Pericarditis in exacerbation of ulcerative colitis

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Abstract

Acute pericarditis occurs very rarely in association with inflammatory bowel disease (IBD). It has been described both as an extraintestinal manifestation of IBD and as an adverse drug reaction in IBD treatment. We present a case of a 26-year-old female patient with a severe exacerbation of ulcerative colitis, who was previously under long-term treatment with mesalazine and low-dose prednisone. The literature on pericardial involvement in IBD is reviewed. (Acta gastroenterol. belg., **2006**, 69, **390-392**).

Introduction

Extraintestinal manifestations of inflammatory bowel disease (IBD) are common and well-described. However, cardiac involvement is rare. The most frequent cardiac complication is pericarditis, with or without associated myocarditis or pleuritis. Cardiac conduction abnormalities (1) and myocarditis alone (2,3) have been described as well. Pericarditis can also develop as an adverse reaction to mesalazine or sulfasalazine therapy. We present a patient with simultaneous occurrence of acute pericarditis and pyoderma gangrenosum in acute flare-up of ulcerative colitis.

Case report

A 26-year-old woman with a 14-year history of ulcerative colitis presented to our hospital with a severe relapse. Ten months before current admission she started to notice blood in the stool and had occasional tenesmus, after a period of remission eight years long. Worsening of bowel symptoms occurred three months later, when colonoscopy in another clinic identified active pancolitis (Mayo endoscopic subscore of 3) (4) and therapy with prednisone 5 mg daily and mesalazine 2,5 g daily was started. The symptoms were worsening despite therapy.

At admission she presented with abdominal pain and cramping, bloody diarrhea, fever with chills, dyspnea, right chest pain that was pressure-like and pleuritic in nature, and multiple ulcerative lesions with exudates on both legs. She also reported anorexia and polakisuria.

On physical examination she was tachypnoic, pale, with heart rate of 100 beats/min, blood pressure of 120/70 mmHg, and body temperature of 38.0°C. Pericardial friction rub was audible and there was abdominal tenderness in right lower quadrant and epigastrium, with lesions on the legs as described above. No jugular venous distension or paradoxal pulse were present. Lungs were clear bilaterally.

The erythrocyte sedimentation rate was 43 mm/h (normal value < 15), and C-reactive protein 121,6 mg/L (n.v. < 6). Hemoglobin level was 115 g/L (normal range 119-158) with erythrocyte count of $4{,}09 \times 10^9$/L (n.r. 3.86-5.08) and hematocrite of 34% (n.r. 35-47), leukocyte count was $13{,}8 \times 10^9$/L (n.r. 3.4-9.7), and platelets count 469×10^9/L (n.r. 158-424). Potassium level was 3,4 mmol/L (n.r. 3.9-5.1), and iron level 2 µmol/L (n.r. 11-32). Prothrombin time was 69% (n.r. 70-130) and total protein level 58 g/L (n.r. 66-81). Oxygen saturation was 94,6%. ECG showed no other abnormalities than incomplete right bundle branch block. Chest radiograph finding was normal, without cardiomegaly or pleural effusion. Urinalysis was normal, but urine culture showed significant bacteriuria (Proteus mirabilis). Blood cultures were negative.

After admission to interventional gastroenterology unit, central venous line was placed, the patient was rehydrated and total parenteral nutrition was introduced. Therapy was started with broad-range antibiotics, intravenous methylprednisolone 60 mg daily, mesalazine 4x1 g orally, with other supportive therapy and local treatment of skin lesions with a topical corticosteroid. Because of dyspnea, pleuritic chest pain and audible pericardial friction rub, echocardiography was performed, showing large pericardial effusion (Fig. 1) and fibrine deposits, with signs of inflow impediment to both ventricles, but without signs of impending cardiac tamponade. Serologic studies for viral pathogens known to cause pericarditis (echovirus, coxsackie, adenovirus, cytomegalovirus, Epstein-Barr, herpes simplex 1 and 2) were negative, as was immunoserology (ANA, ANCA, rheumatoid factor, complement levels). Thyroid-stimulating hormone level was normal. Consecutive follow-up echocardiograms performed over the following two weeks showed gradual withdrawal and eventual disappearance of pericardial effusion. Ten days after admission sigmoidoscopy was performed, showing mucosal

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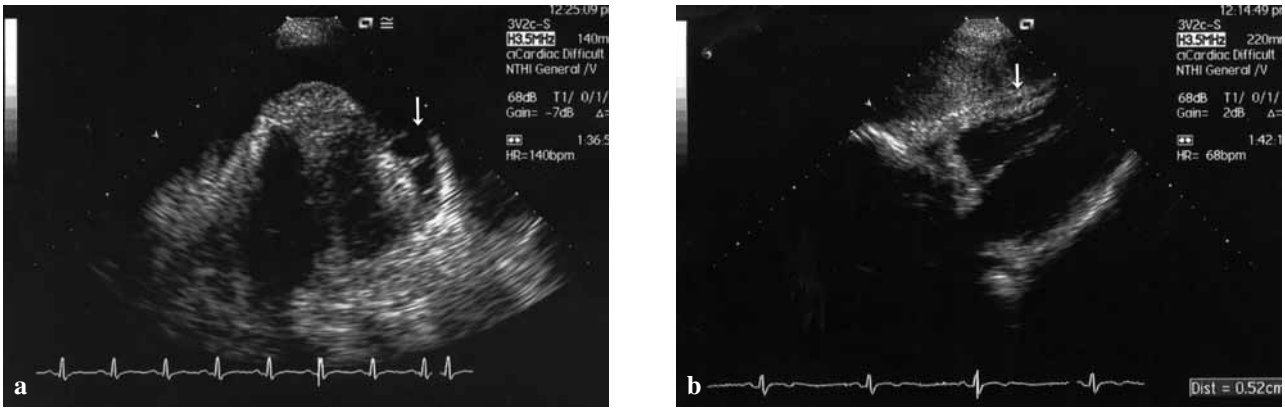

Fig. 1. — a. Echocardiogram at diagnosis, showing pericardial effusion ; b. Follow-up echocardiogram obtained a week later, showing regression of the effusion.

edema and friability, multiple ulcers and pseudopolyps. Four weeks after admission the patient was transferred, significantly improved, to Department of Dermatology for further treatment of skin lesions. Therapy at discharge was 32 mg of methylprednisolone orally with suggested gradual tapering to 20 mg daily, mesalazine in a total dose of 1,5 g daily and mesalazine enema 2 times daily. Eventually remission was achieved. Cushing's syndrome was observed during follow-up.

Discussion

Extraintestinal manifestations of IBD involve most organ systems and include pyoderma gangrenosum, erythema nodosum, arthritis, ankylosing spondylitis, conjunctivitis, uveitis, fatty liver, primary sclerosing cholangitis, pericholangitis, and thromboembolic disease.

Pericarditis, sometimes associated with myocarditis or pleuritis, is a rare manifestation in patients with inflammatory bowel disease (IBD). After the first report in 1967 (5), about 80 cases have been described to date.

Pericarditis occurs as an extraintestinal manifestation of ulcerative colitis and Crohn's disease, but it can also occur as an adverse reaction to mesalazine or sulfasalazine therapy. One case of azathioprine-induced pericarditis has been described as well (6).

Pericarditis as an extraintestinal manifestation of IBD occurs more commonly in the active phase of disease, but it occurs during remissions as well, regardless of disease activity (7). It may also be the initial manifestation of IBD (8). Although most cases are associated with colonic involvement of IBD and are described more frequently in patients with ulcerative colitis, pericarditis associated with isolated small bowel disease has been described as well (9). Pathogenesis of pericarditis as an extraintestinal manifestation of IBD remains unclear.

Pericarditis as an adverse reaction to mesalazine therapy typically occurs within 2 to 3 weeks after starting the treatment. Reactions to various doses of mesalazine have been reported. A drug-induced cell-mediated hypersensitivity has been suggested as the underlying pathogenetic mechanism (10). Mesalazine enemas have also been reported to cause pericarditis (11).

In several described cases of sulfasalazine-induced pericarditis it presented as a lupus-like syndrome, with positive anti-nuclear antibody, sometimes also positive anti-dsDNA antibodies and other features of systemic lupus erythematosus present (9). It presented in a range from months to years after drug initiation.

Pericarditis in IBD is most commonly treated with corticosteroids, though there have been reports of cases responsive to non-steroidal antiinflammatory drugs (12). In cases of suspected drug-induced pericarditis, discontinuation of the drug and initiation of corticosteroid therapy are recommended (13).

In our patient laboratory studies excluded viral or other possible causes of pericarditis, and a cause other than underlying ulcerative colitis seems unlikely. Possible mesalazine-induced pericarditis is ruled out concerning the history of 7-month mesalazine therapy prior to pericarditis manifestation and continuous therapy throughout the hospital stay and afterwards. With corticosteroid therapy, pericarditis subsided in several days.

Although it is common for patients with IBD-associated pericarditis to have other extraintestinal manifestations at the same time, to our knowledge pyoderma gangrenosum was reported only twice (9,14). Simultaneous occurring of pyoderma gangrenosum and pericarditis in an acute flare-up of IBD in our patient could suggest the same underlying pathogenetic mechanism for both manifestations, and confirm the notion of inflammatory bowel disease as a systemic disorder.

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