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Late Dressler's Syndrome at 6 Months after Myocardial Infarction: A Case Report

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Abstract Case Report

Fixed drug eruption is a recurrent rare-onset children disease. The etiology seems to be exclusively due to drug intake. However, several cases of food origin have been reported in the literature. We report an observation of a six-year-old girl had presented with typical bullous fixed drug eruption recurrent at the genital, with no concept of drug intake. Detailed interview with the parents describing the chronology of the appearance of skin lesion 24 hours after the consumption of farm eggs, in all episodes, evoking the possibility of the occurrence of the lesions following the ingestion of eggs of hen. However, the traceability of the egg was not possible. We interviewed poultry farmers who confirmed that they were administering drugs to chickens including sulfonamides, especially sulfadiazine.

Keywords: Food Contaminants, Child, Fixed drug eruption.

INTRODUCTION

Dressler's syndrome, also called postcardiac injury syndrome, is a form of secondary pericarditis, with or without pericardial effusion. Its incidence has been declining in recent years. Dressler's syndrome is a secondary form of pericarditis that can occur after acute myocardial infarction, cardiac surgery, traumatic injury or routine endovascular procedures. The etiology is not well understood, but it is believed that an initial injury to mesothelial pericardial cells, combined with blood in the pericardial space, triggers an immune response. This results in immune complex deposition in the pericardium, pleura and lungs, which causes an inflammatory response.

CASE REPORT

A 59 year-old male patient with a medical history of hypertension and diabetes; was admitted to the emergency department due to pericarditis chest pain associated with dyspnea (NYHA class III) that has been evolving for 20 days, worsening progressively. Patient had a past history of (CAD-AWMI) 6 months ago, who underwent percutaneous transluminal coronary angioplasty of the anterior interventricular artery 6 months previously. Patient was on medication for diabetes mellitus (DM), dyslipidemia (DLP) and double antiplatelet therapy. The physical examination demonstrated a blood pressure of 120/75 mmHg, tachycardia (103 bpm), temperature of 38 C°, and no jugular venous distension. Heart sounds were decreased at cardiac auscultation. Moreover, the electrocardiogram showed a diffuse microvoltage with flat T waves (Fig 1), the chest x-ray showed cardiomegaly with clear lung fields suggestive of a significant pericardial effusion (Fig 2). Transthoracic echocardiogram revealed an abundant circumferential anechoic pericardial effusion (diastolic diameter of 18mm), without collapse of the cardiac chamber; but with significant respiratory variations. Laboratory tests revealed elevated acute-phase reactants (mild leucocytosis and C reactive protein of 68 mg/L). Sterile blood cultures and myocardial necrosis markers were negative.

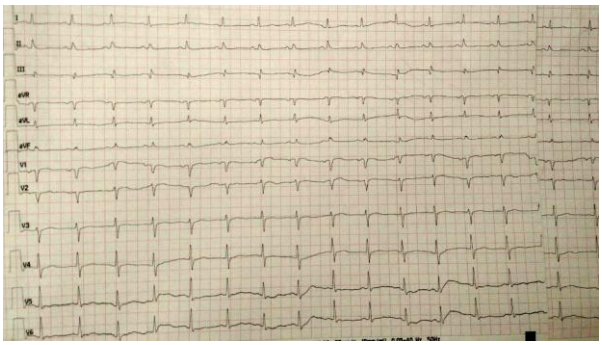

Fig-1: ECG at admission showing sinus rhythm and diffuse microvoltage with flat T waves

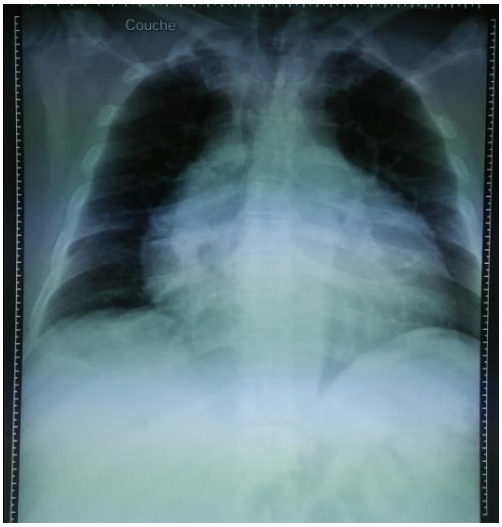

Fig-2: Chest X-ray revealed prominent cardiomegaly

The patient's clinical presentation (pericarditis chest pain and fever), the recent cardiac injury, the increase in the inflammatory markers, the ECG findings compatible with acute pericarditis, and the alterations found in the thoracic images support the diagnosis of Dressler's syndrome.

The diagnosis of infective endocarditis was considered less likely, given the negative sterile blood cultures, the absence of vegetations on transthoracic echocardiogram and the improvement of the patient's condition without the institution of antimicrobial therapy.

The patient was hospitalised with anti-inflammatory therapy (aspirin 1000mg every 8 hours and colchicine 0.5mg every 12 hours orally) under the presumptive diagnosis of Dressler's syndrome.

During the 6 days of hospitalisation, the patient presented a clinical improvement and C reactive protein decreased to 20mg/L. The patient's transthoracic echocardiography at the discharge showed reduced pericardial effusion.

After 2 months, the patient remained asymptomatic, and a transthoracic echocardiography showed a complete remission of pericardial effusion.

DISCUSSION

Dressler syndrome (DS) was first described by William Dressler in 1956, after observing the late period development of an acute myocardial infarction [1, 2]. It is an immuno-inflammatory disease similar to others that occur after myocardial injury, such as post-pericardiectomy syndrome and post-traumatic pericarditis [1, 3]. After myocardial infarction (MI), injuries to the cardiac myocytes expose myocardial antigens to the immune system, stimulating immune complex formation. This immune system activation triggers a systemic immune-inflammatory response that may involve other organs, such as the pleura, due to immune cross-reactions [1, 4]. The symptoms tend to occur 2 - 3 weeks after MI, and may include pleuritic chest pain and fever. A direct relationship between systemic inflammation and myocardial ischemia has been demonstrated by several authors [3, 4].

The disease is characterised by a persistent low-grade fever, thoracic pain (usually pleuritic in nature), pericarditis (usually evidenced by a pericardial friction rub) and a pericardial effusion and/or pleural effusion [5, 6]. The symptoms tend to occur 1–6 weeks following the initial damage to the pericardium but can also be delayed for a few months [5, 7]. 1 Dressler's syndrome is difficult to diagnose because its symptoms are similar to those of many other conditions, such as pneumonia, pulmonary embolism, angina, congestive heart failure or acute myocardial infarction.

Diagnostic tests for investigation of Dressler's syndrome should include a complete blood count that will demonstrate leucocytosis and an increase in C reactive protein level; blood cultures to exclude an infectious process; ECG that may show global ST segment elevation and T-wave inversion, such as with pericarditis; echocardiogram to look for the presence of fluid near the heart or thickening in the pericardium; thoracic radiography to see if there is any inflammation/effusion in the lungs and/or thoracic CT or cardiac MRI scan, which produces detailed images of the lungs, heart and the pericardium [5, 8].

The clinical course is most often benign, responding to a conservative management with anti-inflammatory therapy, including non-steroidal anti-inflammatory drugs (aspirin, ibuprofen or naproxen) and colchicine. In some non-responsive cases, corticosteroids (prednisone essentially) can be given for a course of one week, being tapered over a four week period, but are usually avoided in the first month due to the high frequency of impaired ventricular healing leading to an increased rate of ventricular rupture [7].

Rarely, Dressler's syndrome can cause more severe complications, including cardiac tamponade and constrictive pericarditis. Such complications can require invasive treatments, including pericardiocentesis or pericardiectomy [5, 9].

Although not a common condition, Dressler's syndrome should be considered in all patients with persistent fever and pericarditis chest pain or pleuritic thoracic pain, especially if symptoms begin 2weeks after cardiac injury. With the advancements in treatment of heart lesions, this condition is less frequent. However, early diagnosis of Dressler's syndrome is crucial since favourable prognosis depends on how quickly the condition is treated.

CONCLUSION

This case highlights the importance of the clinical assessment of patients admitted with chest pain. Although rare, Dressler Syndrome should be considered in the differential diagnosis of chest pain, especially in patients with CAD .This becomes especially important in developing countries, where many patients receive no reperfusion therapy due to difficulties in accessing the healthcare system.

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