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Serious cardiac morbidity in infectious mononucleosis

Myocarditis is a recognised complication of infectious mononucleosis, but it is generally believed that there is "little to fear from cardiac complications."¹ We report a case of serious cardiac morbidity as a result of infectious mononucleosis.

Case report

A 16-year-old schoolgirl was admitted to hospital because she had suddenly collapsed to the floor, without losing consciousness, and felt weak in the left arm and leg; her speech was slurred. Two weeks previously she had had a sore throat and painful, enlarged glands in her neck. Her mother had noticed that she had recently been breathless on exertion. On examination she was confused and dysarthric with a left hemiparesis. Her throat was inflamed and there were two palpable lymph glands, in the neck and axilla. Pulse was regular at 34 beats/min, and blood pressure was 155/85 mm Hg. Jugular venous pressure was not raised. The apex beat was displaced towards the anterior axillary line with both a left ventricular thrust and a right ventricular heave, and a pansystolic murmur was audible over the whole precordium.

Chest x-ray examination showed generalised cardiomegaly (cardiothoracic ratio 0.66), and an electrocardiogram showed complete heart block with

total absence of atrial activity. A temporary pacing wire was inserted with immediate improvement in her conscious level. Anticoagulants were given because of the probability that a cerebral embolus from a left ventricular thrombus had occurred. Haemoglobin concentration was 10.9 g/dl and white cell count $4.7 \times 10^9/l$ with 43% lymphocytes, some atypical. A monospot test was positive, as was a Paul-Bunnell test at a titre of 1/224 (this was unchanged after absorption with guinea-pig antigen but fell to 1/7 after absorption with ox-cell antigen). Computed tomography showed an area of infarction in the right internal capsule. Cardiac catheterisation showed normal intracardiac pressures and no evidence of an intracardiac shunt, but ventricular angiograms showed dilated left and right ventricles with normal contractility. Histological examination of a left ventricular biopsy specimen (figure) showed enlarged myocardial fibres of up to 30μ and extensive cytoplasmic degeneration with some nuclear damage, and an oedematous interstitium containing a few inflammatory cells. These appearances suggested an acute process affecting the myocardial fibres compatible with myocarditis.

To confirm a diagnosis of recent infectious mononucleosis, further tests were undertaken at the Central Public Health Laboratory, Colindale. IgG antibody to Epstein-Barr virus capsid antigen was present to a titre of 1/256, confirming that infection with Epstein-Barr virus had occurred at some time; specific IgM to Epstein-Barr virus capsid antigen was also present, indicating current or recent infection.

She was treated with steroids, but, as she remained totally dependent on the pacemaker, a permanent system was implanted. Seven months later she was well apart from breathlessness on moderate exertion, and physical examination was essentially normal apart from cardiomegaly, which was unchanged. A repeat left ventricular biopsy specimen on light microscopy showed no cytoplasmic degeneration, a few lymphocytes, and no apparent increase in fibrosis. Electron microscopy showed non-specific degenerative change with myofibrillar disorganisation and proliferation of mitochondria and moderate interstitial fibrosis.

Comment

Early reports of various complications in infectious mononucleosis have been criticised for lack of firm documentation of current infection.² In our case there was sound haematological and serological confirmation of recent infection.

Electrocardiographic abnormalities in the form of non-specific ST and T-wave changes and various degrees of heart block occur in up to 6% of patients with infectious mononucleosis.³ Few patients have symptomatic cardiac disease, and those who do appear to recover completely.¹ Histological examination of hearts in these patients⁴ shows small, focal collections of lymphocytes in the myocardium. In the occasional case in which death has been attributed to heart disease⁴ interstitial oedema and myofibrillar degeneration are seen in addition to lymphocytes, as in our case.

Mason *et al* suggested that the presence of lymphocytes in an endocardial biopsy specimen may be an indication for steroid treatment,⁵ but our patient showed no benefit in terms of reduction of heart size, recovery of conduction tissue, or disappearance of lymphocytes in the second biopsy specimen. The near-normal appearance of the myocardium in the repeat biopsy specimen on light microscopy may reflect patchy disease of the heart tissue and sampling error.

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Left ventricular endomyocardial biopsy specimen, showing cytoplasmic degeneration, interstitial oedema, and a few inflammatory cells; myofibrils are enlarged up to 30μ . Haematoxylin and eosin; plastic-embedded section $\times 23$ (original magnification).