

Methotrexate Induced Pericarditis and Pericardial Effusion in Psoristic Patient

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Abstract

We describe a 62-year-old woman with Psoriasis who presented as Methotrexate - induced pericarditis and pericardial effusion. Aspiration of the pericardium was required and the patient made a satisfactory recovery. At six - months follow-up, she remained well, psoriasis plaques was controlled by topical crude coal tar and topical corticosteroid. These complications are extremely rare, but have been described as isolated phenomena associated with methotrexate therapy.

Methotrexate is one of the most effective drugs for systemic therapies of psoriasis^(1,2). The recognised features of methotrexate (MTX) toxicity are serious hepatic, hematologic, gastrointestinal symptoms, malaise and other toxicities^(3,4). Many reports indicate that pleurisy and/or pneumonitis can develop in MTX treated patients⁽⁵⁻⁷⁾ and occur in 5-12 per cent^(5,8) of treated patients, whereas, pericarditis and pericardial effusion are extremely rare. We describe a patient with psoriasis that was treated in short term with low-dose methotrexate in whom pericarditis and pericardial effusion developed, leading to symptoms of breathlessness.

CASE REPORT

A 62-year-old psoriatic woman developed small plaque psoriasis vulgaris when she was 54 years old, physical examination revealed approximately 30 per cent total body surface area involvement. Her medications included 5 per cent crude coal tar in 0.05 per cent betamethasone dipropionate cream applied twice a day to psoriatic plaques. She was admitted with a six-day history of flu-like symptoms and pleuritic-type central chest discomfort, for which she had pressure 90/60 mmHg. Past history was normal. Two months before this admission she was started on 20 mg methotrexate weekly⁽⁹⁾ because the patient's psoriasis had

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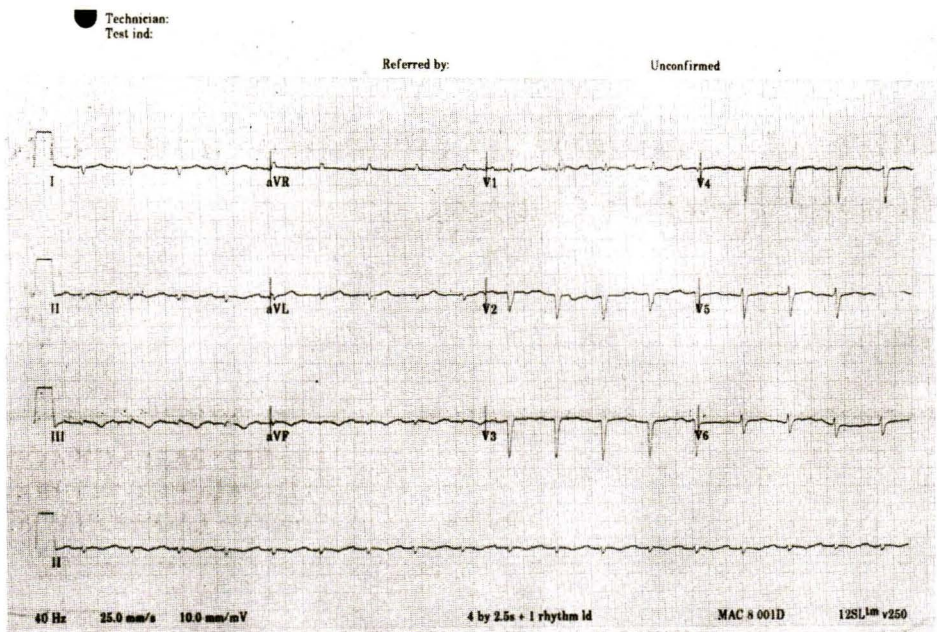


Fig. 1. Electrocardiogram demonstrates sinus tachycardia and generalized low voltage.

worsened. Nine courses were given over a 16 week period (total methotrexate dose 180 mg). After 16 weeks of treatment with methotrexate, she presented with pleuritic type central chest discomfort. She appeared ill with low grade fever, but she had no cough and sputum. Skin lesions showed significant improvement with approximately 2 per cent total body surface area involvement. On examination, pleural rub was noted at the left base without other symptoms. Methotrexate - associated pleurisy and pneumonitis were suspected. Methotrexate was discontinued and she was advised to ensure adequate hydration and symptoms responded to treatment with naproxen. One month after treatment, she had recurrence of her symptoms of pleuritic chest pain and was also dyspneic on exertion. On physical examination she had low grade fever. Jugular venous pressure was normal. Heart sounds were normal and examination of chest and abdomen were also normal. Chest X-ray revealed moderate cardiomegaly. The ECG showed sinus tachycardia with generalized low voltage (Fig. 1). Trans-thoracic echocardiogram showed a 0.7 cm pericardial effusion (Fig. 2) without evidence of impaired myocardial function at rest. The working diagnosis was pericardial effusion. Computed tomography (CT) scan did not show any mass lesions within the mediastinum but did confirm cardiomegaly with a small amount of pericardial effusion and bilateral pleural effusion.

Pericardial aspiration produced 150 ml of a straw-coloured exudate, containing polymorphs, scattered eosinophils and reactive mesothelial cells. Screening for a wide range of infective agents including mycobacteria were negative. Erythrocyte sedimentation rate (ESR) and C-reactive protein were normal. Full blood count, urea, electrolytes, liver function and immunological studies were normal. Total haemolytic complement and antibody to double-stranded DNA were normal. Rheumatoid factor and antibodies to nuclear and related antigens were negative. After 150 ml of straw-coloured fluid was removed, there was immediate improvement in blood pressure to 120/80 mmHg. Her symptom of breathlessness also resolved.

Six weeks after the initial presentation, she developed a recurrent low-grade fever, breathless on exertion together with pleuritic pain in the region of the left base accompanied by a pleural rub and an effusion. The ESR and c-reactive protein rose to 100 mm in the first hour and 308 mg/l, respectively. Repeat echocardiogram showed only

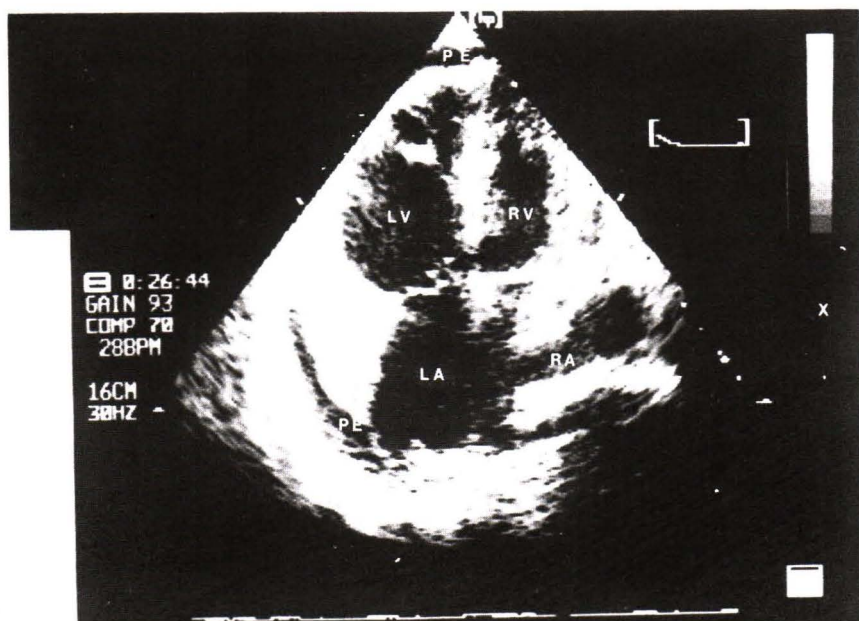


Fig. 2. Transthoracic echocardiogram; ra = right atrium, rv = right ventricle, pe = pericardial effusion, la = left atrium, lv = left ventricle.

a small rim of pericardial fluid and a ventilation/perfusion lung scan showed no evidence of pulmonary embolism.

The patient's condition improved with rest and symptomatic treatment, and at review 2, 4 and 6 months later, she was feeling well and ESR and C-reactive protein had returned to normal. Psoriatic skin lesions had been controlled by 5 per cent crude coal tar and 0.05 per cent betamethasone dipropionate cream applied twice a day to psoriatic plaque.

DISCUSSION

The cardiac manifestations of psoriasis with MTX treated presenting as pericardial disease are very rare; while pericardial effusion is detectable by echocardiography. The occurrence of subsequent pericarditis is even less frequent with only occasional reports in the literature. It is particularly notable in this case that pericardial effusion was the presenting feature of the MTX induced pericarditis and that it appeared to have rapid onset.

The mode of presentation in our patient was particularly interesting. The initial diagnosis was methotrexate-associated pleurisy and pneumo-

nitis. In this patient the diagnosis was made by transthoracic echocardiography which revealed mild to moderate pericardial effusion and the right-sided structures were normal.

Pericardial aspiration was an adequate measure without the use of steroids. It is interesting that in the few cases reported, low-dose steroids did not prevent the development of constrictive pericarditis⁽⁹⁾.

Methotrexate induced pericarditis and pericardial effusion are unusual but have been reported⁽¹⁰⁾. The mechanism of methotrexate in psoriasis is unknown, its effects may be both anti-inflammatory and immunosuppression⁽¹¹⁾. The syndrome of methotrexate pleural toxicity is invariably of pleuritic pain with normal chest X-ray and ventilation/perfusion scan. X-ray changes if present are non-specific and diagnosis may require bronchoscopy and lavage^(7,12) or lung biopsy⁽¹³⁾. The differential diagnosis is infection, pulmonary infarction and tumour-related complications. This patient had methotrexate chemotherapy followed by two episodes of pleurisy, the second being associated with pericarditis and a mild to moderate amount of pericardial effusion. As in the majority

of cases with pleural toxicity, there was no peripheral eosinophilia; however, eosinophils were present in the pericardial fluid. An unrecognised viral infection or a reaction to an unsuspected allergen remains a possible cause; however, we believe that the pericardial effusion was most likely a complication of methotrexate chemotherapy and that, as with methotrexate pneumonitis, this is

likely to be an immune-mediated serositis⁽¹²⁾. The possibility of pericarditis and significant pericardial effusion should be considered as a potentially severe complication of methotrexate therapy. The regular use of echocardiography could provide information on the incidence of isolated asymptomatic pericardial effusion and that concomitant with methotrexate pneumonitis/pleurisy.

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เยื่อหุ้มหัวใจอักเสบและน้ำในช่องเยื่อหุ้มหัวใจซึ่งเป็นผลจากการใช้เมโธเทรกเสท
รักษาผู้ป่วยสะเก็ดเงิน

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ผู้เขียนได้รายงานผู้ป่วยหญิง อายุ 62 ปี ป่วยเป็นโรคสะเก็ดเงิน ได้เกิดภาวะการอักเสบของเยื่อหุ้มหัวใจและ
น้ำในช่องเยื่อหุ้มหัวใจจากยาเมโธเทรกเสท อาการของผู้ป่วยดีขึ้นหลังการเจาะน้ำจากช่องเยื่อหุ้มหัวใจ และมีอาการปกติหลัง
ติดตามการรักษานาน 6 เดือน ผื่นจากโรคของผู้ป่วยดีขึ้นด้วยการรักษาโดยใช้ยาทา
ภาวะแทรกซ้อนในรายงานนี้พบได้น้อยมาก แต่มีรายงานว่าพบได้บ่อยในผู้ป่วยที่ได้รับยา
เมโธเทรกเสท

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