COMMUNICATIONS TO THE EDITOR

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Tolazamide-induced Chronic Eosinophilic Pneumonia

To the Editor:

Drug-induced eosinophilic lung disease characteristically presents as a brief, mild illness with transient roentgenographic shadows. Less commonly, several drugs, including chlorpropamide among the sulfonylurea drugs, have been reported to produce a subacute disease, the syndrome of chronic eosinophilic pneumonia (CEP), with fever, night sweats, dyspnea and weight loss. Here we present a case of CEP caused by another sulfonylurea, tolazamide.

CASE REPORT

A 70-year-old man was admitted for weakness, weight loss, nonproductive cough and temperature to 39°C of three weeks' duration. There was no response to a 14-day course of erythromycin therapy. The patient had had normal findings on a previous chest roentgenogram, no antecedent pulmonary disease, atopy or occupational exposure, and a nine-year history of diabetes mellitus treated with tolazamide. On admission, he appeared chronically ill and had a temperature of 38°C; otherwise, results of physical examination were normal. Laboratory values were normal except for a hemoglobin of 11 g percent, a leukocytosis of 15,000/ml and 9 percent eosinophilia. Chest roentgenogram revealed bilateral upper lobe infiltrates (Fig 1). Blood cultures, PPD (5 TU) test, and sputum culture grew normal flora. Ova and parasites were not found in the stool. Fiberoptic bronchoscopy was normal, bronchial brushings and washings were negative for acid-fast bacilli, fungi and malignant cells, and the patient refused a transbronchial lung biopsy.

Tolazamide was omitted, as the diabetes was controlled with diet alone. The patient became afebrile one day after admission, and remained asymptomatic for the rest of the four-day hospitalization. Isoniazid and rifampin were started pending culture results, but were discontinued two weeks after discharge due to anorexia. Three weeks after discharge, the chest roentgenogram revealed almost complete resolution of the infiltrates. The patient had glycosuria and he was restarted on tolazamide. Intermittent fevers, cough and weakness recurred, and six weeks later, the chest roentgenogram revealed recurrence of bilateral upper lobe infiltrates. Tolazamide was discontinued, as there was a 32 percent eosinophilia. Within one week, he became afebrile, felt well, regained weight and the chest roentgenogram became normal. His hemoglobin increased to 14 g percent, the eosinophil count dropped, and subsequent chest roentgenograms remained normal. The cultures taken at bronchoscopy for acid-fast bacilli and fungi were negative.

Figure 1. Initial chest roentgenogram showing bilateral upper lobe infiltrates.

DISCUSSION

Our patient presented with a subacute illness typical of CEP. The chest roentgenogram in CEP characteristically has a "reverse pulmonary edema" pattern, although upper lobe infiltrates typical of tuberculosis have been reported. Initially, a drug reaction was not considered because the clinical history and roentgenograms strongly suggested tuberculosis. Furthermore, tolazamide has not been reported to have caused CEP and had been used unevenly for nine years. However, rapid clinical and roentgenographic improvement after discontinuation of the drug and eosinophilia suggested a drug-induced illness. This was confirmed by reappearance of the illness and roentgenographic findings after an inadvertent challenge with the drug and by sustained improvement after a second discontinuation of the drug.

In summary, tolazamide appears to have caused CEP in our patient. Prolonged use of any drug should not preclude consideration of drug reaction as an etiologic possibility in the syndrome of CEP.

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