



Secondary Hypertrophic Osteoarthropathy (HOA) Mimicking Primary HOA (pachydermoperiostitis or Touraine-Solente-Golé Syndrome)

Mark N. Lowenthal FRCPE, Alexander Tombak MD and Alexander Lowenthal M Med Sci

Meonot Maccabi Facility for Complicated Nursing, Ganei Omer, Omer 84965, Israel

IMAJ 2004;6:64

A 64 year old former laborer was admitted to the facility from a general hospital for continuation of antibiotics for post-obstructive pneumonia, and for pain control.

The diagnoses were lung (non-small cell, stage 4) and prostatic carcinoma (markedly elevated prostate-specific antigen at 11.78 ng/ml), with (presumptive) bone metastases (serum calcium initially 12.9 mg/100 ml). Plain radiology of the chest, spine and pelvis did not, however, reveal metastases. The patient had been a life-long tobacco and alcohol abuser. The main complaint was incapacitating joint pain, which prevented walking. The Karnofsky performance level was 40.



Figure 1. Facial skin coarsely furrowed and glistening with sweat.

The skin of the face sweated excessively and was coarsely furrowed at the forehead, nasolabial folds and chin [Figure 1]. The skin of the lower legs and ankles was thickened and coarse and the surface resembled fine sandpaper. Very marked clubbing of the fingers and toes [Figure 2] accompanied severe pain and tenderness adjacent to the large joints, predominantly wrists, knees and ankles. On isotopic bone scan, increased uptake along the cortical surfaces of the long bones of the limbs and diffusely increased uptake in the large joints confirmed hypertrophic osteoarthropathy, but not secondaries. The above are the dermatologic and skeletal features of pachydermoperiostitis, which, together with clubbing of the digits, constitute the Touraine-Solente-Golé syndrome, a genetic condition mainly affecting pubertal boys [1]. This condition is also called the primary form of hypertrophic pulmonary osteoarthropathy [1]. The presence of

HOA = hypertrophic osteoarthropathy



Figure 2. Marked clubbing of the toes.

pulmonary carcinoma, a classic cause of HOA, renders this case a very unusual one in that it combines the features of primary and secondary HOA. Clearly this cannot be a case of genuine Touraine-Solente-Golé syndrome in view of the patient's age and lack of a genetic history.

For pain relief, morphine-controlled release 60 mg daily was instituted, but relief was unsatisfactory. The non-steroidal cox-2 inhibitor, etodolac, in standard dosage, was then substituted, but effective analgesia was achieved only when morphine-controlled release was recommenced, in place of etodolac, in doses rising rapidly to 60 mg twice daily. This resulted in an improved Karnofsky status of 60, enabling the patient to be discharged to the care of his family, in whose care he died a few months later.

Acknowledgments. We thank Drs. S. Agronovich and I. Rachinsky, Department of Nuclear Medicine, Soroka University Hospital, Beer Sheva, Israel, for the bone scan.

Reference

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Correspondence: Dr. M.N. Lowenthal, Meonot Maccabi, Ganei Omer, Omer 84965, Israel. Phone: (972-8) 629-5107 Fax: (972-8) 646-7366 email: markll@bgumail.bgu.ac.il