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Case Report

A rare outcome of myocardial infarction by the development of peri-infarction pericarditis: a case report

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ABSTRACT

Peri-infarction pericarditis is a rare complication of myocardial infarction (MI), which can mimic the symptoms of recurrent infarction. In this report, we present the case of a 52-year-old male admitted to the emergency department (ED) with the symptoms of chest pain and hypertension. The patient presented with 10 hours of worsening chest pain. Upon admission to ED, his blood pressure was uncontrolled (165/105). The patient had a normal physical examination. Chest X-ray and computed tomographic angiography (CTA) results were non-significant. An electrocardiogram (ECG) showed sinus rhythm and ST elevation in lead II, III, and aVF, indicating acute inferior wall ST-elevation MI (STEMI). An emergency coronary angiogram procedure was performed and the patient was found to have 95% stenosis of the mid portion of the right coronary artery with a thrombus. An intracoronary stent was placed at the location without complications. Subsequently, the patient continued to report 10/10 chest pain. Another emergency coronary angiography procedure was performed and a patent right coronary artery stent was noted. At 36 hours after the procedure, the patient continued to have symptoms, therefore another ECG was performed. A new anterior injury pattern was noted, suggesting acute MI/STEMI and the patient was taken to a third coronary angiography procedure. No new findings were noted with widely patent stent and no evidence for distal embolization. After reviewing all findings, a diagnosis of post-MI pericarditis was made. The patient was started on colchicine and was discharged the following day. On subsequent follow-up, his symptoms improved.

Keywords: Complications of myocardial infarction, Peri-infarction pericarditis, STEMI

INTRODUCTION

Pericarditis refers to inflammation of the pericardium. There are a number of etiologies that can lead to the development of pericarditis including systemic disease (e.g., Systemic lupus erythematosus), chronic kidney disease, infections (e.g., tuberculosis), HIV/AIDs, COVID-19 infection etc. Post-cardiac injury syndrome (PCIS) is one of the most common causes of pericarditis among all the etiologies.[1] PCIS refers to a group of autoimmune-mediated conditions of pericardial, epicardial, and myocardial inflammation.1 PCIS is further divided into post-myocardial infarction pericarditis, post-pericardiotomy syndrome (PPS), and post-traumatic pericarditis.[1] In this case report, we will focus on post-myocardial infarction pericarditis; specifically early infarction-associated pericarditis, referred to as peri-infarction pericarditis. Peri-infarction pericarditis typically presents within 5 days after MI with an incidence rate of less than 5% in developed countries with the introduction of reperfusion therapy.[1] Dressler's syndrome which is also a rare complication of MI refers to pericarditis caused by an immune response to myocardial necrosis distinguished from peri-infarction

pericarditis by late onset (typically develops 2-8 weeks after MI), with very rare occurrence in modern clinical settings due to the early reperfusion intervention.[1]

Aims and objectives

Despite the scarce occurrence of peri-infarction pericarditis and/or Dressler syndrome, it is necessary for clinicians to be aware of the possibility of developing peri-infarction pericarditis/Dressler syndrome in patients who recently had MI to prevent progression to fatal complications including cardiac tamponade and sudden death (Figure 1) and treat the condition medically once the diagnosis is made.[2]

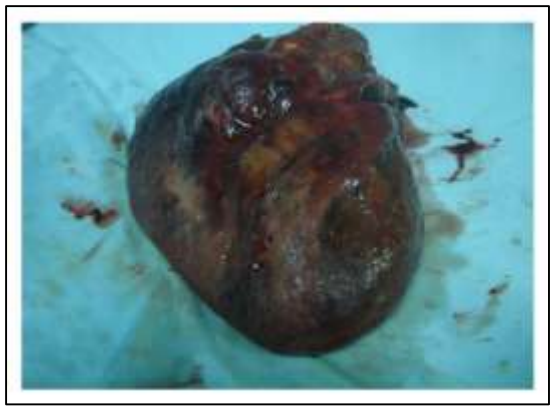

Figure 1: Macroscopic view of the heart from autopsy of a patient with Dressler syndrome.[3]

CASE REPORT

A 52-year-old male with a past medical history of diabetes, hyperlipidemia, and hypertension presented to ED with chest pain which started from the previous night. The patient stated that he went to sleep with chest pain and woke up with worsening pain. The patient reported that he had been non-compliant with his medications. On the route to ED, the patient was given 4 tablets of aspirin 81 mg by emergency medical service and his blood pressure was 165/105 with a heart rate of 94 upon arrival. A chest X-ray was ordered and the result was non-significant. The labs were ordered and the patient had a slight elevated WBC level. The high sensitivity troponin level came back 2700. ECG was completed and showed ST elevation in lead II, lead III, and aVF, indicating acute inferior wall STEMI (Figure 2).

Considering the patient's hypertension, aortic dissection needed to be ruled out first before proceeding with cardiac catheterization. CTA chest was ordered and the result was negative for aortic dissection. Physical examination was normal. The patient was taken for emergency coronary angiogram. A thrombotic 95% area of stenosis in the mid portion of a right coronary artery (RCA) was noted. The left anterior descending artery (LAD) and its branches, the left main (LM) coronary artery, and the circumflex coronary artery (LCx) were normal. A run-through wire was used to cross the occlusion in the RCA and then direct stenting was performed with a 4.5×20 mm boson scientific drug-eluting stent with several inflations up to 18 atmospheres, reducing the lesion to 0%. Thrombolysis in myocardial infarction (TIMI) flow 3 was achieved. A transthoracic echocardiogram (TTE) revealed normal systolic function with a normal ejection fraction (55-60%) and a normal left ventricular diastolic function.

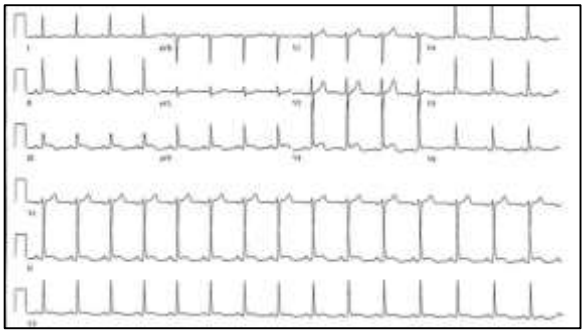

Figure 2: Inferior wall STEMI shown in ECG.

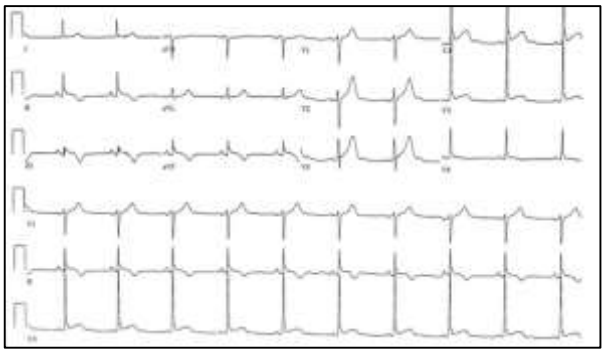

Figure 3: Anterior ST-elevation in ECG.

After the procedure, the patient was hemodynamically stable and transferred to the intensive care unit (ICU). The patient's hospital course included dual anti-platelet therapy (aspirin 81 mg, daily and ticagrelor 90 mg, q12 hours), beta-blocker (carvedilol 12.5 mg, BID), high dose statin (atorvastatin 80 mg, nightly), and angiotensin-receptor blocker (losartan 100 mg, daily). Heparin 5,000 units sub-Q was provided as well for DVT prophylactic purposes. Smoking cessation and abstinence from drug use (the patient reported that he occasionally consumed edible marijuana) were suggested. Approximately 8 hours after the catheterization procedure, the patient developed chest pain (severity: 10 out of 10) which was un-relieving. Heart rate and blood pressure were stable. The patient was transferred to a catheterization lab for an emergent right coronary angiography procedure. A widely patent RCA stent and distal vessel were noted. Nitroglycerin drip was continued overnight. The patient was stable the following day. Nitroglycerin drip was discontinued and oral isosorbide mononitrate 30 mg, daily was added to his regimen. ECG was done and revealed normal sinus rhythm and minimal voltage

criteria for left ventricular hypertrophy (LVH). It also showed anterior ST elevation, suggesting early repolarization, pericarditis, or injury along with T wave abnormality (Figure 3).

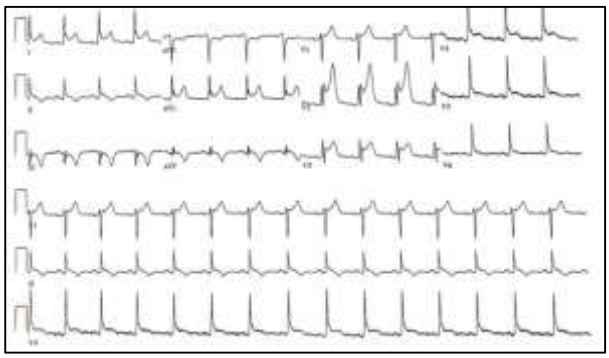

Figure 4: A new acute anterior infarct in ECG.

The following day, the patient complained of severe chest pain (10 out of 10 in severity) and another ECG was done. The result showed ST elevations in the anterolateral leads, suggesting a new acute anterior infarct/acute MI/STEMI (Figure 4). The patient was transferred to the Cath lab for a third coronary angiogram procedure and was found to have a patent RCA stent and normal LAD, LM, and LCx coronary arteries with TIMI 3 flow. There was no evidence of distal embolization. The ECG and procedure findings were consistent with pericarditis. A bedside echocardiogram was performed which demonstrated preserved left ventricular systolic function and no significant pericardial effusion.

The patient was started on colchicine 0.6 mg PO, BID with a diagnosis of post MI pericarditis. The patient was hemodynamically stable after the procedure and discharged the following day with the following medications: Aspirin 81 mg daily, atorvastatin 80 mg nightly, carvedilol 6.25 mg BID, isosorbide mononitrate 30 mg daily, losartan 25 mg daily, and ticagrelor 90 mg q12 hours.

The importance of medication compliance with dual platelet therapy was explained and the patient was instructed to follow up with a cardiologist after discharge. On subsequent office evaluation, his symptoms had improved. The table below is the summary of hospital course over the 3 days of the patient's hospital admission (Table 1).

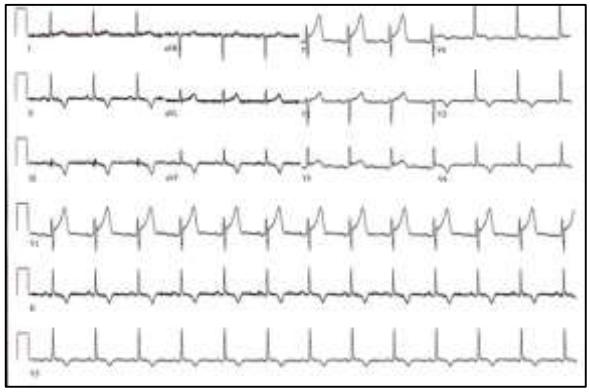

Figure 5: Improved ST elevation in the anterior and anterolateral leads.

The patient was admitted about a month after the discharge for a non-cardiac complaint and an ECG at that time showed improvement of the ST elevation (shown in Figure 5).

Table 1: Summary of the patient's hospital course.

	Admission day 1 (morning)	Admission day 1 (Night)	Admission day 3
Presenting symptoms	10/10 chest pain	10/10 chest pain 8 hours after a stent placement	Continuing chest pain 36 hours after the second coronary angiogram
ECG findings	ST elevation in lead II, III, and aVF	No acute changes	ST elevations in the anterolateral leads
Pre-op diagnosis	Inferior STEMI	Ongoing Chest pain	Anterior STEMI
Findings during a coronary angiogram	95% stenosis of the mid portion of the RCA with a thrombus	A widely patent RCA stent and distal vessel	A patent stent in RCA and normal LAD, LM, and LCx coronary arteries
Intervention	An intracoronary stent placement in RCA	No intervention needed	No intervention needed
Post-op diagnosis	RCA occlusion	A patent RCA stent and no acute findings	Peri-infarction pericarditis

DISCUSSION

Pericarditis is an inflammatory disorder of pericardium that can be caused by infectious or non-infectious etiology and generally is a self-limited condition.[4] Peri-infarction pericarditis (PIP) is one of the subsets of pericarditis which is a rare but potentially serious complication of MI with the characteristic of early onset which typically presents within 5 days after the occurrence of MI.[1]

Diagnosing pericarditis heavily depends on the clinical presentation of pleuritic chest pain, pericardial friction

rubs on auscultation, and the results of ECG and echocardiogram.[5,6] However, diagnosing PIP is challenging due to overlapping features between the electrocardiogram of pericarditis and that of MI.[7] Widespread and diffuse ST-segment elevation shown in PIP is similar to ST-segment elevation featured in STEMI. Acute pericarditis may present with pleuritic chest pain, pericardial effusion, pleural effusion, pericardial friction rubs, and less commonly with vague, non-specific features including malaise, diarrhea, dyspnea, and fever.[4,8,9]

The most common complication of pericarditis by the literature is indicated as recurrence.[4] Constrictive pericarditis and cardiac tamponade leading to sudden cardiac death are rare, but most feared complications of untreated severe acute pericarditis.[1,10,11] However, it was found that the development of cardiac tamponade in patients with pericarditis was significantly associated with malignancy ($p<0.05$) related to lymphatic drainage.[4,12]

Therefore, developing cardiac tamponade as a complication of PIP is significantly low and rare. Additionally, pericardial effusions are one of the most frequently appeared complications of PIP which do not alter the overall prognosis but are associated with a higher morbidity and mortality rate mostly because they occur more frequently with larger MIs that tend to be transmural.[13,14] A study conducted by Figueras et al also found out that patients with early small pericardial effusions are more likely to develop late moderate-to-severe effusions and two-thirds of those patients with moderate-to-severe effusions developed tamponade or free wall rupture.

Aspirin and the adjunct use of colchicine is the first choice of treatment for patients already under antiplatelet therapy in patients with PIP and careful monitoring of renal and cardiac monitoring is essential while a patient is on aspirin therapy.[1] Non-steroid anti-inflammatory drugs other than aspirin and glucocorticoids should be avoided for 7 to 10 days after an acute MI because impairment of collagen deposition and scarring can lead to further fatal complications.[15]

CONCLUSION

Peri-infarction pericarditis is one of the potentially fatal complications of MI and is an inflammation of the pericardium which occurs within 5 days of MI onset and has become scarce with the implementation of percutaneous coronary intervention in the modern era. Even though the most common complication of PIP is recurrence, it can lead to feared and rare outcomes including constrictive pericarditis and cardiac tamponade, leading to sudden cardiac death. Despite the challenges which come from the overlapping features of echocardiogram between MI and pericarditis, it is critical for clinicians to be aware of presenting features of PIP and treat patients accordingly to prevent further complications and feared outcomes.

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