Right Atrial Compression in Postoperative Cardiac Patients: Detection By Transesophageal Echocardiography

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Four patients developed hypotension after heart surgery. Hemodynamic measurements revealed elevated right atrial pressure with normal pulmonary capillary wedge pressure. Conventional transthoracic two-dimensional echocardiography was technically suboptimal for detection of pericardial effusion. In each patient transesophageal echocardiography demonstrated significant compression of the right atrium by a localized mass. At reoperation atrial compression by an organized hematoma was found and in each instance successfully drained.

Case Reports

Case 1
A 77 year old woman with a body surface area of 1.86 m² was admitted for emergency coronary artery bypass grafting for 99% occlusion of the left main coronary artery. Preoperatively a percutaneous intraaortic balloon pump was placed to stabilize the patient's condition after an episode of rest angina with persistent anterolateral ischemic changes on the electrocardiogram (ECG).

Postoperative course. Immediately after operation mediastinal bleeding at the rate of 200 mHb was noted. The bleeding persisted despite a normal platelet count and normal prothrombin time and partial thromboplastin time. Surgical exploration after 6 h of continued bleeding did not show any evidence of clots. Eighteen hours later the patient remained in unstable condition with a systolic blood pressure of 90 mm Hg and a low cardiac index of 1.85 liters/min per m² despite intraaortic balloon pumping at a 1:1 ratio and maximal inotropic support. Hemodynamic measurements revealed an elevated mean right atrial pressure of 19 mm Hg and a normal capillary wedge pressure of 9 mm Hg. A diagnosis of right ventricular infarction with right ventricular dysfunction was considered.

Echocardiography. A transthoracic echocardiogram was performed to determine the cause of the hemodynamic instability. Compression of the right atrium by a localized mass that decreased right atrial volume was found in each case. Surgical exploration confirmed atrial compression by loculated hematoma in all four patients. With removal of the hematoma, the patients' clinical status improved dramatically.
ESOPHAGEAL ECHOCARDIOGRAM IN LOCALIZED TAMPONADE

Case 1

A 65 year old man with a body surface area of 1.76 m² underwent elective triple coronary artery bypass grafting for severe angina pectoris.

Postoperative course. The epicardial pacing wires were removed on the 5th postoperative day and until then the hospital course had been uneventful. On the 6th postoperative day the patient developed shortness of breath. Pulmonary embolism was diagnosed by pulmonary arteriogram and the patient was started on anticoagulant therapy. A transthoracic echocardiogram at this time showed normal left ventricular systolic function and a small anterolateral pericardial effusion. On the 10th postoperative day the patient suddenly became diaphoretic and hypotensive; systolic blood pressure fell to 70 mm Hg. A repeat transthoracic echocardiogram showed a small to moderate pericardial effusion and a questionable diastolic right atrial inversion sign. A transesophageal echocardiogram was recommended for better visualization of the pericardial effusion and assessment of cardiac compression. In addition the patient had fulminant postoperative hemorrhagic pancreatitis with serum amylase levels of 1,237 IU/liter and lipase levels of 237 IU/liter. This episode of hypotension and diaphoresis was attributed to hemorrhagic pancreatitis.

The next day the hemoglobin concentration fell to 5.7 g/dl with a partial thromboplastin time >100 s. He did not improve despite infusion of fluid and inotropic support. The systolic blood pressure remained at 85 mm Hg and urinary output decreased to 10 ml/h. Hemodynamic measurements revealed elevated mean right atrial pressure (18 mm Hg), a normal capillary wedge pressure (12 mm Hg) and a low cardiac index (1.57 liters/min per m²).

Transesophageal echocardiography. After two nondiagnostic conventional transthoracic echocardiograms were made, a transesophageal echocardiogram revealed a large mass measuring 6.5 x 5.8 cm severely compressing the entire anterior surface of the right atrium (Fig. 2). This mass also extended to the inferior and posterior aspects of the right atrium.

Surgical exploration was performed and a large hematoma removed. The hemodynamic variables improved immediately with systolic blood pressure increasing to 120 mm Hg and cardiac index to 2.3 liters/min per m². However, the patient developed sepsis and acute metabolic acidosis and died of multisystem organ failure on the 13th postoperative day.

Case 2

A 66 year old woman with a body surface area of 1.63 m² underwent five vessel elective coronary artery bypass grafting for severe coronary artery disease. The first 5 days were uneventful except for a limited inferior wall myocardial infarction on the 3rd postoperative day. There was no

Findings on reexploration. A repeat bedside thoracotomy was performed and a large loculated hematoma compressing the right atrium anteriorly was found. The clot was evacuated and the cardiac index improved over the next 4 h. A repeat transesophageal echocardiogram after 48 h of evacuation of clot revealed reexpansion of the right atrium (Fig. 1, bottom). The epicardial pacing wires were removed on the 5th postoperative day. The remaining hospital course was uneventful.

**Figure 1.** Case 1. Top, Transesophageal echocardiogram (TEE), four chamber view, showing a large pericardial clot compressing the right atrium (RA). Bottom, Reexpansion of the right atrial cavity is noted after (POST) evacuation of the clot. LA = left atrium; LV = left ventricle; PRE = before; RV = right ventricle.
Figure 2. Case 2. Transesophageal echocardiogram showing a massive clot (6.5 x 5.8 cm) severely compressing the right atrium. Note the slitlike appearance of the right atrial cavity. Abbreviations as in Figure 1.

Evidence of right ventricular dysfunction. At the time of the removal of the Swan-Ganz catheter on the 4th postoperative day, all hemodynamic measurements were normal. The patient was transferred from the coronary care unit to the regular floor for rehabilitation. On the 6th postoperative day, immediately after removal of an epicardial pacing wire, the patient suddenly became diaphoretic, hypotensive (systolic blood pressure 70 mm Hg) and tachycardiac at a heart rate of 120 beats/min.

Echocardiography. A conventional transthoracic two-dimensional echocardiogram to evaluate tamponade was technically suboptimal. A transesophageal echocardiogram revealed a normal to hyperdynamic left ventricle and normal right ventricular systolic function. The right atrium was partially compressed anteriorly by a mass measuring 3.5 x 3.0 cm. The study was interpreted as showing isolated compression of the right atrium by clot and hemodynamic measurements were recommended.

Hemodynamics. Repeat right-sided catheterization revealed elevated mean right atrial pressure (28 mm Hg) and pulmonary capillary wedge pressure (20 mm Hg). Cardiac index measured 1.7 liters/min per m².

Reexploration. The patient was immediately transferred to the operating room for exploration of the pericardium. A large hematoma compressing the right atrium anteriorly and laterally was found. An arterial bleeder was found adjacent to the sinoatrial node in the area of placement of the pacing wire. The hematoma was evacuated and the arterial bleeder was sutured. After evacuation of the hematoma the blood pressure returned to normal and the right atrial pressure decreased from 28 to 6 mm Hg. The remainder of the hospital course was uncomplicated.

Case 4

A 75 year old man with a body surface area of 1.83 m² underwent emergency bypass grafting to the left anterior descending artery after acute closure during balloon angioplasty. On the 1st postoperative day the patient became hypotensive with the systolic blood pressure decreasing to 70 mm Hg. There were no new ECG changes and arterial blood gas values were normal.

Hemodynamics. Elevation of the right atrial pressure to 21 mm Hg and the pulmonary capillary wedge pressure to 16 mm Hg was noted. The cardiac index decreased from 2.8 to 1.9 liters/min per m². A presumptive diagnosis of cardiogenic shock of unclear etiology was considered. The patient was given inotropic agents and an intraaortic balloon pump was inserted.

Echocardiographic findings. A transthoracic two-dimensional echocardiogram showed a small pericardial effusion without any signs of tamponade. The study was technically suboptimal for evaluating left ventricular systolic function. Transesophageal echocardiography revealed normal left and right ventricular systolic function and a 3 x 3 cm mass compressing the right atrium anteriorly. There was a small pericardial effusion. In addition, during intravenous injection of medication microbubbles were observed in the left heart consistent with a patent foramen ovale.

Reoperation. Surgical exploration was performed directly after transesophageal echocardiography. A loculated hematoma was found compressing the anterior surface of the right atrium. The finding of a patent foramen ovale was also confirmed. Removal of the hematoma resulted in an immediate return to normal of the systolic blood pressure, the right atrial pressure and the cardiac index. The epicardial pacing wires were removed on the 4th postoperative day without complications. The remaining hospital stay was uneventful and the patient was discharged on the 10th postoperative day.

Discussion

Pathophysiology. All four patients showed compression of the right atrium by a moderate to large hematoma. Whether or not compression occurs depends on the amount of fluid in the pericardial cavity, the rate of accumulation and the diastolic stretch of the pericardium (3). In chronic effusion the pericardium becomes stretched and may have the capacity to contain several liters of fluid with minimal hemodynamic compromise. However, in acute tamponade a relatively small amount of fluid, 150 to 250 ml, may be sufficient to quickly utilize pericardial reserve volume and cause a
marked increase in intrapericardial pressure (3). A rapid accumulation of only 60 to 100 ml of strategically located blood clot may be enough to produce profound hemodynamic effects after open heart surgery and penetrating injuries to the heart (4). With increase in intrapericardial pressure the transmural distending pressure gradient between the ventricular cavity and the intrapericardial space falls. This results in impairment of cardiac filling and reduction in stroke volume and cardiac output (5). Fowler et al. (6) described a similar mechanism responsible for causing isolated right atrial tamponade in experimental animals. A negative transmural right atrial pressure owing to the right ventricular diastolic suction was observed. In addition, a significant pressure gradient between the superior vena cava and right atrium suggesting caval compression during right atrial tamponade was noted in this study. Vena caval compression was not observed when animals were subjected to tamponade of the entire heart (7). Thus, presumably both the loss of negative transmural right atrial pressure gradient and the caval compression are responsible for causing hemodynamic effects in isolated right atrial compression.

Etiology and Incidence of postoperative tamponade. Pericardial tamponade after cardiac surgery may arise from multiple causes such as excessive anticoagulant therapy, leakage from a rupture of a graft, mediastinal bleeding and even after a seemingly innocuous procedure such as removal of an epicardial pacemaker wire (8). All these different causes for tamponade in the immediate postoperative period were observed in our patients.

Pericardial effusion leading to tamponade occurs in ≤6% of patients after cardiac surgery (9). It usually occurs immediately after operation in association with excessive bleeding and hemodynamic compromise (10). Late tamponade (after 2 weeks) is usually the result of the postpericardiotomy syndrome (9). In a more recent series of 39 and 122 patients, respectively, by Stevenson et al. (11) and Weitzman et al. (12), pericardial effusion after cardiac surgery was found in 56% and 85% of the patients by the 10th postoperative day. However, the incidence rate of cardiac tamponade in those series was only 1% to 2.5%. Despite this low incidence of tamponade after cardiac surgery, it is extremely important to diagnose this condition promptly in the appropriate clinical setting because it is curable; the outcome is directly related to timely reoperation (2).

Localized pericardial hematoma. A localized postoperative pericardial hematoma produces its own diagnostic problems and differential diagnosis (13). It is essential to appreciate that tamponade after cardiac surgery may not always manifest the typical features such as pulsus paradoxus and elevation and equalization of right atrial, pulmonary artery diastolic and pulmonary capillary wedge pressure. Using two-dimensional transthoracic echocardiography D'Cruz et al. (14) studied 11 patients with postoperative pericardial tamponade including 7 with a loculated pattern. Pulsus paradoxus and equalization of right and left heart pressures were seen in only two of the seven patients with a loculated effusion, although these classic signs were present in all the patients with a generalized effusion that compressed all chambers equally. The absence of these hemodynamic signs in loculated pericardial effusion has also been demonstrated in animal studies (6). Single chamber tamponade of the right atrium (15), right ventricle (16), left ventricle (17), left atrium (18) and superior vena cava (19) has been reported.

Diastolic right heart compression is presumably more common than left heart compression because of thinner right atrial and right ventricular walls relative to thicker left ventricular walls (10,20). As illustrated in our cases and those of others (16), right atrial hematoma after heart surgery is commonly localized to the anterior and lateral walls. However, in left atrial tamponade a clot is seen more commonly behind the left atrium because it can easily become encysted in the potential space in the oblique sinus (13).

Role of transthoracic echocardiography in cardiac tamponade. Various noninvasive methods for diagnosing pericardial tamponade include M-mode and transthoracic two-dimensional and Doppler echocardiography. M-mode echocardiography has a very low sensitivity in the diagnosis of loculated pericardial hematoma after cardiac surgery (21). Transthoracic two-dimensional echocardiography has been extensively studied in the diagnosis of cardiac tamponade (22-25). Early and late diastolic collapse of the right ventricle and right atrium by two-dimensional transthoracic echocardiography has been found to be a fairly reliable sign of hemodynamically compromised cardiac tamponade (22,23).

Right atrial inversion is more sensitive than right ventricular collapse (23).

Doppler echocardiography can also be helpful in assisting in the diagnosis of cardiac tamponade if good quality studies can be obtained (23-25). Leeman et al. (24) described the exaggerated respiratory variation in Doppler flow velocities across cardiac valves in patients with cardiac tamponade. A marked inspiratory increase (81%) in right-sided velocities was noted, and corresponding left-sided velocities decreased by at least 35%. Marked inspiratory prolongation of the left ventricular isotropic relaxation time has been reported (25) in patients with clinical tamponade.

An appreciable decrease or total loss of diastolic forward flow in the hepatic veins has been reported in patients with cardiac tamponade (26). Doppler echocardiography is not only cumbersome to perform but can also be misleading in postoperative cardiac patients who are on mechanical ventilators as a result of exaggerated respiratory variation.

Because of the limitations of M-mode and Doppler echocardiography, two-dimensional transthoracic echocardiography has been considered the study of choice in detecting postoperative pericardial hematomas (14,21). However, loculated clots can often be difficult to detect with conventional
transesophageal two-dimensional echocardiography in these patients because of poor image quality. Surgical wounds and dressings, mechanical ventilators and the intraaortic balloon pump limit the ability to obtain high quality echocardiograms. Transesophageal echocardiography may provide an alternative acoustic window with excellent image quality because of the proximity of the transducer to the cardiac structure.

Role of transesophageal echocardiography in the diagnosis of loculated pericardial hematoma. Transesophageal echocardiography can provide critical information in hemodynamically unstable patients after cardiac surgery. The procedure takes only 10 to 15 min to perform (27). Although it is semi-invasive, transesophageal echocardiography is well tolerated in critically ill patients (27). In a study of 30 patients in an intensive care unit, Oh et al. (27) reported no complications from the procedure. Transesophageal echocardiography has an excellent safety record and is feasible in almost all patients (98% to 99%). Daniel et al. (28) analyzed data from 1,300 patients evaluated with transesophageal echocardiography since 1984; a satisfactory study was performed in 98% of patients. On the basis of high quality transesophageal echocardiographic images, medical or surgical treatment can be instituted promptly without further diagnostic procedures.

To date we are not aware of any reports on the use of transesophageal echocardiography in detecting loculated pericardial hematomas after cardiac surgery. The safety of the procedure and its ability to image structure in detail in less than 10 to 15 min at the bedside makes transesophageal echocardiography an attractive alternative for the evaluation of life-threatening complications in patients after cardiac surgery. However, a large series of patients with suspected tamponade after cardiac surgery should be studied to determine the sensitivity and specificity of transesophageal echocardiography in the diagnosis of loculated cardiac tamponade. A normal high quality transesophageal echocardiogram is reassuring in that it excludes tamponade in critically ill patients in the postoperative setting.

Conclusions. Cardiac tamponade presents in an atypical fashion after cardiac surgery because clots may be localized by adhesions. The development of tamponade after cardiac surgery is a rare but serious complication. When cardiac tamponade is suggested by the clinical setting but is not supported by transthoracic echocardiography, the presence of loculated tamponade should be considered and evaluated by transesophageal echocardiography.

References


