Infective endocarditis in Crohn’s disease

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SUMMARY  Infective endocarditis developed in a patient with Crohn’s disease. This case highlights a hazard of immunosuppressive treatment in inflammatory bowel disease.

I report a case of infective endocarditis occurring as a complication of Crohn’s disease. The case highlights the hazard of immunosuppressive treatment in the management of inflammatory bowel disease.

Case report

A 65 year old woman was admitted to our medical unit with a six week history of general malaise and lethargy after an episode of unexplained acute left ventricular failure that had been treated by her general practitioner at home. Myocardial infarction had been excluded by serial electrocardiograms and measurement of cardiac enzymes at that time and her general practitioner had noted an apical midsystolic murmur. Although she had responded to diuretic treatment, she continued to feel unwell with a loss of appetite and concomitant weight loss of 12 kg over this period. She had had Crohn’s disease that predominantly affected the large bowel diagnosed three years before and had initially responded poorly to prednisolone and mesalazine. She had subsequently required azathioprine in addition to steroids to control her bowel symptoms. Apart from a recent exacerbation preceding her current illness, Crohn’s disease had otherwise remained quiescent. She had not had sigmoidoscopy or any other investigations of her large bowel for more than eight months. There was no history of rheumatic fever or valvar heart disease.

At admission she looked generally well and was not dyspnoeic at rest. She was moderately obese and there was mild finger clubbing. No splinter haemorrhages were evident and she was not feverish. She had sinus tachycardia of 120 beats/minute, blood pressure of 160/90 mm Hg, an apical and basal midsystolic murmur, and an early diastolic murmur localised to the left sternal edge. There was no clinical or radiological evidence of left ventricular failure. A combined M mode and cross sectional echocardiogram showed a normal mitral valve with mild left atrial dilatation. Large vegetations were identified on the aortic valve (figs 1 and 2), and aortic incompetence was easily confirmed on Doppler echocardiography. All six blood cultures grew *Streptococcus faecalis*.

She was treated with intravenous benzylpenicillin (3 megaunits four times daily) and gentamicin (80 mg twice daily) and transferred to the regional cardiothoracic centre at the Western Infirmary, Glasgow for further assessment. She initially appeared stable but subsequently recurrent pyrexia developed with

![Image](https://example.com/image.png)

**Fig 1** M mode echocardiogram of the patient’s aortic valve. The vegetation (arrow) was best shown during systole as an echo producing mass attached to the non-coronary cusp that did not restrict valve motion.

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Fig 2  Cross sectional echocardiogram of the patient in the long axis left parasternal view. Vegetation (arrow) was attached to the right coronary cusp. Ao, aorta; LA, left atrium; al, anterior mitral valve leaflet; LV, left ventricle.

Further rises in her C reactive protein. Diuretics did not control her cardiac failure and she required emergency aortic valve replacement. Her aortic valve was found to be covered with large vegetations resulting in a mixture of stenosis and incompetence. Cultures of the vegetations were sterile. Her post-operative progress was satisfactory and she remains well six months later despite a further admission with a relapse of Crohn’s disease when steroid treatment was inadvertently stopped. Her bowel symptoms resolved when treatment with prednisolone was restarted and there was no evidence of recurrence of infection.

Discussion

Immunosuppression increases the risk of infective endocarditis. Infection, particularly when the causal organisms include *Streptococcus faecalis* and *Streptococcus bovis*, may also complicate gastroenterological disorders. So far these have been reported in patients with liver disease, gall bladder disease, diverticulitis, and colorectal carcinoma, but not in patients with Crohn’s disease.

It is likely that the exacerbation of Crohn’s disease in this case led to a constant bacteraemia which resulted in infection of the aortic valve, possibly as a direct sequel of suppression of the patient’s native immune response by prednisolone and azathioprine. None the less, the risk of infective endocarditis in such circumstances must be small because immunosuppressive treatment is regularly used to treat chronic inflammatory bowel disease.

I thank Dr J Morrow, consultant gastroenterologist, for permission to report this case.

References


Notices

**British Cardiac Society**

The Annual General Meeting will take place in Torquay on 22 to 25 May 1990.

**Comparative electrocardiology**

A workshop on Comparative Electrocardiology will be held in Amsterdam on 12 and 13 October 1989. Inquiries to Dr Frits L Meijler, Interuniversity Cardiology Institute of The Netherlands, PO Box 19258, 3501 DG Utrecht, The Netherlands.