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CASE REPORT

Coronary Artery Perforation Complicated With Acute Aortic Valve Regurgitation During Percutaneous Coronary Intervention: Report of Two Cases

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ORONARY artery perforation (CAP) is a rare, catastrophic complication of percutaneous coronary intervention (PCI). CAP during PCI procedure is invariably associated with high risk patients with complex coronary artery disease such as coronary calcified lesions, multi-vessel lesions, coronary chronic total occlusion and so on, with an incidence of 0.1%-3.0%. ¹⁻³ However, in the event of occurrence, the condition is extremely serious and fatal. If detection and therapy is not timely and efficacy, acute cardiac tamponade, cardiac arrest and other life-threatening complications will be rapidly developed.^{4, 5} The overall mortality rate of CAP is up to 5%-10%.^{6,7} In this article, we refer to two cases of CAP during PCI procedures to mean to display a special new phenomenon of CAP that complicated with acute aortic valve regurgitation.

CASES DESCRIPTION

Case 1

A 66-year-old male was admitted to our hospital with

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exertional chest pain, which had lasted for more than 3 months and aggravated for 1 week. The patient had a history of hypertension and stroke, and denied a history of diabetes. Physical examination showed temperature 36.1°C, respiratory rate 19 breaths per minute, blood pressure 140/90 mm Hg, and no obvious cardiopulmonary abnormalities. Laboratory tests showed blood glucose 10.3 mmol/L. Electrocardiography showed a sinus rhythm and myocardial damage. Holter revealed ST-T changes in the anterior leads and echocardiogram indicated slightly larger left atria and reduced left ventricular diastolic function. The diagnoses of coronary heart disease, unstable angina pectoris, and Grade 3 hypertension were established. The coronary angiography at admission showed he had no significant stenosis in the left main trunk (LM); significant calcification in the left anterior descending branch (LAD), diffuse long-segment stenosis in the middle segment after second diagonal branch, and the most severe part narrowed about 95%; diffuse long-segment stenosis in the middle segment of the left circumflex artery (LCX), and the most severe part narrowed about 98%; tubular stenosis in the middle segment of the right coronary artery (RCA), and the most severe part narrowed about 75%.

The scheme of PCI was LCX \rightarrow LAD \rightarrow RCA. We manipulated a 6-Fr JL3.5 guiding catheter (Cordis, NJ, USA) to

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the ostia of left coronary artery (LCA) before stentinstallment in the LCX was performed as planned. Then, we advanced the PILOT50 guide wire (Abbott Vascular, IL, USA) to the distal segment of the stenosis in the LAD. Then we predilated the stenosis with a 1.5 mm/15 mm balloon before dilating the lesion with a 2.0 mm/20 mm balloon. Subsequently, we implanted a 3.0/33 mm Firebird drug eluting stent [MicroPort Medical (Group) Co. Ltd., Shanghai, China] at the remaining stenosis inflated with 12-18 atm pressure for 6 seconds. The subsequent coronary angiogram showed the residual stenosis was about 30%. Then post-dilatation was performed with a 3.25 mm/12 mm noncompliant balloon (Boston Scientific, MA, USA). When pressure had been elevated to 22 atm/6 seconds, the contrast agent leaked from the middle segment of implantation stent (Fig. 1).

The patient suffered from severe precordial pain immediately with declined blood pressure. Electrocardiography showed widespread ST-segment elevation, T-wave inversion and frequent premature ventricular contractions. Occlusion with a 2.0 mm/20 mm balloon delivered at low-pressure (6-8 atm) was performed immediately, but the efficacy was badly poor. Adams-stokes syndrome occurred and fluoroscopy showed pericardial tamponade. Emergency pericardiocentesis was performed, and 600 ml fresh blood withdrawn from the pericardium was slowly injected back into the body through the radial artery. The patient's consciousness was gradually recovered after pericardial tamponade was corrected. After emergency coronary artery bypass grafting at cardiac surgery department, the recovery of his hemodynamics and cardiac function was so slow that assisted intra-aortic balloon pump was kept for 2 weeks. Ultrasound electrocardiogram showed widespread weakness of the left ventricular wall motion (including anterior wall, lateral wall and the distal 2/3 part of ventricular septal) with a ejection fraction value being only 30%.

Case 2

A 79-year-old male was admitted to our hospital owing to episodes of chest tightness, palpitation for 2 years and palpitation getting worse for more than 10 days. He had a history of hypertension more than 10 years and received regular oral medication treatment, but his blood pressure control was unknown. On admission, physical examination showed temperature 36°C, respiratory rate 20 breaths per minute, blood pressure 150/70 mm Hg, pulse rate 65 beats a minute, and normal findings of cardiopulmonary physical examination. His blood glucose was 11.6 mmol/L. Electrocardiogram revealed sinus rhythm and frequent atrial premature beats. Holter monitoring indicated ischemic ST-T changes in the anterior leads. Echocardiography showed reduced left ventricular diastolic function. Admission diagnosis was coronary heart disease, stable angina pectoris; Grade 3 hypertension. Coronary angiography showed he had no significant stenosis in the LM; diffuse long-segment stenosis in the middle segment of the LAD, and the most severe involved segment narrowed about 85%; the middle segment of the LCX narrowed about 75%. The middle RCA had 55% stenosis and significant ischemic ST-T changes on electrocardiogram before PCI.

The implementation of PCI procedure was as follows: LAD→LCX. First, we sent a XB3.0 guiding catheter (Cordis) to the ostia of the LCA. Then, a BMW guide wire (Abbott Vascular) passed directly to the distal segment of the LAD stenosis before a 3.5 mm/36 mm Firebird DES was installed in the remaining lesion. Then we advanced the PILOT50 guide wire (Abbott Vascular) to the distal segment of the LCX stenosis and implanted a 4.0 mm/33 mm Firebird DES. Repeated angiography revealed no significant residual stenosis in the LCX, but showed leakage of contrast agent at the implantation site in stent in the LAD (Fig. 2, type II coronary artery perforation).



Figure 1. Coronary angiogram showing type III coronary artery perforation. Abrupt leakage of the contrast material from the middle segment of stent happened when post-dilatation was performed with a non-compliant balloon. Angiogram displays regurgitation of contrast material through aortic valve into the left ventricle. The patient complained of pain and a sensation of fullness in his chest. Meanwhile, multi-lead electrocardiogram





showed ST-segment elevation. Blood pressure decreased significantly. Fluoroscopy and bedside echocardiography showed pericardial tamponade. Emergency pericardiocentesis was performed immediately. Accompanied with 150 ml fresh blood drained, pericardial tamponade was swiftly corrected and symptoms of chest pain gradually improved. Then, he was transferred to the heart surgery for successful coronary artery bypass grafting after his general condition was stable. He had no exertional dyspnea after one year of follow-up.

DISCUSSION

According to the property of drug eluting stent, compliance and pressure of balloon dilation, characteristics of guide wire, perforation generated during PCI procedures are varied. ^{6, 8} CAP was divided into three types based on the severity of CAP according to Ellis classification. ⁹ If leakage of contrast media into the pericardial space from the coronary exceeds 1 mm in diameter, pericardial effusion will easily progress to symptomatic cardiac tamponade.¹⁰ Varied types of CAP lead to totally different prognosis. ^{3, 10, 11} Type II perforation with a block deal based on intermittent low-pressure balloon inflation, rarely causes myocardial infarction or progresses to cardiac tamponade. But most of the type III perforations progress rapidly, and are easy to complicate with pericardial tamponade which often requires emergency surgery.^{7, 12} Incidence of pericardial tamponade for type I, II, and III CAP are serially as follows: 8%, 13%, and 63%. Among them, about 37%-63% of patients require emergency coronary artery bypass grafting, especially the type III CAP. Patients with type II and type III CAP often have chest pain, heart rate exaltation, systolic blood pressure exaltation, central venous pressure exaltation and other hemodynamic changes and symptoms of pericardial tamponade. Early symptoms of cardiac tamponade are always atypical; it may be similar to the vagal reflex, or manifest as low heart rate and intractable blood pressure drop. Anyway, the increased heart rates are really rare. A fluoroscopy with 45-degree left anteriorposterior view exhibits visible translucent ring at the margin of the heart shadow which favors the clinical diagnosis of cardiac tamponade.

Non-surgical rescue measures for percutaneous coronary angiography (PTCA) complicated CAP include pericardiocentesis, intermittent balloon occlusion, protamine reversal of antithrombotic therapy, preloaded tubular coronary stent (JOSTENT) or polytetrafluorethylene covered stent implants and micro-embolization therapy. ¹³⁻¹⁵ Which kind of treatment adopted should be based on the type of CAP and diameter of blood vessels and hemodynamic status of patients. ¹⁶ Mortality rate of type III perforation with non-surgical procedures is proximity to 20%. ^{4, 17} Thus, most scholars have suggested that type III CAP at the site of stent implantation invariably need to be surgical intervention.⁵ In any case, timely pericardiocentesis is the basis for various types of rescue measures and key points, also conducive to the timing of surgical treatment.⁶ We drew four conclusions from above two cases that are demonstrated as follows. First of all, preoperative echocardiography and angiography of the two patients showed no aortic regurgitation. However, CAP complicated by acute cardiac tamponade was found when significant aortic valve regurgitation occurred. Therefore, we regard that aortic regurgitation may be one of the important signs of acute cardiac tamponade. The exact mechanism of this phenomenon is incompletely understood. We consider it is related to the severe left atrial compression after coronary perforation. ^{1, 18, 19} Second, the blood pressure and hemodynamic status of case one maintained normal after a rapid re-injection of fresh blood taken from pericardial sac through the radial artery access. ²⁰ In addition, the recovery of cardiac function and hemodynamic status of the case one was not good due to balloon occlusion leading to a large area of myocardial necrosis. However, thank for intermittent occlusion balloon therapy, the second patient did not suffered from impaired left ventricular ejection fraction. Related studies have shown that delayed balloon occlusion would lead to myocardial infarction and increased mortality. A delayed period beyond 20 minutes will undoubtedly lead to persistent myocardial necrosis, refractory lower blood pressure or shock, malignant arrhythmia, ventricular asystole and other serious complications.^{2, 19} Therefore, most scholars have suggested that if employment of interval balloon occlusion for CAP, the optimal time for each reperfusion time is 5 minutes, especially for perforation occurring in the large coronary arteries.^{2, 21, 22} Furthermore, regarding coronary artery lesions with severe calcification, high plague burden and saphenous vein bypass graft, if balloon diameter of non-compliance dilatation is too large, stent larger than the standard ratio of preliminary balloon should be avoided being placed, for an oversized stent will easily lead to fatal complications such as coronary perforation.

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