Short reports

Toxoplasma gondii – an unusual cause of myocarditis in old age

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Summary

We report the case of an 86-year-old man who was admitted with congestive cardiac failure and chronic renal failure. He was previously known to have a thoracic aortic aneurysm and chronic bronchitis. There was no history of myocardial infarction but his heart failure was assumed to be due to ischaemic heart disease. Despite treatment of the heart failure the patient died. At post-mortem he was found to have *Toxoplasma gondii* myocarditis.

Keywords: *Toxoplasma gondii*, myocarditis, elderly

Introduction

Infection with *Toxoplasma gondii*, a coccidian parasite, manifests itself in humans in a variety of fashions.^{1–3} In recent years a particularly virulent form of the infection has emerged in patients with a compromised immune system.^{4–9} The syndrome represents reactivation of an infection often acquired years previously, in which bradyzoites have remained dormant in tissue cysts until immunosuppression activates the infection.⁴

Although the proportion of individuals with a positive serological test for *T gondii* increases with age, there have been no reports of toxoplasmosis causing severe organ damage in elderly patients.² It is for this reason that the present report of myocarditis due to *T gondii* is of particular interest.

Case report

An 86-year-old man was admitted with a one-week history of breathlessness associated with cough and mucopurulent sputum. There was a long history of recurrent episodes of respiratory infection previously diagnosed as chronic bronchitis. Six months earlier he had been referred as an out-patient suffering from hoarseness of several weeks' duration. It was established that this was due to a left recurrent laryngeal palsy associated with an aneurysm of the thoracic aorta. At the same time it was noted that he had serum urea and plasma creatinine concentrations of 2.5 mmol/l and 414 µmol/l, respectively. There was no history of blood transfusions in the recent past, and no contact with cats or any other pet. On examination he was co-operative and well orientated. His fingers were clubbed, he was afebrile and there was no lymphadenopathy. There was jugular venous congestion and considerable sacral and lower limb oedema. His pulse was regular at 80 beats per minute, his blood pressure was 100/60 mmHg, and heart sounds were normal. There was dullness and diminished air entry at both lung bases. His liver was enlarged to 5 cm below the right costal margin, and was smooth and non-tender. There were no focal neurological signs. Laboratory investigations showed a normal white cell count. The serum sodium was 146 mmol/l, potassium 6.3 mmol/l, bicarbonate 15 mmol/l, urea 39.7 mmol/l, and creatinine 607 µmol/l. Creatinine clearance was subsequently found to be 1.3 ml/min. An electrocardiograph (ECG) showed left bundle branch block and generalised T-wave inversion. Features noted on chest X-ray were cardiomegaly, bilateral pleural effusions, and a large thoracic aneurysm. There was no evidence of lung consolidation.

The provisional diagnoses at this stage were chronic bronchitis, congestive cardiac failure, aortic aneurysm and chronic renal failure.

He was treated with calcium resonium and parenteral frusemide. There was progressive deterioration in his condition and he died three days later.

Autopsy

Examination of his heart revealed marked biventricular dilatation. There was only moderate atherosclerosis of the coronary arteries and no evidence of an old or recent myocardial infarction. The left ventricular myocardium

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Accepted 8 September 1994

Clinical features of <i>Toxoplasma gondii</i> infection	
Healthy children or adults	Asymptomatic; or malaise, fever and anterior cervical/suboccipital lymphadenopathy. Uveitis (rare). Atypical lymphocytes on blood film.
Pregnancy	Fetal intracerebral calcification, hydrocephalus and choroidoretinitis
Immunosuppressed (eg, AIDS, transplant recipients, elderly)	Brain, lung, heart involvement

was thinned to 0.9 cm on the anterior wall and to 0.7 cm on the posterior wall (normal 1.3–1.5 cm). Microscopic examination of the myocardium, with sections taken from the interatrial septum and anterior and posterior walls of the left ventricle, revealed features of myocarditis with occasional myocytes containing *T. gondii* cysts (figures 1 and 2). A thoracic aneurysm, with a maximum diameter of 10 cm, had its origin 6 cm above the aortic valve and there was a small fusiform aneurysm of the abdominal aorta. The kidneys were shrunken and scarred. There was histological evidence of benign nephrosclerosis and pulmonary emphysema, but no evidence of toxoplasmosis at either site.

Discussion

This man presented with multiple pathologies consisting of congestive cardiac failure, an aortic aneurysm, chronic bronchitis and chronic renal failure. Even with the benefit of hindsight, the most likely diagnosis was congestive cardiac failure due to ischaemic heart disease, with the chronic renal failure accentuating the fluid retention. Without a clear history of ischaemic heart disease the possibility of a cardiomyopathy should have been considered, but the autopsy diagnosis of toxoplasmosis was completely unexpected.

The probable explanation of the condition was reactivation of a latent infection acquired many years previously. There was no opportunity to use stored serum to check for toxoplasma antibodies or for infection with human immuno-deficiency virus (HIV). There was nothing in the history of this octogenarian with severe respiratory disease to suggest that HIV infection was a real possibility.¹⁰ In these circumstances it seems much more likely that the reason for his immunosuppression was severe renal failure secondary to nephrosclerosis.

An unanswered question in this patient remains the precise cause of the pulmonary congestion. It is possible that it related to the toxoplasmosis, but an alternative is that it was due to the severe renal impairment. Most reports of myocarditis associated with toxoplasmosis suggest that the clinical features are non-specific.⁵

While there have been several reports of toxoplasma cysts in the myocardium, it is wrong to assume that their identification is easy, even in cases of myocarditis known to be

Figure 1 The myocardium contains a mononuclear inflammatory infiltrate composed of lymphocytes and some macrophages. There is some interstitial oedema (H + E, $\times 100$)

Figure 2 Toxoplasma cysts within myocytes (arrows). The lack of an inflammatory response surrounding the cysts is characteristic: rupture of a cyst incites an inflammatory reaction (H + E; $\times 400$)

due to toxoplasmosis. It remains uncertain what proportion of cases of so-called idiopathic myocarditis is due to toxoplasma, but clearly some may be. A diligent search for toxoplasma cysts in tissue sections is required in cases of apparently non-specific myocarditis.

It should be noted that the diagnosis in this case would not have been made without an autopsy since none of the ECG changes were specific for myocarditis. The use of autopsies as an effective form of audit has been sadly neglected in recent years. It is our policy to request autopsies on all deaths occurring in our assessment unit. A review has recently established that this has been extremely effective in reducing complacency and encouraging high standards of clinical practice.¹¹

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