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Case Report

Peri-Myocardial Infarction Pericarditis After Single Stent Early PCI on a STEMI Patient: A Rare Case of Outpatient in Urban Setting

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ABSTRACT

Peri-Myocardial Infarction Pericarditis (PMIP), can occur a few days following a myocardial infarction (MI). PMIP frequently exhibits the auscultatory findings of a pericardial friction rub and pericardial effusion. On the other hand, relatively little is known about the management approach. This study aims to report a patient with PMIP following successful revascularization via Percutaneous Coronary Intervention (PCI) for a recent MI. A 50-year-old male was brought to our Outpatient Clinic with chest pain of two days onset. The patient experienced radiating chest pain, cold sweat, heartburn, and general fatigue. ECG shows extensive anterior ST-elevation myocardial infarction (STEMI), and Troponin T was elevated >2000 ng/L. The patient underwent PCI with single stent deployment. Three days later, the patient experienced dyspnea with Transthoracic Echocardiography (TTE) findings consistent with Early PMIP. The patient was discharged after completing full conservative therapy with good results. The clinical findings of PMIP alone may be subtle and go unnoticed. Clinicians should maintain a high level of suspicion in the era of revascularization and develop a strategic plan for timely diagnosis and management. While anti-inflammatory medical therapy is aimed at reducing inflammation and preventing recurrences and progression of the disease, further research is needed to establish prognostic significance and management strategy since clinically diagnosed PMIP has a benign and self-limiting nature.

INTRODUCTION

Peri-Myocardial Infarction Pericarditis (PMIP), has traditionally been thought of as a rather benign illness that develops a few days following a myocardial infarction (MI). The discovery of

pericardial friction rub in a post-MI patient prompts a careful review of post-MI symptoms and test results, including dysrhythmia recordings, electrocardiogram (EKG), and the Transthoracic Echocardiogram (TTE), to rule out potentially fatal post-MI mechanical complications like free wall rupture, even though the condition typically does not require specific treatment[1]. Cardiac and pericardial injury syndromes are often referred to as post-cardiac injury syndrome (PCIS) and are classified into PMIP (Early and Late), post-pericardial syndrome (PPS), and post-traumatic pericarditis. Acute pericarditis can complicate the course of acute myocardial infarction.

Etiologies of postinfarction are classified according to onset, with Early PMIP usually occurring shortly after MI (less than seven days) and is transient and self-limiting, whereas delayed postinfarction pericarditis would be addressed as Late PMIP or also known as Dressler Syndrome (more than seven days after MI). Peri-Myocardial Infarction Pericarditis frequently exhibits the auscultatory findings of a pericardial friction rub and pericardial effusion. In MI studies without reperfusion, 68% of pericardial friction rubs were heard on day one or two, of which 85% were heard for less than three days, and pericardial friction rub was rarely persistent. On the other hand, relatively little is known about the management approach[2,3]. Peri-Myocardial Infarction Pericarditis was assumed to occur in <5% of post-acute MI patients in the current revascularization era, and recent studies suggest an even lower incidence of less than 1% for Dressler Syndrome[4,5]. This study aims to report a patient with PMIP following successful revascularization via Percutaneous Coronary Intervention (PCI) for a recent MI.

CASE PRESENTATION

A 50-year-old male brought over by family members had visited our Outpatient Clinic with recent chest pain of two days onset. The patient experienced chest pain, cold sweat, heartburn, and general fatigue. The chest pain was described as feeling pressure from the front of the chest and spreading to the back as well as the left arm, the patient felt his complaints worsen accompanied by dyspnea for up to two days despite taking pain relievers and decided to be checked further.

The patient was transferred to the emergency ward. Physical examination stated blood pressure was 132/80 mmHg, heart rate of 82 bpm, and respiration rate of 19x/min. The patient's other physical examination was normal. Chest X-ray did not show any abnormalities. Blood examination results showed a marked increase in Troponin T of >2000 ng/L and leukocytosis of 10.700/µL. He didn't have any underlying diseases such as diabetes, dyslipidemia, high uric acid, and kidney disease. The patient was an active smoker who smokes three packs daily. The electrocardiogram (EKG) results in Figure 1 showed sinus rhythm, right axis deviation, and

right bundle branch block, with >1 mm ST elevation in leads V2–V6, I, and aVL. The patient was diagnosed with Extensive Anterior ST-elevation Myocardial Infarction (STEMI).

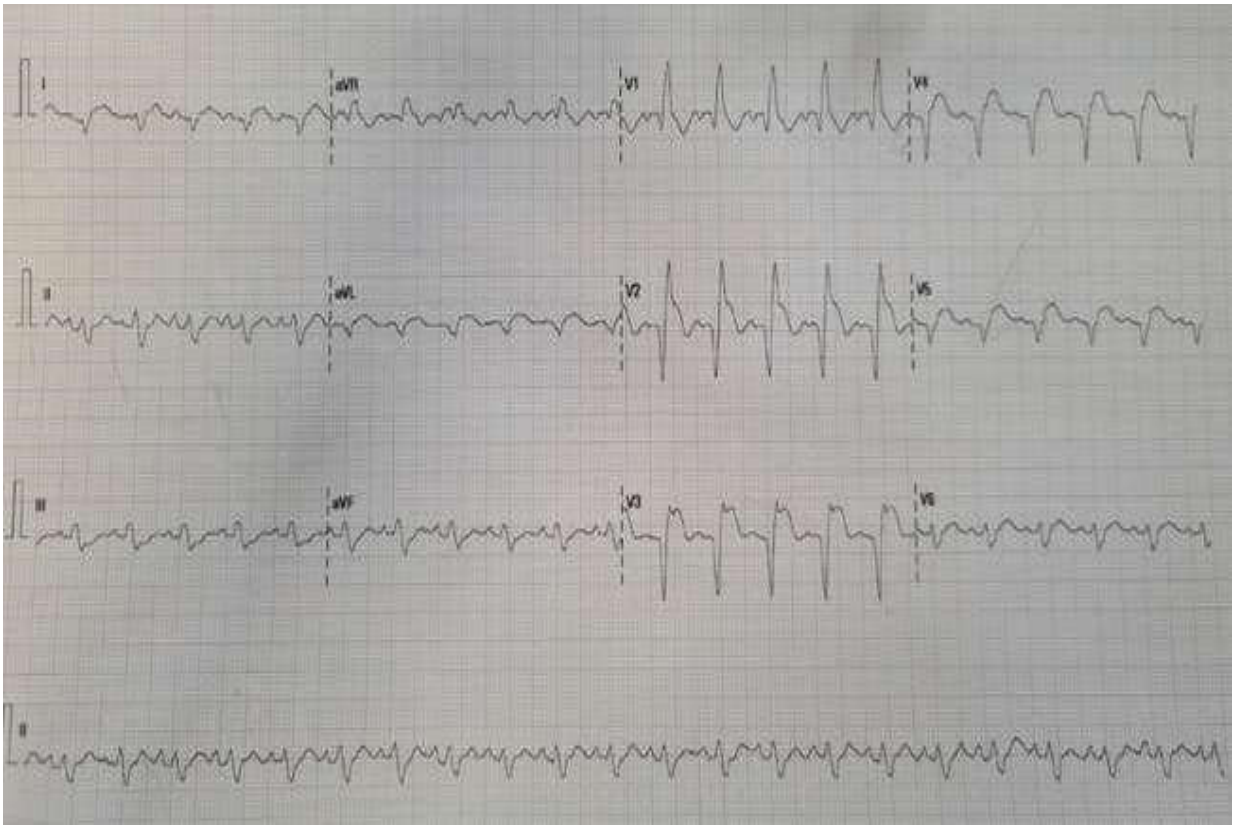

Figure 1. Results of EKG before PCI showed sinus rhythm, right axis deviation, and right bundle branch block, with >1 mm ST elevation in leads V2–V6, I, and aVL.

The patient was initially treated with Aspirin 1x80mg, Clopidogrel 1x80mg, and Isosorbide dinitrate 3x5mg. Treatment with early percutaneous coronary intervention (PCI) was soon carried out because the Cardiologist and Team evaluated that the outcome would worsen over time owing to the 90% stenosis of MID LAD, shown by coronary angiography results before PCI in Figure 2(A). The revascularization process via PCI with the deployment of a single stent in LAD was successful, with angiography results post PCI shown in figure 2(B). The patient was transferred to be monitored in the Intensive Coronary Care Unit (ICCU) and given Aspirin 1x80mg, Ticagrelor 2x90mg, and Nitroglycerin 2x2,5mg for his medications.

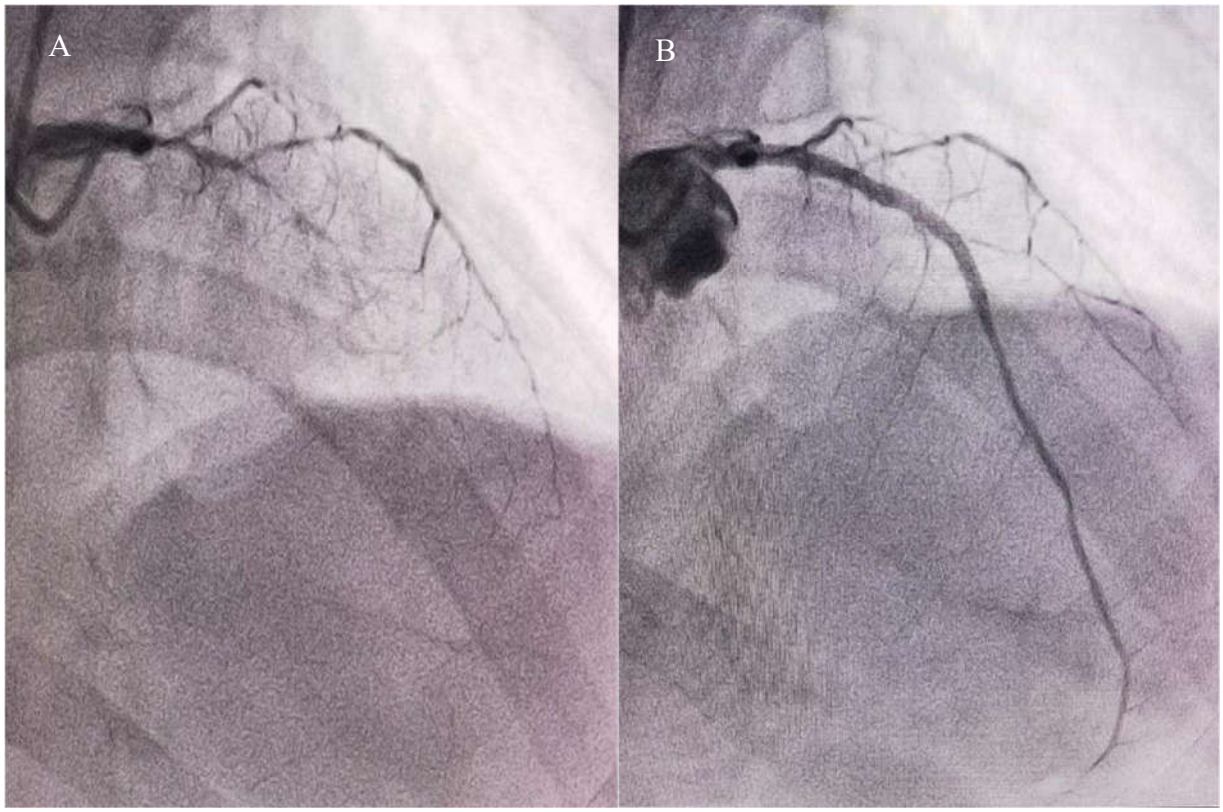

Figure 2. (A) Results of Coronary Angiography before PCI showed 90% stenosis in MID LAD. (B) Results of Coronary Angiography after successful revascularization via PCI, with the deployment of a single stent in LAD.

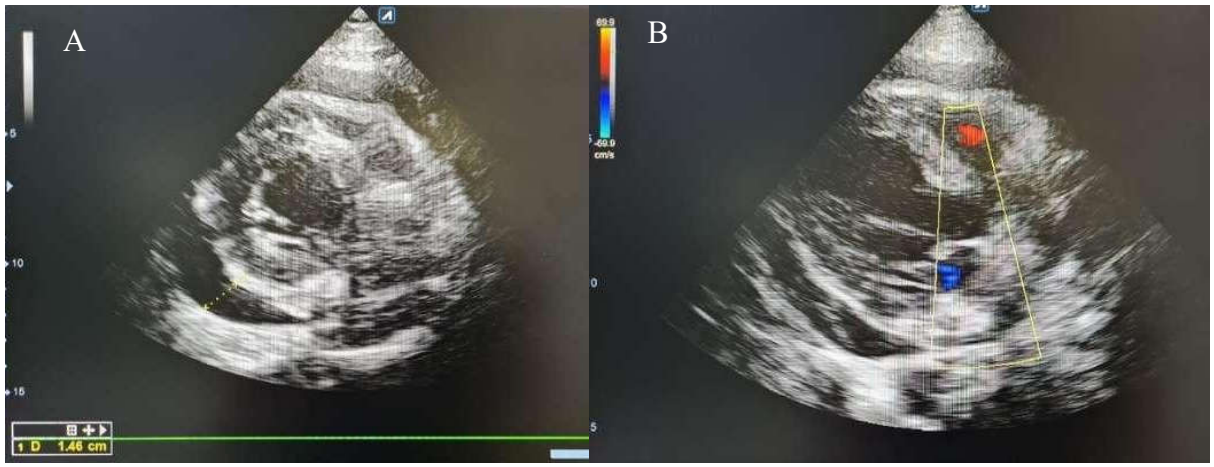

Figure 3. Results of Transthoracic Echocardiogram (TTE) 3 days after PCI. Transthoracic Echocardiography results showed normal left ventricular ejection fraction, no regional wall motion abnormalities, moderate pericardial effusion, and thickening of the pericardium. (A). Short Axis View. (B) Parasternal long axis view.

However, three days post PCI the patient had been experiencing dyspnea that worsened over time, accompanied by pericardial friction rub findings through auscultation. Then we did a Transthoracic Echocardiography exam, the result shown in Figures 3(A) and 3(B). Transthoracic Echocardiogram results showed normal left ventricular ejection fraction, no regional wall motion abnormalities, moderate pericardial effusion, and thickening of the pericardium. These findings

were expected, and symptoms were consistent with a diagnosis of Early PMIP. The Cardiologist and team agreed to proceed towards full conservative therapy with close monitoring over the continuing days, therefore we added high dose Colchicine 2x2,5mg and Ibuprofen 3x400mg to the therapy. After two days of full conservative therapy, the symptoms showed marked improvement and the patient was hemodynamically stable. The patient had clinically improved at day six post PCI and was allowed to be discharged and prescribed to continue Colchicine with a tapered dose expected to follow up after one month.

DISCUSSION

The normal pericardium is composed of two layers. It is a specialized layer that covers the surface of the heart chamber with a fibrous wall-side layer and visceral layer. The parietal layer together with the visceral layer forms a closed sac around the heart. The parietal pericardium attaches to the sternum and diaphragm. The pericardial sac usually has a small amount of serous fluid (less than 25-50 mL). Regardless of the etiology, damage to the pericardium generally results in a non-specific response with the formation of fluid and inflammatory cells with the formation of fibrous adhesions during the recovery phase[1,6].

The pathogenesis of pericarditis after acute MI is ascribed to the release of cardiac antigens during myocardial ischemia, which results in a hypersensitive immunological response in genetically susceptible people. Through molecular mimicry, these antigens activate complement. Following a period of dormancy, the immune system attacks the pericardium by activating an inflammatory cascade, resulting in the creation of autoantibodies and gene and/or microRNA expression. Anti-heart antibody levels have been found to correlate with post-cardiac injury disease activity, lending credence to these views[7,8].

An inflamed pericardium has a dry, rough surface, as well as the development of yellow to brown fluid comprising leukocytes, red blood cells, and fibrin. Microscopically, neovascularization and fibroblast proliferation may occur, leading to fibrosis. Delay in reperfusion, higher cardiac biomarkers, bigger infarction size, younger age, anterior site of ischemia, inferior infarcts accompanied by right ventricular involvement, and, more commonly, lower left ventricular ejection fraction are risk factors for the development of PMIP[2,8].

Post-cardiac injury syndrome is diagnosed when at least two of the five following criteria are satisfied, according to the European Society of Cardiology diagnostic criteria for PCIS: 1) Fever without a plausible alternative, 2) pericarditis or pleuritic chest pain, 3) pericardial or pleural friction rub, 4) pericardial effusion 5) pleural effusion with high C-reactive protein. Whatever the

cause, the usual EKG findings of PCIS include extensive ST-segment elevation, typically sparing the aVR and V1, as well as PR segment depression. These EKG alterations, however, are dynamic, changing over time and eventually would be normalizing[9].

Table 1. Diagnostic criteria for post-cardiac injury syndrome (PCIS) - At least two of the five criteria should be fulfilled. European Heart Journal (2015).

Diagnostic criteria for post-cardiac injury syndrome (PCIS)
1. Fever without alternative causes.
2. Pericarditis or pleuritic chest pain.
3. Pericardial or pleural rubs.
4. Evidence of pericardiaI effusion.
5. Pleural effusion with elevated CRP.

The majority of patients report severe, pleuritic chest pain that is centrally situated in>80% of cases, a low-grade fever >50-60% of cases, and dyspnea in 50-60% of cases. Leukocytosis, high inflammatory markers such as erythrocyte sedimentation rate and C-reactive protein >80% of cases, the appearance of a pericardial effusion >80% of cases, new pleural effusion with or without pulmonary infiltrates >60% of cases, and signs of a pericardial or pleural rub 30-60% of cases, and an increase of Troponin would indicate a myocardial involvement[9,10].

Within this case we observed at least two of the diagnostic criteria (Pericardial friction rub + pericardial effusion with pericardial thickening in TTE) with an onset of <7 days post-MI, therefore clinically diagnosing the patient with Early PMIP is accepted. Additionally, our lab results showed a marked increase in Troponin and leukocyte count. This was also the case in one recent study by Massalha et al., where they showed that PMIP individuals usually also had considerably greater levels of inflammation and biomarkers of myocardial damage (leukocyte count, Troponin, CPK, and CRP)[4,11].

The typical approach to patients with PMIP, whether identified clinically or by advanced cardiac imaging like cardiac MRI, is supportive, with no specific therapy suggested because symptoms and signs usually recover without intervention. Acetaminophen is still the favored analgesic for people with severe symptoms, with high-dose aspirin coming in second. Early and late-onset PMIP is mainly treated with supportive care, which includes nonsteroidal anti-inflammatory medications (NSAIDs), Colchicine, and perhaps Corticosteroids as last resort. Except for Indomethacin which should be avoided, as it decreases coronary blood flow[1,12].

Acetaminophen is beneficial to take 650 mg every six to eight hours for up to 10 days. If symptoms persist, aspirin 650 up to 800 mg every six to eight hours can be started, with reduction starting after symptoms resolve, generally after seven to 10 days. Because of the patient's need for

antiplatelet treatment, aspirin should be taken first in patients with PMIP, and because the anti-inflammatory effects of other NSAIDs may interfere with myocardial repair and scar formation[13,14].

Ibuprofen, which has been demonstrated to enhance coronary flow, can be taken at a dosage of 600 to 800 mg every six to eight hours, with the total daily dose declining by 400 to 800 mg per week for around three to four weeks. Colchicine has been demonstrated to be beneficial in reducing discomfort in patients with acute pericarditis and avoiding recurrences, and it may help prevent PMIP (0.5 mg BID for patients >70 kg or 0.5mg/day if <70kg for four to six weeks). Prednisone 0.2-0.5 mg/kg/day for four weeks followed by a taper has been demonstrated to be beneficial in treating individuals for whom aspirin or NSAID use is contraindicated as well as those who have not responded to more conventional treatment[1,12]. In our case patient has responded well with Ibuprofen, and a high dose of Colchicine of 2.5mg BID which we then continued with a tapered dose at discharge.

In most cases, PMIP patients do not require inpatient hospitalization, but those with high-risk traits should be sent to the hospital. Leukocytosis, fever, immunosuppression, subacute course, concomitant oral anticoagulant usage, high troponin levels, massive effusion or tamponade, and aspirin or NSAID failure are some of the conditions that fall under this category[1,12]. In the recent study by Massalha et al., the presence of a pericardial involvement might be a good prognostic value in ST-segment elevation myocardial infarction patients. In their study, the incidence of PMIP even supports a decrease in the major adverse cardiac events (MACE) rate as well as hazard ratio on follow-up[4]. However, although the majority of patients with PMIP have a good prognosis there is still a 10-15% recurrence rate[12].

CONCLUSION

The clinical findings of PMIP alone may be subtle and go unnoticed. It could be challenging to differentiate and treat pericarditis from other clinical conditions in the post-MI setting. Detailed evaluation with clinical history, EKG, biomarkers, and echocardiography may provide the diagnosis. Clinicians should maintain a high suspicion in the era of revascularization and develop a strategic plan for timely diagnosis and management. While anti-inflammatory medical therapy is aimed at reducing inflammation and preventing recurrences and progression of the disease, further research is needed to establish prognostic significance and management strategy since clinically diagnosed PMIP has a benign and self-limiting nature.

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