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Intramyocardial haemorrhage: a rare sequela of acute myopericarditis

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ABSTRACT

INTRODUCTION

Acute myopericarditis is an inflammatory disease of myocardium and pericardium. The disease has variable presentation ranging from mildly symptomatic cases to severe disease manifested by cardiomyopathy with associated risk of sudden cardiac death. However, the association with intramyocardial hemorrhage (IMH) has not been previously described.

CASE REPORT

We describe an IMH detected by cardiac magnetic resonance imaging, in a patient with classical ‘de novo’ myopericarditis. To the best of the authors’ knowledge, this has not been previously described in patients without associated haematological or connective tissue disorders.

CONCLUSION

The adverse prognostic signal of IMH in acute myocardial infarction may also be reflected by IMH in acute myopericarditis and lead to adverse ventricular remodeling. The pathophysiology of IMH in myopericarditis is unknown and warrants further exploration.

Keywords: Intramyocardial hemorrhage;Myopericarditis;Cardiac magnetic resonance imaging

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INTRODUCTION

Myopericarditis is a condition characterized by inflammation of the pericardium with concurrent involvement of the adjacent myocardial layer. Myopericarditis typically presents with chest pain, fever, and shortness of breath of varying severity. Diagnosis is often based on electrocardiogram (ECG) [1], echocardiogram [2] and cardiac magnetic resonance imaging (CMR) findings [3,4]. The presence of intramyocardial hemorrhage on CMR in the context of myopericarditis and in the absence of connective tissue disease or hematological conditions has not been previously described.

CASE REPORT

A 39-year-old man, with no past medical history of note, presented with severe central chest pain radiating to the jaw and left arm. The pain started when the patient was exercising, had a pleuritic component and was accompanied by dyspnoea and light headedness. His presenting ECG revealed anterior ST segment elevation (figure 1), which led to immediate transfer to the regional cardiothoracic centre for primary percutaneous coronary intervention.

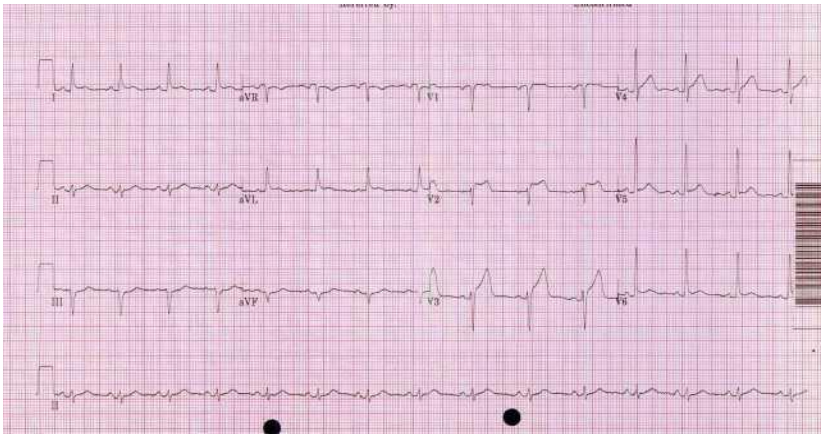

Figure 1: ECG demonstrating ST elevation on anterior leads V1-V4.

A bedside echocardiogram revealed preserved left ventricular systolic function with no regional wall motion abnormalities and a small pericardial effusion. The attending interventional cardiologist felt that in the context of non - ischemic sounding chest pain with preserved left ventricular function that immediate invasive coronary angiography was not mandated and requested urgent CT coronary angiogram. This demonstrated normal coronary arteries. There was no evi- dence of pulmonary embolism or aortic dissection. However, the basal to mid inferolateral and anterolateral segments of the left ventricle were thickened significantly when compared to other myocardial walls (20mm compared to 8mm anterior wall; Figure 2) with pericardial effusion along the left ventricular wall. The exact cause of myocardial wall thickening was not clear and led to request for in-patient cardiac magnetic resonance imaging.

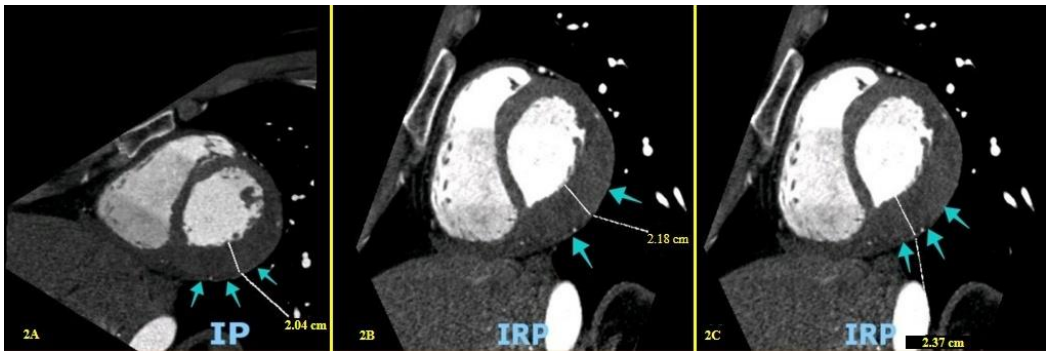

Figure 2 (A, B and C): CT coronary angiogram; Short axis demonstrating basal to mid inferolateral and anterolateral segment thickening.

Initial high sensitivity troponin (hsTnT, elecsys Roche) was 500 nanograms/l (upper reference limit 14ng/L), with a subsequent rise to 1200ng/L. Initial CRP (c-reactive protein) was 6 rising to 226 mg/L. Based on elevated CRP and troponin, pericardial effusion, ECG changes and pleuritic chest pain a diagnosis of myopericarditis was made.

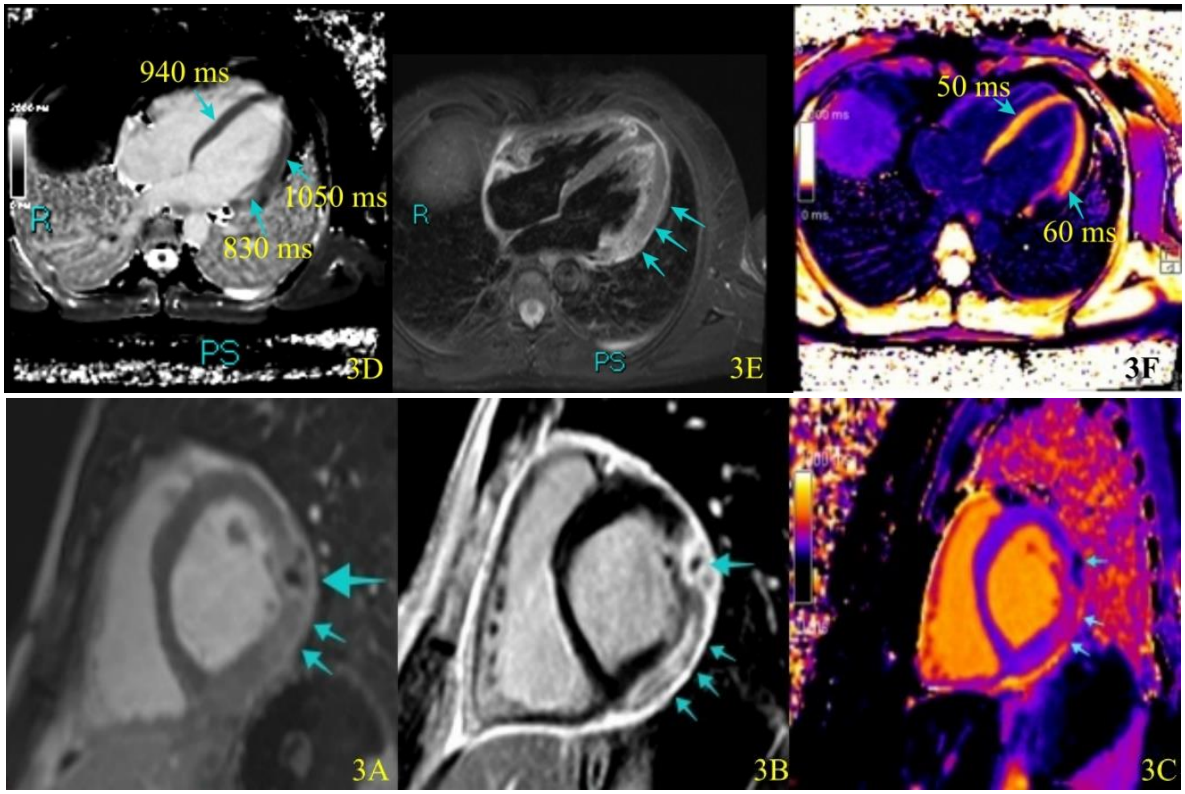

Figure 3: CMR in the acute phase;
(A) Early gadolinium enhancement of short axis left ventricle demonstrating circular low signal legion with surrounding area of increased signal intensity in the inferolateral and antero-lateral wall.
(B) Late gadolinium enhancement of short axis cut of left ventricle with hyperenhancement in inferior, inferolateral and antero-lateral wall surrounding low signal circular lesion.
(C) T1 map of left ventricular short axis.
(D) 4-chamber view of left ventricle demonstrating low T1 values in low intensity lesion (830ms; arrow) characteristic of intramyocardial haemorrhage, with surrounding myocardial oedema (1050ms) compared to normal myocardium (interventricular septum 940ms).
(E) Increased signal on T2 weighted STIR images in keeping with myocardial oedema.
(F) Increased T2 values in the inferolateral and anterolateral wall (60 ms; arrow) compared to septal wall (50 ms; arrow).

The patient was treated with paracetamol, ibuprofen, colchicine, and morphine, for acute myopericarditis associated with persistent chest pain. Subsequently he developed clinical signs of impending tamponade including hypotension and tachycardia (he was not treated with anticoagulants nor antiplatelets). A repeat echocardiogram demonstrated significant increase in pericardial effusion. Given a rapid increase in pericardial effusion and hypotension, an emergency pericardiocentesis was undertaken with extraction of 465ms dark blood-stained fluid. Fluid analysis confirmed it to be an exudate, rich with leucocytes and demonstrated no growth after 48 hours of incubation. Cardiac magnetic resonance imaging, undertaken following a period of clinical stability (Figure 3), revealed evidence of oedema and intramyocardial bleed in the basal and mid lateral walls (with a characteristic hypointense core on T1 mapping) (Figure 3A, C). T2 values were increased in this region (60 ms vs 50 ms in interventricular septum) and there was increased signal on T2-weighted STIR images, in keeping with oedema (Figure 3F). In addition, T1 values were reduced on the basal, antero-lateral and

infero-lateral wall (compared to the unaffected interventricular septum) consistent with an intramyocardial bleed (Figure 3D). The T1 values surrounding the hypo-intense core were higher than other myocardial segments (1050 ms vs 940 ms in the septum) (Figure 3D). This is in keeping with myocardial oedema surrounding an area of myocardial bleed. Early enhancement revealed an hypointense core in this region and late enhancement images are in keeping with myocardial oedema and intramyocardial hemorrhage. The pericardium was of normal thickness, there was adhesion between the pericardium and the epicardium in the basal lateral wall. Rheumatology input was requested. Subsequent serological and immunological tests for connective tissue diseases and viral infections (HIV, CMV) respectively were all negative.

The patient's subsequent course was characterized with repeated episodes of chest discomfort, leading to hospital admissions. There was persistent hypokinesia of the anterolateral and basal inferolateral wall.

Repeat cardiac MRI, one year after his initial presentation, revealed severe localized hypokinesia at the previous site of the IMH with subepicardial and mid-wall enhancement in the basal lateral wall with a small area of near transmural hyperenhancement of the basal anterolateral wall (Figure 4). A diffuse pericardial enhancement of the pericardium at the basal and mid-LV level was also present suggesting pericardial fibrosis.

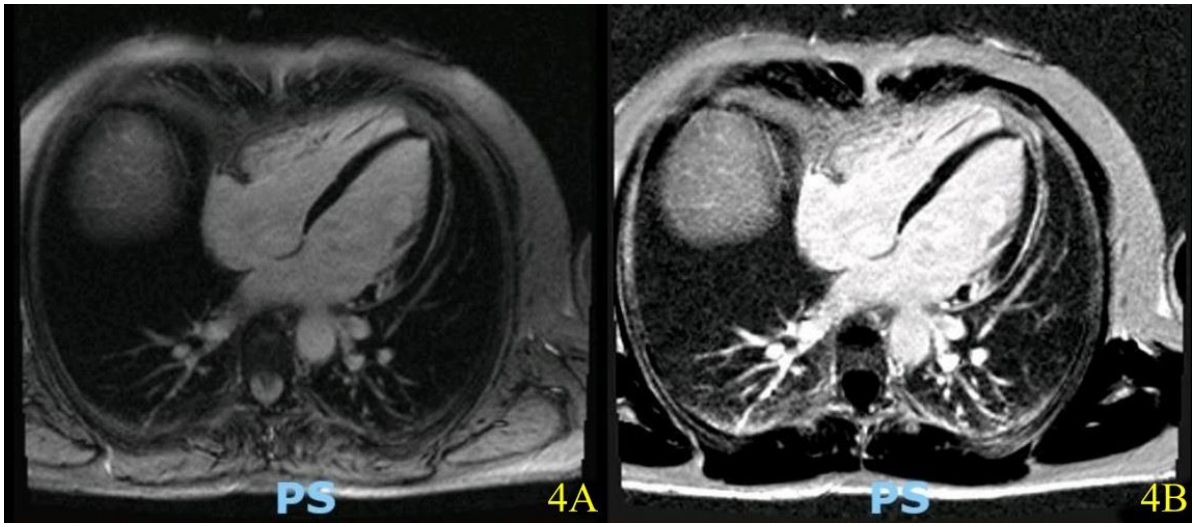

Figure 4: Follow-up CMR;
Repeat cardiac MRI at 1 year: late gadolinium enhancement 4-chamber view revealing diffuse enhancement of the pericardium at the basal and mid-level suggesting pericardial fibrosis.

DISCUSSION

Intramyocardial bleed leads to an increased deposition of hemoglobin related iron in the myocardial tissue. Increased iron levels lead to magnetic field perturbances which can be readily detected by CMR. Although, T2 * imaging is considered the gold standard for iron deposition assessment and for IMH imaging, the T1 mapping was in fact shown to be superior in certain 'iron overload' conditions. An area of microvascular obstruction without a bleed would be expected to have higher native T1 mapping values [8]. In contrast, hypointense myocardial region with very low T1 values, compared with T1 values in surrounding myocardium, is consistent with a myocardial bleed.

To the best of our knowledge, there is no previous description of intramyocardial hemorrhage in acute myopericarditis in the absence of concomitant pathology. Only two cases of intramyocardial bleeding were reported in the literature due to vascular Ehlers-Danlos syndrome [6] and thrombotic thrombocytopenic purpura [5]. Intramyocardial haemorrhage in the context of acute myocardial infarction is widely reported. It is associated with an adverse

prognosis and adverse ventricular remodeling [7]. The presence of IMH should alert the physician to potentially more serious and lasting adverse effects on LV function in acute myocarditis [7]. The mechanism of intramyocardial hemorrhage, in acute myocardial infarction, is postulated to be thrombotic occlusion and reperfusion injury after thrombolysis or mechanical reperfusion [7]. However, the pathophysiology of intramyocardial hemorrhage in acute myopericarditis is unknown. It is hypothesized that a similar pathophysiology occurs in myopericarditis as to acute myocardial infarction. The presence of extensive oedema and ongoing inflammation is likely to lead to injury of the microcirculation with the possibility of intramyocardial bleed in severe cases. This proposed mechanism is a reasonable basis for further research and exploration.

CONCLUSION

IMH in acute myopericarditis has not been previously described in the absence of connective tissue disease or haematological disorders. The pathophysiology in this context remains uncertain but it is associated with adverse left ventricular remodelling. Recognition of IMH in myopericarditis is important and further exploration of pathophysiology and outcomes is warranted.

CONFLICT OF INTEREST

The Authors declare that there is no conflict of interests regarding the publication of this paper.

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Consent to publish the case was obtained from the patient.

AUTHOR'S CONTRIBUTIONS

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GROUP 1: Conception of the work **AND/OR** Design of the work **AND/OR** Acquisition of data **AND/OR** Analysis of data **AND/OR** Interpretation of data

GROUP 2: Drafting the work **AND/OR** Revising the work critically for important intellectual content

GROUP 4: Agree to be accountable for all aspects of the work in ensuring that questions related to the accuracy or integrity of any part of the work are appropriately investigated and resolved

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GROUP 3: Final approval of the version to be published

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