

Perimyocarditis – uncommon extraintestinal manifestation of ulcerative colitis

Zapalenie osierdza i mięśnia sercowego – niecodzienna pozajelitowa manifestacja wrzodziejącego zapalenia jelita grubego

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Abstract

Ulcerative colitis (UC) is an example of inflammatory bowel disease that can be manifested by extraintestinal complications including cardiac disorders. The most commonly reported – pericarditis – occurs in 0.23% of all UC patients. The knowledge about the etiology of pericarditis is important to implement accurate therapy. However, the diagnosis is not always clear and can be connected with diagnostic and therapeutic challenges. In this case, we present a perimyocarditis in the course of UC exacerbation.

Key words: ulcerative colitis, perimyocarditis, pericarditis

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Introduction

In clinical practice pericarditis is the most common disorder of the pericardium. The aetiology of acute pericarditis include infectious and non-infectious causes. Pericarditis may be isolated disorder or may occur as a part of systemic disease [1]. Ulcerative colitis (UC) is an example of inflammatory bowel disease (IBD) that not only affects the gastrointestinal tract but also can be associated with extraintestinal complications. Cardiac disorders seem to be uncommon but potentially serious manifestations. They can range from mild inflammations to pericardial tamponade, cardiogenic shock and myocardial infarction [2, 3]. The most commonly reported – pericarditis – occurs in

0.23% of all UC patients [4]. Nevertheless, the diagnosis is not always clear and can be related with diagnostic and therapeutic challenges. Therefore, we present a case of perimyocarditis in the course of UC which caused some diagnostic problems.

Case report

A 21-year-old woman with newly diagnosed ulcerative colitis, treated with small dose of sulfasalazine, was admitted urgently to the hospital complaining of fever, weakness, chest pain and bloody diarrhoea (< 4 bloody stools/day). Blood analysis revealed anaemia (hemoglobin [Hb] 7.8 g/dL), hypokalemia (3.2 mmol/L), slightly elevated troponin

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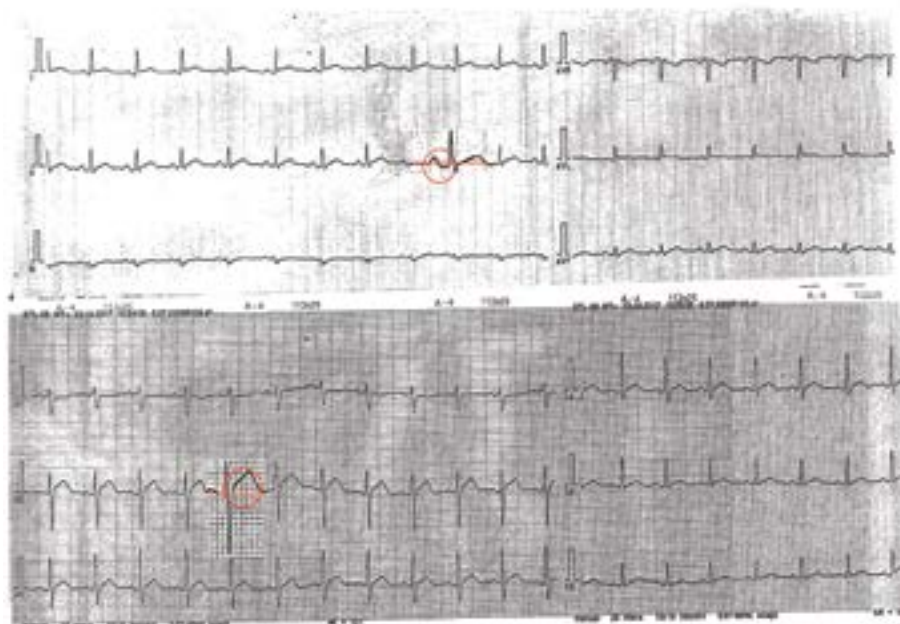


Figure 1. Electrocardiogram showing ST elevations and PR depressions

(0.028 µg/L) and D-dimers (1.94 mg/L). Laboratory test revealed increased inflammatory markers: white blood count (WBC) – 14 ths/µL, C-reactive protein (CRP) – 230 mg/L and procalcitonin – 22 ng/mL. A chest radiograph showed no pulmonary infiltrative changes. Angio-CT excluded pulmonary embolism. Abdominal and pelvic CT revealed no significant deviations apart from inflammatory features of the colon as in the UC. Patient received red cell concentrate transfusion. Optimal treatment with mesalazine (4 g/day) was instituted. After that the chest pain decreased, the stool frequency decreased with no bleeding, but patient's condition was not improving. Due to persistent fever up to 41 °C, hypotension (84/40 mm Hg), tachycardia (up to 120/min), neutrophilic leukocytosis, increased CRP and high level of procalcitonin, the suspicion of septic shock was raised and combined wide spectrum antibiotic therapy (vancomycin and imipenem) was administered. Nevertheless patient's condition deteriorated, chest pain reoccurred. Physical examination revealed pericardial friction rub and ankle oedema. In the following tests the levels of D-dimers (2,62 mg/L) and troponin I (0,73 µg/l) increased. NT-proBNP was significantly elevated to 15082 pg/mL. Electrocardiogram showed ST segment elevation in I, II, aVL and V2–V4 and PR depressions (Figure 1). Transthoracic echocardiography revealed pericardial effusion with fibrin strands and fluid in the pleural cavity (Figure 2A, B). Systolic function of the left ventricle was decreased – left ventricular ejection fraction (LVEF) was reduced to 38%. The diagnosis of perimyocarditis was made. Cardiac magnetic

resonance imaging (MRI) was performed to assess the degree of myocardial involvement. Due to negative blood cultures and probable autoimmune aetiology of pericarditis, successful treatment with prednisolone 50 mg per day was started. In a couple of days alleviation of fever and chest pain was observed. Markers of inflammation and NT-proBNP level were normalizing. Control echocardiography showed reduction of pleural and pericardial effusion and improvement of left ventricular contractility of the heart (Figure 2C, D). The patient was discharged in a good condition, free of symptoms. Prednisolone was prescribed orally with the recommendation of dose reduction. On the basis of overall clinical picture, echo and MRI results, myocarditis and pericarditis was confirmed, most likely caused by the main disease.

Discussion

Cardiac manifestations of UC may occur before, simultaneously or after the diagnosis of underlying disease. It should be emphasized that extraintestinal manifestations of IBD not always parallel the activity of the underlying disease. Temporal relationship between onset of UC flares and the course of cardiovascular manifestation may be difficult to define and can cause a significant challenge to physicians managing these patients. A multidisciplinary team approach is often needed for effective management.

In the process of differential diagnosis UC related pericarditis the drug-induced aetiology has to be taken into

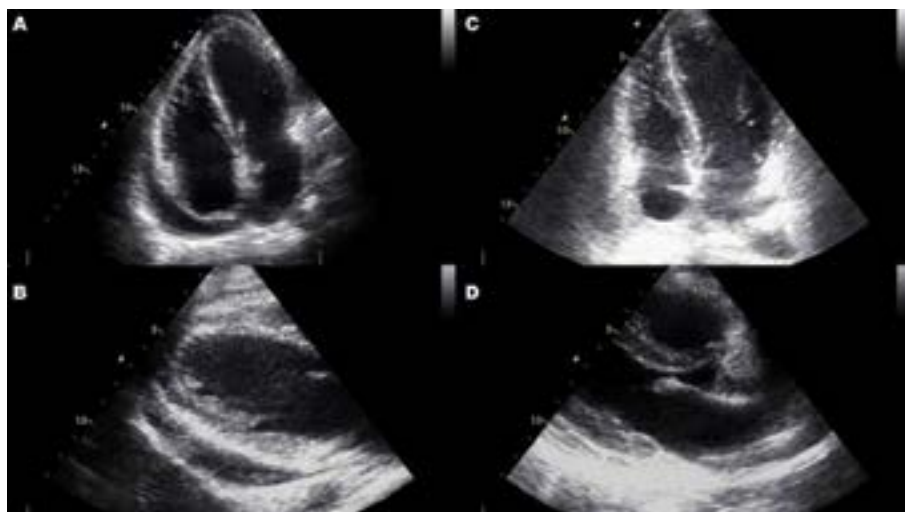


Figure 2A–D. Transthoracic echocardiography: **A, B.** Echocardiographic findings before implementation of steroid therapy: 1.1–1.5 cm of pericardial effusion with features of fibrin and right atrial collapse; **C, D.** Regression of pericardial effusion at discharge

account (toxic effects of – mesalazine and azathioprine). In the literature, cases of mesalazine-induced pericarditis have been reported [3, 5, 6]. One study recommend that among patients treated with mesalazine who present symptoms of perimyocarditis, mesalazine should be immediately discontinued [3]. In our case, perimyocarditis occurred early after diagnosis of the UC, however severity of intestinal symptoms was disproportional to the systemic involvement. Implementation of steroid therapy was problematic owing to highly increased inflammatory markers and possibility of sepsis. It has been reported that the intensive-care unit admissions of UC patients in 22% resulted from septic complications [7]. Finally, patient was treated successfully with mesalazine and prednisolone.

The knowledge about the aetiology of pericarditis is important to implement accurate therapy [8].

Conclusions

Myocarditis is rarely taken into account as one of the possible extraintestinal manifestations of UC. Any patient with UC who develops symptoms of pericarditis require prompt diagnosis and accurate therapy with glucocorticosteroids and consideration of mesalazine cessation.

Conflict of interest(s)

None declared.

Streszczenie

Wrzodziejące zapalenie jelita grubego (UC) jest przykładem nieswoistej choroby zapalnej jelit, która może się manifestować objawami pozajelitowymi, w tym objawami ze strony układu sercowo-naczyniowego. Najczęściej stwierdzane zapalenie osierdzia występuje u 0,23% pacjentów z UC. Wiedza na temat etiologii zapalenia osierdzia jest niezbędna do wdrożenia właściwej terapii. Mimo to diagnoza nie zawsze jest łatwa i może być związana z wyzwaniem zarówno w zakresie diagnostyki jak i leczenia. Przedstawiony opis przypadku ilustruje problem zapalenia osierdzia i mięśnia sercowego w przebiegu UC.

Słowa kluczowe: wrzodziejące zapalenie jelita grubego, zapalenie osierdzia i mięśnia sercowego

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