Case report

Pain in the knee associated with osteoporosis of the patella

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SUMMARY Four patients, all of whom complained of pain in the knee, were found to have radiological evidence of osteoporosis particularly marked in the patella. In 3 a neurological lesion at the appropriate spinal segment was present, and the fourth patient, who was frankly hysterical, had an autonomic disturbance. If radiological porosis is isolated to the patella a distant cause such as nerve irritation should be sought.

Osteoporosis of the patella was diagnosed by radiology in 4 patients. Symptoms in all 4 patients were similar. The radiological changes were unexpected and it may be that the pain so accurately localized to the patella itself is due to reflex bone dystrophy.

Case reports

CASE 1

Diagnosis: L3-4 root irritation due to a disc lesion
A 53-year-old housewife presented to a rheumatology department with a 3-week history of a nagging ache in the right knee. This was worse on weight-bearing and there was no history of trauma.

Full clinical examination was normal; in particular the right knee had a full painless range of active and passive movement though some crepitus was present. Anteroposterior and lateral views of both knees showed minor degenerative change in keeping with her age. 4 months later the pain was worse and disturbing her sleep. For the first time tenderness in the patella was elicited. Further x-rays were taken (Fig. 1) and as the appearance of the patella was unusual, she was referred to an orthopaedic surgeon who diagnosed and operated upon an L3-4 disc lesion. At no stage had she admitted to having pain in the back.

Fig. 1 X-rays of patellae of Case 1.

The x-rays showed a spotty porosis throughout the patella with subcortical lucent bands, but little evidence of porosis in the femoral condyles. Her knee pain settled immediately after surgery but she was lost to long-term follow-up.

CASE 2

Diagnosis: diabetic amyotrophy
This patient, born in 1897, was well until he developed low back pain accompanied by dull symptoms in 1968. He rapidly lost 13 kg in weight and was found to have diabetes mellitus. Back pain disappeared after wearing a plaster jacket but he then developed weakness of both legs and pins and needles in both feet. There was proximal wasting and weakness with absence of both knee and ankle jerks.

Vibration sense in the legs was absent but pin-prick intact. Cerebrospinal fluid was normal and electrical testing showed denervation in the left extensor digitorum brevis and both quadriceps muscles. While being treated with diet alone for diabetes, he developed a painful flexion deformity of the left knee. The knees were dry, cool, and not tender. X-rays showed osteoporosis of both patellae with minor changes in the femoral condyles.

He was readmitted in May 1969 with a neurogenic bladder by which time the osteoporosis in the patellae was more marked though he had no knee pain. Skyline views of both patellae taken in September 1970 (Fig. 2) showed spotty porosis as described by Jones (1969). The following year he developed a red, cold ischaemic left foot and underwent a below-knee amputation. He died in 1973.

Fig. 2 Skyline views of patellae of Case 2.

CASE 3

Diagnosis: femoral nerve palsy

A 56-year-old clerk was admitted to hospital in March 1971 with a myocardial infarction. He was treated with heparin and 4 days later developed a retroperitoneal haematoma accompanied by weakness of hip flexion, knee extension, and painful paraesthesiae over the front of the right thigh.

He was readmitted in July with a painful effusion of the right knee. Knee extension was weak, the right knee jerk absent, the quadriceps wasted and electrical activity in this muscle nearly absent. X-rays (Fig. 3) showed a widespread spotty osteoporosis with subcortical lucent bands, particularly on the femoral articular surface of the patella, though there

Fig. 3 X-rays of patellae of Case 3.

was minor spotty porosis in the femoral condyles. The right knee jerk was still absent a year later but he was no longer bothered by his knee and no further x-rays were taken.

CASE 4

Diagnosis: hysterical personality disorder

An ambulance man, born in 1936, was sitting in the back of his ambulance in February 1972 when it was hit in the rear by a car. He was shot through the open sliding door into the driving cab, knocking his head and grazing his left knee. He did not lose consciousness and was fully orientated when seen in casualty. An hour later he became drowsy, denied all memory of the accident which he had previously clearly described, and claimed not to remember his name. X-rays of knees and skull were normal.

On being transferred to the ward, he complained of headache and pain in his apparently uninjured right knee. 6 days later x-rays of the right knee were taken but tunnel views and skyline views were not obtained as he claimed to be unable to flex his knee. The other views were normal.

Three weeks after the accident he developed signs of a deep vein thrombosis and then a pulmonary embolus. Having recovered from this he developed hysterical amnesia and bilateral blindness. By June 1972 his right knee would not bend actively nor passively but he walked normally under hypnosis.

He was seen a year later in the rheumatology department. He claimed to be unfit for work and his compensation case was still pending. He walked and 'sat' with the right knee and hip held actively and rigidly in extension. The right foot was sweaty, hairless, swollen, and pink. No pulses were felt. There was a small effusion in the right knee, the right patella was exquisitely tender, and the quadriceps muscle measured 10 cm above the patella and was 2.5 cm less in diameter than the left. X-rays (Fig. 4) of December 1972 showed generalized porotic changes in the patella and minor porotic changes were also seen in the femoral condyles.

Fig. 4 X-rays of patellae of Case 4.

Discussion

We believe that our 4 patients have a neurovascular dystrophy similar to that described by Sudeck in 1900. Sudeck's atrophy appears to be an entity of its own but avascular necrosis also appears to be a component in osteochondritis and perhaps in chondromalacia patellae (CP).

Our patients were too old to have a growth disorder but an osteochondritis of the primary epiphysis of the patella was described in 1908 by Köhler, and Sinding-Larsen (1921) described similar changes in the secondary epiphysis in older children. According to Lewin (1952), osteochondritis interferes with the blood supply and leads to avascular necrosis.

Our patients did not have clinical evidence of CP. They were all older than one would expect and x-rays showed a generalized abnormality affecting the patella. Most authors in any case consider CP to be primarily a disorder of articular cartilage. Chalkin (1939) thought that the bone changes were secondary. Cox (1945) found areas of necrosis in the subchondral bone. Wiles *et al.* (1960), staging the pathological changes, mentioned changes in the subchondral bone but, like Outerbridge (1961), reported no other abnormality in the patella. Recently, Darracott and Vernon Roberts (1971), examining 11 patellae excised for CP, found histological evidence of focal or diffuse osteoporosis of trabecular bone most marked in the region of the blood supply. They suggested that CP is not a primary disease of cartilage but that both cartilaginous and bony changes are secondary to a disturbance in the blood supply.

Mechanical factors may also play a part in the aetiology of CP. Insall *et al.* (1976) suggested that the state of patella alta (a high-riding patella where the length of the patellar tendon exceeds the length of the patella by 1 cm or more) predisposes to CP. 3 of our 4 patients, excluding the one with diabetes, had patella alta.

We found that the report by Rostock (1929) was the only one which specifically described aseptic bone necrosis of the adult patella. He studied 4 patients, one of whom was a 16-year-old girl. The other 3, in 2 of whom the patella was explored surgically, were working men with pain in the knee, loss of movement and muscle wasting after injury. At operation there were cavities filled with necrotic bone surrounded by osteoid material. The x-rays of Rostock's second patient are remarkably similar to our patients' x-rays in that the patella was 'spotty'.

King (1935) suggested that the initiating factor in cases of localized bone rarefaction is an injury. A neurovascular disturbance of some sort then

produces the peculiar change of 'spotted atrophy' which distinguishes it from ordinary disuse atrophy. Jones (1969) studied the hands or feet of 100 patients whose limbs had been immobilized in plaster. There was general or diffuse rarefaction in 34 and a spotted porosis in 46. He did not study the more proximal bones.

One of us (A.K.T.), therefore, looked at the films of 100 patients whose fractured legs had been immobilized in plaster for periods of up to one year. None had complained of knee pain and in no case was porosis isolated to the patella. Rarefaction of the patella was common but was always accompanied by general decalcification of the other bones, particularly marked in the femoral condyles.

Three of our patients had a peripheral neurological disorder. King (1935) stated that peripheral nerve lesions give rise to rarefaction in those bones whose movements depend on the muscles which are weak. Smillie (1974) stated that diabetic patients may develop wasting and weakness of the quadriceps muscle with sensory changes, indicating a lesion of the femoral nerve. Neither he nor Garland (1955) in his description of diabetic amyotrophy mentioned bony changes.

We suggest that our first 3 patients had a neurovascular dystrophy secondary to root or nerve irritation. The fourth patient clearly had some disturbance of the autonomic system. The x-rays of all 4 show a spotted disuse atrophy of the patella with little change in the femoral condyles. When such an appearance is seen a careful neurological assessment is indicated.

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