THE JOURNAL OF BONE & JOINT SURGERY (BRITISH)

Pain related to the psoas muscle after total hip replacement

V. Jasani, P. Richards, C. Wynn-Jones

From North Staffordshire Royal Infirmary, Stoke-on-Trent, England

Residual pain after total hip replacement may be due to a number of causes both local to and distant from the hip. We describe pain related to the psoas muscle after total hip replacement in nine patients. All presented with characteristic symptoms. We describe the key features and management. Gratifying results were achieved with treatment. This diagnosis should be considered when assessing patients with pain after total hip replacement.

J Bone Joint Surg [Br] 2002;84-B:991-3.
Received 30 January 2002; Accepted 14 May 2002

A primary aim of total hip replacement (THR) is relief from pain. In most cases this is achieved, but residual pain is disheartening both for the clinician and the patient. Infection and aseptic loosening need to be excluded.[1,2] Various other causes have also been reported.[1,2,3,5-11]

Residual pain after THR was attributed to the psoas muscle by the senior author (CWJ) because of symptoms and signs related to pain on active flexion and external rotation of the hip. Pain resulting from psoas tendonitis after uncemented hip replacement has been described in two cases.[4] In both patients revision of the acetabular component was performed. We describe a series of patients with both cemented and uncemented prostheses who had pain in the groin without loosening or infection. None required revision of a component to relieve symptoms. All presented with characteristic symptoms implicating the psoas muscle. We have developed a protocol for the investigation and treatment of these patients.

V. Jasani, FRCS Orth, Specialist Registrar in Orthopaedics and Trauma
P. Richards, FRCR, Clinical Lecturer in Radiology
C. Wynn-Jones, FRCS, Consultant Orthopaedic Surgeon
North Staffordshire Royal Infirmary, Princes Road, Stoke-on-Trent, Staffordshire ST4 7LN, UK.

Correspondence should be sent to Mr V. Jasani at 3 Bannacks Close, Willaston, Nantwich, Cheshire CW5 6RP, UK.

0301-620X/02/713194 $2.00

Patients and Methods

Nine patients were identified and retrospectively reviewed. All had pain in the groin after THR. There were seven women and two men with a mean age of 59 years (40 to 75). Seven had had cemented THRs, one a hybrid THR and one an uncemented arthroplasty. The seven cemented THRs had Ogee acetabular cups and flanged 45 mm femoral prostheses (DePuy, Leeds, UK). The single hybrid THR had a Trilogy cup (Zimmer, Warsaw, Indiana) and a C stem femoral implant (DePuy). The uncemented THR was an ABG arthroplasty (Stryker Howmedica, Newbury, UK).

The mean time to onset of symptoms was 12.4 weeks (2 to 32) after THR. All patients presented with constant pain in the groin which was aggravated by activity. It was worse on active flexion such as when attempting to lift the affected limb out of a car. Clinical examination showed no evidence of abductor deficiency, leg-length discrepancy, limitation of passive range of movement or abnormal neurological signs. Active straight-leg raising reproduced the symptoms in all patients, as did attempted active external rotation. Active and resisted flexion of the hip at 90° in the sitting position also reproduced the symptoms.

Routine evaluation for evidence of aseptic loosening and infection using clinical, radiological and laboratory investigations was undertaken. Any patient who underwent further surgery had intraoperative assessment for loosening and tissue biopsy for culture.[1-3]

The first patient was evaluated further by the injection, guided by the image intensifier, of 20 mg of triamcinalone hexacetonide and 10 ml of bupivicaine 0.5% into the psoas muscle. In six subsequent patients, CT of the pelvis was performed using a Picker PQS000 scanner (Marconi Medical Systems, Stevenage, UK). After intravenous contrast (100 ml of ultravist 300 mg I/ml) had been given spiral axial 8 mm contiguous slices were taken through the pelvis and the proximal femur and imaged on soft tissue and bone windows. In all six patients CT-guided injections of triamcinalone hexacetonide and bupivicaine 0.5% were also given under local anaesthesia.

Seven patients had further surgery. The original anterolateral approach was used in all except one in whom a separate anterior (Smith-Petersen) approach was used.

All patients were followed up postoperatively.

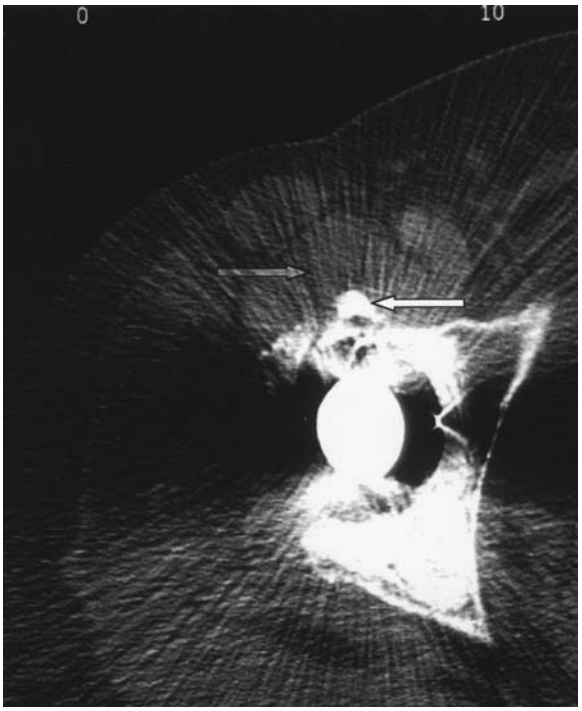

Fig. 1

Axial CT scan showing the impinging lesion (white arrow) related to the deep surface of the psoas muscle (shaded arrow).

Results

The symptoms improved in the patient who had an image-intensifier-guided injection of steroid and bupivicaine in the region of the psoas muscle. In the six patients in whom CT scans had been performed there was evidence of impingement on the deep surface of the psoas muscle by cement and/or prominent flanges of the acetabular implant (Figs 1 and 2). CT-guided injection was beneficial in all patients (Fig. 3).

The resolution of pain after injection was temporary in all but one patient. The mean time to recurrence of pain was 3.6 months (3 to 5).

Seven patients underwent further exploration. All were evaluated for infection by fluid and tissue culture and histological examination. No evidence of infection or loosening of the implant was found.

In five with cemented prostheses there was a prominence of the anterior flange of the acetabular component, often with an extruded piece of cement. In the region of this impingement, the psoas muscle was palpably and visibly thickened and irregular reactive tissue was present. Histological examination showed this to be fibrous tissue. Operative management involved removal of the cement and trimming of the flange where appropriate.

In the two patients with uncemented cups, there was no direct impingement, and no uncovering of the cup. The psoas was thicker, however, and appeared to be under tension. This was lengthened using an interstitial tenotomy.

In all the patients who had operative intervention the symptoms resolved immediately and remained so over a follow-up of ten months (2 to 14).

One patient has requested further injections rather than operative intervention and the final one remains free from pain after injection.

The patient who had a Smith-Petersen approach had a mildly hypertrophic and tender scar. No other complications occurred. There was no clinically detectable deficit of the psoas muscle in the two patients who had a tenotomy.

Discussion

Pain after THR can be due to many causes apart from aseptic loosening or infection. These may be related to the spine, peripheral nerves (sciatic and obturator), vessels, herniae, stress fractures, compartment syndromes and neoplasms.[1,2,3,5-11] Occasionally, despite investigation, no cause can be found, and the outcome remains unsatisfactory. We present another possible cause of persistent pain which is related to the psoas muscle.

In this condition there is characteristically a constant pain in the groin which starts in the early postoperative period. All patients described an exacerbation of symptoms on activities involving active flexion and external rotation, in particular lifting the affected limb out of a car. This

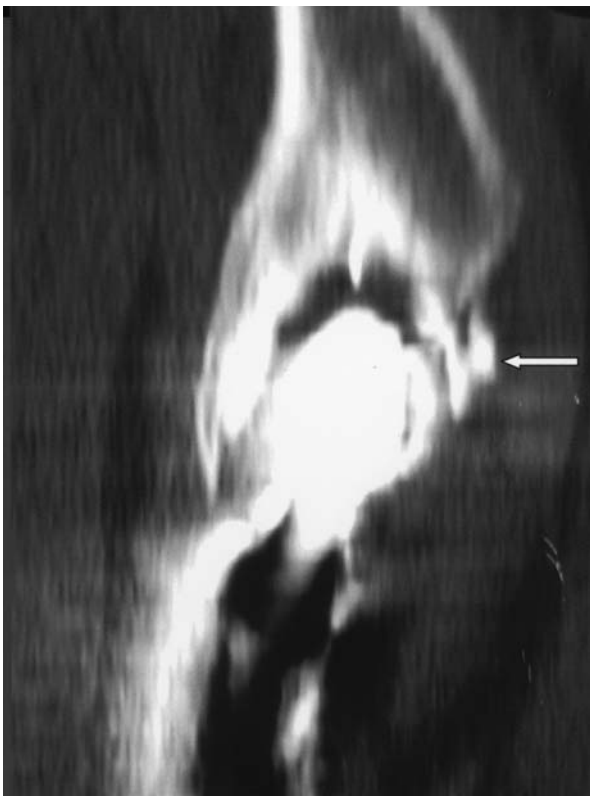

Fig. 2

Sagittal reconstruction of a hip showing the impingement lesion (arrow).

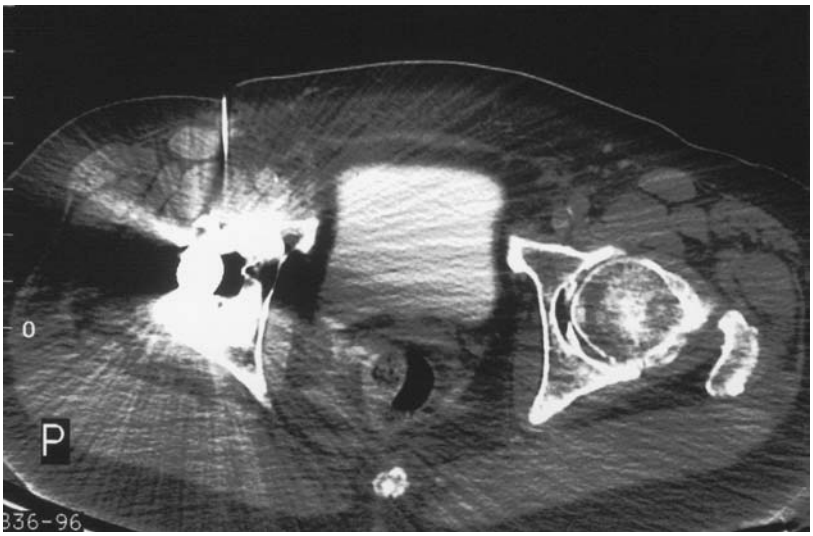

Fig. 3

CT-guided injection.

symptom was reported by every patient. Clinical examination reproduced pain on active straight-leg raising and external rotation. Another sign implicating the psoas muscle was an inability to lift the thigh actively against resistance while in the sitting position. This is similar to the movement required to lift the leg out of a car. We suggest that although the psoas is relaxed with the hip flexed and at rest, it impinges when contracting to flex the hip actively while in the sitting position.

After relief of symptoms had been obtained using an image-intensifier-guided injection in one patient, we attempted to place the injection more accurately under CT guidance after scans had shown an 'impinging' lesion. This injection was beneficial in all patients.

Further management seemed to identify two separate possible causes of this condition. The first, which was found in all patients with a cemented THR, was a true impingement of the psoas muscle. There was fibrotic thickening and granulation tissue on the undersurface of the muscle in the region of the impingement, which was caused usually by a combination of a prominent flange and extruded cement. Removal of these lesions resulted in resolution of symptoms.

The second, involving uncemented cups, had identical clinical features, but no impinging lesion. However, the psoas muscle was thickened and the tendon tight. Lengthening by tenotomy was beneficial in these cases.

In the patients with impingement the cause is obvious. In the others, the underlying pathology is more obscure. There was no gross fixed contracture preoperatively, or significant leg-length discrepancy. All implants were effectively a 45 mm horizontal femoral offset. There was an increase in the offset in these cases as compared with that seen on the preoperative anteroposterior radiographs. However, comparison of the postoperative offset with that of the normal contralateral hip did not show a significant difference. The only other distinguishing feature in these two patients was their relatively young age.

The results of intervention were gratifying, with resolution of pain in all patients. This therefore is an important, treatable cause of pain after THR. There is no need for major revision of the acetabular implant in these cases. We suggest that the characteristic symptoms and the presence of the 'car sign' can identify this condition. Confirmation by CT and image-guided injection is now our preferred initial management. In those in whom pain recurs, operative exploration and management of an impingement lesion or lengthening of the psoas tendon may be needed.

No benefits in any form have been received or will be received from a commercial party related directly or indirectly to the subject of this article.

References

1. **Horne G, Rutherford A, Schemitsch E.** Evaluation of hip pain following cemented total hip arthroplasty. *Orthopaedics* 1990;13:415-9.
2. **Harris WH, Barrack RL.** Contemporary algorithms for evaluation of the painful total hip replacement. *Orthop Rev* 1993;22:531-9.
3. **Harris WH, Barrack RL.** Developments in diagnosis of the painful total hip replacement. *Orthop Rev* 1993;22:439-47.
4. **Trousdale RT, Cabanela ME, Berry DJ.** Anterior iliopsoas impingement after total hip arthroplasty. *Arthroplasty* 1995;10:546-9.
5. **Mallory TH, Halley D.** Posterior buttock pain following total hip replacement: a case report. *Clin Orthop* 1973;90:104-6.
6. **Floman Y, Bernini PM, Marvel JP, Rothman RH.** Low back pain and sciatica following total hip replacement: a report of two cases. *Spine* 1980;5:292-4.
7. **Marmor L.** Stress fracture of the pubic ramus stimulating a loose total hip replacement. *Clin Orthop* 1976;121:103-4.
8. **Gaunt ME, Tan SG, Dias J.** Strangulated obturator hernia masquerading as pain from total hip replacement. *J Bone Joint Surg [Br]* 1992;74-B:782-3.
9. **Mai DD, MacDonald SJ, Bourne RB.** Compartment syndrome of the right anterior thigh after primary total hip arthroplasty. *Can J Surg* 2000;43:226-7.
10. **Siliski JM, Scott RD.** Obturator nerve palsy resulting from intrapelvic extrusion of cement during total hip replacement: report of four cases. *J Bone Joint Surg [Am]* 1985;67-A:1225-8.
11. **Oleksak M, Edge AJ.** Compression of the sciatic nerve by methylmethacrylate cement after total hip replacement. *J Bone Joint Surg [Br]* 1992;74-B:729-30.