

MITRAL VALVE PROLAPSE, HYPOTHYROIDISM AND RHEUMATOID ARTHRITIS

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SYNOPSIS

The coexistence of autoimmune thyroid disease and rheumatoid arthritis, although known, is uncommon. Both of these conditions may affect the heart. A patient with myxoedema, rheumatoid arthritis and mitral valve prolapse is presented.

INTRODUCTION

In mitral valve prolapse, the pathological features which have been reported include 'myxomatous' (Read et al, 1965), 'myxoid' (Kern and Tucker, 1972), and mucinous (Frable, 1969) degeneration of the valve. In myxoedema, pathological changes in the heart include interstitial oedema, mucoid infiltration and vacuolation, necrosis and degeneration of the muscle fibres. (Robbins, 1974; DeGroot and Stanbury, 1975). Interestingly, myxoedema has not been listed as a cause for mitral valve prolapse (Sloman and Hunt, 1975; Aranda et al, 1976).

Although it is recognised that rheumatoid arthritis may cause valvular lesions (Williams et al, 1968; Barfiglio et al) we are not aware of any report of its association with mitral valve prolapse.

We report here a patient with mitral valve prolapse co-existing with myxoedema and rheumatoid arthritis.

Case History

A 70 year old Chinese woman presented in June 1978 with multiple joint pains and stiffness for the last 15 years.

During the last 4 years, her friends noted her increasing lethargy and mental dullness. She gave symptoms of increased sensitivity to cold and constipation. There was no history of angina, palpitations or exertional dyspnoea.

Clinical examination showed the classical features of myxoedema. She was pale, lethargic, with dull coarse facial features

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and a hoarse voice. The ankle jerks showed delayed relaxation phase. The thyroid gland was not palpable. A bradycardia of 48/min, cardiomegaly, and a late systolic murmur at the mitral area, grade 3/6 were present. There was no mid-systolic click.

Joint involvements were typical of that in rheumatoid arthritis. There were symmetrical bilateral swellings and tenderness of the fingers, wrists, knees and ankles. There were synovial thickening of both wrists with subluxation of the right inferior radio-ulnar joint.

Investigations

Haemoglobin was 9.8 gm/dL; red blood cells normocytic with tendency to macrocytosis, mean corpuscular volume 104 fl; serum folate 9.9 ug/L (N: 3-20 ug/L); serum B12 600 ng/L (N: 150-960 ng/L); anti-parietal cell antibody negative, ESR 85 mm/hr.

Thyroid function tests (Yeo et al, 1976, 1978) confirmed the hypothyroidism consequent to autoimmune thyroid disease:-

T4I 1.6 (N: 3-7.3 ug%); T3 resin uptake 84 (74-124%), Free thyroxine index 1.3 (2.7-7.5); TSH 32 units/L (up to 6 units/L); antithyroglobulin antibody positive 1:40; automicrosomal antibody positive 1:5; Rose Waaler test (RA factor) was positive 1:640; ANF and LE cells were negative.

A chest X-ray showed cardiomegaly (Fig. 1A). X-rays of wrists, hands, and knees showed bony erosions consistent with rheumatoid arthritis with secondary osteoarthritis changes.

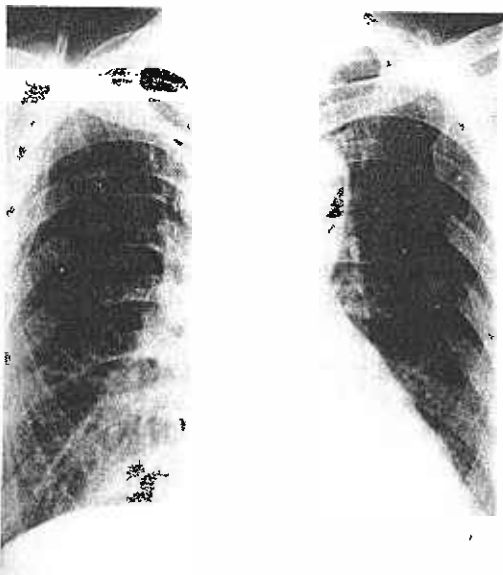


Figure 1A (16.6.78) Chest X-ray before treatment.

ECG (Fig. 2A) showed sinus bradycardia of 50/min with low voltages, T wave inversion with flattened ST segments in most of the leads. Echocardiography (Fig. 3A) showed both anterior and posterior pericardial effusions and a posterior mitral valve leaflet prolapse. The interventricular septum was thickened.

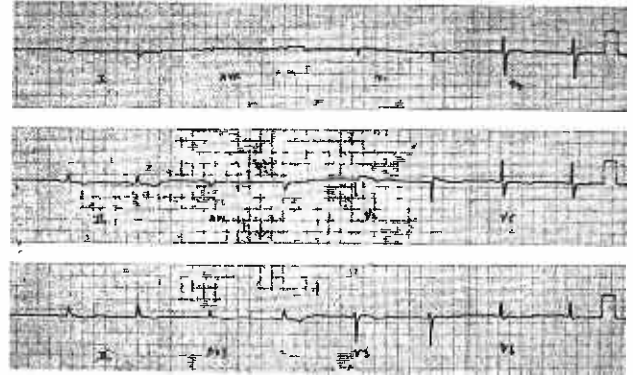


Figure 2A (16.6.78) ECG before treatment.

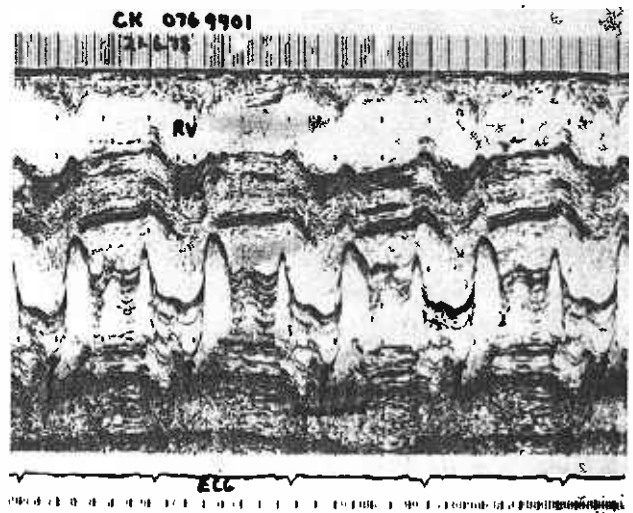


Figure 3A (21.6.78) Echocardiography before treatment.

An electromyogram (EMG) showed mild right carpal tunnel syndrome; ankle jerk relaxation time recorded 640 msec (N: 230-310 msec).

Progress

With replacement therapy, patient became clinically and biochemically euthyroid. There was a reduction in the cardiac silhouette (Fig. 1B) and ECG abnormalities reverted to normal (Fig. 2B). Repeat echocardiography (Fig. 3B) showed resolution of the pericardial effusion. The mitral valve prolapse and thickened interventricular septum remain unchanged. Her joint pains improved with aspirin.

DISCUSSION

The coexistence of autoimmune thyroiditis and other diseases of autoimmune aetiology is well known. (De Groot and Stanbury, 1975). The association between rheumatoid arthritis and autoimmune thyroiditis has been reported but is uncommon. 6 cases of rheumatoid arthritis were reported among 34 unselected patients with Hashimoto's disease. Thyroid autoantibodies were also demonstrated in a significantly higher number of women with rheumatoid arthritis than in the control series (Buchanan et al, 1961). In a report of 506 cases of Hashimoto's disease in Mayo Clinic, 4% has rheumatoid arthritis (Becker et al, 1963).



Figure 1B (20.6.78) Chest X-ray after treatment.

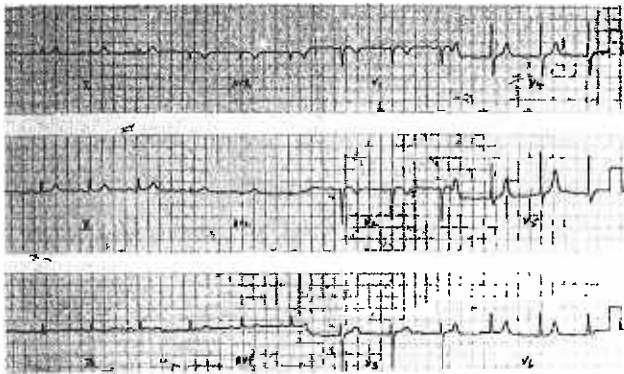


Figure 2B (20.6.79) ECG after treatment.

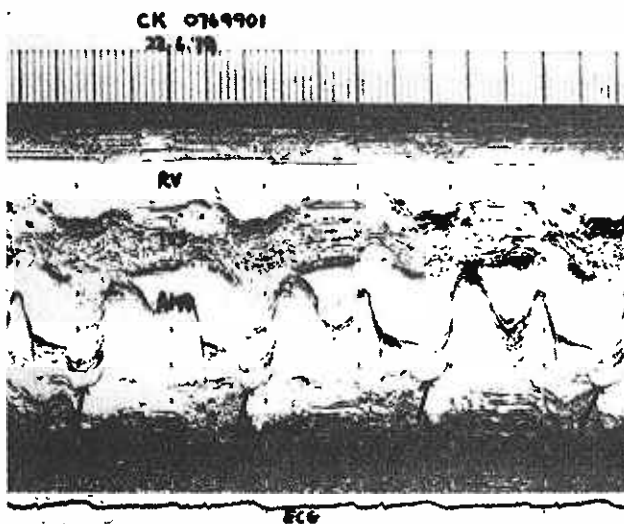


Figure 3B (22.6.79) Echocardiography after treatment.

The conditions associated with mitral valve prolapse are multiple. On the other hand, it is known that in routine necropsies, mitral valve prolapse can be found, particularly in subjects over the sixth decade of life (Davies et al, 1978). Pathological features of mitral valve prolapse includes myxomatous, myxoid and mucinous degeneration of the valve. However, we are not aware of any report of myxoedema with mitral valve prolapse.

Because of the age of the patient, coronary heart disease has to be considered as a possible cause.

Although, the ECG in our patient may suggest coronary artery disease, these changes can occur in myxoedema or mitral valve prolapse alone. Moreover, subsequent ECG done after replacement thyroxine therapy showed reversion of T wave and ST segment abnormalities to normal. Besides our patient did not have symptoms of ischaemic heart disease. The thickened interventricular septum shown on the patient's echocardiography may suggest hypertrophic obstructive cardiomyopathy (HOCM) as an underlying cause. However, there is no other evidence on echocardiography (e.g. systolic anterior motion of the mitral valve, small left ventricular cavity, midsystolic closure of the aortic valve) to support this diagnosis. Moreover, classical clinical signs of HOCM are absent in this patient. We would like to postulate that the thickened septum may be due to hypothyroid cardiomyopathy. This contention is supported by a very recent report from New York (Santos et al, quoted by International Cardiovascular News, 1980). These workers in their study of cardiomyopathy linked with hypothyroidism, discovered, on echocardiogram, asymmetric septal hypertrophy similar to that seen in our patient. This septal hypertrophy may be associated with other features which mimic HOCM.

Valvular involvements in rheumatoid arthritis had been reported but they are rare. The valvular lesions result from rheumatoid nodules in the valve leaflets. Autopsy studies have reported 5% frequency of fibrinoid granuloma in the heart of patients with rheumatoid arthritis. (Borfiglio et al, 1969). However, mitral valve prolapse in rheumatoid arthritis is not a recognised feature.

The precise cause for the mitral valve prolapse in our patient of course remains unclear. However, in view of the clinical setting, it is tempting to speculate that the mitral valve prolapse in this patient is causally related to myxoedema or rheumatoid arthritis, or both. On the other hand, the mitral valve prolapse could well be an incidental finding in an elderly patient, coexisting with these two other conditions.

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