Hypertrophic Osteoarthropathy in Association with Pulmonary Tuberculosis


Three white male patients with advanced cavitary pulmonary tuberculosis presented with HOA. No other pathology to explain the osteoarthropathy was detected. The osteoarthropathy responded symptomatically to NSAID drugs and treatment of tuberculosis but resolved radiologically in only one patient.

(Chest 1991; 99:769-70)

Table 1 — Main Clinical Features of Three Patients with HOA

<table>
<thead>
<tr>
<th>Age, yr</th>
<th>History of Tuberculosis</th>
<th>Symptoms</th>
<th>Finger Clubbing</th>
</tr>
</thead>
<tbody>
<tr>
<td>1-35</td>
<td>1 TU + None</td>
<td>Weight loss; night sweats; chest pain</td>
<td>No</td>
</tr>
<tr>
<td>2-48</td>
<td>10 TU + 1 yr previously</td>
<td>Cough; weight loss; fever; malaise</td>
<td>No</td>
</tr>
<tr>
<td>3-44</td>
<td>1 TU + 20 yr, 2 yr, 1 yr previously</td>
<td>Cough; weight loss; fever</td>
<td>Yes</td>
</tr>
</tbody>
</table>

staining and culture of sputum for Mycobacterium tuberculosis, and the isolates were fully sensitive to first-line antituberculosis drugs. The patients complained of pain and stiffness in the wrists and ankles, and roentgenograms showed subperiosteal new bone formation. All three patients had a history of excessive alcohol intake, but only in cases 2 and 3 did this disrupt their life-style.

All three patients had advanced cavitary pulmonary tuberculosis. None of the three patients had neurovascular changes or increased thickness of subcutaneous tissue. Finger clubbing was present in case 3, and all patients had tenderness over the lower forearms. Symptoms of HOA resolved with NSAID drugs, analgesia, and response of tuberculosis to treatment. Radiologically, the HOA resolved in case 1. Details of the patients are given in Table 1.

The patients were investigated for underlying bronchogenic carcinoma or other pathology to explain the presence of HOA. In no case was any other pathology identified. Patient 1 is alive and well six years after presentation. Patient 2 was alive and well when last seen 2 1/2 years after presentation, and patient 3 died three years after initial presentation following craniotomy for a pyogenic brain abscess.

**DISCUSSION**

There were no clear pointers to the pathogenesis of HOA in these patients. The pulmonary tuberculosis was extensive, with involvement of both lungs, cavitation, and extensive disease. This has been previously discussed.

Why the HOA resolved in case 1 and not in the other two cases is unexplained, but both patients 2 and 3 had been noncompliant and hence may have had active tuberculosis for a considerably longer time. This may have allowed the HOA to become irreversible or the provoking agent to continue to be secreted for a prolonged period of time.

The pathogenesis of HOA is unknown, but the earliest changes are an increase and growth of vascular connective tissue in association with subperiosteal new bone formation. There is increased blood flow to the extremities, but this is shunted through arterial venous communications close to the areas of the osteoarthropathy. The increase in blood flow to the extremities is due to a reflex mechanism with the vagus nerve as the afferent pathway. The efferent pathway is unknown but is probably hormonal, rather than neuronal. Abnormal intrathoracic masses or tissue, or ischemic tissue distal to these masses or pulmonary damage may be the stimulus to initiate this reflex; however, vagotomy may not always improve blood flow to the extremities, even when HOA is seen in association with intrathoracic disease.

Hypertrophic osteoarthropathy affecting mainly the lower

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Hypertrophic osteoarthropathy is a systemic disorder of bones, joints, and soft tissue which develops in association with another disease process, most commonly intrathoracic disease. Hypertrophic osteoarthropathy is characterized by several or all of the following abnormalities: (1) clubbing of the digits; (2) periosteal new bone formation; (3) symmetrical arthritis-like changes in the joints and periarticular tissues, most commonly of the ankles, knees, wrists, and elbows; (4) increased thickness of the subcutaneous soft tissue in the distal one-third of the arms and legs; and (5) neurovascular changes of the hands and feet, including chronic erythema, paresthesia, and increased sweating.

Most cases of HOA seen today are associated with bronchogenic carcinoma, other intrathoracic neoplasms, and Hodgkin's disease of the mediastinum. It is also seen in severe cystic fibrosis, bronchiectasis, chronic empyema, and lung abscess, occasionally in hepatic disease, and rarely in pregnancy, and purgative abuse.

In the last two decades, HOA has been described in only two cases of severe pulmonary tuberculosis. It has been stated that HOA does not occur in tuberculosis, and its presence suggests another underlying pathology.

**CASE REPORTS**

We present three case histories of HOA in association with pulmonary tuberculosis. All three patients were positive on direct

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limbs has been described in association with abdominal aortic grafts (Dacron grafts).22-24

REFERENCES
16 Fraser RG, Pare JAP, Pare PD, Fraser RS, Genereux GP. Diagnosis of disease of chest. 3rd ed. Philadelphia: WB Saunders, 1988; 1:404 (ch 3)

Spontaneous Regression of Cardiomyopathy in a Patient with the Acquired Immunodeficiency Syndrome*

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Cardiac involvement is common in patients with the acquired immunodeficiency syndrome (AIDS) and, when symptomatic, it portends a poor prognosis. We present a case of marked spontaneous regression of cardiomyopathy in a patient with AIDS. To our knowledge, this is the first reported case of spontaneous recovery of ventricular function in an AIDS patient.

(Chest 1991; 99:770-72)

\[ \text{VEF} = \text{ventricular ejection fraction} \]

Cardiovascular abnormalities are commonly found in patients with the acquired immunodeficiency syndrome (AIDS). We present a case of idiopathic cardiomyopathy in a patient with AIDS who experienced spontaneous resolution of congestive heart failure associated with improvement in ventricular function. To our knowledge, this is the first reported case of such a recovery.

CASE REPORT

A 32-year-old woman was found to have human immunodeficiency virus (HIV) infection (Walter-Reed stage 5, CDC group IV-B) in November 1987 when she developed Guillain-Barré syndrome. A history of intravenous cocaine and heroin abuse was obtained. Echocardiography was normal. Her clinical course was stable until June 1988, when she presented with onset of peripheral edema, abdominal fullness, and dyspnea. Her only medication was methadone.

Physical examination revealed a thin woman, afebrile, with normal vital signs. Marked jugular venous distention with prominent V waves was noted without pulsus paradoxus. The lungs were clear. Cardiac examination revealed a right ventricular heave, summation gallop, and a grade 2/6 pansystolic murmur over the xyphoid with inspiratory augmentation. Pulsatile hepatomegaly and 2+ bipedal edema were present.

Laboratory data at the time of hospital admission showed hemoglobin of 11.4 g/dL, WBC count of 4,800 cu/mm (normal differential), and normal creatinine. Room air arterial oxygen saturation was 92 percent. Chest roentgenography showed clear lung fields with a markedly increased cardiac silhouette (Fig 1). Electrocardiography demonstrated sinus tachycardia with left atrial abnormality, decreased voltage, and diffuse T-wave flattening.

Echocardiography showed biventricular dilatation with generalized hypokinesis, paradoxic septal motion, and small pericardial effusion. Doppler studies demonstrated pulmonic and tricuspid insufficiency. Gated radionuclide ventriculography revealed a left ventricular ejection fraction (VEF) of 36 percent; right VEF was 37 percent by first pass technique. Gallium and perfusion lung scan results were normal. All cultures, antibody titers, rheumatologic studies, and endocrine studies were normal.

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