

Hypertrophic osteoarthropathy after cerebrovascular insult

Sirs,

We report the case of a patient who developed HO after several episodes of cerebrovascular insult (CVI). Since 1972 the patient had increased blood pressure and occasionally took antihypertensive therapy. In 1990 several CVI appeared, first with left and then with right hemiparesis, talking disturbances and headache. He had been treated in a Department of Neurology with the diagnosis of "Insuffientia vascularis cerebri recidivans". In less than 4 months, after the last CVI affected the left cerebrum hemisphere, clubbed fingers on the hands and feet started to develop (Fig. 1).

The patient had left facial paresis (central type) and sometimes could "hardly remember some words". His RR was 170/95 mm Hg and pulse 80/min. He could not walk on his toes and heels and fell on the right side. Romberg was negative. He could hardly elevate the right leg from the ground with tremor and performed the heel-knee test with failure, markedly on the right side. Sensibility was not changed. Own reflexes were lively, Babinski was positive on both sides. The hands were moistened and sweaty with furrowed nails, periungual erythema, and tight and shining skin. The distal parts of the fingers were hyperextended. The volume of the hands was enlarged. Fine finger movements had become clumsy. The patient had finger clubbing characterized by nails that were convex toward the dorsal side and looked like watch glass. The distal parts of the lower legs were swollen with perimalleolar edema. The patient felt tingling sensations and skeletal pain in the distal part of the lower legs. Measurement of the finger circumference across the middle of the nail by a goldsmith's ring showed an enlarged circumference. A Schamroth's test was positive.

Scintigraphy showed intensified accumulation in the region of the right parietal bone, the dorsal leg of the 10th right rib, both upper arms and forearms, radiocarpal joints, metacarpal bones and some phalanges, acetabulums, the upper legs, lower legs and feet. Color Doppler of the carotid trunk showed swollen and uneven walls of the common carotid arteries (CCA) and obliterated left CCA. Transcranial Doppler showed asymmetric circulation of the Willis's circle.

This patient fulfills the criteria for HO (1). He developed clubbed fingers during the first 2 months after CVI. After that, the fingers continued to enlarge, so that hyperextension of the distal phalanx of all the fingers and

other signs of clubbed fingers developed. Scintigraphy showed an abundant radioisotope accumulation in the long bones (tibia, fibula, radius and ulna) and the flat bones (calvaria, pelvis and radiocarpal joint), and after 4 months a periosteal reaction of both tibias was found on radiological images. Thermography was positive in the face and the hands.

Clubbed fingers are the main symptom of primary hypertrophic osteoarthropathy (HO), which was first described by Hippocrates. Many centuries later, Bamberger (1, 2) and Marie (3) recognized them as a part of the typical clinical and pathological picture that Marie termed "pulmonary HO". This form is different from the primary form because it develops over a lifetime, mainly in pulmonary diseases (inflammation, tumor, fibrosis, bronchiectasis) (4), cyanotic congenital heart disease (5), liver diseases (primary biliar cirrhosis, carcinoma of biliar ducts, chronic active hepatitis, posthepatic cirrhosis, alcoholic cirrhosis) (6), ulcerative colitis (7), and Crohn's disease (8). However, HO has very rarely been described in other states or diseases (9).

The etiology and pathophysiology of the syndrome is not clear. Full development of HO appeared with the improvement of hemiparesis. The potential role of CNS in the generation of HO is not understood. There is a theoretic possibility that released growth factors may induct HO, just as in cyanotic heart disease (10). To conclude, SHO in this patient developed after hemiparesis, which has not been described in literature.

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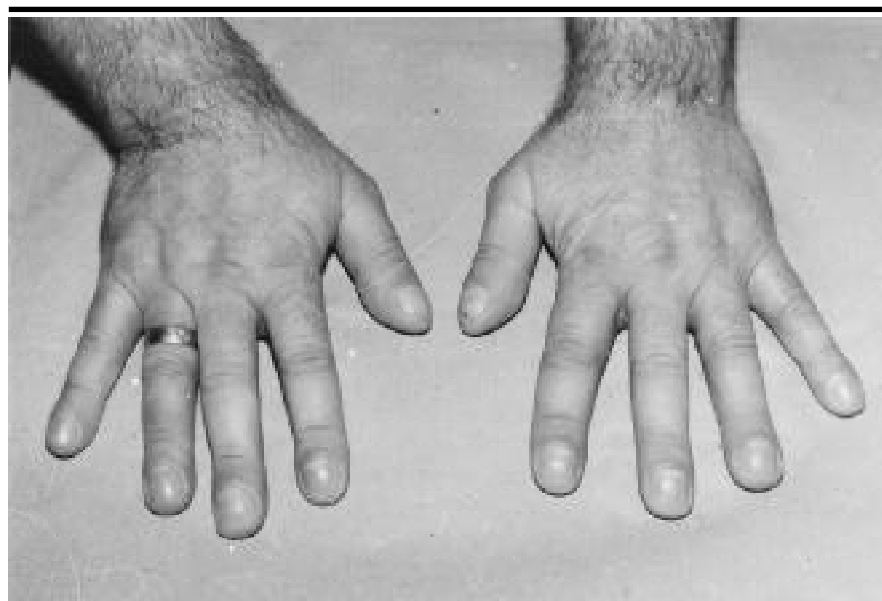


Fig. 1. Clubbed fingers in a patient after cerebrovascular insult.