A modern cause of an old disease. Constrictive pericarditis after percutaneous coronary intervention: A case report

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A 70-year-old patient developed severe constrictive pericarditis with exertional dyspnea, lethargy and marked peripheral edema four months after percutaneous coronary intervention for acute coronary syndrome. The diagnosis of constrictive pericarditis was confirmed by cardiac catheterization and computed tomography. Despite the short duration of the disease, the pericardium showed

fibrosis and had a thickness of approximately 6 mm. The patient underwent pericardiectomy, with a satisfactory outcome. Hemopericardium due to coronary microperforation was suspected to be responsible for the development of constrictive pericarditis. This unusual mid-term complication of percutaneous coronary intervention has rarely been reported and should be suspected in this particular clinical setting.

Key Words: *Angioplasty; Constrictive pericarditis; Coronary intervention; Coronary perforation; Peripheral edema*

Coronary perforation is a rare complication after percutaneous coronary intervention (PCI). Acutely, coronary perforation may result in significant hemopericardium, and may lead to cardiac tamponade and death. Although acute complications of coronary perforation and their treatment are well recognized, intermediate- or late-term complications have rarely been reported (1).

The present report describes an elderly man who developed constrictive pericarditis four months after a multivessel PCI. Although there was no evidence of coronary perforation noted during the PCI procedure, we suspected hemopericardium due to a coronary microperforation to be responsible for this complication.

CASE PRESENTATION

A 70-year-old nonsmoking man, who was recently diagnosed with adult-onset diabetes mellitus and essential hypertension, presented in December 2005 with a six-week history of progressive exertional angina. There was no evidence of acute myocardial infarction. A coronary angiogram performed at that time revealed severe two-vessel coronary artery disease involving the proximal segments of the left anterior descending (LAD) and left circumflex (LCX) coronary arteries. He underwent multivessel coronary angioplasty with deployment of drug-eluting stents in the proximal segments of the LAD and LCX coronary arteries. There were no complications encountered during or immediately after the procedure. Post-PCI cardiac marker analysis revealed no evidence of myocardial injury or necrosis. He had no further recurrences of angina since that time. He was sent home in stable condition.

In April 2006, he presented to a local hospital because of progressive exertional dyspnea, fatigue, increasing lower extremity edema and increasing abdominal girth. He was

found to have marked peripheral edema, bilateral pleural effusion and ascites. A physical examination on admission to Regina General Hospital revealed an elderly man in mild respiratory distress. His heart rate was 70 beats/min and the rhythm was regular. His blood pressure was 125/65 mmHg, with a pulsus paradoxus of 15 mmHg. His jugular venous pulse was elevated up to the angle of the jaw. There was marked lower-extremity pitting edema up to the mid-thighs. A cardiac examination revealed soft heart sounds, with no gallop rhythm, murmurs, pericardial rub or pericardial knock. An abdominal examination revealed a markedly distended abdomen, normal bowel sounds, no localized tenderness and no organomegaly. Shifting dullness was positive for ascites.

An electrocardiogram revealed a sinus rhythm of 88/min and nonspecific ST-T wave abnormalities.

An echocardiogram showed that the pericardium appeared thickened, with no pericardial effusion. The left atrium was mildly dilated, with abnormal diastolic septal motion. The inferior vena cava was dilated, with insignificant respiratory variation. A Doppler study revealed mild respiratory variation in the mitral and tricuspid velocities.

An abdominal ultrasound showed moderate ascites, no hepatosplenomegaly and normal kidneys. A chest x-ray revealed normal heart size and bilateral pleural effusion. Chest computed tomography showed moderate left pleural effusion and a thick pericardium (Figure 1).

Cardiac catheterization and coronary angiograms

A hemodynamic examination revealed a cardiac output of 4.9 L/min, a cardiac index of 2.5 L/min/m², a left ventricular ejection fraction of 55%, a mean right atrial pressure of 14 mmHg, a right ventricular pressure of 40/13 mmHg, a pulmonary arterial pressure of 43/24 mmHg, a pulmonary capillary wedge pressure of 18 mmHg, a left ventricular pressure of

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Figure 1) Computed tomography of the heart (H) showing a thick pericardium (P; arrow). [P] Posterior

131/15 mmHg and a left ventricular end-diastolic pressure of 22 mmHg.

The left and right ventricular pressure curves showed a 'dip and plateau' pattern, with equalization of the right and left ventricular diastolic pressures, suggestive of constrictive physiology (Figure 2).

Coronary angiograms revealed no significant coronary artery disease. The LAD and LCX coronary artery stents were patent.

He was initially treated with intravenous furosemide and spironolactone, without any significant improvement. He also underwent abdominal paracentesis and removal of large amounts of clear, transudative fluid.

Pericardiectomy was performed in July 2006. During surgery, a fibrotic, thick, adherent pericardium (Figure 3) was stripped. Biopsy specimens revealed a thick (6 mm) and fibrotic pericardium.

The patient's postoperative course was uneventful, and he was discharged in stable condition.

On follow-up, the patient remained asymptomatic and had no further recurrence of peripheral edema or ascites. He lost approximately 11 kg after surgery.

DISCUSSION

In the modern era, the three most common identifiable causes of constrictive pericarditis are cardiac surgery (18%), pericarditis (16%) and mediastinal irradiation (13%). Thirty-three per cent of cases are idiopathic (2).

Constrictive pericarditis is a well-recognized complication following open heart surgery; the incidence has been reported to be between 0.2% and 0.3%. Although the actual cause is unknown, several factors are suspected, including surgical trauma, hemopericardium, infection, postpericardiotomy inflammation, cold injury, various pericardial irritants (povidone-iodine, antibiotics) and air desiccation. The onset of symptoms has been

Figure 2) Left ventricular (LV) and right ventricular (RV) pressures recorded simultaneously, showing that both pressures are equal throughout diastole. A long diastole (large arrow) following a premature beat shows equalization of the LV and RV diastolic pressures and a 'dip and plateau' pattern

Figure 3) This photo was taken during surgical stripping of the pericardium (P). It illustrates the markedly thick and adherent pericardium. H Heart

reported to be from two weeks to 30 years after cardiac surgery (3,4).

The incidence of coronary perforation in the general interventional population has been reported to be between 0.1% and 0.4%. Hemopericardium due to coronary perforation can lead to cardiac tamponade and death. Prompt recognition and early management of coronary perforation is critical. The mortality rate in the setting of cardiac tamponade is 10%.

Constrictive pericarditis caused by hemopericardium is rare and generally appears as an intermediate- or late-term complication. Bonello et al (1) reported a case of constrictive pericarditis diagnosed six months after PCI. However, their patient's PCI was complicated by a type 3 coronary rupture associated with well-tolerated hemopericardium. The coronary rupture was successfully sealed with a polytetrafluoroethylene-covered stent (Jostent GraftMaster Coronary Stent Graft System, Abbott Laboratories, USA).

To our knowledge, constrictive pericarditis occurring several months after clinically uncomplicated PCI has not been previously described. Our patient had no obvious coronary perforation during the PCI procedure. However, we suspect that a microperforation with a small, unrecognized blood leakage (which may have included contrast material) into the pericardial sac was likely the cause of our patient's pericardial constriction.

Constrictive pericarditis should be suspected in patients who present with generalized edema and ascites several months after PCI.

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