Delayed Postoperative Cardiac Tamponade Mimicking Severe Tricuspid Valve Stenosis*

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A patient developed acute, severe hemodynamic deterioration five days after an aortic valve replacement. Cardiac catheterization revealed a markedly elevated right atrial pressure but a normal right ventricular end-diastolic pressure. Angiography revealed an extrinsic mass causing compression of the right atrium and the tricuspid anulus. A large clot overlying the right atrium and ventricle was found at emergency surgery. Postoperative cardiac tamponade may result in an atypical hemodynamic presentation when there is selective compression of one chamber or of a valve anulus.

Cardiac tamponade following cardiac surgery occasionally occurs late in the postoperative course after the patient has stabilized and has left the intensive care unit.14 Delayed tamponade is frequently overlooked or misdiagnosed as congestive heart failure, chronic lung disease, or pulmonary emboli.15-7 High mortality rates from delayed cardiac tamponade have been reported.1-4 The risk for developing delayed tamponade is increased in patients who are receiving anticoagulation therapy for prosthetic heart valves or in patients who have had the postpericardiotomy syndrome.8-9 An unusual hemodynamic pattern is reported in a case of cardiac tamponade occurring five days after an aortic valve replacement. In this case, a localized thrombus caused compression of the A-V groove, resulting in obstruction of flow through the tricuspid valve.

CASE REPORT

A 66-year-old white man was admitted for angina and symptoms of mild congestive heart failure. Physical examination suggested aortic stenosis. The chest x-ray film, ECG, and echoangiogram were consistent with severe aortic stenosis. Cardiac catheterization revealed critical aortic stenosis with an estimated orifice area of 0.4 cm, and the left ventriculogram showed generalized hypokinesis with an overall ejection fraction of 27 percent. Coronary arteriography revealed complete occlusion of the proximal right coronary artery and no significant disease in the left coronary artery. Collaterals from the left filled the distal right coronary artery.

He underwent aortic valve replacement with a 21 mm Bjork-Shiley prosthesis, and a single saphenous vein graft was inserted into the posterior descending branch of the right coronary artery. Postoperatively, he was stable, and his initial hospital course was routine. Chest tube drainage was approximately 2 L on the first day, but decreased to less than 10 ml/hr on the second and third day. The chest tubes were removed on the fourth postoperative day. Several hours after the chest tubes were removed, anticoagulant therapy was instituted with heparin, 5,000 unit bolus, followed by 1,000 units/hr by continuous infusion. Also, oral anticoagulation therapy was instituted with an initial dose of 10 mg of warfarin. Approximately 12 hours following initiation of heparin anticoagulation therapy, early on the fifth postoperative day, while sitting in a chair visiting with the nurses, he suddenly became markedly short of breath. On examination, he was diaphoretic and cool peripherally; his heart rate was 110, and the systolic blood pressure was 60 mm Hg. The jugular venous pressure was difficult to evaluate. Breath sounds were diminished at both bases. Cardiac examination was unremarkable except for the prosthetic aortic valve sounds. The chest x-ray film showed no change in the cardiac silhouette. The ECG showed left ventricular hypertrophy with strain and was unchanged from previous ECGs. Several liters of plasma and high levels of epinephrine and dopamine infusions were necessary to maintain a systolic blood pressure of 70 mm Hg. His arterial oxygen saturation deteriorated markedly, and he required endotracheal intubation, mechanical ventilation, and an FiO2 of 1.0 to maintain an arterial PO2 greater than 70 mm Hg. An arterial line was placed, and a Swan-Ganz catheter was inserted through the right internal jugular vein. The right atrial pressure was 16 mm Hg; the right ventricular pressure was not recorded. The pulmonary artery pressure was 24/12 mm Hg, and the PCWP was 5 mm Hg. The thermodilution cardiac index was 1.2 L/min/m². A PTT drawn at the time of hemodynamic decompensation was greater than 200 seconds.

Initially, the diagnosis of right ventricular infarction due to an occluded saphenous vein graft was considered, but there was no ECG evidence for an inferior wall infarct. The patient was then taken to the cardiac catheterization laboratory. A 7 F Swan-Ganz catheter was advanced from the right femoral vein into the right atrium without difficulty. The right atrial mean pressure was 24 mm Hg (Fig 1). Despite multiple attempts, it was not possible to cross the tricuspid valve with this catheter. After considerable manipulation, a 7 F Courand catheter was passed across the superior portion of the tricuspid anulus into the right ventricle. The systolic right ventricular pressure was 36 mm Hg with an end-diastolic pressure of 6 to 8 mm Hg (Fig 1). Thus, a transvalvular gradient of 16 mm Hg was documented. It was not possible to manipulate the catheter into the pulmonary artery. A selective right atrial angiogram was then performed to evaluate the tricuspid valve. There were two considerations: a thrombus, partially occluding the tricuspid valve, or an extracardiac hematoma compressing the tricuspid anulus and producing the tricuspid stenosis. The cineangiogram revealed an extracardiac density compressing the lateral wall of the right atrium and causing severe narrowing of the tricuspid valve (Fig 2). Contrast material passed from the right atrium into the right ventricle only through the most superior aspect of the tricuspid anulus and opacified an otherwise normal right ventricle and pulmonary outflow tract. Additionally, a small amount of contrast material crossed

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FIGURE 1. Pressure recordings from the right atrium and right ventricle obtained during cardiac catheterization. A 16 mm Hg gradient across the tricuspid valve during diastole.
postoperative bleeding, and he was therefore probably at a higher risk for complications of anti-coagulation. In such a patient, anti-coagulation should be performed slowly, with extremely close monitoring of the level of anti-coagulation. When tamponade occurs in the setting of postoperative anti-coagulation, adequate treatment usually requires surgical exploration for pericardial drainage and removal of thrombus.

Recently, Ofori-Krakye et al reported a different subset of patients at risk for delayed tamponade. They found that most cases of delayed tamponade occurred in patients who had developed the postpericardiotomy syndrome. Only one of their 11 patients was receiving anti-coagulation therapy. In this group, cardiac tamponade was due to a collection of serosanguinous fluid; pericardiocentesis and medical management were adequate therapy in all but one patient.

In nearly every report of delayed tamponade, a comment is made about the difficulty of diagnosing this condition. In general, patients have been stable and out of the intensive care unit when decompensation occurs, and tamponade is not initially a prime consideration. Frequently, congestive heart failure or pulmonary emboli are considered more likely. In our case, a right ventricular infarct secondary to an occluded saphenous vein graft was considered. Deterioration may be rapid, as in this case, or subacute. Symptoms are usually nonspecific, but dyspnea is most common. Physical examination reveals hypotension, tachycardia, and elevated central venous pressure. The classic sign of paradoxical pulse is frequently absent. An increase in the cardiac silhouette on chest x-ray film is frequently present, but this finding may be subtle and apparent only in retrospect when the diagnosis has been made by other means. Low voltage on ECG was absent in our patient, and is not reliably present in tamponade patients. Echocardiography has been found to be useful by some, but there are technical limitations with this technique in postoperative patients.

Most authorities have emphasized the diagnostic importance of hemodynamic evaluation utilizing bedside Swan-Ganz catheterization. Pericardial tamponade should be suspected when there is decreased cardiac output with elevated and closely approximated right atrial, right ventricular end-diastolic, and pulmonary capillary wedge pressures. Our case is highly unusual in that tamponade was associated with normal right ventricular end-diastolic, pulmonary artery, and pulmonary artery wedge pressure. The marked gradient across the tricuspid valve was not initially recognized in the intensive care unit because the right ventricular pressure was not recorded during the bedside catheterization. Failure to recognize this gradient delayed the diagnosis of cardiac compression. At the same time, the low pulmonary artery and pulmonary artery wedge pressures suggested alternative diagnoses. This gradient was well demonstrated in the catheterization laboratory. An intracardiac mass was momentarily suspected when we encountered difficulty crossing the tricuspid valve. The etiology for the gradient was apparent only after the cineangiogram revealed an extracardiac density with compression of the tricuspid anulus.

It is interesting that a small amount of contrast material promptly passed from the right atrium into the left atrium, presumably through a patent foramen ovale. The severe

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**Figure 2.** Frame from selective right atrial angiogram. Right atrium (RA) is compressed, superior vena cava (SVC) is dilated, and right ventricle (RV) shows normal trabeculations. The tricuspid orifice (arrow) is markedly restricted due to extrinsic compression by thrombus.

Promptly into the left atrium, presumably through a patent foramen ovale. Because of the likelihood of an extracardiac hematoma causing localized cardiac tamponade and compression in the A-V groove, emergency exploratory surgery was performed. Immediately after opening the sternum, the systolic arterial pressure rose from 70 to 170 mm Hg. A large thrombus overlying the right atrium and A-V groove was removed. Following relief of the tamponade, pullback of the Swan-Ganz catheter from the pulmonary artery to the right atrium disclosed no residual pressure gradient across the tricuspid valve (Fig 3). The remainder of the patient's hospital course was unremarkable, and he was discharged home 11 days after the repeat sternotomy.

**Discussion**

Delayed cardiac tamponade, or tamponade occurring relatively late in the postoperative course, is a well-described complication of open heart surgery. Occurrence rates of 1 to 3 percent have been reported. In the majority of cases, tamponade appears related to anti-coagulation. In many cases, as in this one, the level of anti-coagulation was poorly controlled. The time to initiate heparin anti-coagulation in the early postoperative valve replacement patient is controversial. Our patient had somewhat excessive early
arterial oxygen desaturation in our patient was certainly multifactorial, and the intracardiac shunting probably played only a minor role. However, Miller et al reported a case of delayed postoperative tamponade in which localized right atrial and right ventricular compression resulted in a significant right-to-left shunt through a patent foramen ovale and cyanosis. In that case, there was no tricuspid valve gradient.

A significant tricuspid valve gradient due to localized thrombus compressing the right atrium or A-V groove in postoperative patients has been reported recently. Others have recognized localized cardiac compression in postoperative tamponade. Tricuspid valve stenosis has been reported as a rare manifestation of constrictive pericarditis; in these cases, it is caused by scarring of the A-V groove.

This case demonstrates that postoperative tamponade may have a varied hemodynamic presentation due to localized compression of the heart and illustrates the need to consider tamponade whenever hemodynamic deterioration occurs in the postoperative cardiac patient. Furthermore, the importance of careful measurement of pressures in each chamber during bedside catheterization is illustrated well by this case. Finding a marked gradient across the tricuspid valve in a postoperative patient should immediately suggest cardiac tamponade. Early recognition of this entity will lead to more appropriate management and lower mortality and morbidity.

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**Esophageal Compression of the Heart Presenting as an Extracardiac Mass on Echocardiography**

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Echocardiography is helpful in the identification of extracardiac masses. The role of echocardiography in evaluating the effects of the masses on the heart is not well established. The case presented details the identification of and the evaluation by echocardiography of structural and functional effects of a massively dilated esophagus on the heart. Two-dimensional echocardiography revealed anatomic distortion due to a mass effect of the dilated esophagus on the left heart chambers. M-mode echo recordings demonstrated decreased left atrial and ventricular dimensions with increased atrial and ventricular fractional shortening. These findings normalized after Heller myotomy, without a change of estimated stroke volume.

Echocardiography has been shown to be useful for the investigation and diagnosis of extracardiac mediastinal masses. The case reported illustrates the identification and also the evaluation of the structural and functional effects of a mediastinal mass on the heart by echocardiographic techniques.

**CASE REPORT**

A 42-year-old man from Haiti presented to the general surgical service of the University Hospital of Jacksonville with a left inguinal hernia. Review of symptoms revealed a six- to eight-year history of dysphagia and intermittent vomiting. There was no history of weight loss, diarrhea, rectal bleeding, dyspnea, or chest pain. A physical

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