ACUTE CALCIFIC TENDINITIS OF THE RECTUS FEMORIS

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We report six caucasian patients who had acute pain in the hip and marked limitation of all movements of the joint. Plain radiographs and CT of the pelvis showed calcification within the reflected head of rectus femoris. All six responded to accurate CT-controlled injections of corticosteroid and local anaesthetic with dramatic and prolonged pain relief, although one required a second injection for recurrence of symptoms after two months.

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Painful periarticular calcification is most commonly seen within the rotator cuff of the shoulder (Neviaser 1983), although it may develop around the wrist, elbow, hip, knee, foot and rarely in the neck (Benanti et al 1986; Cox and Paterson 1991; Dilley and Tonkin 1991). The deposits may be located within the tendon or in the soft tissues adjacent to the tendon or ligament near its attachment to the bone. There is an acute inflammatory reaction, with pain, exquisite tenderness, local swelling and redness. Misdiagnosis is

common and leads to delay in treatment and recovery (Johnson and Guly 1994).

At the hip, trochanteric tendinitis with calcification is well described (LeCocq 1931). It may affect the tendons of the abductors of the hip or even the origin of the vastus lateralis at the distal margin of the trochanter. It usually causes pain in the posterolateral aspect of the thigh which may simulate radicular pain from a prolapsed intervertebral disc.

We report six patients with calcific tendinitis in the reflected head of rectus femoris. This has been described previously (Archer et al 1992; Pope and Keats 1992; Holt and Keats 1993), but the clinical and radiological response to CT-guided injection of steroid and local anaesthetic has not.

PATIENTS AND METHODS

From January 1993 to May 1994 six patients (Table I) were seen at the Lister Hospital with an acute onset of pain in the hip and inability to bear weight on the affected side. None had a history of previous problems in the hip or of any injury of note. The affected hips were held in 20° to 30° of flexion and all movements were limited by pain. The ESR,

Table I. Details of six patients with calcific tendinitis of rectus femoris

Case	Age (yr)	Sex	Injury	Time to presentation (days)	Admitted to hospital	Response to injection	Absence of calcification	Comments
1	43	F	None	2	No	Partial resolution of symptoms	12	Needed reinjection at 6 weeks
2	30	F	None	<1	Yes	Immediate	6	
3	41	M	Picking up shopping	<1	No	Immediate	12	
4	36	F	None	<1	Yes	Relief of pain but discomfort in thigh for 3 days	6	
5	49	M	None	4	Yes	Immediate	6	
6	45	F	Climbing stairs	2	No	Immediate	8	

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the Rheumatoid Latex test, and the serum uric acid and C-reactive protein levels were within normal limits. Radiographs of the hip revealed an area of calcification close to the superior lip of the acetabulum in all cases (Fig. 1). Three of the patients required admission to hospital for control of pain, which was achieved with intramuscular pethidine and oral non-steroidal anti-inflammatory agents. Subsequent CT confirmed the presence of calcification

Fig. 1

AP view showing calcification near the superior lip of the acetabulum.

within the reflected head of rectus femoris in all six patients (Fig. 2), and excluded an intra-articular lesion. Under CT control, a 22-gauge spinal needle was positioned within the calcification and 80 mg of methylprednisolone in combination with 2 ml of 0.5% bupivacaine were injected (Fig. 3).

RESULTS

After injection, all patients obtained dramatic relief from pain and were discharged the next day. One had a recurrence two months later and received a second injection with complete relief; she was free from pain one year later. The others had no recurrence after six months. Further radiographs revealed complete disappearance of the calcification by six weeks in four patients (Fig. 4) and by 12 weeks in the other two.

DISCUSSION

Goldenberg and Leventhal (1936) reviewed the radiographs of 550 adult hips and found calcified deposits near the greater trochanter in 30 (5.4%). They concluded that the calcification occurred at three sites, namely in the tendon of gluteus medius, in the bursa between the tendon of gluteus medius and the greater trochanter and in the undersurface of gluteus medius but not connected to the trochanter. Blundell Jones (1955) reported seven patients with an acute onset of pain in the hip and periarticular calcification. All were managed conservatively with persistence of symptoms for at least two weeks. Using stereoscopic radiographs he identified the site of calcification as the gluteus medius in one patient and beneath it or in the capsule in the others. Review of the radiographs shown in his paper suggests that some of these lesions may have been within rectus femoris.

Fig. 2

CT of the right hip showing calcification within the reflected head of rectus femoris.

Fig. 3

CT-guided injection into the calcific deposit within the tendon.

Fig. 4

AP view of the same hip as in Figure 1 showing complete resolution of the calcification at six weeks.

In our series, calcification was noted in the reflected head of rectus femoris close to the capsule of the hip. The physical signs were those of acute synovitis of the hip, suggesting that the deposit in the tendon may have ruptured into the hip to precipitate the acute episode (Blundell Jones 1955).

Calcific tendinitis of rectus femoris has been reported previously (Archer et al 1992; Pope and Keats 1992; Holt and Keats 1993) and has usually been treated with oral non-steroidal anti-inflammatory agents, although radiotherapy has also been used (King and Vanderpool 1967). The symptoms have usually resolved over the course of a few weeks (Blundell Jones 1955; Pope and Keats 1992; Holt and Keats 1993).

The differential diagnosis of calcification in the region of rectus femoris includes os acetabuli, avulsion fractures, sesamoid bones in the tendon of rectus femoris and myositis ossificans. A comet-tail appearance of the calcific deposit suggests that it lies within a tendon. Malignancies such as juxtacortical chondroma, chondrosarcoma and synovial sarcoma must be considered but CT allows proper visualisation and localisation of the lesion (Hodge et al 1993) and facilitates accurate injection if required.

The exact mechanisms of the origin and of resorption of the calcium deposits are not clearly understood. Schein and Lehmann (1941) proposed that a preceding injury to the tendon could provoke healing by calcification rather than by scarring. Carroll, Sinton and Garcia (1955) suggested 'local stress necrosis' as the cause of the deposition of calcium salts, either directly or through fatty-acid and soap intermediaries. Uhthoff, Sarkar and Maynard (1976), after histological studies of patients treated by operation, proposed local hypoxia secondary to either mechanical or vascular factors as the causative factor. Degeneration of tendon substance into fibrocartilage and subsequent calcification, mediated by chondrocytes, are followed by vascular proliferation and subsequent resorption of the deposits by macrophages, which restores normal perfusion and oxygen tension to the tissue. After resorption of the calcium, the tendon probably regains its original architecture by the synthesis of new matrix, so that no functional impairment remains. It is suggested that chemical dissolution of the inorganic material is not responsible for the acute phase of resorption which probably depends on a change in the

bonding capacity of the organic molecules. This initiates phagocytosis in the resorptive phase (Gärtner and Simons 1990).

We are unable to explain the clustering of six patients with a rare condition in one area; we treated only one further case in the year after the study. Prompt diagnosis and early treatment with local injection of corticosteroid under CT control were successful in relieving symptoms in all these patients.

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