Charcot’s joints: a case report

Osteonecrosis may be a predisposing condition for Charcot’s joints

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Summary. In the case of a patient who had Charcot’s joints in her left hip and right shoulders, multiple osteonecroses in the joints were disclosed by MR imaging. This suggested that osteonecrosis may be a predisposing condition for Charcot’s joints.

Charcot’s joint is an insidiously destructive joint disease. It has sites of predilection such that in tabes dorsalis it affects mostly lower limb joints and in syringomyelia, mostly upper limb joints [11, 14]. The pathomechanism of Charcot’s joint is still controversial. There are two main hypotheses. First, the neurotraumatic theory [3] is based on the assumption that minor trauma causes the destruction of a joint in which pain sensation is disturbed. Secondly the neurovascular theory [2] proposes that a disturbed blood supply changes the bony metabolism to a catabolic state.

We had a patient with an unusual manifestation of Charcot’s joints [12], insofar as both upper and lower extremities were affected. Magnetic resonance imaging (MRI) of one shoulder and the contralateral hip joint and of both diaphyseal femora revealed osteonecroses, which, it is suggested were the predisposing condition for Charcot’s joint.

Case report

A 78-year-old woman attended our out-patient clinic on 5 May 1989. She had fallen out of bed 7 days before, and complained of gait disturbance due to leg length discrepancy and instability of her left hip. She also had mild pain in her right shoulder. She could walk with a walking frame in the nursing home where she lived. Physical examination showed slight swelling and an almost full range of motion in the involved joints. Neurological findings included positive Argyll-Robertson pupil (right side), Westphal’s sign and impaired vibration sense. Proprioception appeared normal. Romberg’s sign was negative. Serological tests revealed a positive Wassermann’s reaction and normal fasting blood sugar. Cervical and thoracic MRI did not support the suspicion of syringomyelia. A diagnosis of tabetic Charcot’s joints was therefore made. X-ray films revealed typical Charcot’s joints in her left hip (Fig. 1) and right shoulder (Fig. 2). Her sequential radiographs showed that her shoulder and hip seemed to have been normal before her fall. After the fall, her hip and shoulder showed an early stage of Charcot’s joint, accompanied by subcapital fracture in her left hip. Typical destruction developed in the next 3 months (Figs. 1, 2). MRI suggested soft tissue hypertrophy and joint fluid in the left hip, as shown in Fig. 3. Interestingly, the subcapital area of the right hip and the anterior portion of the left humeral head, which appeared normal on the plain X-ray, showed low signals in T1-weighted and combined high and low signals in T2-weighted MR images (Figs. 4, 5). The diaphyses of both femora also showed low signals in T1-weighted images (Fig. 6a, b). These data suggested that the right femoral head was necrotic with repair already in progress, while the left shoulder and the diaphyses of both femora were in the early stages of necrosis [6, 8]. Of course, no particular information could be gained from plain X-ray films of these joints. The bone scintigram showed high uptake in the right shoulder and the left hip and also in the adjacent bones of the involved joints (Fig. 7). A total hip arthroplasty was performed because of the gait disturbance and the standing incapacity due to the instability of her hip. Histologically, hypertrophic synovial tissue incorporating bone and cartilage debris with giant cells and capillary proliferation were seen (Fig. 8), which typical for Charcot’s joint [1].

Discussion

The pathomechanism of Charcot’s joint has already been discussed in some detail [2], but there is no generally accepted opinion on this as yet. In our patient, MRI disclosed osteonecroses of the femoral and humeral heads, which appeared normal on plain X-ray films. Bilaterality of osteonecroses in the femoral heads [7, 9, 10] and diaphyses supported the speculation that the same condition may have predisposed the left hip and also the right shoulder to transformation into Charcot’s joints. Minimal trauma caused the fracture of the femoral neck [4, 5, 13] because of the weakness in the subcapital region. An impaired sense of pain led the patient to continue walking, resulting later in destruction of the hip joint. It seems that the same process was going on in her right shoulder. This supports the neurotraumatic theory. But the high uptake in the bones adjacent to the involved joints in the bone scintigram could also be interpreted as vascular disturbance and increased metabolism, which would sup-
Fig. 1a–c. Sequential roentgenograms of the left hip joint. a On 22 March 1989, before the patient had fallen out of bed, the hip joint appeared normal. b On 5 May 1989, after this fall, subcapital fracture of left hip and upper and lateral migration of femoral neck were seen. c On 24 August 1989, upward migration of left femoral neck was increased and the femoral head was resorbed.

Fig. 2a–c. Sequential roentgenograms of right shoulder. a On 26 March 1989, the shoulder joint seemed to be normal. b On 5 May 1989, humeral head had begun to be resorbed. c On 24 August 1989, humeral head and glenoid were resorbed but reactive bone formation was minimal.

Fig. 3a, b. Coronal MRI of both hips shows that the left hip joint was filled with low signal on T1-weighted and high signal in T2-weighted images; it was suggested that this was due to joint fluid. Right hip showed low signal on T1-weighted image, which suggested osteonecrosis. (MRI apparatus: Phylips Gyrosan, 1.5 Tesla)

Fig. 4a, b. Axial MRI of the right hip joint. T1-weighted image (a) showed low signal in the subcapital area, and T2-weighted image (b) showed high and low signal in the same area. These findings suggest osteonecrosis and repair processes in the subcapital region of the right femoral head (1.5 Tesla, Phylips Gyrosan)