Case Report

Sequential development of cardiac tamponade and subacute stent thrombosis after primary percutaneous coronary intervention for acute ST-segment elevation myocardial infarction: A case report

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Summary A 42-year-old male suffered from ST elevation myocardial infarction and underwent successful percutaneous coronary intervention (PCI) of the left anterior descending coronary artery (LAD) with drug-eluting stent using intravenous glycoprotein IIb/IIIa inhibitor (Clotinab®, Isu Abxis). Five hours after PCI, the patient developed cold sweating and went into a stupor. Urgent 2D-echocardiography showed a large amount of pericardial effusion and akinesia in the LAD territory. A repeat emergent angiography (CAG) was done to ascertain whether acute stent thrombosis or coronary perforation had occurred after PCI. The CAG showed, however, no leakage of dye or thrombus in any coronary arteries with a patent stent in the middle LAD. Approximately 200 cm³ of bloody pericardial effusion was drained, and his blood pressure returned to normal immediately after pericardiocentesis. Seven days later, he again developed sudden hypotension, bradycardia, and loss of consciousness. The ECG showed ST elevation in V1—V6 and 2D-echocardiography showed scanty pericardial effusion. Emergent CAG showed total occlusion of the LAD due to subacute stent thrombosis. He was successfully treated with balloon angioplasty and was discharged with dual anti-platelet therapy. A follow-up CAG after 9 months showed good flow without residual stenosis across the stented segment.

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Introduction

Bleeding and thrombosis are always a dilemma during the treatment of acute coronary syndrome. Especially, in patients with drug-eluting stent implantation, appropriate anti-platelet therapy is important to prevent stent thrombosis, but there is a potential risk for serious hemorrhagic complications. We experienced a case of sequential development of pericardial tamponade and subacute stent thrombosis after primary percutaneous coronary intervention (PCI) for acute ST-segment elevation myocardial infarction.

Case report

A 42-year-old man presented to the Emergency Department with continuous chest pain of 4 h duration. He had no risk factors for coronary artery disease, and he was not on any medication at the time of admission. Upon physical examination his blood pressure was 150/90 mmHg and his heart rate was 104 beats per minute, with regular heartbeat and normal S1 and S2 sounds. On auscultation, his breathing sound was clear. The initial electrocardiogram (ECG) showed ST-segment elevation (STE) in lead V1 through V6 with reciprocal ST-segment depression in leads II, III, and aVF (Fig. 1). The levels of cardiac enzymes were elevated: creatine kinase (CK) 688 U/L (35—172), CK-MB 20.6 U/L (2.3—9.5), and troponin-I 6.18 ng/mL (0—0.05). Conventional unfractionated heparin was administered at an initial dose of 5000 units, followed by continuous infusion of 1000 units per hour, subsequently targeting an activated partial thromboplastin time of 1.5—2.0 times baseline. Additionally, loading doses of aspirin (300 mg) and clopidogrel (300 mg) were given and abciximab, a glycoprotein IIb/IIIa inhibitor (Clotinab®, Isu Abxis) was infused (0.25 mg/kg bolus and 10 µg/min infusion for 12 h). One hour later, emergent coronary angiogram (CAG) revealed thrombotic total occlusion of the middle left anterior descending coronary artery (LAD) with no collaterals from the left circumflex or right coronary artery (Fig. 2A). After wiring into the LAD, balloon angioplasty was performed with a 3.0 mm balloon, followed by deployment of a 3.5 mm × 24 mm zotarolimus-eluting stent (Endeavor®, Medtronic) at 14 atm. But there remained residual thrombi and additional balloon dilation was performed. The final CAG showed thrombolysis in myocardial infarction III antegrade flow but residual stenosis distal to the stent (Fig. 2B). The vital signs were stable during and following the PCI and the patient did not complain of any symptoms such as chest discomfort or dyspnea. Five hours after PCI, however, the patient developed cold sweating and went into a stupor. Subsequently, his blood pressure decreased to 70/40 mmHg. Urgent 2D-echocardiography showed a large amount of echo dense pericardial effusion and akinesia in the LAD territory (Fig. 3). A repeat emergent angiography was done to ascertain whether acute stent thrombosis or coronary perforation had occurred after PCI. The CAG showed, however, no leakage of dye or thrombus in any coronary arteries with a patent stent in the middle LAD. Emergent pericardiocentesis was performed via apical approach. Approximately 200 cm³ of bloody pericardial effusion was drained, and his blood pressure returned to normal immediately after pericardiocentesis. Peak levels of elevated cardiac enzymes: CK-MB 198.3 IU/L (2.3—9.5) and troponin-I 104.1 ng/mL (0—0.05). Heparin was not administered after pericardiocentesis, but dual anti-platelet therapy was maintained. A follow-up 2D-echocardiography 3 days after pericardiocentesis showed scanty pericardial effusion. Seven days later, he again developed sudden hypotension, bradycardia, and loss of consciousness. The ECG showed ST elevation in V1—V6 and 2D-echocardiography showed scanty pericardial effusion. Emergent CAG was done, which showed total occlusion of the LAD due to subacute stent thrombosis (Fig. 4A). Balloon angioplasty using 2.0 mm and 3.5 balloons was performed, followed by thrombus aspiration. A huge amount of thrombus was aspirated from that lesion. Then, additional balloon angioplasty was done several times using a 3.5 mm balloon in the LAD. The final CAG showed good distal flow without residual thrombus burden (Fig. 4B). He was discharged with dual anti-platelet therapy (aspirin 300 mg and clopidogrel 75 mg). A follow-up CAG after 9 months showed good flow without residual stenosis across the stented segment.

Discussion

There are several reasons that could possibly explain the development of pericardial tamponade and myocardial
Coronary angiogram revealed thrombotic total occlusion of the mid-left anterior descending artery (A, white arrow). Balloon angioplasty was performed with a 3.0 mm balloon, followed by deployment of a 3.5 mm × 24 mm zotarolimus-eluting stent at 14 atm. The final coronary angiogram showed thrombolysis in myocardial infarction III antegrade flow but residual stenosis distal to the stent (B).

Figure 2

Our patient had no angiographic appearance of perforation that is no extraluminal crater, extravasation, or contrast streaming into the pericardium or anatomic cavity chamber [1]. It has been described that tamponade due to perforation may occur up to 24 h after the angioplasty procedure, but the absence of any obvious sign of coronary perforation in our case makes this diagnosis very unlikely. Hemorrhagic pericarditis was also considered as the cause of cardiac tamponade in our patient. It developed because of several causes such as complication of anti-thrombotic therapy (thrombolytic therapy, glycoprotein IIb/IIIa inhibitor) or hemorrhagic infarction followed by late reperfusion therapy. 

Abciximab is currently used for prevention of ischemic complications in patients undergoing PCI and in patients with unstable angina when PCI is planned within 24 h. Despite the proven effect of glycoprotein IIb/IIIa inhibitors, their use is associated with bleeding complications such as pseudoaneurysm (0.5—0.9%), arteriovenous fistula (0.2—2.1%), and retroperitoneal hematoma (0.15—0.44%) [2]. Also, hemorrhagic pericarditis leading to cardiac tamponade is rare, but there were several reports after PCI associated with the use of abciximab [3]. Hemorrhagic infarction and subsequent hemorrhagic pericarditis due to late reperfusion therapy may be considered, but symptom onset to balloon time was only 4 h, making this diagnosis unlikely in our patient.
Cardiac free wall rupture following acute myocardial infarction may lead to hemopericardium and cardiac tamponade. There are two distinct types, based on the clinical course: acute (blow out type) and subacute (oozing type) free wall rupture [4]. When the acute cardiac rupture occurs, it usually results in an abrupt hemodynamic collapse with cardiac tamponade and electromechanical dissociation. On the other hand, the rupture can be sealed by the epicardium or by a hematoma on the epicardial surface of the heart. This situation — subacute or "oozing type" rupture, may evolve over hours or even days, and presents mainly with pericardial effusion-related signs and symptoms [5]. In our patient, clinical signs of cardiac tamponade, pericardial effusion greater than 5 mm thickness, echogenic pericardial effusion, and bloody pericardial effusion by pericardiocentesis may suggest subacute type cardiac rupture [6]. Emergency cardiac surgery may be usually the available therapeutic option in the cardiac rupture. However, because initial aspiration of pericardial fluid leads to rapid improvement of the clinical condition and scanty pericardial effusion by follow-up 2D-echocardiography, we consider a conservative approach (prolonged bed rest, beta blocker).

Stent thrombosis is an uncommon but serious complication of coronary artery stents that often presents as death or myocardial infarction. Several factors were identified to be associated with stent thrombosis, including older age, black race, diabetes mellitus, bifurcation lesion, in-stent restenosis lesion, procedure-related factors such as stent malapposition, greater stent length, postprocedure acute renal failure, and non-compliance to anti-platelet therapy [7]. It was thought that following subacute stent thrombosis might be due to inappropriate anti-thrombotic therapy because of bleeding complication in our patient. We treated this patient with dual anti-platelet therapy (aspirin 100 mg daily, clopidogrel 75 mg daily), but heparinization was not used due to pericardial tamponade. Subacute stent thrombosis may be caused by high thrombotic burden and early stopping of heparinization.

Bleeding complications and thrombosis are always a dilemma during the treatment of acute coronary syndrome. Especially, in patient with drug-eluting stent implantation, appropriate anti-thrombotic therapy is important to prevent stent thrombosis but there is a potential risk for serious hemorrhagic complications. This case suggests that we should consider early cardiac tamponade and stent thrombosis when the patient with ST-segment elevation myocardial infarction develops hypotension and loss of consciousness after apparently successful PCI. Careful monitoring of post-infarction complications should be performed in acute coronary syndrome after PCI.

References
