

Arthropathy in Dressler's syndrome

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Summary

Three patients developed a polyarthritis in association with Dressler's (post-myocardial infarction) syndrome. Joint involvement was more pronounced in the upper limbs and persisted many months after other features of Dressler's syndrome had settled. In 2 cases the findings in the joints were slight and might have been overlooked but for the persistently raised ESR. There was a prompt symptomatic response to prednisone.

Introduction

The post-myocardial infarction syndrome described by Dressler (1956) is characterized by fever, high ESR, pericarditis, pneumonitis and pleurisy. Joint involvement has not previously been recognized in this syndrome, but was striking in the first case described below. Two milder cases were subsequently seen with similar features.

Case 1

A 55-year-old man was admitted with an anterior myocardial infarct after a period of crescendo angina. Ten days later he developed fever, tachycardia and a right pleural effusion. He was treated with digoxin and frusemide and anticoagulants were added a few days later when he developed ill-defined calf tenderness. In the 3rd week he suddenly developed an inflammatory arthritis affecting shoulders, elbows, wrists, knees and small joints of the hand. The metacarpophalangeal and interphalangeal joints were visibly swollen and red, and the grip weak. The white count rose to $19.8 \times 10^9/l$ and the ESR to 68 mm/hr and there was a reversed albumin globulin ratio (0.29 : 0.31 g/l); rheumatoid and antinuclear factors were negative and serum uric acid was 0.41 mmol/l. The painful joints were treated with splinting and analgesics.

In the 4th week he developed haemoptysis and the chest X-ray showed shadowing in the left mid zone. No organism was isolated but the WBC rose to $27.4 \times 10^9/l$ and the ESR to 110 mm/hr and amoxicillin was given. In the 5th week a pericardial friction rub was heard and, with a belated diagnosis of Dressler's syndrome, prednisolone 60 mg/day was

prescribed. Five days later his sputum became purulent and the chest X-ray showed cavitation in the left mid zone. A penicillin-resistant *Staphylococcus pyogenes* was isolated and the abscess slowly resolved on flucloxacillin.

Over the next 2 months the chest X-ray and WBC returned to normal, and the steroids were tailed off. However, he continued to have pain in his shoulders, hands, knees and ankles with 1-2 hr morning stiffness and this more than all else prevented him from returning to work. There were few objective findings; some joint tenderness, pain at the extremes of movement and weak grip. There were no subcutaneous or tendon nodules; the ESR remained between 20-40 mm/hr but the rheumatoid factor, ANF and uric acid were normal, and there were no joint erosions on X-ray.

Eighteen months later he was admitted with acute inferior myocardial infarction, developed cardiogenic shock and died. At post-mortem he had an old anterior and recent postero-septal infarct with a large mural thrombus. There was fibrinous pericarditis and adhesive pleurisy at the bases of both lungs. Unfortunately the joints were not examined histologically.

Case 2

A 59-year-old man was admitted with an inferior myocardial infarction. He had a pericardial friction rub, raised venous pressure and a gallop rhythm. The blood urea rose transiently to 41.8 mmol/l. He was given diuretics and prophylactic anticoagulants.

On the 5th day he developed purulent sputum and some shadowing in the right lower zone on chest X-ray. No organism was isolated but he received a course of amoxicillin. He continued to have a low grade fever and on the 10th day the chest X-ray showed a large pleural effusion on the right and a smaller one on the left. The pleural fluid was sterile but contained some polymorphs. His condition slowly improved on an increased dose of frusemide but the ESR rose to 88 mm/hr. He complained of stiffness in both shoulders and there was restricted movement, more on the left than the right.

Two weeks later he developed left pleuritic chest

pain. The chest X-ray still showed small bilateral pleural effusions but the heart size was normal and the ESR was 100 mm/hr. The pain settled quickly and anticoagulants and diuretics were tailed off. He continued to have pain in the shoulders and 3 months later when he returned to work, he developed stiffness in the hands and knees, especially in the mornings. He was tender over the metacarpophalangeal joints, shoulder movements were restricted and small bilateral effusions were present in the knees. The ESR was still raised at 54 mm/hr. Uric acid was 0.45 mmol/l, rheumatoid factor negative and X-rays of hands and feet normal. He was given prednisone 15 mg/day with dramatic improvement. Steroids were gradually withdrawn over 6 months and now, 2 years later, there has been no recurrence in his symptoms.

Case 3

A 60-year-old man was admitted with an anterior myocardial infarct. He had a low grade fever from the 2nd to the 11th day post-infarction, associated with pallor, malaise, stiffness in both shoulders and a raised ESR (77 mm/hr). No pericardial friction rub was heard and the chest X-ray remained clear.

Mobilization was slow with some continuing pains in his shoulders. Three months after the infarct he returned to work, but shortly afterwards developed generalized 'rheumatism' with aching in the shoulders and both arms. Chest X-ray at this time was clear, but the ESR was 80 mm/hr.

Over the next month he deteriorated with involvement of shoulders, knees, and the small joints of the thumb and index finger on both hands. As a result his grip was poor, and considerable morning stiffness was present. There was some tenderness of the affected joints but no visible swelling or inflammation, no rheumatoid nodules, or tenosynovitis. For the first time a soft pericardial rub was heard at the apex. Full blood count, uric acid and immunoglobulins were normal, rheumatoid factor and antinuclear factor negative, but the ESR was 77 mm/hr. He was given prednisolone 15 mg/day with resolution of his symptoms. Steroids were gradually withdrawn over the next 6 months and his symptoms have recurred to a milder degree. There are still few findings but the ESR has risen to 44 mm/hr. At present the arthralgia is controlled on anti-inflammatory drugs alone.

Discussion

These cases have several features in common. Joint symptoms appeared soon after the myocardial infarct at a time when there were other findings suggesting Dressler's syndrome. In the more severe case,

widespread joint involvement was present from the outset, whereas in the milder cases pain was initially confined to the shoulders and later spread to other joints. The symptoms persisted for several months and the resulting disability seemed out of proportion to the clinical findings. The temporal relation of the arthropathy to the myocardial infarct, the rapid response to steroids and the lack of chronic sequelae suggests the arthropathy was part of Dressler's syndrome.

Despite the numerous surveys and case reports of Dressler's syndrome joint involvement has only been recorded once. Brock and Ofstad (1960) described 2 patients with a clinical picture suggesting atypical Dressler's syndrome. One developed arthritis of the knee and elbow and one the knee alone; few clinical details are given and the second may have had chondrocalcinosis. In his own cases Dressler (1956) noted that pleuropericardial pain when present may radiate to the arms or shoulder tip, but did not suggest that the joints themselves were involved.

The pattern of joint involvement in the cases reported here, affecting mainly shoulders and upper limbs, is reminiscent of the shoulder-hand syndrome following myocardial infarction (Edeiken, 1957). However, the clinical picture was of a polyarthropathy and there was no swelling of the hands or later trophic change. There is a much closer similarity between these cases and several patients described by Ernstene and Kinell (1939) in which transient 'rheumatoid arthritis' followed myocardial infarction. Unfortunately there is not enough clinical information to know whether any of these might have had Dressler's syndrome.

The occurrence of joint symptoms in Dressler's syndrome is not surprising in view of current opinion (Bernstein, 1977) which favours an auto-immune cause. The fact that these 3 cases were seen by one physician over a 3-year period suggests that joint involvement in Dressler's syndrome is not uncommon and has perhaps been overlooked.

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References

- BERNSTEIN, A. (1977) The post myocardial infarction syndrome of Dressler. *British Journal of Hospital Medicine*, **17**, 560.
- BROCK, O.J. & OFSTAD, J. (1960) The post myocardial infarction syndrome. *Acta medica scandinavica*, **166**, 281.
- DRESSLER, W. (1956) A post-myocardial infarction syndrome. *Journal of the American Medical Association*, **160**, 1379.
- EDEIKEN, J. (1957) Shoulder hand syndrome following myocardial infarction with special reference to prognosis. *Circulation*, **16**, 14.
- ERNSTENE, A.C. & KINELL, J. (1939) Pain in the shoulders as a sequel to myocardial infarction. *Archives of Internal Medicine*, **66**, 800.



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