A 49-Year-Old Woman With Progressive Peripheral Edema and Jugular Venous Distension After Bypass and Defibrillator Placement

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Case History

A 49-year-old woman with a history of hypertension and insulin-dependent diabetes mellitus presented to an outside medical institution with the chief complaint of retrosternal chest pressure. The chest pain occurred at rest, radiated to both arms, was associated with shortness of breath, and was relieved with sublingual nitroglycerin. The presenting ECG showed an inferior and anterolateral infarct pattern without evidence of acute ST segment changes. Acute myocardial infarction was excluded by the absence of cardiac enzyme changes (creatinine kinase-MB and lactate dehydrogenase). Cardiac catheterization revealed three-vessel coronary artery disease (90% proximal right coronary artery, 70% left anterior descending, 60% proximal first obtuse marginal) with a left ventricular ejection fraction of 35%. She was referred to Hermann Hospital, Houston, for further evaluation and therapy and underwent three-vessel saphenous vein grafting to the left anterior descending, obtuse marginal, and right coronary arteries. The left internal mammary artery could not be used because of its small caliber. The patient received an automatic implantable cardiac defibrillator device as per research protocol (coronary artery bypass grafting patch trial). An echocardiogram in the postoperative period showed the presence of a thrombus in the apex of the left ventricle. The patient therefore received anticoagulation therapy with warfarin and was maintained on this agent. The patient was discharged and on follow-up 2 weeks later was noted to have peripheral edema; treatment was begun with furosemide, producing mild improvement in symptoms. In addition, the patient was prescribed ciprofloxacin for erythema in the left upper quadrant at the site of the automatic implantable cardiac defibrillator generator. Four weeks after discharge from the hospital, the patient presented again with the complaint of dull, nonradiating retrosternal chest pain that was acute in onset, moderate in intensity, and of 12 hours' duration. The pain waxed and waned for several hours, was associated with nausea and diaphoresis, but was not relieved by sublingual nitroglycerin. The pain was not altered by change in body position or respiration and was different from the chest pain she had experienced previously. She denied orthopnea, paroxysmal nocturnal dyspnea, fever, chills, hemoptysis, or hematemesis.

Her past medical history revealed hypertension for 20 years, adult-onset diabetes mellitus for 25 years, and hypothyroidism. She was a cigarette smoker but had stopped smoking before her cardiac surgery. Her medications included enalapril 5 mg PO qd, furosemide 40 mg PO qd, levothyroxine 0.05 mg PO qd, aspirin 325 mg PO qd, warfarin 5/7.5 mg PO on alternate days, digitalis 0.25 mg PO qd, and NPH insulin 40 U/15 U subcutaneously every morning and evening. Her mother died at the age of 85 years of myocardial infarction, and her father died at 53 years of age of the sequelae of rheumatic heart disease. Relevant laboratory data are shown in the Table.

Physical Examination

Clinical examination revealed a pleasant, obese white woman who appeared to be mildly tachypneic and with edema of the lower limbs to the midthigh. There was no pallor of the mucous membranes, icterus, or clubbing. Her temperature was 98.1°F.

Cardiovascular examination showed a pulse rate of 120 beats per minute; pulses were of normal amplitude and symmetrical throughout; there was no pulsus paradoxus. The blood pressure was 133/90 mm Hg. The jugular veins were distended to the angle of the mandible. Carotid upstrokes were full and brisk, and no bruits were auscultated. The apex beat was displaced to the sixth intercostal space 1 cm lateral to the midsclavicular line. The first and second heart sounds were normal, and a third heart sound was present. No pericardial friction rub or heart murmur was noted.

The respiratory rate was 20 breaths per minute. Breath sounds were diminished over the left lower lobe with dullness to percussion. Moist rales were present over the right lower lobe; no rhonchi or wheezes were noted. The abdominal examination showed an enlarged liver palpable 12 cm below the costal margin. There was a small nonhealed partially open, erythematous surgical wound in the left upper quadrant with a bloody purulent discharge. The rectal examination was normal, and the stool was negative for occult blood. The patient was alert and oriented, and the neurological examination

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was normal. Her extracocular movements were intact, with pupils round, equal, and reactive to light. Examination of the eyes revealed sharp discs bilaterally and no hemorrhages, exudates, or arteriovenous nicking.

Electrocardiogram

The ECG (Fig 1) showed a sinus tachycardia with low-voltage QRS and q waves in leads II, III, aVF, and V₃ through V₆ with generalized nonspecific ST-T-wave changes.

Chest Radiograph

The chest radiograph showed mild cardiomegaly, pulmonary venous redistribution, no evidence of pulmonary edema or parenchymal lung disease, and a left pleural effusion.

Hospital Course

The patient was initially treated with intravenous furosemide and nitrates, with improvement of symptoms of heart failure. On day 2 of hospitalization, the patient was found to be hypotensive, with tachypnea and tachycardia. The respiratory rate was 26 breaths per minute. The heart rate was 130 beats per minute, and the blood pressure was 90/60 mm Hg. There was no pulsus paradoxus. The jugular veins were distended to the angle of the mandible with the patient sitting upright. The heart sounds revealed a normal S₁ and S₂, and an S₃ was also noted. Pulmonary examination showed dullness to percussion and reduced breath sounds over the left chest posteriorly. A diagnostic procedure was performed.

Case Discussion (Dr Francis Thandroyen)

The critical findings in this patient were the onset of marked peripheral edema, jugular venous distension, absence of pulmonary edema, and maintenance of normal arterial pressure within 6 weeks of surgery. Acute decompensation, manifested by hypotension, occurred within 48 hours of hospitalization. The above constellation of clinical findings raises the differential diagnosis of pericardial disease (pericardial effusion with cardiac tamponade or constrictive pericarditis), pulmonary embolic disease, or right ventricular myocardial infarction in the setting of depressed left ventricular function.

Was There Evidence of Myocardial Infarction?

Perioperative myocardial infarction has been reported to occur in approximately 5% to 6% of patients undergoing elective coronary artery bypass graft surgery.¹ ² Saphenous vein occlusion occurs in 8% to 12% of patients before discharge from hospital and in 12% to 20% of patients within 1 year of coronary artery bypass graft surgery.³ ⁴ Technical problems relating to the graft (excessive tension or kinking of the vein graft, anastomoses of the vein graft to a small distal coronary artery), coronary embolism, coronary vasospasm, or thrombus formation of the graft or native coronary artery may each predispose to the development of myocardial infarction.

The history, ECG, and cardiac enzyme analysis revealed no new evidence of myocardial infarction. Specifically, the perioperative course was uncomplicated by the acute onset of chest pain, and the chest pain manifest 6 weeks after cardiac surgery was atypical for myocardial infarction. The ECG before coronary artery bypass graft surgery showed q waves over the inferior and anterolateral distribution. Serial ECGs showed no new evidence of q-wave development of myocardial infarction or of evolutionary ST-T-wave changes of ischemia or injury. Serial cardiac enzymes were not elevated. Before coronary artery bypass graft surgery, there was moderate impairment in ventricular function. Acute myocardial infarction of the left ventricle would probably have resulted in further deterioration of ventricular function and thereby predisposed to pulmonary congestion and edema. In this patient, neither symptoms of orthopnea or paroxysmal nocturnal dyspnea nor radiographic evidence of pulmonary edema was present.
Could this patient have had right ventricular infarction? Significant right ventricular infarction is characteristically associated with jugular venous distension and hypotension, with a low cardiac output. The initial arterial pressure was well preserved, there was no evidence of right ventricular injury pattern on the ECG, and the marked peripheral edema and jugular venous distension were disproportionately greater than what would be expected for acute right ventricular infarction.

Was There Evidence of Pulmonary Embolus and Development of Pulmonary Hypertension?

The patient was obese, had depressed left ventricular function and a thrombus in the apex of the left ventricle, and had recently undergone saphenous vein removal and cardiac surgery. These features increase the likelihood for thromboembolic disease. A massive pulmonary embolus predisposes to systemic hypotension, acute pulmonary hypertension, right ventricular dilatation, acute right heart failure, and hypoxemia. A common presenting symptom is dyspnea. Rarely, myocardial ischemia may manifest because of either decreased arterial pressure in the setting of coronary artery disease or reduced transmural myocardial perfusion pressure secondary to lowered aortic diastolic pressure and increased right atrial pressure. In this patient, however, the clinical findings did not suggest a massive pulmonary embolus. Specifically, the clinical presentation occurred gradually over 4 weeks, and dyspnea was not the presenting complaint. Furthermore, the dull retrosternal chest pain was more in keeping with a pericardial origin; the marked extent of the peripheral edema and the jugular venous distension were disproportionate to that expected for an acute event, and the initial blood pressure was well maintained. It is important to note that on discharge from the hospital after cardiac surgery, the patient was receiving treatment with warfarin and the prothrombin time was within the therapeutic range for anticoagulation, making it less likely for thromboembolism to occur.

Could the patient have had recurrent episodes of smaller pulmonary emboli? The signs and symptoms of recurrent small pulmonary emboli may be subtle and nonspecific, and generally the clinical picture is one of chronic pulmonary hypertension and the development of cor pulmonale. In this patient, there was no clinical, ECG, or radiographic evidence of pulmonary hypertension. Specifically, there was no clinical evidence of an accentuated pulmonary component of the second heart sound or of a right ventricular parasternal lift. There was no ECG evidence of a rightward shift of the QRS axis, right ventricular hypertrophy, right bundle branch block, or an S, Q3, T3 pattern. Furthermore, chest radiography did not reveal evidence of either parenchymal changes (such as a wedge-shaped opacity) or pulmonary arterial changes (such as dilatation of the proximal pulmonary arteries or abrupt termination of a more peripheral pulmonary vessel). However, it should be noted that in the Urokinase-Streptokinase Pulmonary Embolism Trial and the PRIOPET trial, clinical
symptoms and signs, the ECG, and the chest radiograph did not differentiate patients with pulmonary embolism from those without emboli. Thus, recurrent pulmonary embolic disease predisposing to pulmonary hypertension and progressive right heart failure, although of low probability, cannot be excluded as a cause of this patient’s problem.

Was There Evidence of Pericardial Disease?

The presentation of progressive peripheral edema, jugular venous distension, a mildly dilated heart, absence of heart murmurs, lack of pulmonary edema, and low-voltage QRS on the ECG is in keeping with pericardial disease. Pericardial effusion associated with cardiac tamponade, constrictive pericarditis, or effusive/constrictive pericarditis in which there is coexistence of effusion plus constriction might each have led to the symptoms and signs found in this patient.

Pericarditis occurs frequently within the first 2 days after cardiac surgery, and pericardial effusion may occur in up to 80% of patients within 10 days of a cardiovascular operation. Postpericardiotomy syndrome is reported to occur in 10% to 40% of patients within 1 to 4 weeks after a cardiac operation. The incidence of cardiac tamponade after cardiac surgery ranges from 1% to 2.5%; tamponade occurring immediately after surgery is usually due to bleeding, whereas tamponade occurring 2 weeks after surgery usually results from a postpericardiotomy syndrome. Constrictive pericarditis has been reported to occur as early as 2 weeks after cardiac surgery. Thus, the initial presentation of peripheral edema within 2 weeks of cardiac surgery and the severe right heart failure within 6 weeks of cardiac surgery do not negate the diagnosis of effusive/constrictive pericarditis. The incidence of constrictive pericarditis within 7 weeks of cardiac surgery is approximately 0.3%.

There were at least three factors that could predispose this patient to develop a pericardial effusion or hemorrhagic pericardium. First, the patient was receiving anticoagulation therapy with warfarin for treatment of a thrombus in the left ventricle; one complication of this therapy is bleeding. Second, an automatic implantable cardiac defibrillator patch electrode was inserted into the epicardium as part of a multicenter research protocol; one complication is bleeding. Third, a bloody purulent discharge was noted at the site of the generator in the left upper quadrant. Infection of the generator can track to the pericardial space and predispose to a pyogenic pericardial effusion. The absence of a history of febrile episodes or chills is surprising but may be due to antibiotic therapy and/or localization of the pericardial infection. The presence of a polymorphonuclear leukocytosis further suggests the presence of infection. The results of cultures from the generator site and from the blood are not provided; if pyogenic pericardial effusion is present, surgical drainage of the pericardial space would probably be required in the management of this patient.

The absence of pulsus paradoxus and the presence of mild cardiomegaly on chest radiograph are more suggestive of constrictive pericarditis or effusive/constrictive pericarditis. However, it is important to note that the finding of a normal heart size or mild cardiomegaly does not preclude a diagnosis of cardiac tamponade.
Right ventricular pressure trace shows an elevated pressure of 78/18 mm Hg within the body of the right ventricle (top panel). As the catheter was advanced to the region of the right ventricular outflow tract, a pressure of 37/15 mm Hg was obtained (bottom panel). The pulmonary artery pressure was 23/18 mm Hg. Thus, systolic gradients were detected within the body of the right ventricle and also at the outflow tract. The cardiac output was 2.64 L/min, and the cardiac index was 1.45 L·min⁻¹·m⁻².
Fig 6 and 7. Intraoperative transesophageal echocardiogram illustrates a markedly dilated right atrium with the atrial septum bulging toward the left, findings indicative of right atrial hypertension (left panel). Anteriorly, a large cystic mass compresses the body (left panel) and outflow tract (right panel) of the right ventricle, markedly reducing the cavitary size of the ventricle and producing a slittlike right ventricular outflow tract. Doppler color flow analysis illustrated markedly turbulent flow in the right ventricular outflow tract, indicative of obstruction (right panel). RA indicates right atrium; LA, left atrium; RV, right ventricle; LV, left ventricle; RVOT, right ventricular outflow tract; PA, pulmonary artery; AO, aorta.

The rapid accumulation of a small volume (for example, 100 mL) of blood, especially if loculated within the pericardial space, may induce hemodynamic compromise and evoke a reduction in stroke volume and cardiac output. In the postsurgical patient, localized pericardial hematoma or loculated pericardial effusion are more prone to occur for the following reasons. First, bleeding may occur from suture sites and result in hematoma formation; the administration of anticoagulants may cause excessive bleeding. Second, pericardial adhesions can predispose to loculation of the effusion. Loculated effusions or localized hematoma may cause localized compression of the right atrium, right ventricle, or superior vena cava and result in cardiac tamponade.

No information is provided as to the nature of the X or Y descent of the jugular venous pressure waveform, findings that are used in inferring the presence or absence of cardiac tamponade or constrictive pericarditis. The presence of sinus tachycardia (=120 to 130 beats per minute) may have precluded accurate analysis of the jugular venous pressure waveform. Recent reports11-13 have indicated that pericardial hematoma producing localized cardiac compression of right atrium or right ventricle may not manifest with the classic clinical or hemodynamic findings of cardiac tamponade (pulsus paradoxus, rapid systolic X descent of the jugular venous waveform, presence of respiratory variation of the right atrial pressure trace, or equalization of right atrial, pulmonary artery diastolic, or pulmonary capillary wedge pressures). Rather, the clinical and hemodynamic findings detected in patients with localized cardiac compression may be those of constrictive pericarditis (absent pulsus paradoxus, rapid diastolic Y descent of the jugular venous waveform, loss of respiratory variation in right atrial pressure trace, and the “dip and plateau” diastolic ventricular pressure trace).

In a recent study,14 in only two of seven patients were the classic clinical and hemodynamic features of cardiac tamponade detected postoperatively, even though cardiac compression was present. Constrictive pericarditis or a loculated pericardial effusion or hematoma may each have predisposed to right heart failure in this patient. The onset of hypoten-
Alkaline phosphatase 102 U/L
Total protein 7.4 g/dL
Albumin 3.0 g/dL
Aspartate aminotransferase 16 U/L
Alanine aminotransferase 34 U/L
Lactate dehydrogenase 347 U/L
Total bilirubin 1.1 mg/dL
Cholesterol 142 mg/dL
Triglycerides 96 mg/dL
White blood cell count 22.6 mm³
(89% polymorphs, 6% lymphocytes)
Hemoglobin 12.1 g/dL
Platelets 619 mm²
Prothrombin time 20.0 s
Partial thromboplastin time 35.8 s

sion and further increase in sinus tachycardia 48 hours after hospitalization may have resulted from the development of cardiac tamponade or volume depletion aggravating preexisting constrictive pericarditis. The critical information required in this patient is the presence or absence of (1) cardiac tamponade, (2) pericardial thickening, (3) pericardial infection, and (4) pulmonary hypertension. The diagnostic test that would provide the greatest information would be a transthoracic echocardiogram.

Transthoracic Echocardiogram

This study was technically difficult. In the parasternal long-axis view, the right ventricle was not visualized, and in the space anterior to the site where the right ventricle is normally situated, there was a cystic mass. In the apical views, the right ventricle was found to be markedly reduced in size, suggesting compression from an extrinsic mass. The right atrium appeared to be dilated. The left ventricle was normal in size, with moderate global impairment in systolic function.

Right Heart Catheterization

Right heart catheterization revealed right atrial hypertension with a and v waves of 23 and 22 mm Hg, respectively; the mean pressure was 19 mm Hg. Rapid X and Y descents were noted in the right atrial pressure waveform; a lack of respiratory variation was also present (Fig 2). The pulmonary capillary wedge pressure showed a and v waves of 16 and 14 mm Hg, respectively; the mean pressure was 14 mm Hg. Respiratory variation was present in the tracing (Fig 3). The right ventricular pressure trace showed an elevated pressure of 78/18 mm Hg within the body of the right ventricle (Fig 4). As the catheter was advanced to the region of the right ventricular outflow tract, a pressure of 37/15 mm Hg (Fig 5) was obtained. The pulmonary artery pressure was 23/18 mm Hg. Thus, systolic gradients were detected within the body of the right ventricle and also at the outflow tract. The cardiac output was 2.64 L/min, and the cardiac index was 1.45 L·m⁻¹·m⁻².

Intraoperative Transesophageal Echocardiogram

An intraoperative transesophageal echocardiogram illustrated a markedly dilated right atrium with the atrial septum bulging toward the left, findings indicative of right atrial hypertension (Fig 6). Anteriorly, a large echolucent mass compressed the body (Fig 6) and outflow tract (Fig 7) of the right ventricle, markedly reducing the cavity size of the ventricle and producing a slitlike right ventricular outflow tract (Fig 7). Doppler color flow analysis illustrated markedly turbulent flow in the right ventricular outflow tract, indicative of obstruction (Fig 7). The left ventricle was normal in size with moderate global impairment in systolic function, and mild mitral regurgitation was present.

Surgical Findings

During the operation, a hematoma was found to be compressing the right ventricle. The mass was drained, and there was immediate reexpansion of the right ventricle and the right ventricular outflow tract (Fig 8). On removal of the hematoma, the saphenous vein graft to the left anterior descending coronary artery was found to be actively bleeding. This was controlled with a single monofilament suture. The orifice in the saphenous vein graft was created by the corner of the patch electrode of the automatic implantable cardiac defibrillator device, which was in close proximity. The contained pericardial hematoma acted as a compressive force on the vein, temporarily preventing further bleeding. Culture of the hematoma yielded Staphylococcus nonaureus species.

Final Case Discussion

In this patient, pulsus paradoxus was absent, there was absence of respiratory variation of the right atrial pressure trace, and a rapid Y descent of the right atrial pressure trace was noted. These findings are more in keeping with constrictive pericarditis than cardiac tamponade. However, the right atrial pressure was elevated and significantly higher than pulmonary capillary wedge pressure; right ventricular systolic pressure showed evidence of obstruction within the body of the ventricle, and the pulmonary capillary wedge pressure revealed respiratory variation. The above combination of findings indicates localized compression of the right ventricle. The transesophageal echocardiogram demonstrated a large echolucent mass producing marked compression of the right ventricle and the outflow tract. At surgery, a pericardial hematoma was found and decompressed; the source of the bleeding was the saphenous vein graft to
the left anterior descending coronary artery. Culture of the pericardial hematoma revealed Staphylococcus non-
aureus species. This infection originated from the gener-
ator site of the automatic implantable cardiac defibrillator
device, since culture from this site also yielded Staphylococcus nonaureus species.

Localized pericardial hematomas may occur after car-
diac surgery. They are more commonly localized to the
anterior aspect of the heart and may cause compression of
the right atrium and right ventricle. The physiology of
such compression should be that of cardiac tamponade.
However, this case, as well as recent reports, shows that
localized hematoma may not produce the expected clinical
and hemodynamic findings of cardiac tamponade but
rather produce findings that simulate constrictive pericar-
ditis. A hematoma produces a noncompliant localized
mass in the pericardium; in contrast, a pericardial effu-
sion is under pressure and compliant. These differences in
compliance presumably account for the variation in the
clinical and hemodynamic findings between tampon-
ade and hematoma.

In this case, compression of the right ventricle pro-
duced right ventricular obstruction resulting in severe
right heart failure. A localized pericardial hematoma
causing localized cardiac compression, hypotension, and
right heart failure after cardiac surgery may be easily
detected by transesophageal echocardiography. The
value of transesophageal echocardiography in the
hemodynamically compromised patient after cardiac sur-
gery is highlighted in this case.

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