Acute Pericarditis After Percutaneous Coronary Intervention Mimicking Inferolateral ST-Elevation Myocardial Infarction

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Abstract: Acute pericarditis is a rare complication of percutaneous coronary intervention (PCI). Here, we describe a case of PCI complicated by guidewire perforation and contrast extravasation. Acute pericarditis developed 36 hours after PCI procedure with fever and severe chest pain. Electrocardiogram showed ST elevation in inferior-lateral leads. However, the follow-up coronary angiography showed negative result and the symptom improved dramatically with the treatment of nonsteroidal anti-inflammatory drug treatment. Therefore, it is important for the clinician to differentiate acute myocardial infarction/acute stent thrombosis from this rare complication after PCI.

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Case report
An 86-year-old woman presented to the hospital emergency department with a 9-day history of progressively worsening exertional chest tightness and mild dyspnea. She had past history of hypertension but denied history of diabetes mellitus, hyperlipidemia, and smoking. In the emergency department, her initial electrocardiogram (ECG) showed T-wave inversion in leads V1 to V3 and borderline ST elevation in lead II, III, and aVF (Figure 1A). The physical examination was unremarkable. Complete blood count, electrolyte, and renal function were also within normal limits. Cardiac enzymes were not elevated, with a troponin-I of 0 ng/mL, CK of 35 U/L, and CK-MB of 3 U/L. She underwent cardiac catheterization 2 days later for unstable angina. Left ventriculogram showed mildly depressed left ventricular function with inferior wall hypokinesis. Coronary angiography revealed double-vessel disease with a complex severe stenosis at the bifurcation of the left anterior descending artery (LAD) and second diagonal branch (D2) (Figure 2A), severe stenosis in the distal LAD, and borderline severe stenosis in the middle left circumflex artery. The right coronary artery (RCA) appeared normal. Her culprit lesion was identified in the proximal LAD and PCI of the LAD bifurcation lesion was performed. Procedural anticoagulation was accomplished with a bolus of 8000 units of heparin. A Fielder FC 0.014” floppy guidewire (Asahi Intecc) was advanced without difficulty to the distal part of the LAD; after several unsuccessful attempts with a Runthrough guidewire (Terumo Medical), we were able to cross the tortuous segment containing the D2 branch lesion using a Pilot 200 guidewire (Abbott Vascular). Drug-eluting stent (DES) placement was performed from the proximal to the distal part of the LAD (Xience Prime; Abbott Vascular): 3.0 x 28 mm, 2.75 x 18 mm, and 2.25 x 23 mm, respectively and balloon angioplasty was used to treat the ostium of the D2 (2.5 x 15 mm Apex balloon catheter [Boston Scientific Corporation]). However, contrast extravasation was observed in the distal part of the D2, suggesting a guidewire perforation (Figure 2B). The patient remained asymptomatic and hemodynamically stable. Nevertheless, an urgent echocardiogram was performed and showed no evidence of pericardial effusion. Therefore, the heparin was not reversed with protamine. Final angiography revealed TIMI 3 flow through the LAD and D2 branch with spontaneous resolution of contrast
extravasation. The patient was transferred back to the coronary intensive care unit for close observation. Thirty-six hours after the procedure, the patient complained of severe chest pain and mild dyspnea. She developed a low-grade fever (38°C) and a pericardial friction rub was noted on auscultation. Laboratory data revealed leucocytosis (14 x 10^9/L) and an elevated C-reactive protein level (40.6 mg/dL), although cardiac biomarkers remained within normal limits (troponin-I of 0 ng/mL, CK of 24 U/L, CK-MB of 3 U/L). Furthermore, her ECG showed ST-elevation in inferior leads, ST-depressions in V1-2 and elevations in V4-V6 (Figure 1B), suggesting acute ST-elevation myocardial infarction of the inferior-lateral walls with extension to the posterior wall. Emergent coronary angiography revealed no evidence of coronary artery occlusion and no evidence of contrast extravasation in the distal D2 (Figure 3). Repeat echocardiogram demonstrated a small pericardial effusion localized to the posterior-lateral compartment (Figure 4). Non-steroidal anti-inflammatory drugs (NSAIDs) were started because of suspected acute pericarditis and chest pain improved significantly by the second day of treatment. Blood cultures were sterile and serum cardiac biomarkers remained within normal limits during the follow-up. On the fifth day after the procedure, the ECG showed virtual complete resolution of the ST-segment elevation without pathologic Q-wave (Figure 1C). The patient was discharged 6 days later with routine follow-up scheduled.

Discussion

Postcardiac injury syndrome (PCIS), also known as postpericardiotomy syndrome and Dressler’s syndrome, commonly occurs after cardiac surgery or following myocardial infarction, but has also been reported following temporary/permanent pacemaker implantation and radiofrequency ablation, and, as reported here and by others, can be a rare complication following PCI. According to Troughton et al, the incidence of pericardial complications after PCI is less than 0.2%. The clinical features of PCIS include fever, dyspnea, pleuritic chest pain, pericardial friction rub, pericardial effusion, leukocytosis, and elevated CRP level, congruent with our patient’s clinical presentation. Although the pathogenesis of PCIS following PCI remains unclear, immune-mediated mechanisms involving anti-heart autoantibodies as well as irritable blood accumulating in the pericardial space inciting pericardial injury have both been proposed as possible explanations. Escaned et al reported PCIS in a patient the day following coronary perforation during balloon angioplasty and Huang et al reported of acute pericarditis following coronary artery dissection during PCI. These authors suggested that coronary dissection/perforation and subsequent pericardial hematoma formation might be the cause of the acute pericarditis inciting PCIS. In the present case, coronary perforation by guidewire tip during PCI procedure likely caused injury to the local pericardium with a resultant focal type of PCIS, as evidenced by the inferior-lateral and posterior injury pattern that developed on her ECG mimicking myocardial infarction. This contrasts with the ECG finding of diffuse ST elevation in previous case reports. Furthermore, the localized nature of our patient’s syndrome argues against the immune-mediated mechanism proposed by others.

Of note, bacteria such as Staphylococcus and Pseudomonas aeruginosa can cause purulent pericarditis after coronary stenting. However, negative blood culture and dramatic improvement of symptoms after treatment with NSAIDs also favor the diagnosis of PCIS rather than purulent pericarditis.

Here, we describe a rare case of PCIS manifesting as acute pericarditis with localized pericardial effusion (mimicking acute myocardial infarction) following epicardial coronary artery perforation during PCI procedure. Although PCIS is a rare complication of PCI, given the similar clinical features, we hope this report helps to underscore the importance of prompt differentiation of PCIS from acute myocardial infarction/stent thrombosis so that appropriate treatment can be instituted.
References

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**Figure 1.** 12-lead electrocardiogram (ECG) of the patient. (A) ECG before percutaneous coronary intervention. (B) ECG after percutaneous coronary intervention. The ECG showed ST-segment elevations in inferior leads and V4-V6 with no evidence of PR segment depression. (C) ECG on the fifth day after the procedure. The ECG showed nearly complete resolution of the ST-segment elevation without pathologic Q-wave.
Figure 2. (A) Pre-intervention image: left coronary angiogram showed a complex severe stenosis at the bifurcation of left anterior descending artery and second diagonal branch (arrow). (B) Post-intervention image: the stent was successfully deployed to the middle left anterior descending artery; note contrast extravasation from the D2 branch (arrow), consistent with a guidewire perforation.

Figure 3. Repeated coronary angiography showed left coronary artery stent was patent without contrast extravasation.
Figure 4. Echocardiogram showed small pericardial effusion with posterior-lateral localization (arrow).