Suspicious mind: the association between \textit{Streptococcus bovis} endocarditis and carcinoma of the colon

Sir,

The association between \textit{Streptococcus bovis} endocarditis and carcinoma of the colon is well documented.\textsuperscript{1-5} We report a patient in whom, following treatment of \textit{S. bovis} endocarditis, a right hemicolectomy was carried out for a caecal lesion which, although biopsies had shown benign disease, the subsequent postoperative histology revealed the presence of an adenocarcinoma.

An 83 year old man was admitted and treated for an infective exacerbation of his chronic obstructive airways disease. He had a past medical history of myocardial infarction, congestive cardiac failure, mitral regurgitation and pernicious anaemia. In 1989 a sigmoid colotomy had been performed for a villous adenoma found following investigation of an iron deficiency anaemia. Five days after discharge he was readmitted complaining of malaise, backache and headache. He was pyrexial. Blood culture grew \textit{S. bovis} and transesophageal echocardiogram demonstrated vegetations on the mitral valve. Following commencement on intravenous teicoplanin, the patient improved rapidly, with no recrudescence of his initial pyrexia.

The patient denied any bowel symptoms and faecal occult bloods were negative, but at colonoscopy a large polypoid lesion of malignant appearance was seen just above the ileo-caecal valve. A number of smaller polyps were also seen. Caeliac biopsies showed no malignancy.

In view of the macroscopic appearance of the caecal lesion, the association of malignancy with \textit{S. bovis} endocarditis, and the possibility of recurrence of the endocarditis, an elective right hemicolectomy was carried out. At operation three polyps were found in the caecum. Histological examination of the largest polyp revealed a moderately differentiated adenocarcinoma Dukes stage A. The other two polyps were tubulo-villous adenomata and three lymph nodes were free from tumour.

This case emphasizes the need for a high index of suspicion of malignancy and thorough gastrointestinal investigation, even in the absence of gastrointestinal symptoms, in cases of \textit{Streptococcus bovis} endocarditis or bacteraemia. Colonoscopy is the investigation of choice as barium enema failed to detect the presence of carcinoma in some reports.\textsuperscript{6,7}

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References


Successful treatment by gonadotrophins of a patient with genetic haemochromatosis and infertility

Sir,

Phlebotomy provides a normal life expectancy to patients with genetic haemochromatosis (GH) if it is started before the establishment of cirrhosis and diabetes,\textsuperscript{1,2} so the problem of infertility in young patients with GH is no longer an academic one. In GH, infertility results mainly from hypogonadotropic hypogonadism\textsuperscript{3} and is considered irreversible.\textsuperscript{4,5} There are now rare reports of gonadal hormonal\textsuperscript{6} and spermatogenesis\textsuperscript{7} recovery after iron depletion. Gonadotrophin use to replace the deficient pituitary function seems logical and has permitted recovery of spermatogenesis but not pregnancy.\textsuperscript{8,9} We report an infertile patient with GH in whom, after iron depletion, gonadotrophins induced spermatogenesis and allowed three paternities.

A 25 year old man was admitted in 1978 for congestive cardiomyopathy. Genetic haemochromatosis was diagnosed, serum iron 40.2 \textmu mol/l, serum iron binding capacity 45.6 \textmu mol/l, serum ferritin 1,300 \mu g/l. Liver biopsy showed micronodular cirrhosis with massive iron deposition in hepatocytes (411 \textmu g/dry liver tissue, normal <18). Complications also included impotence of 3 years' duration with hypogonadotropic hypogonadism (total testosterone 0.33 nmol/l, normal 8.6–38; luteinizing hormone (LH) 0.86 ng/ml, normal 1.1–2.9; follicle stimulating hormone (FSH) 0.28 ng/ml, normal 1.2–2.5; LH and FSH unresponsive to LH-releasing hormone (RH). Familial investigation disclosed two relatives with GH. Phlebotomy (120 in 30 months) resulted in complete reversal of cardiac symptomatology but hypogonadism persisted.

The wish of this patient and his wife to conceive a child, together with the response to iron depletion, prompted us to undertake treatment by gonadotrophins (human chorionic gonadotrophin 5,000 U twice a week and human menopausal gonadotrophin 150 U three times a week). This obtained spermatogenesis and, after 22 months, paternity. Two subsequent courses of gonadotrophins allowed two other paternities. We thought it unethical to propose paternity testing for the couple only for scientific purposes.

This case is one of hope for young people suffering from GH, who expect not only normal life expectancy but also to be cured of phlebotomy-resistant complications of the disease.
Aspirin and risk of fatal colon cancer

Sir,
Recent studies have demonstrated that regular use of aspirin at low doses may reduce significantly the risk of fatal colon cancer. However, how aspirin might produce this beneficial effect is unknown. I suggest two possible mechanisms.

First, it has been recently demonstrated that aspirin enhances the production of interferon-α and interferon-γ and these two compounds have been shown to produce beneficial effects in the treatment of colon cancer. In addition, it was recently found that the level of interferon-γ in extracts of human colorectal adenocarcinomas decreased progressively with the advance of clinical stage, and the levels of interferon-γ of the patients with distant metastases were significantly lower than those of the patients without distant metastases.

Second, production of tumour necrosis factor-α from the macrophages increases with the addition of aspirin, and the tumoricidal activity of this monokine is well established.

Thus, the suggested above two mechanisms for aspirin reducing the risk of fatal colon cancer seem to be reasonable.

References