Transient electrocardiographic changes during two episodes of relapsing brucellosis

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Summary
Cardiac involvement in the course of acute brucellosis is rare and, when present, is usually manifested by endocarditis. Myocarditis is very infrequent and in the few reported cases, the course of the disease was fulminant. A patient with recurrent brucellosis who presented transient electrocardiographic T wave changes during two episodes of acute illness is reported. It is suggested that the patient had minor asymptomatic myocarditis in the course of recurrent brucellosis.

KEY WORDS: brucellosis, myocarditis, electrocardiogram.

Introduction
Cardiac involvement in acute brucellosis is infrequent and when present, it is usually manifested by endocarditis (Smith, 1979). Myocarditis and pericarditis are very rare, and the reported cases had a fulminant course (Peery and Belter, 1960). The present report describes the occurrence of transient T wave changes in the electrocardiogram of a patient with acute relapsing brucellosis.

Case report
A 25-year-old woman was admitted with high remittent fever of 10 days' duration. Three weeks before admission she had eaten goat cheese. The main physical findings were fever (40°C) and splenomegaly. There were no heart murmurs, friction rub or gallop sounds. Her haemoglobin was 10 g/dl, leukocytes 5.9 x 10^9/l, ESR 15 mm after 1 hr (Westergren). The Hood creatine kinase, aspartate aminotransferase, electrolyte and calcium concentrations were within normal limits. Brucella melitensis was cultured repeatedly from the blood. The agglutination titre for brucellosis rose from zero to 1:6400. A chest radiogram showed no cardiomegaly and the lung fields were clear. The patient had neither chest pain nor dyspnoea, and there were no signs of heart failure or changes in the heart findings during the whole hospital period.

The electrocardiogram on admission showed sinus rhythm, P-R interval 0.16, negative T waves in lead III, AVF, V₁ and biphasic or flat T waves in V₂-₄ (Fig. 1A). Streptomycin, 1.0 g, was given daily for 10 consecutive days and tetracycline, 3.0 g, for 30 days. After 5 days of treatment the fever subsided and the patient was discharged on the 10th day. The electrocardiogram on discharge showed no change from that on admission, although the patient was afebrile and clinically healthy. One month later the electrocardiogram was normal (Fig. 1B). Four months later, the patient was readmitted with a relapse. Brucella melitensis was once again cultured from her blood and she was given a course of tetracyline and streptomycin. The electrocardiogram on admission showed again transient T wave changes similar to those during the first hospitalization (Fig. 1C), which disappeared after recovery (Fig. 1D).

Discussion
Although brucellosis is a systemic disease, cardiac involvement is rare. O'Meara collected only 15 case reports of brucella endocarditis between the years 1936–1948 (O'Meara et al., 1974) and Hart reported 12 cases until 1951 (Hart, Morgan and Lacey, 1951). Buchanan reviewed 160 cases of brucellosis in the U.S.A. between 1960–1972 and was unable to find cardiac involvement in any of them (Buchanan, Faber and Feldman, 1974). Dalrymple-Champneys (1950), who reviewed 983 cases of brucellosis in England, could not find a report of endocarditis. Only a few cases of brucellosis with myocardial or pericardial involvement have been reported (Peery and Belter, 1960; Hart et al., 1951; Dalrymple-Champneys, 1950; Hunt and Bothwell, 1967). The electrocardiographic changes appeared in fulminant
disease and included atrioventricular or right bundle branch block; they were also observed in patients whose course was complicated by rupture of the sinus of Valsalva (Hart et al., 1951) and were, in most cases, associated with endocarditis (Peery and Belter, 1960). Peery and Belter (1960) described three patients with myocarditis without endocarditis, but the course of the disease was nevertheless fulminant and fatal. Granulomata and myocardial necrosis as well as endocarditis were found on autopsy. The histological changes were sometimes similar to Ashoff bodies (Call, Baggenstoss and Merritt, 1944). The patients with myocardial involvement had a relatively high prevalence of rheumatic heart disease and calcific aortic stenosis (Peery and Belter, 1960; Hart et al., 1951; Call et al., 1944; Cleveland, Suchar and Dagleie, 1978). It is of importance that electrocardiographic changes were, in general, very rare and mild, even in cases with post-mortem evidence of cardiac involvement. Call et al. (1944) described two cases of fatal myocarditis associated with endocarditis. The first case had a normal electrocardiogram, even though the myocardium was infiltrated with lymphocytes, fibroblasts and polymorphonuclear cells. The second patient had rheumatic aortic stenosis, endocarditis, perforation of the aorta to the right ventricle and myocarditis but the electrocardiogram showed only prolongation of the P-R interval.

It is obvious that in the few reported cases of brucellosis with cardiac involvement, the disease was fulminant or fatal. Most cases with myocarditis had associated endocarditis and the electrocardiographic changes were of minor importance and affected mainly the conduction system. The case reported herein manifested electrocardiographic changes in the T waves early in the course of the disease. The patient was a young and healthy woman and had no clinical signs of previous cardiac involvement. The changes were not caused by electrolyte disturbances, or any other obvious causes, and appeared on two occasions during the acute stage of the disease. We assume, therefore, that the patient had two episodes of minor asymptomatic myocarditis in the course of recurrent brucellosis, manifested only by electrocardiographic changes.

References


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