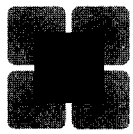

Superior vena cava syndrome after open heart surgery

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■ Superior vena cava syndrome is an uncommon complication of open heart surgery. While both cardiac tamponade and superior vena cava syndrome may present as elevated central venous pressure accompanied by decreased mean arterial pressure and cardiac output, the presence of upper body cyanosis is unusual with tamponade. We report the diagnosis and successful emergency treatment of a patient with acute superior vena cava syndrome and dyspnea apparently caused by a pericardial hematoma developing after aortic valve replacement and coronary artery bypass grafting.

□ INDEX TERMS: SUPERIOR VENA CAVA SYNDROME; CORONARY ARTERY BYPASS; HEART VALVE PROTHESIS; CARDIAC TAMPONADE
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Superior vena cava (SVC) syndrome is a rare complication following open heart surgery, generally presenting as a subacute problem weeks after surgery. We report a case of SVC syndrome accompanied by cardiac tamponade that presented as acute dyspnea, upper body venous engorgement, and cyanosis within 24 hours of aortic valve replacement and coronary artery bypass grafting.

CASE REPORT

A 74-year-old man with unstable angina was transferred to The Cleveland Clinic Foundation for coronary artery bypass surgery and aortic valve replacement. Three weeks prior to admission, a myocardial infarction complicated by congestive heart failure had led to the discovery of severe triple vessel coronary artery disease and severe aortic stenosis with a gradient of 65 mm Hg. The patient's past medical history included peripheral vascular disease with claudication, chronic renal failure, chronic hypertension, and non-insulin-dependent diabetes mellitus. Medications at the time of admission included bumetanide, transdermal nitroglycerin, diltiazem, glyburide, captopril, and intravenous heparin sodium. Preoperative laboratory values were within normal limits except for a partial thromboplastin time of 69.9 seconds (secondary to heparin therapy), blood urea nitrogen of 46 mg/dL, and serum creatinine of 2.3 mg/dL. Preoperative chest radiographs were unremarkable.

Aortic valve replacement with a #23 St. Jude valve, and aortocoronary bypass grafting to the left anterior descending, diagonal posterior descending artery, and lateral circumflex were accomplished uneventfully. Atrial and ventricular pacing were required for temporary complete heart block and inotropic therapy with dobutamine and dopamine for low cardiac output immediately following separation from cardiopulmonary bypass. Continued bleeding was noted prior to chest closure, despite protamine reversal of heparin and administration of fresh frozen plasma, 5 units of packed red cells, and 14 units of platelets. Epsilon aminocaproic acid was administered as a 5-gm loading dose followed by continuous infusion of 1 gm/hr beginning at chest closure for continued diffuse bleeding.

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The postoperative course was remarkable for new onset right bundle branch block, borderline urine output (40 to 50 cc/hr), and chest tube drainage of 100 to 200 cc/hr for the first 12 hours. The patient was extubated uneventfully on the morning of the first postoperative day. Throughout the day, heart rate was 100 bpm paced, and hemodynamics were stable with a mean arterial pressure ranging from 65 to 85 mm Hg, central venous pressure of 10 to 13 mm Hg, systemic vascular resistance (SVR) of 1,100 to 1,500 dyne-sec/cm^4, and cardiac index of 1.8 to 2.7 L/min/m^2.

Hemodynamic status changed markedly 24 hours after surgery: the mean arterial pressure fell transiently to 50 mm Hg without pulsus paradoxus; the cardiac index decreased to 1.7 L/min/m^2; and central venous pressure (measured at the SVC) increased from 12 to 30 mm Hg. Pulmonary artery pressures were unchanged at 21/13 mm Hg. Chest tube blood drainage became negligible. Chest radiography did not show widening of the mediastinal shadow or increased heart size compared to preoperative or early postoperative films. The patient complained of sudden onset of dyspnea, and was noted to have prominent jugular venous distention, differential cyanosis limited to the head and neck, and fullness at the suprasternal notch. Faint inspiratory stridor was noted on auscultation. Arterial blood gases sampling revealed pH of 7.36, PCO_2 of 40 mm Hg and PO_2 of 103 mm Hg on 5 L of oxygen via nasal cannula.

The patient was immediately intubated orally and brought to the operating room for suspected cardiac tamponade. Anesthesia was induced with fentanyl, vecuronium, and scopolamine. At exploration, findings included an apical pericardial thrombus, and a large extrapericardial blood clot causing obstruction of the SVC. Evacuation of these clots resulted in immediate improvement in mean arterial pressure from 60 to 100 mm Hg, and cardiac index from 1.7 to 2.8 L/min/m^2. The central venous pressure fell to 13 mm Hg and the physical findings of SVC obstruction disappeared. Following exploration and control of bleeding, the patient was returned to the intensive care unit. He was extubated the next morning and discharged to home on the seventh postoperative day after an otherwise uneventful course.

DISCUSSION

SVC syndrome is uncommon as a complication of open heart surgery. Two English-language reports describe late localized tamponade causing SVC syndrome 5 to 6 weeks after aortic valve replacement,[1,2] and there is one report of early extravascular obstruction of the SVC following mitral valve replacement.[3]

More than 80% of obstructions of the SVC are due to malignancy,[4] with small-cell lung cancer the most common cause.[5] Other reported causes include substernal goiter,[6] filarial mediastinal lymphadenitis,[7] thrombotic complications of Hickman-Broviac catheters,[8] and bronchogenic cyst.[9] Prior to this patient's initial surgical procedure, a pulmonary artery catheter had been inserted via the right internal jugular vein, but the timing and location of the hematoma compressing the SVC make it unlikely this event was related to line placement.

Untreated SVC syndrome in itself is not necessarily fatal.[5] However, a mass that is causing compression of the SVC can result in hemodynamic compromise and possibly airway obstruction and must be managed emergently when the presentation is acute.

When SVC syndrome is due to neoplastic disease, radiation therapy is palliative in most cases.[4,5] Conservative therapy such as diuretics, oxygen, phlebotomy, repositioning into the sitting position, and sedation may be used to alleviate some of the symptoms of SVC obstruction, but are for the most part ineffective.[4,5,10] Surgical treatment using axillo-femoral bypass grafting or SVC reconstruction with a Dacron prosthesis has been successfully utilized.[4,10]

This patient exhibited classic signs of SVC obstruction, including marked venous engorgement, swelling of the head and neck, and conjunctival edema. In addition, the patient complained of shortness of breath, which was worse in the supine position. Airway obstruction has been reported to occur concurrently with SVC obstruction in neoplastic disease.[10] While it was impossible to determine whether this patient's dyspnea was primarily due to airway compression or low cardiac output, the presence of stridor and the acuity of onset were sufficient to warrant emergent endotracheal intubation.

Cardiac tamponade is relatively common after open heart surgery; in our experience, re-exploration for bleeding is required in 1% to 2% of patients. Most cases present with elevation of central venous pressure, decreased cardiac output, and decreased arterial pressure. Arterial hypoxemia may be seen, but cyanosis and stridor are unusual. Classic equalization of cardiac chamber pressures may not occur in postoperative tamponade because of localized compression with an open pericardial sac, or due to changes in chamber compliance as a result of valvular or ischemic disease.

Low output syndrome following open heart surgery is commonly caused by volume depletion, ventricular dysfunction due to ischemia or infarction, or tamponade. Differentiation of low output syndrome can sometimes be accomplished with technetium scanning[3] or Doppler echocardiography and venography[1]; however, the tempo of this patient's hemodynamic decompensation mandated emergent return to the operating room before such studies could be performed. Thus, recognition of the syndrome by clinical findings alone may be important, particularly when the syndrome occurs within the first 24 hours of operation.

This case is unusual because of the association of SVC syndrome with cardiac tamponade. Although because of the urgent nature of this case diagnostic contrast or ultrasound studies were not performed, the findings at surgery at the level of the heart were such that SVC obstruction rather than cardiac tamponade appeared to be the most likely etiology of the clinical findings. However, tamponade itself may cause compression of the SVC as it passes through the pericardium.[11]

Important points in the management of this patient included recognition of respiratory distress with immediate reintubation to ensure a patent airway, and emergency exploration with removal of blood clot compressing the SVC.

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