digoxin (0.25 mg daily) and hydrochlorothiazide (50 mg daily) for the past two years for organic heart disease. These medications were continued throughout this course of hospitalization. The serum uric acid level on admission was 6.55 mg/100 ml.

Early in the course of hospitalization, a smear of sputum positive for mycobacteria was obtained. Also, the skin test with intermediate-strength purified protein derivative of tuberculin was positive. On June 5, therapy with isoniazid (300 mg daily) and ethambutol (1,100 mg or 15 mg/kg daily) was started for possible tuberculosis. The course of hospitalization was then unremarkable until July 11, when the patient complained of onset of a warm, swollen, painful right wrist and thumb. The serum uric acid level was 10 mg/100 ml. Relief was obtained with administration of colchicine and later, because of better tolerance by the patient, indomethacin.

CASE 3

A 78-year-old man was being treated for tuberculosis with ethambutol and isoniazid. Before chemotherapy for tuberculosis was initiated, the serum uric acid level was 7.2 mg/100 ml. After five weeks, therapy with streptomycin and pyrazinamide replaced the use of isoniazid and ethambutol. Acute onset of inflammation of the dorsum of the left foot was noted four days later. The serum uric acid level reported four days after the onset of inflammation was 15.1 mg/100 ml.

DISCUSSION

Ethambutol was the only obvious possible precipitating factor identified in the first case we describe. In the second patient, hydrochlorothiazide was being administered concurrently, but the uric acid level was within normal limits on admission, and the patient had been taking the drug two years prior to admission. Therapy with isoniazid has been reported to cause arthritis, but the clinical picture of our patient was not typical of the previously described cases. In the third case, therapy with pyrazinamide was probably contributory.

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REFERENCES


Coexistence of Congenital Bicuspid Aortic Valve and Rheumatic Heart Disease

To the Editor:

In their report of a case of combined acute rheumatic fever and congenitally bicuspid aortic valve, McReynolds et al. stated that this is a hitherto unconfirmed combination; however, we have previously reported the findings in a patient with severe polyvalvular disease necessitating quadruple valvar commissurotomy. This patient suffered from repeated documented attacks of unequivocal rheumatic fever from an early age. Cardiac catheterization showed severe stenosis, as well as mild to moderate incompetence of the mitral, aortic, and tricuspid valves, and mild stenosis of the pulmonic valve. The aortic valve was seen to be bicuspid on angiocardiographic studies; and at operation the valve, although stenotic, was clearly seen to be composed of two distinct valvar cusps and two distinct commissures. Typical rheumatic mitral stenosis was present. This patient, therefore, represented a case of combined rheumatic heart disease and congenitally bicuspid aortic valve. Moreover, the structure of the stenotic tricuspid valve was strongly suggestive of a congenital lesion, and pulmonic stenosis was also present, thus providing further evidence in favor of a combination of congenital and rheumatic heart disease.

Both rheumatic and congenital heart disease are not uncommon conditions, and there is no reason why the two should not occasionally occur together. Lack of previous published reports, the sole purpose of which is to point out the coexistence of two common diseases, should not be construed as an indication of the rarity of the combination.

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