Case reports

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Myocardial biopsy in diagnosis of endomyocardial infarction in patient with electro- and vectorcardiographic signs of myocardial infarction

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SUMMARY A 52-year-old woman is described with the clinical picture of congestive heart failure, electro- and vectorcardiographic evidence of myocardial infarction, combined with angiographically hypokinetic left ventricle, but with patent coronary arteries. Endomyocardial biopsy disclosed changes consistent with 'congestive cardiomyopathy'. Thus, the study shows that the Q wave pattern on the electrocardiogram provides insufficient evidence for the diagnosis of myocardial infarction, and may be misleading in patients with cardiomyopathy. In such circumstances, endomyocardial biopsy from the left or right ventricle appears to be a helpful diagnostic method, and further use of this technique may permit a more precise diagnosis in patients with a history of myocardial infarction, but with normal coronary arteries.

Various electrocardiographic changes have been associated with primary or secondary cardiomyopathy. The most common electrocardiographic findings are non-specific. Occasionally, Q waves or QS complexes simulating the patterns of myocardial infarction have been reported (Gau *et al.*, 1972). In the present case electro- and vectorcardiographic abnormalities were consistent with left ventricular myocardial infarction and would usually indicate underlying coronary artery disease. Ventricular biopsy, however, showed endomyocardial fibrosis consistent with diffuse endocardiomyopathy.

Case report

The patient was a 52-year-old woman without earlier signs of heart disease. After a febrile episode of approximately 4 weeks' duration she developed congestive heart failure. A grade 2/6 systolic murmur localised to the apex of the heart suggesting mitral regurgitation was heard. Chest x-ray film showed an enlarged heart (710 ml/m² BSA). An electrocardiogram showed atrial fibrillation and changes consistent with acute anterior myocardial

infarction. She was treated with digitalis and diuretics. The atrial fibrillation was converted to sinus rhythm.

On admission 6 months later there were no clinical signs of heart failure. The heart rate was regular. The systolic murmur had disappeared. The heart volume was reduced (540 ml/m² BSA). An electrocardiogram (Fig. 1) showed deep, pathological Q or QS waves in leads V1-3. Vectorcardiographically, the initial forces were directed posteriorly and to the right (Fig. 2), suggesting anteroapical myocardial infarction. At left- and right-sided cardiac catheterisation, the cardiac output was slightly reduced at rest and failed to show a normal response to exercise. The increase in the left ventricular end-diastolic pressure during exercise was reflected in a moderate increase in pulmonary arterial pressure (Table).

Left ventricular angiography showed anterior and apical dyskinesia with calcification of the free wall of the left ventricle. Selective coronary angiography showed completely normal coronary arteries. Right ventricular endomyocardial biopsy ('Kings' bioprobe) was performed percutaneously from the right femoral vein and the left ventricle was biopsied retrogradely via the left femoral artery.

Fig. 1 Twelve lead electrocardiogram showing anteroseptal infarction. NB paper speed 50 mm/s.

Fig. 2 The vectorcardiogram in the transverse view shows counterclockwise rotation of QRS with early forces displayed posteriorly.

Table Results of cardiac catheterisation

	Rest	Exercise (250 kpm/min for 5 minutes)
Pressures (mmHg)		
Main pulmonary artery	28/10-16	60/24-40
Pulmonary artery wedge	12/16-9	28/14-20
Left ventricular end-diastolic (LVED)	13	26
Blood flow (Pick)		
CO/CI	4.8/2.6	9.3/5.1

HISTOLOGICAL EXAMINATION

Histological examination of the right ventricular biopsy (Fig. 3) showed fibrotic thickening of the endocardium with an increase of the elastic fibrils. The cardiomyocytes were moderately hypertrophic with irregular, enlarged, and hyperchromatic nuclei. Some of the muscle fibres showed myocytolysis with loss of fibrillar material. Lipofuscin pigment was not particularly increased. The interstitium showed fibrosis and sparse mononuclear cell infiltration. There was some hyalinisation of the small vessels. The left ventricular biopsy was fragmented and lacked the endocardial layer. The myocardial fibres and the interstitium showed changes identical with those found in the right ventricular biopsy.

Discussion

The association of myocardial infarction patterns in the electrocardiogram and vectorcardiogram together with localised left ventricular dyskinesia suggested a diagnosis of coronary arterial disease. However, the coronary arteriogram, which is widely regarded as 'the final court of appeal', was normal. A normal coronary tree has been reported in a small number of cases with myocardial infarction (less than 1%) (Arnett and Roberts, 1976). The explanations given have been acute coronary embolism with subsequent clot lysis, retraction, or recanalisation, or else arterial spasm. Rider *et al.* (1974) reported abnormalities in the arterioles and capillaries, with either endothelial, smooth muscle, or basement membrane thickening, in myocardial biopsy specimens from patients with ischaemic heart disease and normal coronary arteriograms, but have since failed to confirm this observation. Since the inner third of the myocardium has been shown to contain most of the small vessels and this is the region from which the biopsy was taken, small vessel disease is unlikely to have been missed in the present case. Richardson *et al.* (1974) were also unable to show small vessel disease from endomyo-

Fig. 3 The right ventricular biopsy shows prominent endocardial and some myocardial fibrosis. The fibrillar appearance of the cardiomyocytes is preserved, but the nuclei are enlarged and irregular as seen in myocardial hypertrophy. Some few mononuclear cells can be seen between the muscle fibres. (Approx. $\times 100$.)

cardial biopsy specimens in 7 patients with angina pectoris and normal coronary arteriograms.

Abnormally deep Q waves in myocardial infarction are explained on the basis of the resultant vector concept (Chung, 1974). Patients with idiopathic cardiomyopathy or chronic myocarditis, and abnormal Q waves, may be erroneously diagnosed as having coronary arterial disease. Increasing fibrosis of the septal wall might cause gradual electrocardiographic dominance by the left ventricular free wall through lessening of the electrical forces of septal depolarisation (Coyne, 1968). Diffuse fibrosis might also alter the inter-ventricular conduction with loss of positive vectors (Goodwin, 1970).

The light microscopy findings in the ventricular biopsies are strongly indicative of a diffuse pathological process affecting the whole heart. Ordinarily, the endocardium and myocardium of the right ventricle are not altered in infarction of the left ventricle. Endomyocardial fibrosis is a common finding in cardiomyopathies and has been described in several surveys of heart diseases diagnosed by right ventricular biopsy (Sekiguchi and Konno, 1971; Somers *et al.*, 1971; Inoh and Takeshita, 1972). Our findings are consistent with the group designated 'congestive cardiomyopathy' (Goodwin, 1970). The clinical picture of congestive heart failure with radiologically hypokinetic left ventricular wall and electrocardiographic evidence of ischaemic heart disease with normal coronaries has been reported earlier, even associated with calcification of the left ventricular wall. The causes have been progressive muscular dystrophy (Storstein, 1962), sarcoidosis (Chun *et al.*, 1975), rheumatic fever (Aryanpur *et al.*, 1975), or unknown (Franch and Shepherd, 1975; Wise and Conrad, 1975). The clinical onset and course of the disease in our patient might have suggested the diagnosis of myocarditis, which was excluded by biopsy.

The present case shows that endomyocardial right ventricular biopsy may be helpful in obtaining a more precise diagnosis in patients with suspected ischaemic heart disease, in particular when the coronary arteries are patent.

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