Crohn's disease and pericarditis

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Summary

A patient is described who developed acute pericarditis during a severe first attack of Crohn's disease. The opportunity was taken to measure levels of circulating immune complexes, both during the acute phase of the illness and during convalescence; no significant change in these levels from normal values was found. The Crohn's disease and the associated pericarditis responded to systemic corticosteroid therapy, and no relapse has occurred during 14 months of follow-up.

KEY WORDS: Crohn's disease, pericarditis, immune complexes.

Introduction

An appreciable proportion of patients with chronic inflammatory bowel disease develop non-intestinal complications of their illness at some time during its course. Hepatobiliary, cutaneous, ocular and skeletal disorders are all encountered with relative frequency, but a number of other possible, much less common, associations have been described.

The authors have recently seen a patient with a severe first attack of Crohn's disease who developed acute pericarditis and who was investigated in some detail both in the acute attack and during follow-up.

Case report

A 24-year-old woman was admitted to hospital with a history of diarrhoea, weight loss and episodic colicky abdominal pain for several months.

On examination she was very thin and looked extremely unwell. Her temperature was 39.5°C. She was clinically anaemic and had finger clubbing. Her abdomen was tender along the course of the colon.

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The anal margin was surrounded by large fleshy ulcerated skin tags and there was a small recto-vaginal fistula.

Investigations revealed an iron deficiency anaemia, with a haemoglobin of 8.7 g/dl and a sedimentation rate of 88 mm in the first hour. A plain abdominal X-ray suggested thickening and irregularity of the descending colon with a grossly distorted mucosal pattern.

Sigmoidoscopy revealed patchy inflammation of the distal rectal mucosa. Above about 8 cm, discrete linear ulcers were seen, interspersed with areas of cobblestoned mucosa. Histological examination of a number of biopsy specimens showed relative preservation of the glandular architecture but infiltration of the lamina propria by lymphocytes, eosinophils and plasma cells. Many well formed granulomata were present. The appearances were those of Crohn's disease of the large bowel.

The patient was started on an intensive regimen of intravenous steroids, blood transfusion and parenteral nutrition (Truelove and Jewell, 1974). She improved rapidly, her fever settled and stool frequency was much reduced. After 6 days, barium enema showed severe ulceration in the transverse and descending colon with relative distal sparing.

On the 8th day after admission, the patient's fever returned and she complained of severe central chest pain worse on movement and breathing. A loud pericardial rub was heard. Sequential chest X-rays and two-dimensional echocardiograms remained normal. The electrocardiogram showed minor T-wave changes only. ASO titre, RAHA test, LE cells, autoantibody screen, total C₃ complement, throat swabs and stools for viral culture, and serum viral antibody titres (acute and convalescent) were all negative or normal.

The central venous line was removed and treatment with oral prednisolone (40 mg daily) substituted

for the corticosteroid infusion. Three days later the pericardial friction rub was barely audible and the chest pain had subsided. The Crohn's disease continued to improve.

Venous blood samples were collected during the acute illness, and 3 weeks and 6 weeks afterwards. Each sample was allowed to clot at room temperature. The serum was separated, snap-frozen and stored at -70°C . When all the serum samples had been obtained, immune complexes were assayed by polyethylene glycol precipitation and single radial immunodiffusion using a commercially available kit [Merrid CIC kit (M201), Mercia-Brocades Ltd].

IgG complex levels remained within the quoted normal range ($0-93\text{ }\mu\text{g/ml}$) throughout the investigation period. Although, at $28\text{ }\mu\text{g/ml}$, IgM complex levels were within the normal range ($0-41\text{ }\mu\text{g/ml}$) at the start of the illness, they rose to $67\text{ }\mu\text{g/ml}$ at 3 weeks, but had fallen again to $32\text{ }\mu\text{g/ml}$ when last measured.

A later barium follow-through examination showed the presence of jejunal Crohn's disease. The patient's steroid treatment was completely withdrawn over 6 weeks and she has remained entirely well during 14 months of follow-up on oral sulphasalazine only.

Discussion

Pericarditis is such a rare accompaniment to inflammatory bowel disease, that suggestions have been made that it may be a chance finding (Kern, 1980). There are only 16 other case reports, 13 concerning patients with ulcerative colitis and three with Crohn's disease (Thompson *et al.*, 1979; Becker *et al.*, 1981).

All four patients with Crohn's disease and pericarditis have had predominant large bowel involvement (Mukhopadhyay *et al.*, 1970; Breitenstein, Salel and Watson, 1974; Goodman *et al.*, 1976). In our patient, as in two of the other cases, the pericarditis developed while the Crohn's colitis was active rather than in remission. The three previously recorded patients all had additional non-intestinal complications of their inflammatory bowel disease (arthritis in two, and a bullous skin rash in one) but no such associations have been seen in our case, either before the pericarditis developed or during follow-up.

Raised levels of circulating immune complexes may accompany inflammatory bowel disease (Jewell and MacLennan, 1973), and are particularly common when the disorder is active and when non-intestinal complications are present (Hodgson, Potter and Jewell, 1977). Immune complex levels have been measured in only one previous case of pericarditis

associated with inflammatory bowel disease; in this patient with ulcerative colitis, circulating immune complexes were only assayed during the acute phase of the pericarditis and were within normal limits (Becker *et al.*, 1981).

In our case all the values obtained, with the exception of one moderately elevated IgM complex level 3 weeks after the acute episode, were similarly within the expected normal range. This was, perhaps, surprising in view of the severity of the patient's illness, the predominant colonic involvement, and the presence of a non-intestinal complication of Crohn's disease (Jewell, 1978). In any event, as in the majority of the previously reported cases, high dose systemic corticosteroid treatment resulted in a rapid resolution of the pericarditis. Although pericardial effusion and even tamponade have been described in other patients (Breitenstein *et al.*, 1974; Rheingold, 1975), no fluid accumulation was demonstrated in this case and no additional therapy was required. Some previous reports suggest that relapse may not be uncommon in patients with pericarditis and inflammatory bowel disease (Goodman *et al.*, 1976). However, our patient has to date remained extremely well, with both aspects of her disease staying in complete remission despite withdrawal of corticosteroid treatment.

This case report provides further evidence of a definite association between acute pericarditis and active inflammatory bowel disease. Pericarditis should certainly be excluded in a patient with ulcerative colitis or Crohn's disease who complains of chest pain because of the possible dangerous consequences of the condition should tamponade develop.

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