(Benkeddache and Gottesman 1982, Evanchick et al. 1986, Watts and Lifeso 1996, Vohra et al. 1997). The involvement of metacarpal bones is rare (Benkeddache and Gottesman 1982, Halsey et al. 1982, Evanchick et al. 1986, Martini 1988). Tuberculosis can also destroy tendons (Bush and Schneider 1984).

There is often a long delay between the onset of tuberculosis and the diagnosis (Bush and Schneider 1984, Shannon et al. 1990, Vohra et al. 1997). Musculoskeletal tuberculosis can mimic other infectious and inflammatory diseases like reumatoid arthritis, gout, chronic pyogenic osteomyelitis, granulomatous lesions, Brodie's abscess, tumors and foreign body lesions (Bush and Schneider 1984, Boulware et al. 1985, Evanchick et al. 1986, Shannon et al. 1990, Maffulli 1992, Vohra et al. 1997). Signs of pulmonary tuberculosis are not always present and the main complaints are swelling and pain, not responding to analgetic medication (Bush and Schneider 1984, Vohra et al. 1997).

A bone scan is recommended for detecting possible multiple bone involvement (Shannon et al. 1990). However, a negative bone scan does not exclude tuberculosis (Watts and Lifeso 1996). Surgery may be necessary to obtain a biopsy, but only seldom to eradicate the lesions (Evanchick et al. 1986, Martini 1988).

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HLA-B27-negative ankylosing spondylitis resulting in panclavicular ligament ossification—a 28-year follow-up

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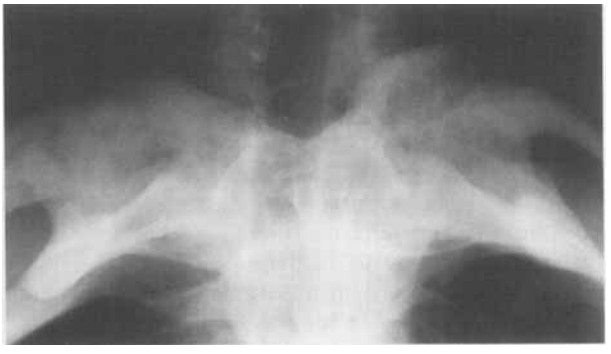
A 32-year-old Japanese auto-mechanic spontaneously developed pain in his right clavicular region in 1968. He also had low back pain of insidious onset. Clinical investigations in 1975 revealed limitation of cervical and lumbar spinal motion, swelling and redness over the right clavicle, and dilatation of cutaneous veins over the anterior chest. CRP was 4.3 mg/dL and ESR was 104 mm/h, while rheumatoid factor was negative. Serum calcium was 7.8 mg/dL and serum alkaline phosphatase was 250 IU/L. Culture from an aspiration from the right clavicle yielded a few *Staphylococcus aureus;* however, the bacteriologists interpreted this finding as contamination from the skin.

He was seen, on average, once a year during the next 20 years with pain and redness in the right clavicular region. Each time non-steroidal anti-inflammatory drugs were given, resulting in temporary relief. Since 1992, he has had painless stiffness of the right shoulder joint. When examined by the authors in 1995, the elevation of the right shoulder was limited to 80°. In the Schober test, the mobility of the lumbar spine was 0 cm, and the chest circumference on deep inspiration was only 1.5 cm greater than that on expiration. Repeated blood tests invariably showed a high titer of CRP and elevated ESR. HLA antigen was positive for A24, A30, B61, CW3 and CW7, but negative for B27. Throughout the clinical course during 28 years, the patient had no skin lesions or eye, geni-

The bamboo appearance of the lumbar spine and complete fusion of the sacroiliac joints in 1975.



Marked sternocostoclavicular hyperostosis in 1975.

tourinary or bowel symptoms.

Radiographs revealed that the whole spine and the sacroiliac joints had been ankylotic since 1975. The hips showed joint space narrowing with irregular sclerosis of the acetabulum. The sternocostoclavicular region showed marked hyperostosis bilaterally from 1975 through 1995. The right acromioclavicular joint had a normal appearance in 1975, bony erosions in 1980 and has shown osseous ankylosis since 1992. The coracoclavicular ligament became ossified partly in 1980 and entirely in 1995 (Figure). The glenohumeral joint was radiographically normal. Bone scan, with ^{99m}technetium-diphosphonate in 1975 and 1992, revealed accumulation of the radioisotopes around the lateral end of the right clavicle and in the spine, the sacroiliac and the hip joints and the sternoclavicular region. Venography in 1975 showed complete obstruction of the right subclavian vein.

Discussion

The most likely diagnosis was ankylosing spondylitis (AS). The pain and stiffness in the lower back and the reduced thorax mobility fulfilled the Rome and the New York criteria of AS (Resnick and Niwayama 1995). Moreover, radiographic appearances in the spine and the sacroiliac and hip joints were entirely consistent with those of advanced AS (Resnick and Niwayama 1995). Our patient was negative for HLA-B27; but, among the Japanese, the appearance of HLA-B27 antigenicity has been known to be extremely rare in the normal population and relatively low in patients with AS (Sonozaki et al. 1975).

Other possible diagnoses would have been pustulotic arthro-osteitis (PAO) (Sonozaki et al. 1981) or synovitis, acne, pustulosis, hyperostosis and osteitis (SAPHO) syndrome (Chamot et al. 1987), which often presents with so-called sternocostoclavicular hyperostosis (SCCH) (Köhler et al. 1975) and is usually negative for HLA-B27 (Saghafi and Buchanan 1993). This syndrome usually presents with spondylarthritis simulating AS (Sonozaki et al. 1981, Fritz et al. 1992, Jurik 1992, Saghafi and Buchanan 1993). However, in our case, no

Preserved acromioclavicular joint space and partial ossification of the coracoclavicular ligament in 1980.

Osseous ankylosis of the acromioclavicular joint and the coracoclavicular ligament in 1995.

skin lesions were found during the follow-up period of 28 years. This conflicts with the diagnosis of SAPHO syndrome, although in rare instances the skin manifestations may be delayed more than 20 years after the appearance of bone lesions (Kahn et al. 1991).

Our patient showed progressive ossification around the lateral end of the clavicle years after the spinal and sternoclavicular changes seemed complete. SCCH can occur with AS (Jurik 1992), and the acromioclavicular joint fusion (Emery et al. 1991) or the coracoclavicular ligament ossification (Pritchett 1983) individually may develop in AS. However, to our knowledge, ossification of all the ligamentous connections of the clavicle has not been described. We believe that, in AS, progressive ossification of the peripheral joints and ligaments may occur, even after the hyperostosis of the axial skeleton has seemingly been completed. This, in turn, indicates that a long-term follow-up is required to diagnose the final extent of ossification and to discuss the similarity or dissimilarity of hyperostotic syndromes. In addition, our case indicates that AS may cause painless restriction of shoulder joint mobility due to loss of scapulothoracic motion, not due to the glenohumeral involvement.

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Spontaneous osteonecrosis of the capitate—a case report

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A 26-year-old man presented with a long history of pain in his left wrist. He had no trauma and no symptoms suggestive of systemic arthropathy. There was a history of primary hypothyroidism and possible coeliac disease. He had a short stature and small hands, a markedly reduced range of movement in all directions at his wrist, and he was tender over the dorsum of the wrist.

Radiographs revealed a fragmented and sclerosed capitate and a diagnosis of spontaneous osteonecrosis of the capitate was made (Figure).

At operation, the capitate was soft and the joint surface was abnormally pink. The capitate was subtotally excised, leaving the distal one third, and the space was filled with an iliac cortico-cancellous graft. The surrounding joint surfaces were denuded of articular cartilage and the carpal mass was fixed with staples. Histological analysis showed necrotic bone, but no vasculitis.

4 months postoperatively, he was almost pain-free and the range of wrist movement was from 10 degrees of extension to 40 degrees of palmar flexion. Radiographs showed progressive union of the carpal fusion.