

## CASE REPORTS

# Rupture of Chordae Tendineae during Acute Rheumatic Carditis

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Rheumatic endocarditis may rarely be complicated by rupture of the mitral chordae tendineae. In all such cases reported hitherto, rupture has occurred in the chronic or quiescent phase of the disease. We report a case of rupture of the mitral chordae tendineae in acute rheumatic carditis.

### *Case Report*

K.H.C., a 7-year-old Chinese boy, was admitted in August 1966, with a history of cough for two weeks, swelling of ankles, and puffiness of face for 10 days, fever, and the passage of diminishing amounts of dark urine for 3 days. He had had four previous admissions for exacerbations of acute nephritis over the previous three years. After discharge from his last admission in May 1965, he had failed to return for out-patient follow-up. No abnormalities of the heart were detected in the four previous admissions.

Physical examination showed that the child was febrile, pale, and slightly dyspnoeic. The neck veins were distended. There was bilateral swelling of the legs and puffiness of the eyelids. The pulse rate was 160 a minute and the blood pressure 90/60 mm. Hg. There was a diffuse heaving impulse over the praecordium and an apical systolic thrill at sixth intercostal space half an inch lateral to the left mid-clavicular line. A loud pansystolic murmur was present over the left sternal border and mitral area, radiating to the axilla. No mid-diastolic murmur was detected. There were a few crepitations over the posterior base of the left lung. The liver was enlarged some 4 cm. below the right costal margin in the mid-clavicular line.

Laboratory investigations revealed a mild anaemia and a moderate leucocytosis. The erythrocyte sedimentation rate was raised to 40 mm. in one hour (Westergren). Total urinary protein was 50 mg./100 ml. Microscopical examination of the urine showed 1–2 red blood cells and 20–30 white cells per high power field. The antistreptolysin O titre was 625 Todd units. Serum pyruvic acid level was raised to 3 mg./100 ml. Blood cultures were negative on two occasions. Chest radiography showed an enlarged cardiac shadow, with

evidence of early cardiac decompensation. On admission, the electrocardiogram showed only sinus tachycardia.

The fever subsided with penicillin and streptomycin therapy. The cardiac failure was controlled by digoxin, mersalyl, vitamin B<sub>1</sub>, and low salt diet. Two days after admission, he suddenly went into acute left ventricular failure. His condition improved after emergency venesection. A transient apical mitral mid-diastolic murmur was audible after this episode. There was no change in the loud apical pansystolic murmur. His general condition slowly deteriorated. The electrocardiogram three weeks after admission showed an incomplete right bundle-branch block. A week later he suddenly relapsed with acute pulmonary oedema and died.

*Necropsy.* The pericardial sac contained 100 ml. of straw-coloured fluid. The heart weighed 128 g.; the right ventricular wall was 0.5 cm. thick, the left ventricular wall 0.8 cm. The left atrium was slightly dilated with a rough patch 2 × 1 cm. on the posterior wall above the mitral valve. All the chordae tendineae of the anterior cusp of the mitral valve were torn, the cusp being unattached to the papillary muscle (Fig. a). The apex of the papillary muscle was retracted and showed two minute chordal stumps each measuring 1–2 mm. in length. The mitral cusps were slightly thickened but no vegetations or ulcerations were present.

Other gross findings were acute pulmonary oedema, mild hydrothorax, and a Meckel's diverticulum.

*Histology.* The sections of the heart showed extensive changes in the connective tissue. The collagen fibres, especially those in the endocardium and the valves, were swollen and more eosinophilic than normal. There was an acute inflammatory reaction, consisting mainly of polymorphs, this being very marked in the endothelium. The mitral valve showed conspicuous vascularization. A few typical Aschoff nodules consisting of central fibrinoid degeneration surrounded by Anitschow cells were present (Fig. b). The ruptured ends of the

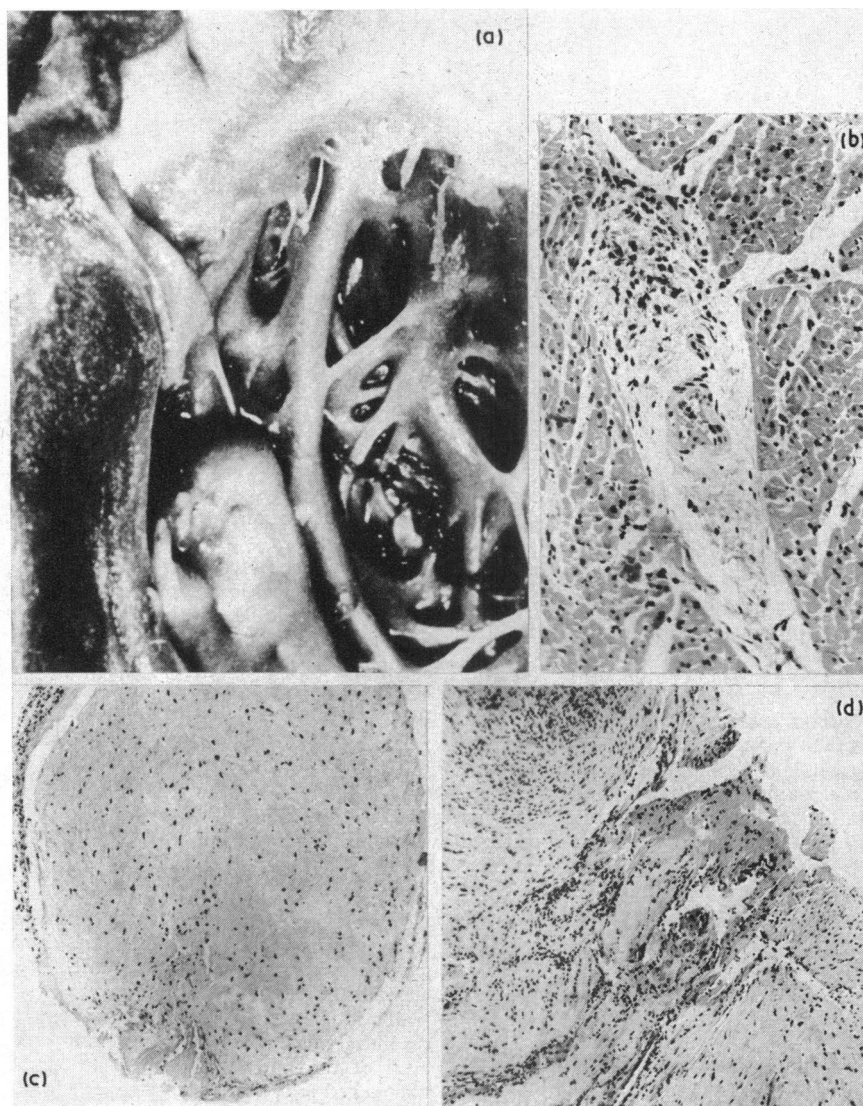


FIG.—(a) Rupture of all the chordae tendineae of the anterior cusp of mitral valve. (b) Aschoff's nodule in myocardium. (H. and E.  $\times 150$ .) (c) Valvular end of a ruptured chorda tendinea, covered by thin layer of fibrin. (H. and E.  $\times 70$ .) (d) Papillary muscle end of a ruptured chorda tendinea. The torn surface is covered by fibrin. The underlying connective tissue shows an acute inflammatory reaction. (H. and E.  $\times 70$ .)

chordae were covered by a thin layer of fibrin indicating an antemortem rupture (Fig. c and d). No bacteria were demonstrable at the point of rupture or elsewhere.

Other microscopical findings were acute pulmonary oedema, acute centrilobular congestion of the liver, a few calcium crystals in the renal tubules, and resolving acute glomerulonephritis.

#### Discussion

Rupture of the chordae tendineae of the mitral or

tricuspid valve is rare. It most commonly occurs in a heart previously damaged by rheumatic disease with superadded subacute bacterial endocarditis (Hepper, Burchell, and Edwards, 1956; Osmundson, Callahan, and Edwards, 1961; Menges, Ankeney, and Hellerstein, 1964). Less commonly it can result from traumatic chest injury (Barber, 1944) or cardiac infarction (Hudson, 1965). Some ruptures appear to be spontaneous (Frothingham and Hass, 1934).

Frew (1931) described rupture of the mitral chordae tendineae in an 8-year-old girl some nine months after the onset of rheumatic fever with evidence of carditis. At necropsy, marginal thickening of the cusps was present in the tricuspid, aortic, and mitral valves with left ventricular hypertrophy. This would be the only published case suggestive of rupture of the mitral chordae tendineae in an exacerbation of rheumatic fever, but unfortunately there was no histological confirmation of an active rheumatic endocarditis or exclusion of superimposed subacute bacterial endocarditis.

In the present case, subacute bacterial endocarditis may be excluded by negative blood cultures, together with absence of embolic phenomena, valvular vegetations, or ulcerations, and bacteria in histological sections. Clinical diagnosis of acute rheumatic fever can be made on the basis of a history of fever, carditis, raised erythrocyte sedimentation rate, and high antistreptolysin titre. Necropsy and histological findings substantiate an early fulminating rheumatic carditis with extensive connective tissue changes, an exudative reaction, and Aschoff nodules beginning to make their appearance.

### Summary

A case of rupture of the mitral chordae tendineae in a 7-year-old Chinese boy with acute rheumatic carditis is presented.

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