4.0 to 9.5)†¶	6.7 (SD 1.7) (3.2 to 10.2)‡**	5.3 (SD 2.3) (2.0 to 9.0)*†‡

* p = 0.026
† p = 0.028
‡ p = 0.048
§ p = 0.006
¶ p < 0.001
** p < 0.001

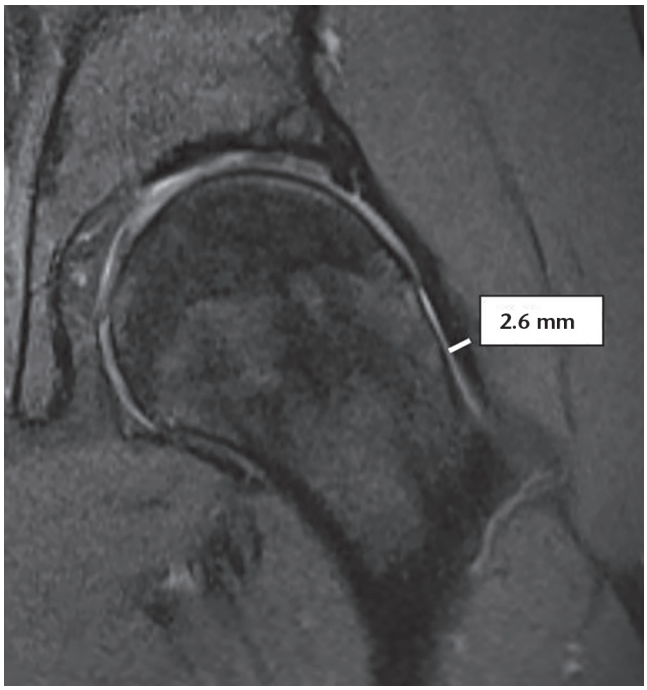

Fig. 1a

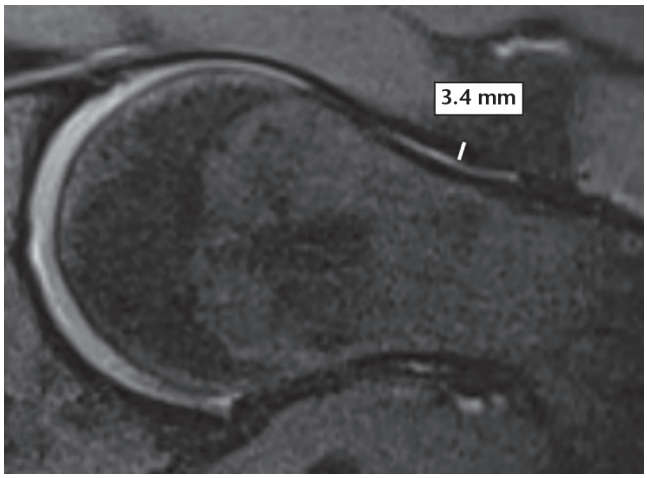

Fig. 1b

Measurement of the (a) superior (12:00) capsule thickness on an oblique coronal MRI image of an asymptomatic control subject and (b) anterior (3:00) capsule thickness on an oblique axial MRI image of an asymptomatic control subject.

For all subjects combined, there was no significant correlation between age and capsule thickness at either the anterior or superior positions.

In the cam-FAI group, all 16 patients were either Tönnis 0 (n = 5) or Tönnis 1 (n = 11). In the non-FAI (labral tear) group, the 25 patients were Tönnis 0 (n = 15), Tönnis 1 (n = 8) and Tönnis 2 (n = 2).

Discussion

Although there has been extensive research on the internal joint derangements of the hip, there has been minimal investigation of the morphology of the periarticular soft tissues. Specifically, it is unknown if the capsule thickness is altered in the setting of hip disease. This study aimed to determine capsule thickness in an asymptomatic hip and in pre-operative diseased hips *in vivo*. In addition, subanalysis of the diseased hip group was performed to compare the hip capsule thickness of subjects with cam-FAI, with that of non-FAI chondrolabral diseased hips and asymptomatic control hips. The non-FAI hips (chondrolabral pathology only) served as a standard with which cam-FAI hips with chondrolabral pathology could be compared, and thus the effect of the cam deformity could be determined. This would further test the previously published theory that capsule thickening in the context of cam-FAI results specifically from engagement with the cam deformity.[3] This study thus also sought to determine whether the thickness can be used as an anatomical marker specific to cam-FAI. The results demonstrate that the hip capsule is thicker in the diseased hip group compared with asymptomatic controls. In addition, the hip capsule thickness was not different between surgically proven cam-FAI and non-FAI diseased hips. Regardless of the aetiology, hip disease was associated with a thicker superior capsule compared with asymptomatic hips. There were no gender differences in hip capsule thickness, there was no correlation between age and capsule thickness, nor was there a correlation between the alpha angle and capsule thickness.

Regardless of the aetiology, it may be that in hips sufficiently diseased or symptomatic requiring surgery,

Table II. Mean alpha angle values as measured on the oblique axial plane image

	Cam-femoroacetabular impingement	Non-femoroacetabular impingement	Control
Mean alpha angle (°)	57.3° (SD 8.9°) (47.8° to 74.0°)*†	46.5° (SD 6.0°) (38.9° to 54.4°)*	44.5° (SD 10.0°) (32.7° to 53.6°)

* $p < 0.001$
† $p < 0.001$
SD, standard deviation

Table III. Mean capsular thickness (mm) at anterior (3:00) and superior (12:00) positions gender analysis

		Hip disease (cam- and non-FAI) (n = 41)	Cam-FAI (n = 16)	Non-FAI (n = 25)	Control (n = 15)
Anterior (3:00)	Men	5.0 (SD 1.6) (3.0 to 7.5)	5.0 (SD 1.5) (3.4 to 7.5)	5.0 (SD 1.9) (3.0 to 6.8)	4.4 (SD 1.2) (2.6 to 5.5)
	Women	5.0 (SD 1.3) (2.3 to 6.6)	5.0 (SD 1.0) (3.6 to 6.4)	4.9 (SD 1.4) (2.3 to 6.6)	4.5 (SD 1.0) (3.3 to 6.1)
Superior (12:00)	Men	6.9 (SD 1.2) (4.9 to 8.5)	7.2 (SD 1.1) (5.4 to 8.5)	6.3 (SD 1.2) (4.9 to 7.4)	5.3 (SD 2.0) (2.9 to 9.0)
	Women	6.7 (SD 1.8) (3.2 to 10.5)	6.7 (SD 1.8) (4.0 to 9.5)	6.8 (SD 1.8) (3.2 to 10.5)	5.1 (SD 3.0) (2.0 to 9.0)

FAI, femoroacetabular impingement; SD, standard deviation

there may be altered biomechanics with increased stress load superiorly, leading to acquired capsular thickening.[9] This finding is important as it may shed light into the soft-tissue physiology of a joint. The results raise the possibility that capsular thickening occurs, even in the absence of direct mechanical abutment. There may be a physiologically mediated process at the joint level, such as synovitis leading to synovial lining thickening. Evidence for this theory has been found in a biomechanical study of the hip capsule ligaments, where a greater cross-sectional area of the capsule, dependent on thickness, was associated with higher load to failure values, suggesting an adaptive and/or advantageous effect of a thicker capsule.[10] Compositional changes of the capsule have been reported, further supporting the suggestion that the hip capsule is not static, but rather dynamic, with the ability to respond to changes in a loading force profile.[11] These changes may also lead to alteration of the macrostructure and thickness of the capsule. The evolution of OA or chronic synovitis may also lead to secondary reactive capsular thickening, given the synovium is attached to the deep capsular surface.[9] Adhesive capsulitis of the hip is a known condition characterised by synovial inflammation leading to capsular fibrosis, and can occur in the setting of any hip pathology, including OA or FAI.[12] Neither the cam- or non-FAI surgical hip groups showed clinical signs of adhesive capsulitis.

Given the lack of significant difference in hip capsule thickness between cam- and non-FAI surgical hips, the current study suggests that capsular thickening is not secondary to the presence of a cam deformity, or to the mechanism of cam-type impingement. This is in distinction to a study limited to symptomatic FAI patients, where the capsule was thickest in the anterosuperior quadrant, and was postulated to be secondary to the cam deformity,[3] given that the magnitude of the cam lesion is known to be greatest in the anterosuperior quadrant.[13] However, the thicker capsule may have been incidental anterosuperiorly, and independent of cam morphology. In addition, that study did not include a comparative group of non-FAI diseased hips, nor controls.

The superior capsule thickness was significantly greater than that of the anterior capsule in the surgical cam- and non-FAI hips. This suggests that in diseased surgical hips, there may be differential topographical, biomechanical or inflammatory changes to the capsule, with a greater effect on the superior region. The superior capsule was also thicker superiorly than anteriorly in the control group, in absolute terms, although the difference was not statistically significant ($p = 0.178$). A naturally thicker capsule superiorly may be an evolutionary, adaptive anatomical feature that provides greater joint stability.

The mean maximal hip capsule thickness in the cam-FAI group was 7.0 mm superiorly (12:00) and 5.0 mm anteriorly (3:00). The capsule thickness superiorly and anteriorly in another published surgical FAI group[3] was 4.2 mm and 4.9 mm, respectively. However, the two groups are not entirely comparable – in the prior study,[3] there was a predominance of mixed-FAI subjects, with only nine of 30 purely cam-FAI, and thus the group may not be comparable with that of the current study which included pure cam-FAI subjects. In addition, that study employed radial imaging, allowing for circumferential interrogation of the hip capsule. In this study, circumferential and specific sectoral assessment was not possible as only standard two-dimensional planar images were evaluated.

In the control hip group, the mean capsular thickness was 4.4 mm anteriorly and 5.3 mm superiorly. There have not been any other studies looking at the thickness in the adult, asymptomatic hip *in vivo*. A study in asymptomatic paediatric subjects using ultrasound found the anterior capsule thickness was found to be 4.70 mm.[14] Cadaveric studies have been performed, although the age, history and status of the hip joints were not entirely know,n[15-17] precluding confirmation that the hips were, in fact, free of disease. One of the studies looked at the hip capsule thickness at variable distances (0 mm to 15 mm) from the acetabular rim; anteriorly it ranged

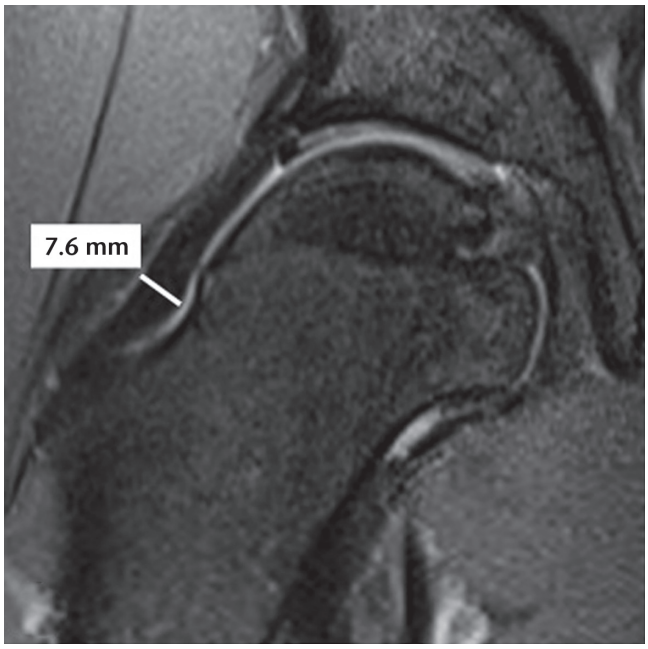

Fig. 2a

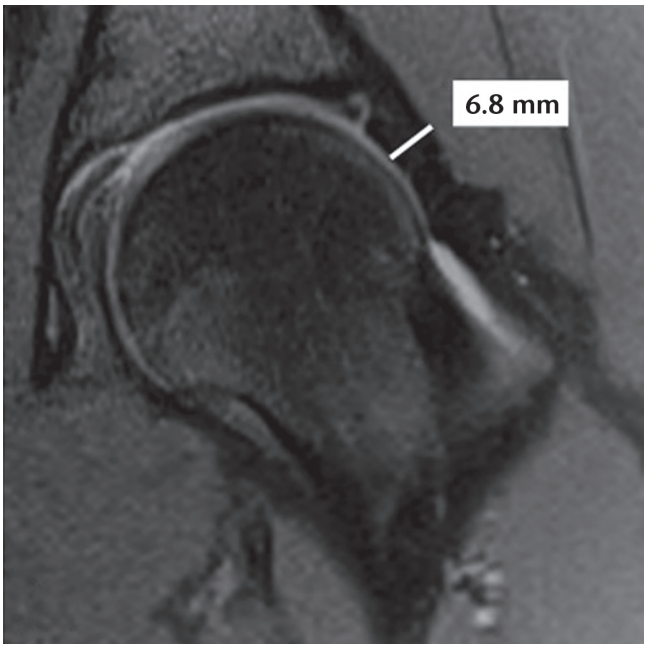

Fig 3a

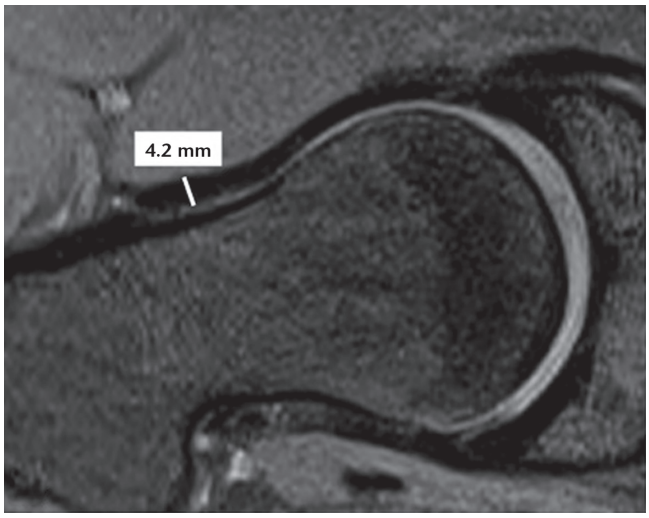

Fig. 2b

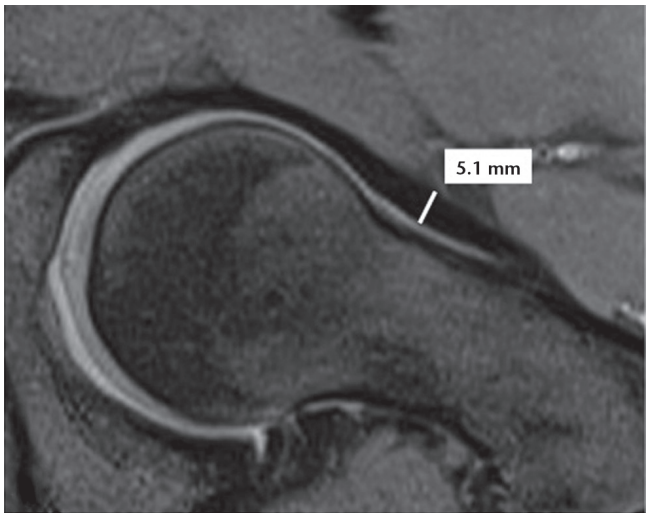

Fig 3b

Measurement of the (a) superior (12:00) capsule thickness on an oblique coronal MRI image of a cam-FAI subject and (b) anterior (3:00) capsule thickness on an oblique axial MRI image of a cam-FAI subject.

Measurement of the (a) superior (12:00) capsule thickness on an oblique coronal MRI image of a non-FAI subject (b) anterior (3:00) capsule thickness on an oblique axial MRI image of a non-FAI subject.

from 5.9 mm to 7.3mm and superiorly from 5.6 mm to 6.9 mm.[15] However, the measurements were made on excised portions of the capsule, which would lose the normal tension present when fixed *in vivo*. Given the known viscoelastic properties of the capsule, this methodology may have resulted in shortening and widening of the capsule, accounting for the significantly higher thickness values compared with the current study. The second study used MRI to measure individual ligaments through their midpoints, compared with the thickest points as performed in the current study.[16] In addition, measurements were made at specific points based on the location of certain ligaments, rather than specific positions on a clock face, as in the current study, and thus it would not serve as a valid comparison. However, the mean thickness at the inferior iliofemoral ligament (approximating the anterior, 3:00 position) and the superior iliofemoral ligament (approximating the superior, 12:00 position) was 6.9 mm and 4.2 mm, respectively. In the current study, the asymptomatic control group also had larger capsule thickness superiorly (5.3 mm) than anteriorly (4.4 mm), although the difference was not significant. The third study examined cadaveric hips and found the capsule to be thicker superoposteriorly (4.0 mm) than anteriorly (1.3 mm), demonstrating a similar trend as in the current MRI study.[17]

In the current study, there was no correlation identified between the magnitude of the alpha angle and hip capsule thickness. This suggests that engagement or abutment of the cam deformity does not directly cause capsular thickening by a direct contact mechanism. This possibility was raised in the prior study evaluating capsule thickness in cam and mixed FAI patients, although admittedly it was not conclusive given the design of that study.[3]

The present study did not identify any significant gender differences at any location, in the FAI, non-FAI or control subjects. The lack of gender differences in capsule thickness specifically in the cam-FAI group is in contradistinction to a prior study on FAI hips, which found a significantly thicker capsule in men compared with women anteriorly.[15] However the two groups are not entirely comparable – in that study, there was a predominance of mixed-FAI subjects, with only nine of 30 purely cam, and thus the group may not be comparable with that of the current study which included pure cam-FAI subjects. It is unknown what the effect of the pincer mechanism (present in some subjects of the previous study) might have on capsular thickness. Although the alpha angle is known to vary between men and women,[18] there is no similar gender difference seen in the hip capsule in the current study. Future studies looking at gender differences should also consider normalising the data to some internal reference measure that would account for potential differences in the patient's size.

The substantial to near perfect inter-reader and intra-reader reliabilities for the hip capsule thickness measurement are important to recognise given no human *in vivo* study has previously evaluated them. Although the hip capsule is a relatively thin structure, high resolution and quality MRI images can allow for reproducible measurements.

This study has limitations. Firstly, the sample size was small when considering the number of subjects in each group. A larger sample size may have identified more subtle differences in capsular thickness between the three groups. However, it is the largest *in vivo* human study performed to date. Additionally, the proportion of men and women differed in each surgical group. The non-FAI group had a disproportionately lower number of men compared with women. The lack of differences seen with some analyses may relate to lack of power of the study. *Post hoc* power analysis suggests that a minimum of 30 subjects per group to be certain that some of the small differences were not significant due to sample size alone. A second category of limitations is related to the MRI protocol. The measurements are subject to volume averaging artifact, especially when measuring a non-linear structure such as the capsule that is somewhat circular. There is greater curvature of the capsular contour superiorly than anteriorly, resulting in some volume averaging artifact, leading to some blurring at the boundaries of the capsule, and thus greater variability in measurement. This may also explain why the inter-reader ICC was lower superiorly than anteriorly. It may also explain the general trend to have larger measurement values, in absolute terms in all groups. Radial imaging would have allowed for reduction in volume averaging artifact, as each image would be perpendicular to capsule, giving more accurate thickness values. However, the images for measurements were selected in the current study such that the images in each of the two planes used would be orthogonal to the superior (12:00) and anterior (3:00) capsule to minimise this effect. In addition, with radial imaging, the capsule thickness could be measured circumferentially, and at multiple positions on the clock face, especially in the anterosuperior quadrant.[13] Radial imaging would also have allowed for the alpha angle to be measured at other positions of the femoral head-neck junction. However, radial imaging would have required an extra, relatively long MRI sequence, that was not performed on the volunteers to minimise the total time in the MRI scanner, as they would have already been there for a considerable length of time for their clinically indicated and two further research scans. MR arthrography, with its distension effect, would in theory increase separation between the capsule and the femoral neck cortex, facilitation determination of the deep margin of the capsule.[19] However, for ethical reasons, this type of invasive examination with intra-articular injection of gadolinium was not performed in controls. Thirdly, asymptomatic volunteers serving as a control group were not age matched, with a mean age significantly greater than that of the other two groups. In addition, an anecdotal review of the control subject images demonstrated labral and/or chondral pathology with the limited two MRI sequences, although perhaps insufficient to cause joint dysfunction or pain. However, a younger, age matched control group, free of any abnormality on MRI would be preferred and might lead to even greater differences when compared with clinically diseased hips. The impact of age on the hip capsule thickness is unknown, and determining the impact of age would require a larger study with wider age ranges in a population of asymptomatic normal hips.

Determination of hip capsule thickness has several applications. It has been suggested in a prior study[3] that the hip capsule is thicker in cam-FAI. However, that study did not include a control group, nor any subjects with non-FAI hip disease. Thus it was unknown prior to this study whether firstly, the hip capsule thickness observed in cam-FAI hips is thicker than in asymptomatic hips, and secondly, if so, capsular thickening is specific to cam-FAI or present in all diseased hips. Knowledge of *in vivo* baseline capsular thickness in asymptomatic hips is essential for any future clinical or research studies in which capsule thickness might be altered, whether thinned or thickened.

There has been relatively limited research on the periarticular soft-tissue structures of the normal and diseased hip. Any variation in capsule thickness might have the potential to serve as an anatomical biomarker specific to hip joint disease. In addition, the disciplines of orthopaedic, rheumatological and rehabilitative medicine deal with various forms of hip disease in which there is altered mobility and joint function, which may be caused by, or associated with, a thickened capsule.

In conclusion, this study demonstrates that the hip capsule is thickened in diseased hips. Both symptomatic cam- and non-FAI surgical hips have capsular thickening superiorly compared with control hips, possibly related to altered biomechanics or synovial physiology in surgical diseased hips. Thus the capsule thickness cannot be used as an anatomic, MRI marker, specific for cam-FAI. There were no gender differences in the hip capsule thickness. Future prospective studies, with a larger sample size, age and gender matching, with multi-group comparisons and biomechanical correlation, are required to explore the cause and effects of location-specific capsular thickening in the diseased hip.

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- R. Nairn: Study design, Subject identification, Ethical application, Manuscript review
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- N. M. Kolanko: Data collection, Manuscript review
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