A 44-Year-Old Mentally Handicapped Institutionalized Man With Acute Circulatory Collapse†

Ward Cassells, MD; Francisco Fuentes, MD; David Mishkel, MD; Wilson E. SooHoo, MD

Clinicopathological Conference

Case Presentation

A 44-year-old man with a history of hypothyroidism and seizure disorder presented to Hermann Hospital for further evaluation after being found unconscious. The patient, who was mentally handicapped and resided in a skilled nursing facility, had been in his usual state of health until the day of admission. Up to that time, he was able to perform limited self-care skills, walk and eat without assistance, and report daily to the nurse’s station to receive prescribed medications. On the day of admission, he did not report as usual and was found in his room unresponsive, pale, and with cyanotic-appearing extremities. No peripheral pulse was palpated, and a blood pressure measurement could not be obtained. Emergency medical services (EMS) personnel arrived and immediately administered a bolus of 2.5 L normal saline; subsequently, a weak and rapid femoral pulse was appreciated, and blood pressure was measured at 110/60 mm Hg. No chest compressions were performed. ECG tracings obtained by EMS personnel were not available or reported.

On arrival at the emergency department, the patient was awake and responsive but disoriented. He received 1 L of normal saline after the emergency placement of a central venous catheter. His femoral pulse was weak at 96 beats per minute, but peripheral pulses could not be felt. His systolic blood pressure measured 72 mm Hg by palpation. Respiratory rate was 20 breaths per minute. Rectal temperature measured 99.8°F. He was mildly dyspneic and tachypneic with change in position. On examination, no jugular venous distension was reported, heart sounds were not well characterized, breath sounds were audible but distant, and there was mild bilateral pedal edema. An emergency ECG revealed normal sinus rhythm, rate of 83 beats per minute, reduced voltage in the limb leads only, and no ST-segment or T-wave changes (Fig 1). Chest radiograph showed a large cardiac silhouette, a left pleural effusion, and pulmonary venous redistribution. Arterial blood gas measurement on 4 L \text{O}_2 \text{ by nasal cannula was } \text{pH} 7.36; \text{PCO}_2, 30; \text{PO}_2, 97; \text{HCO}_3^-, 17; \text{ and BE, 6 (no } \text{O}_2 \text{ saturation reported). After insertion of a radial arterial catheter, we transferred the patient to the intensive care unit for continued management (cuff blood pressure was now 115/64 beats per minute, and pulse was 97 beats per minute).

The nursing home staff denied the patient having any history of fever, chills, nausea, complaints of pain, change in character of stool, change in bowel or bladder habits, or bizarre behavior preceding these events. The patient had a past medical history of mental retardation and seizure disorder, both since childhood and of unknown etiology, and of hypothyroidism, which was diagnosed 5 years before admission. His current thyroid function status was unclear. His surgical history included an appendectomy and cholecystectomy in his young adulthood. He had no history of cigarette smoking or alcohol or drug abuse. His sexual history was unknown. His family history was noncontributory. Medications included clonazepam, 0.5 mg qAC and 0.25 mg qHS; valproic acid, 500 mg at 6:00 AM, 6:00 PM, and 9:00 PM and 250 mg at 12:00 noon; Synthroid, 0.025 mg QD; propranolol, 20 mg every 12 hours; selenium, by mouth daily; erythromycin, 1 g IV; and Rocephin, 1 g IV (one dose) in the emergency department. He had no known drug allergies.

Physical Examination

The patient was well nourished, short in stature, awake, drowsy, and disoriented. He was tachypneic but not in extreme distress. His temperature was 100.4°F (oral); pulse, 100; blood pressure, 100/70 mm Hg; and respirations, 28. Examination of his ears, nose, and throat revealed no contusions or lacerations; pupils were symmetrical and reactive; disc margins were sharp; and conjunctivae and oropharynx were within normal limits. The neck was supple; there was reduced intensity of carotid pulsations; jugular venous pulsations were not visualized; and no thymomegalgy or lymphadenopathy was noted. The chest examination revealed pectus excavatum, diffuse wheezing over the right lower lobe, diminished breath sounds over the left lower lobe, and no crackles. Cardiovascular examination revealed tachycardic, distant heart sounds, and “regular” rate but every third heart beat was inaudible, and no murmur or rub was heard. His abdomen was soft and nontender with bowel sounds.

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present, surgical scars were well healed, and there was no hepatosplenomegaly. Genitourinary and rectal examinations revealed normal genitalia; there was normal sphincter tone (guaiac negative). Extremities had cyanotic digits and were cool to touch with no clubbing; there was mild lower extremity pitting edema with no rashes. On neurological examination, the patient was drowsy but easily aroused to alertness, answered questions, followed commands appropriately, and oriented with prompting; no cranial nerve, motor, or sensory deficits were noted, and the reflexes were not tested.

**Hospital Course**

Hemodynamic and cardiovascular monitoring was intensified (Fig 2). Intravenous crystalloid was continued. Oxygen saturation improved to 95% with application of a 50% face mask. Repeat serum chemistries revealed normal serum sodium and potassium levels. Disseminated intravascular coagulation screen was negative. Blood cultures and urine cultures were submitted, and the antibiotic regimen was changed to intravenous ampicillin/sulbactam. A series of diagnostic and therapeutic procedures were performed.

**Case Discussion**

**Ward Casscells, MD (Department of Internal Medicine, Cardiology Division)**

This 44-year-old man, who had a poorly characterized history of retardation, seizure disorder, and hypothyroidism and was receiving thyroxine, clonazepam, valproic acid, and propranolol (possibly for hypertension), was found unconscious and in shock, with fever, wheezing, distant heart sounds, peripheral edema, and cyanosis. The ECG is notable for low voltage in the limb leads only, raising the possibility of pericardial effusion, although the cause usually is obesity or emphysema. The ECG also has tall R waves in V₁ and V₂, which could indicate (1) an old true posterior myocardial infarction, (2) right ventricular strain or hypertrophy (although usually accompanied by inverted T waves in V₁ and V₂, deep S waves in V₅ and V₆, or an S₁, Q₃, or T₃ pattern or (3) low lead placement (Fig 1).

The chest radiograph shows a large cardiac silhouette consistent with cardiomegaly or pericardial effusion, a left pleural effusion (which is noteworthy because heart failure is more often accompanied by right pleural effusion and pericarditis by left pleural effusion), and pulmonary venous redistribution consistent with either heart failure or tamponade. An epicardial lucency ("fat stripe") is not seen, but this does not rule out the diagnosis of pericardial effusion. The film is underpenetrated, so it is not possible to exclude left atrial enlargement or valvular, coronary, or pericardial calcification. Likewise, underlying lung disease cannot be excluded.

The differential diagnosis of shock, fever, and unconsciousness is long, but the distant heart sounds, wheez-


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Urinalysis: pH 6.0; SPGR, 1.010; trace protein, ketone; nitrite-positive microscopy: 3 to 5 WBCs, 10 to 20 RBCs, few bacteria, few hyaline casts.

ABG (40% face mask): 7.45; Pco₂, 28; Po₂, 76; HCO₃⁻, 20; BE-2; oxygen saturation, 93%.

Chest radiograph (portable): enlarged cardiac silhouette, left pleural effusion, no nodules or infiltrates seen. Computed tomography scan of head without contrast (done in emergency department); dilated ventricles and increased atrophy for age; no masses, edema, or midline shifting.

TG indicates triglycerides; ALT, alanine transaminase; AST, aspartate transaminase; WBC, white blood cell; HGB, hemoglobin; HCT, hematocrit; PLT, platelets; PT, prothrombin time; PTT, partial thromboplastin time; SPGR, specific gravity; RBC, red blood cell; and ABG, arterial blood gas.

...tured aortic aneurysm. If such an aneurysm had dissected retrograde and occluded the ostium of the right coronary artery, it could cause true posterior infarction. An aneurysm of the ascending aorta can also dissect into the pericardium and cause acute tamponade, which can stabilize and give the type of presentation seen here. The rather wide mediastinum on the chest film is consistent with this possibility. I presume the blood pressure was measured in both arms, but equal blood pressures do not rule out the possibility of an aortic dissection.

A true posterior infarction can cause cardiovascular collapse in a previously compromised heart or when complicated by bradycardia, heart block, involvement of the right ventricle, or rupture of the septum or posterior papillary muscle. With rupture, a thrill and harsh murmur should be heard, but the murmur can be surprisingly soft in a hypotensive patient and is easily missed in a noisy emergency department.

Cardiac tamponade is suggested by the distant heart sounds, cardiac silhouette, and low limb lead voltage, but it is usually associated with jugular venous distension (not seen here) unless the patient is hypovolemic. The edema and lack of hemoconcentration of the blood tests argue against dehydration. However, the key finding is that after appropriate initial treatment (oxygen and 3 L of saline), the patient was found to have 48 mm Hg of pulsus paradoxus. Although pulsus paradoxus is regularly seen in chronic obstructive pulmonary disease or asthma, and occasionally with pulmonary embolism, this degree of pulsus paradoxus strongly suggests a diagnosis of pericardial tamponade.¹

The immediate goals are (1) support of the blood pressure with saline; (2) complete blood count and typing and crossing for possible blood transfusion; (3) arterial blood gas analysis and oxygen therapy; (4) stat glucose, thyroxine, creatinine, ammonia, troponin, CK-MB, calcium, sodium, potassium, drug levels, blood cultures, and viral serologies; (5) placement of a pulmonary artery catheter (to aid in volume management, to check for equalization of end-inspiratory pressures in the cardiac chambers, to look for an oxygen step-up indicative of acute rupture of the ventricular septum, and to look for a dip-and-plateau “square-root sign” suggestive of pericardial constriction or restrictive cardiomyopathy); and (6) a transesophageal echocardiogram.

The echocardiogram is less sensitive than a magnetic resonance scan for detection of aortic dissection, but it is faster, can be done without placing the patient in a magnet—which hinders other diagnostic and therapeutic procedures—and also gives a great deal of other information: (1) Doppler estimation of the pulmonary artery pressure and of valvular stenoses, (2) color flow mapping for mitral regurgitation or ventricular septal defect, (3) wall motion analysis indicating infarction, (4) hypertrophy, (5) visualization of an embolus in the main pulmonary artery, (6) a tissue signal suggesting infiltrative disease or scarring, and (7) left main coronary stenosis. Most important, however, the echo will determine the amount of pericardial fluid and the presence of diastolic collapse of the right atrium and right ventricle, indicative of tamponade. The echo may also indicate pericardial thickening and calcification (suggestive of tuberculous pericarditis), a tumor mass, or...
echo-dense fluid and fibrinous strands suggestive of tumor, thrombus, or tuberculosis.

Once the patient has been stabilized, there may be time to get more information from the history and examination. Has anyone else in the institution been sick? Has the patient received immunizations for rubella, hepatitis B, pneumococcal pneumonia, and influenza? Is there a history of a positive tuberculin skin test or of mononucleosis?

With fluid replacement, it may be possible to get a better look at the neck veins. A loss of the Y descent (the collapse of the jugular venous pulse with opening of the tricuspid valve) would be consistent with pericardial tamponade, whereas prominent X and Y descents—the so-called M or W pattern—would be more indicative of pericardial constriction, as would a Kussmaul sign and a pericardial knock (in any case, pericardial constriction does not present with acute hypotension and a large cardiac silhouette). In contrast, in cardiac tamponade, the neck veins generally do not increase with inspiration and no knock is heard, although an S4 gallop may be present if there is underlying myocardial disease. A closer examination may also indicate signs of hypothyroidism, such as diminished deep tendon reflexes, coarse hair, skin, or voice, etc. A murmur of pulmonary stenosis, fixed splitting of A2, or aortic ejection click could suggest a syndrome that includes mental retardation and congenital heart defects (Down’s syndrome, Hunter’s syndrome, Hurler’s syndrome, Williams’s syndrome, de Lange’s syndrome, congenital rubella, etc).2

The laboratory tests are notables for hyponatremia, hyperkalemia, elevated liver enzymes and bilirubin, anemia, thrombocytopenia, a normal white blood cell count with a left shift, a low cholesterol level, and slight elevation of the prothrombin time. These could indicate any number of noncardiac problems. For example, the patient could have adrenal insufficiency, hepatitis, nephritis, or pleurisy. Alternatively, all these organs could be affected by the same process, such as a malignancy, infection, systemic lupus, sarcoidosis, and so forth. These possibilities will all need to be investigated, but there is a good chance that these problems are all due to one underlying problem: malignant or tubercular pericarditis (probably originating at some other site, such as the lung) complicated by tamponade and secondarily by pulmonary congestion, hepatic congestion, malnutrition, anemia, thrombocytopenia, and inappropriate antidiuretic hormone (alternatively, the hyponatremia and hyperkalemia could be due to tubercular or malignant adrenal involvement).

The differential diagnosis of pericarditis is headed by idiopathic and viral causes, followed by cancer, Dressier’s syndrome, lupus, hypothyroidism, sarcoidosis, and tuberculosis or other mycobacteria. However, in chronic pericarditis with tamponade, the most likely cause is malignancy. In a malnourished, immunocompromised, or institutionalized patient, it is also important to consider tuberculosis, particularly given its increasing occurrence in the United States and the rapid increase in drug-resistant strains. Thus, if the initial echocardiogram and PA line confirm the diagnosis of pericardial tamponade and do not indicate primary diagnosis of congestive heart failure or myocardial infarction, it will be important to obtain not only pericardial fluid (which is often falsely negative for cancer and mycobacteria) but also pericardial tissue by an open or closed tech-
nique, even if the echocardiogram does not show a particulate pericardial fluid or a shaggy, thickened, or calcified pericardium.

These latter findings would certainly be expected in a late stage of tuberculous pericarditis, but it would be a mistake to wait for them before considering the diagnosis, particularly if the hemodynamics reveal a constrictive pattern after removal of fluid: “effusive-constrictive” pericarditis is highly suggestive of tuberculosis or cancer. If the clinical suggestion is high, a negative result on an insensitive test does not exclude a diagnosis. Do not delay isolation of the patient and initiation of four antimycobacterial agents. In addition to the smear for acid-fast bacilli, there are recently described tests that use in situ hybridization and polymerase chain reaction to make rapid diagnoses of bacterial and fungal infections.

In addition to obtaining pleural and pericardial fluid and tissue, a tuberculin skin test and serologic tests for hepatitis B, hepatitis C, syphilis, rubella, and HIV should be performed. Thyroid function tests must be checked, as should a morning cortisol. If the patient is not stable after volume replacement and pericardiocentesis (leaving the catheter in place to monitor pressure and permit ongoing drainage), the patient should receive broad-spectrum antibiotics for presumed septic shock.

The patient’s loss of consciousness could certainly be explained by the profound hypotension, clonazepam overdose, hypothryoidism, head trauma, meningitis, stroke, seizures, or low sodium level. The computed tomography scan to rule out intracranial masses (caused by hemotoma, fungus, tumor, etc), hydrocephalus, thickening of the meninges, and stroke was certainly appropriate. If the patient does not wake up completely, a lumbar puncture should be performed and the fluid tested for tuberculosis, fungus, syphilis, cancer, etc.

The peripheral blood smear should be examined, and levels of iron, folic acid, vitamin B12, haptoglobin, and hemoglobin should be checked. A bone marrow biopsy may be necessary. If monitoring of pulse oximetry does not reveal prompt improvement in oxygen saturation, a lung scan should be performed, focusing on the areas of the lung that are relatively normal.

In summary, this patient is most likely to have pericardial tamponade due to malignancy or, less likely, tuberculosis. Either etiology could explain the many systemic findings, although other equally serious and treatable conditions, such as aortic dissection, myocardial infarction, congestive heart failure, pulmonary embolism, adrenal insufficiency, sepsis, and myxedema, must be excluded.

Francisco Fuentes, MD (Department of Internal Medicine, Cardiology Division)

Transthoracic echocardiography revealed a large pericardial effusion with right atrial and right ventricular collapse in diastole. The left ventricle was normal in size and function as were the valves, with the exception of mild tricuspid regurgitation. Mild right atrial and right ventricular dilations were noted.

David Mishkel, MD (Department of Internal Medicine, Cardiology Division)

Pulmonary artery catheterization revealed equal right atrial and pulmonary capillary wedge pressures of 14 mm Hg. The right atrial waveform revealed V waves consistent with tricuspid regurgitation and small X waves consistent with a dilated right atrium (Fig 3). The expected loss of Y descent was not seen, presumably because of the tricuspid regurgitation.

Pericardiocentesis yielded yellowish-clear nonbloody fluid, restored blood pressure to 114/62 mm Hg, and eliminated the pulsus paradoxis and respiratory variation in the R-wave amplitude.

Wilson SooHoo, MD (Department of Pathology and Laboratory Medicine)

Specimens were received in the surgical pathology, cytopathology, and microbiology laboratories. The surgical pathology specimen consisted of two red-brown and yellow fibrofatty tissue fragments of pericardium measuring 3.0×2.0×0.4 and 3.0×1.5×0.7 cm with shaggy surfaces and cut sections that revealed membranous tissue up to 0.4 cm thick. Hematoxylin and eosin-stained sections revealed edematous thickened pericardium with fibrinous material at the surface containing red blood cells, neutrophils, and reactive mesothelial cells (Figs 4 and 5). The blood vessels were surrounded by small to moderate numbers of mature lymphocytes (Fig 6). There were no granulomas, viral inclusions, calcifications, areas of necrosis, or evidence of malignancy. Brown-Brenn, Gomori’s methamine silver, Kinyoun’s acid-fast bacillus, and Ziehl-Neelsen stains were negative for bacteria, fungi, Pneumocystis carinii, and mycobacteria.

The cytopathology specimen consisted of 180 mL of bloody pericardial fluid. Papanicolaou and DiffQuik-stained cytopsin preparations revealed reactive mesothelial cells in a background of acute and chronic inflammation. There were no fungi, viral inclusions, or multinucleated cells.

The microbiology specimens consisted of both pericardial tissue and pericardial fluid. A fluorochrome stain performed on a tissue preparation revealed occasional mycobacteria. Gram and methenamine silver stains performed on tissue preparations were negative for bacteria, fungi and P carinii. Gram, fluorochrome, and methenamine silver stains performed on the fluid specimen were negative for bacteria, mycobacteria, fungi, and P carinii. Final bacterial, viral, fungal, and mycobacterial cultures performed on both pericardial tissue and pericardial fluid specimens were negative.

The combination of fibrinous pericarditis and a positive fluorochrome stain for mycobacteria is diagnostic of mycobacterial pericarditis. One can diagnose mycobacterial pericarditis when one or more of the following are true: (1) pericardial tissue or fluid culture is positive for mycobacteria; (2) acid-fast organisms and/or caseating granulomas are demonstrated in a pericardial biopsy specimen; or (3) there is extrapericardial bacteriological or histological evidence of active tuberculosis in conjunction with a major pericardial effusion or pericardial thickening by imaging studies.

The diagnosis can be made by culturing mycobacteria from pericardial tissue and pericardial fluid. Demonstration of mycobacteria in tissue or fluid using histological stains is also a way to establish the diagnosis. Two types of acid-fast stains are used: carbolfuchsins (Kinyoun and Ziehl-Neelsen) and fluorochrome (auramine O and auramine-rhodamine) stains. Of these,
the fluorochrome stains are considered by some to have greater sensitivity, but this experience probably varies from institution to institution. Indeed, in the present case, the auramine O stain was positive, whereas the Kinyoun stain was negative. In addition, the diagnosis of tuberculous pericarditis can be made in a patient with a pericardial effusion or pericardial thickening by ultrasound who also has known tuberculosis elsewhere.

However, the sensitivities of histological and microbiological examination for mycobacteria are low. Acid-fast bacilli were found in stained smears of pericardial fluid in only 42% of cases in one series. Other series demonstrated sensitivities of 56% and 50% for fluid cultures. These low percentages generally are attributed to low numbers of mycobacteria in the fluid. The latter two series had pericardial biopsies with histological features of mycobacterial infection in 70% and 83% of patients. Measurement of adenosine deaminase activity in pericardial fluid has shown great promise in one small series in detecting mycobacterial pericarditis; however, larger studies are needed to establish its true use.

There are acute, subacute, and chronic stages of mycobacterial pericarditis. The acute stage can be subdivided into an early effusive stage and a later fibrinous stage. In the effusive stage, serosanguinous fluid, some-
times containing lymphocytes, is present in the pericardial space. This effusion can be massive, sometimes more than 2 L. This is followed by the fibrinous stage, when the pericardium is covered by a layer of fibrin, which results in its shaggy appearance. Although the causes of fibrinous pericarditis are many, the combination of the fibrinous inflammation and a positive fluorochrome stain for mycobacteria makes the diagnosis in the present case possible.

The subacute stage is characterized by the classic lesions of tuberculosis—granulomatous inflammation with typical epithelioid histiocytes and Langhans giant cells. Often, caseous necrosis is present, and special stains may reveal mycobacteria. The chronic phase is characterized by a proliferation of fibroblasts, marked thickening of the pericardium with virtual obliteration of the pericardial cavity, and consequent constriction. Calcification can be seen at this stage. Complications in the chronic stage include constrictive pericarditis and acute pericardial tamponade.11

Mycobacterial pericarditis is rare and invariably is secondary to a focus of mycobacterial infection elsewhere in the body. The primary focus usually is the lung, and the pericarditis can be due to hematogenous spread or to contiguous spread from mediastinal or hilar lymph nodes.11 Signs and symptoms are similar to those associated with tuberculosis at any other site; in addition, dyspnea, orthopnea, chest pain, and ankle swelling are more frequent in patients with tuberculous pericarditis than in those with tuberculosis without pericarditis.9 Physical findings include cardiomegaly (95%), pericardial friction rub (84%), tachycardia to more than 100 beats per minute (83%), pulsus paradoxus (71%), hepatomegaly (65%), neck vein distension (61%), pleural effusion (58%), and distant heart sounds (56%) (Fowler). Complications include constrictive pericarditis, pericardial tamponade, and myocarditis,3 which when unprevented and untreated can result in a mortality rate of as high as 40%.9 The treatment includes standard antituberculous drugs, corticosteroids, and, when necessary, pericardiectomy.

*Mycobacterium tuberculosis* is the pathogen in almost all cases of mycobacterial pericarditis; however, cases involving *M. chelonei* and *M. avium-intracellulare* have been reported.11 There is an increased incidence of extrapolunmonary tuberculosis, including mycobacterial pericarditis, in patients with AIDS. Treatment of *M. tuberculosis* appears to be as effective in patients with AIDS as in those without AIDS. Unfortunately, atypical mycobacteria, especially *M. avium-intracellulare*, are poorly responsive to antituberculous therapy and have an increased incidence in AIDS patients.9

**Final Diagnosis**

Cardiac tamponade secondary to tuberculous pericarditis.

**References**

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