Case report

Inspiratory chest pain and fever after acute myocardial infarction: Dressler's syndrome demonstrated on cardiac MRI

ABSTRACT

Introduction: Dressler's syndrome develops in some cases several days to weeks or even months after the myocardial infarction; the incidence also seems to have declined in recent years. This syndrome is caused by an autoimmune reaction to material extracted from necrotic myocytes.
Case Report: In this report, we present a case of a 72- year-old woman who was diagnosed with Dressler's syndrome after 1 month from acute coronary syndrome. A cardiac MRI was performed, which showed features of pericarditis. The patient responded well to treatment with NSAID and colchicine and was discharged home in good general condition.
Conclusion: Due to the significant number of patients living in the community who suffered MI, underwent cardiac surgery or received cardiac implantable device placement, clinical suspicion of PCIS warrants further diagnostic evaluation using imaging modalities especially cardiac MRI for timely diagnosis and treatment

Keywords: Dressler's syndrome, Inspiratory chest pain, Fever, Cardiac MRI*, Case report*

1. INTRODUCTION

Dressler's syndrome also known as post myocardial infarction (MI) syndrome, is a form of secondary pericarditis with or without a pericardial effusion, that occurs because of injury to heart or pericardium [1]. It develops weeks to months after the initial infarction, and rarely within the first week post-MI. It is characterized by the development of inflammation of the pericardium as well as another serosa [2]. If left untreated, inflammation of the pericardium can lead to scarring, thickening, and muscle tightening of the heart, which can be life-threatening [3]. Dressler's syndrome is associated with an immune system response to heart damage [4,5].

2. CASE PRESENTATION

A 72-year-old female with, arterial hypertension, diabetes mellitus and dyslipidemia. She had past history of coronary artery disease - inferolateral myocardial infarction, moderate left ventricular (LV) dysfunction diagnosed 1 month back and Percutaneous transluminal coronary angioplasty (PTCA) with stent to the circumflex artery done 1 month back.

The patient presented to the emergency department, reporting pain of a different pattern: severe constrictive chest pain lasting one hour that worsened with deep inspiration and while in a supine position associated with fever. The physical examination demonstrated a blood pressure of 140/85 mmHg, tachycardia (105 bpm), temperature of 38.6°C, and no jugular venous distension. A pericardial friction sound was easily audible at the lower left sternal border. Moreover, the electrocardiogram showed diffuse ST-segment elevation with a depression of the PR segment, highly suggestive of pericarditis.

Laboratory tests revealed elevated acute-phase reactants (moderate leukocytosis, high erythrocyte sedimentation rate and C reactive protein of 158 mg/L). Sterile blood cultures and myocardial necrosis markers were negative.

Transthoracic echocardiogram revealed a small circumferential anechoic pericardial effusion (diastolic diameter of 5 mm), without collapse of the cardiac chamber.

Cardiac MRI (CMRI) demonstrated an inferior akinesia and medial and basal inferolateral akinesia, leading to a mild deterioration of left ventricular systolic function (ejection fraction 41%, Figure 1).

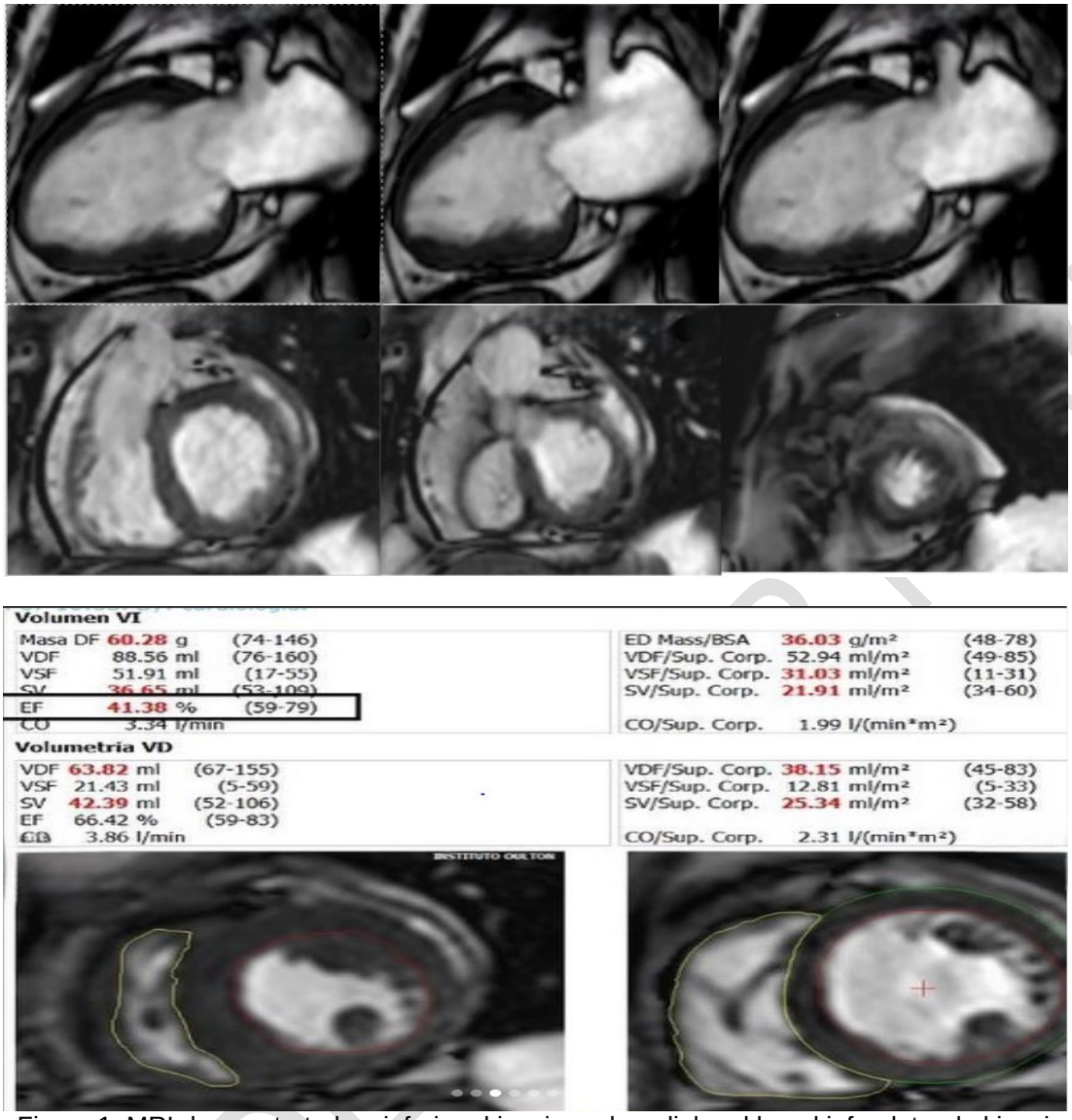

Figure 1: MRI demonstrated an inferior akinesia and medial and basal inferolateral akinesia with LV ejection fraction 41%)

Figure 2 : The late gadolinium enhancement sequence shows: medial infero-lateral transmural necrosis, and infero-medial, inferior and basal infero-lateral subendocardial necrosis

Figure 3 : corresponding to a necrotic mass of 23 grams, equivalent to 28% of the total LV mass

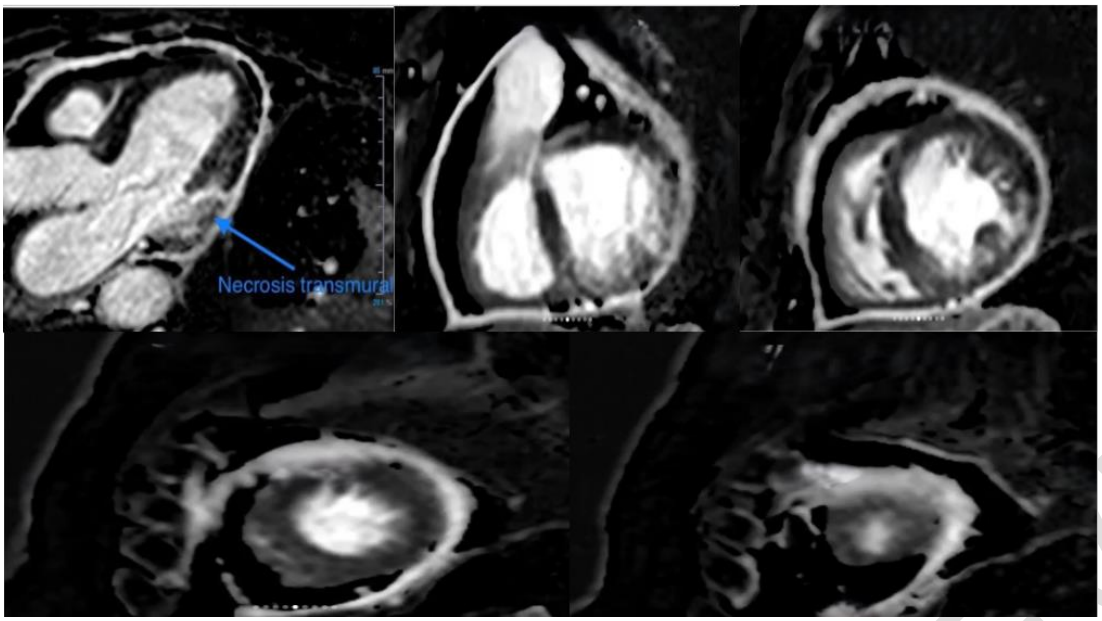

Figure 2

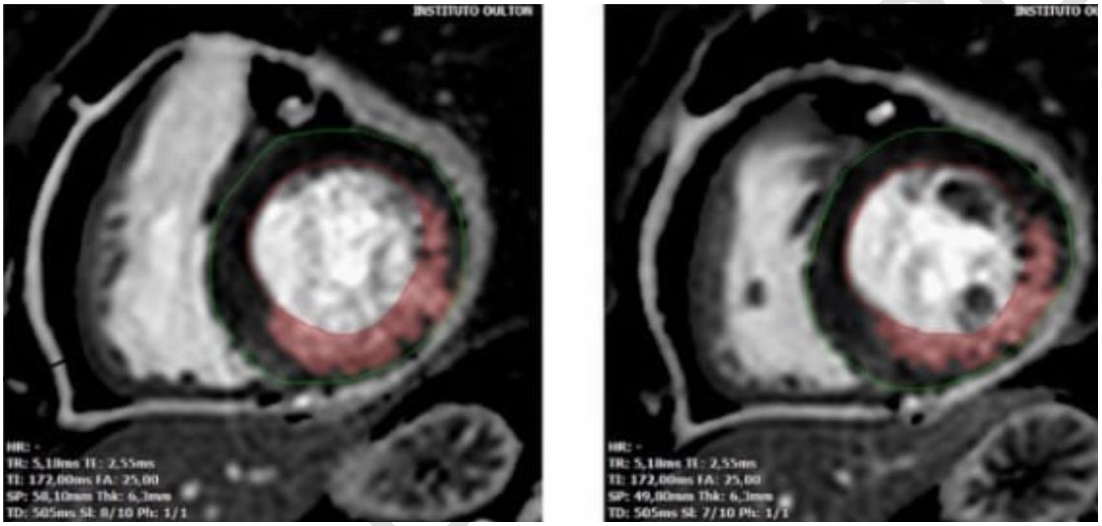

Figure 3

Figure 4 : It is important to note that the pericardium is thickened (4 mm), and also shows hypersignal in the T2 STIR sequence and homogeneous gadolinium uptake (blue arrow), which makes the diagnosis of acute pericarditis.

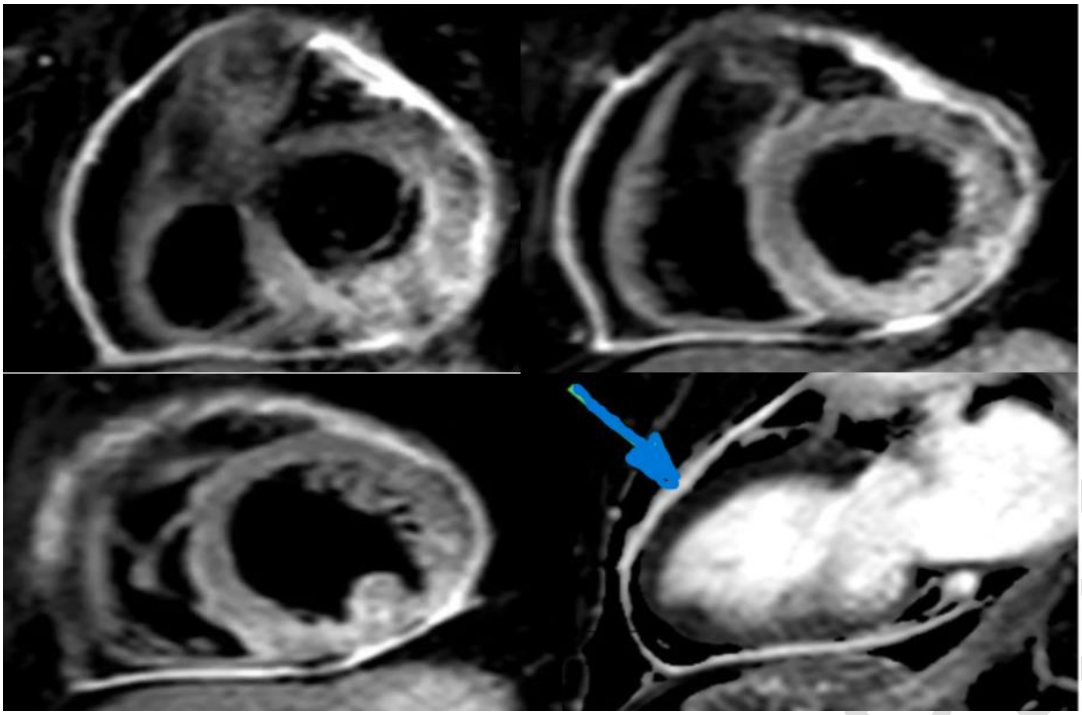

Figure 4

figure 5 : showed a mild left pleural effusion (orange arrow) and considering the clinical context of the patient, corresponds to the so-called Dressler's syndrome.

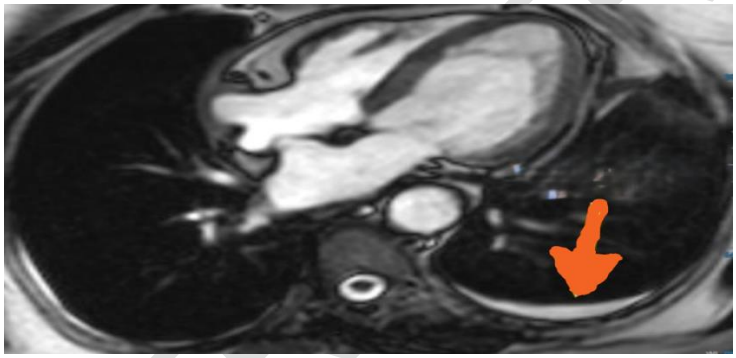

Figure 5

The patient was treated with aspirin 500 mg 3 times daily and colchicine 1 mg once daily. His physical signs and laboratory data improved within several days. Three months later, his pericardial effusion had improved. Colchicine 1 mg/day was continued without any side effects for 6 months, with no recurrence in symptoms noted or examinations repeated.

3. DISCUSSION

Dressler syndrome is part of the post-cardiac injury syndrome (PCIS) an aetiologic heterogenous group of autoimmune-mediated conditions of pericardial, epicardial, and myocardial inflammation. It is important to realize that inflammation is not confined to the pericardium and epicardium but also affects the myocardium to some extent.
The pathophysiology of PCIS is not completely understood. In 1956, Dressler suggested a role of an auto-antigens-mediated hypersensitivity reaction after MI, leading to myocardial necrosis, and subsequent pericarditis and pleuropericardial effusions in predisposed patients (15). The current hypothesis of PCIS stands with autoimmune pathogenesis, inciting the cascade with damage to the pericardial and pleural mesothelial cells, caused by either necrosis of the myocardium, surgical trauma, blunt thoracic trauma, or iatrogenic damage to the pericardium (16).

Nevertheless, symptoms of epicardial and pericardial inflammation, such as pain, effusions, and fever, dominate the clinical picture. Signs of ventricular dysfunction such as heart failure, ventricular arrhythmias or severe conduction abnormalities are very rare (6).

Clinical evaluation usually reveals mild to moderate effusions both in the pericardium (>80%) and in the pleural space (>60%), and sometimes pericardial friction rubs (30-60%) on auscultation. Laboratory analysis shows systemic inflammation with elevation of C-reactive protein (CRP) (74%), and elevated blood leucocytes (9,10).
A diagnosis of PCIS may be considered in patients with chest pain, fever, pericardial or pleural effusions, and a re-emerging systemic inflammatory response syndrome after an appropriate latency period following prior Myo pericardial or pleural injury. Importantly, alternative causes for inflammation and effusion need to be ruled out before a diagnosis of PCIS can be established (6-7)

In PCIS the cardiac MRI (CMRI) has good diagnostic value for pericardial inflammation in patients with post-MI (11). Presence of pericardial inflammation and effusion on CMRI has been reported as marker of myocardial infarction (MI) severity (11,12).

CMRI findings of pericarditis have been reported in up to 40% of patients post-myocardial infarction when imaged early, typically adjacent to the site of infarction (14). CMRI can provide evidence of pericardial inflammation and is clinically useful when trying to establish a cause for atypical post-myocardial infarction chest pain with atypical ECG and/or laboratory data (14).

CMRI findings in a post-MI PCIS (Dressler syndrome) include localised inflammation of pericardium surrounding the infarcted myocardium. T1 and T2 sequences shows moderate to high intensity signal. T1 sequences using fat suppression reveal gadolinium enhancement of pericardium, consequently, suggesting inflammation.
Also, non-haemorrhagic pericardial effusions have low signal intensity of T1 compared with haemorrhagic effusions and vice versa on gradient echo images, respectively (13).

Mainstays of treatment are anti-inflammatory NSAID and the adjunct use of colchicine, albeit that there are no randomized controlled trials addressing specific treatment approaches for PCIS. Treatment decisions must be taken with care given the potential side effects and risks associated with NSAID and colchicine in cardiac and post-surgery patients (8).

4. CONCLUSION

After development of primary percutaneous coronary intervention treatment for STEMI, complications decrease. Nevertheless, occasionally doctors can encounter rare complications. This case report shows that, although very rare, Dressler syndrome should be considered as a possible cause in every post-MI patient presenting with chest pain, fatigue, and signs of inflammation**.**

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WRITTEN INFORMED CONSENT WAS OBTAINED FROM THE PATIENTS FOR PUBLICATION OF THIS CASE REPORT AND ANY ACCOMPANYING IMAGES.

ETHICAL APPROVAL

AS PER INTERNATIONAL STANDARDS OR UNIVERSITY STANDARDS WRITTEN ETHICAL APPROVAL HAS BEEN COLLECTED AND PRESERVED BY THE AUTHOR(S).

AVAILABILITY OF DATA AND MATERIAL

ALL DATA GENERATED OR ANALYSED DURING THIS STUDY ARE INCLUDED IN THIS PUBLISHED ARTICLE**.**

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