A 39-Year-Old Man With Anasarca*

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A 39-year-old man presented to the emergency department with a 2-week history of mild abdominal pain, abdominal distention, and dyspnea. He had a nonproductive cough with sharp chest pain, chills, night sweats, anorexia, and early satiety. A 20-lb weight gain with bilateral lower extremity swelling was also reported. An outpatient course of doxycycline had failed to improve his symptoms. His medical and social history were otherwise unremarkable.

Physical Examination

The patient presented as a thin man who appeared to be chronically ill, with a pulse of 115 beats/min, BP of 107/76 mm Hg, a temperature of 96.7°F, and a respiratory rate of 24 breaths/min. A lung examination was notable for findings of dullness at both bases without crackles. Jugular venous pressure (JVP) was 17 cm H2O, but the emergency physician did not describe its waveform. Heart sounds were distant without gallop, rub, or a murmur. Peripheral pulses diminished on inspiration, and hepatomegaly with ascites and leg edema were noted without the stigmata of advanced liver disease.

Laboratory Data

Baseline hematology and chemistry measurements were notable for the following: WBC count, 13,100 cells/µL (normal differential); sodium level, 124 mmol/L; bicarbonate level, 16 mmol/L; aspartate aminotransferase level, 363 U/L; alanine aminotransferase level, 387 U/L; international normalized ratio, 1.6; total protein level, 7.3 g/dL; albumin level, 3.0 g/dL; and lactate dehydrogenase level, 118 U/L. An arterial blood gas measurement revealed a pH of 7.47, PaO2 of 66 mm Hg, and PaCO2 of 21 mm Hg. The results of a urinalysis were normal. An ECG was notable for sinus tachycardia at a rate of 110 beats/min with diffuse low-voltage and nonspecific ST and T-wave changes. A chest radiogram demonstrated large bilateral pleural effusions. Normal left ventricular systolic function, and a large pericardial effusion with right atrial and right ventricular diastolic collapse were noted on echocardiography, but indexes of diastolic function were not obtained.

Hospital Course

Emergent pericardiocentesis for cardiac tamponade was performed, during which 450 mL bloody fluid was removed and a pericardial drain was placed. The patient then was admitted to a hospitalist service. Subsequently, a diagnostic thoracentesis and paracentesis were performed. These demonstrated that the pericardial effusion was proteinaceous but the pleural effusion was transudative (total protein level, 1.9 g/dL; lactate dehydrogenase level, 39 U/L). Rheumatologic serology tests, bacterial, viral, and atypical mycobacterial cultures, and cytology were all negative. CT scans of the chest, abdomen, and pelvis only noted pleural fluid and ascites. Due to the persistent drainage of fluid from the pericardial catheter, a pericardial fenestration and biopsy were performed. The pericardial specimen was 3 mm thick and showed only nonspecific inflammation. Although a definitive diagnosis had still not been established, the recurrent pleural effusions were treated by bilateral thoracoscopic pleurodesis. However, the patient continued to have signs of systemic congestion, while systolic function of the left ventricle remained normal and pericardial fluid did not recur.

What was the cause of the patient’s anasarca?

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**Answer: Effusive-constrictive pericarditis**

At this point, the 11th day of hospitalization, a cardiology consultation was requested for the first time. On examination, the patient appeared ill with a pulse of 120 beats/min, a temperature of 100.9°F, a respiratory rate of 24 breaths/min, and a BP 115/72 mm Hg. Diffuse bilateral crackles, with normal heart sounds, but no rub, gallop, or murmur were noted. The JVP was elevated at 14 cm H₂O but unexpectedly showed a prominent y-wave descent. Ascites and lower extremity edema also were present.

**Diagnostic and Treatment edema Aspect**

The emergency physician’s initial impression of severe cardiac tamponade, based on the presence of a pericardial effusion, elevated JVP, and diastolic right heart collapse on echocardiography was correct. The rapid accumulation of fluid within the intrapericardial space alters right heart filling and leads to hypotension, pulsus paradoxus, and elevation of the JVP with loss of the y-wave descent. Right atrial collapse and right ventricular diastolic collapse usually precede pulsus paradoxus and severe hypertension. It was highly appropriate to obtain an echocardiogram early for this patient, but at the time of echocardiography it would have been useful to obtain left ventricular diastolic filling parameters, and transtricuspid and transmirtal inflow velocities, as well as pulmonary venous and superior vena caval blood flow velocities. This would have established the degree of ventricular diastolic dysfunction, and increased the respiratory variation of transmirtal and transtricuspid inflow velocities that are characteristic of cardiac tamponade.

Anasarca is an uncommon presentation of cardiac tamponade and should raise the suspicion of associated constrictive pericarditis, myocardial disease, right ventricular infarction, or tricuspid valvular disease. Similarly, the prominent c-wave of the jugular pulse is incompatible with uncomplicated cardiac tamponade. The JVP of 17 cm H₂O on admission to the emergency department would make noncardiac causes of anasarca highly unlikely. Furthermore, despite the drainage of pericardial fluid, the patients’ condition remained unchanged, indicating that his condition could not have been caused solely by cardiac tamponade. Tamponade was likely to have been complicated by concomitant pericardial constriction or coexisting heart disease. The distinction between uncomplicated cardiac tamponade and effusive-constrictive pericarditis is not a matter of esoteric academic interest, but is vital to correct clinical management. The diagnosis is established by persistent elevation and equalization of right atrial pressure and pulmonary capillary wedge pressure after the aspiration of pericardial fluid has reduced intrapericardial pressure to ≤ 0. If pericardiotesis had included right heart catheterization with simultaneous pericardial pressure measurement before and after the drainage, effusive-constrictive pericarditis would have been documented and myocardial or valvular disease ruled out.

Prior to the cardiology consultation, the emergency department physician recognized pericardial disease, obtained an echocardiogram, and performed pericardiotesis for tamponade. Subsequently, the hospitalist service completed an exhaustive workup, which excluded other causes of anasarca, but the diagnosis of pericardial disease or heart disease was not even considered despite all the evidence. Bilateral pleurodesis was performed without establishing a diagnosis, a step that should be reserved for symptomatic patients with a large, chronic idiopathic pleural effusion that fails to respond to therapy.

Restrictive cardiomyopathy is an uncommon condition that results from increased stiffness of the myocardium in mid-diastole and late diastole leading to abnormal ventricular filling, and simulates constrictive pericarditis with the typical dip-and-plateau configuration. As in patients with tamponade and classic constrictive pericarditis, systolic function and cardiac size are normal. In the patient presented above, the presence of a large pericardial effusion, however, made restrictive cardiomyopathy unlikely and favored effusive-constrictive pericarditis. Thus, to definitively evaluate the patient for effusive-constrictive pericarditis, cardiac catheterization (with an endomyocardial biopsy to exclude restrictive cardiomyopathy) was performed by the consultative cardiology service. Diastolic pressures in all four cardiac chambers were, as expected in a case of cardiac tamponade, equal and substantially higher than normal (Table 1), but the pressure waveforms with a prominent y-wave descent and a dip-and-plateau configuration were highly consistent with constrictive pericarditis (Fig 1). The findings of the coronary arteriogram and endomyocardial biopsy were normal. Thus, cardiac catheterization established the diagnosis of effusive-constrictive pericarditis and excluded the unlikely possibility of restrictive cardiomyopathy or other myocardial disease, in which the test results for the endomyocardial biopsy specimen would likely have been abnormal and the pressure equalization may not have been present.

**Table 1—Hemodynamics at Cardiac Catheterization**

<table>
<thead>
<tr>
<th>Cardiac Chamber</th>
<th>Pressure, mm Hg</th>
</tr>
</thead>
<tbody>
<tr>
<td>Right atrial pressure</td>
<td>18*</td>
</tr>
<tr>
<td>Right ventricular pressure</td>
<td>45/18</td>
</tr>
<tr>
<td>Pulmonary capillary wedge pressure</td>
<td>20*</td>
</tr>
<tr>
<td>Left ventricular pressure</td>
<td>110/20</td>
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</tbody>
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*Mean value.
With this evidence of effusive-constrictive pericarditis in hand, the patient was referred for a parietal and visceral pericardiectomy. Pericardiectomy for this condition must include the visceral pericardium, because the recent presence of a large pericardial effusion precludes constriction by the parietal pericardium. Visceral pericardiectomy is a more difficult operation, with a higher morbidity and mortality, but successful surgery usually leads to an excellent long-term outcome. This patient’s hemodynamic parameters improved immediately after surgery, and at the 6-month follow-up he was functioning normally without any residual stigmata of congestive heart failure or restrictive pulmonary disease.

**Health-Care Delivery Aspect**

The management this patient received after admission to the hospital raises the issue of the appropriate role of hospitalists in today’s medical practice. A second, but less important, question concerns the performance of highly specialized procedures by emergency department physicians. The emergency department physician recognized that the patient had a large pericardial effusion with convincing clinical and echocardiographic evidence of cardiac tamponade. The conclusion that pericardiocentesis was indicated was likewise correct. No absolute rule should dictate when it is appropriate for emergency department physicians to perform pericardiocentesis, but, except in the case of acute life-threatening emergencies, it is preferable to have a cardiologist evaluate the echocardiogram and supervise or perform the pericardiocentesis.

An emergency department physician should not be expected to be familiar with the details of the pressure contour of the jugular venous pulse, and to know how quickly and completely the central venous pressure should return to normal after successful pericardiocentesis. In the case presented, the doctors in the emergency department correctly diagnosed and treated a case considered to be uncomplicated but, hospital policy permitting, should have admitted the patient to the cardiology service. Emergency department evaluation may or may not always yield a diagnosis for the patient. In the former case, a principle organ system is involved and has been identified, and if the illness seems straightforward and within the province of the general internist, admission to a hospitalist service is acceptable. However, if the case seems rare, unusual, or complicated, admission to the relevant specialty service is more appropriate. When the diagnosis is not established, admission to a hospitalist service where further consultations can be obtained is also appropriate.

The concept of the hospitalist is relatively new, and although it has been discussed in the literature, further thought should be applied to how these physicians fit into the delivery and financing of health care for hospitalized patients. The model was initially developed in response to the push by managed care companies for better health outcomes with a minimum waste of valuable resources. However, it has not been universally endorsed by internists, and while primary care physicians are demanding better communication from hospitalists, health-care plans
are generally satisfied with the model due to its cost-effectiveness. Regardless, it is now a specialty with its own national organization affiliated with the American College of Physicians, and preliminary studies suggest that this model of care can be effective in delivering less costly health care without compromising clinical outcomes or patient satisfaction. The issues involved are controversial and will remain so until the outcomes of care delivered by hospitalists have been evaluated by high-quality randomized or epidemiologic studies.

**Clinical Pearls**

1. Both pericardial and myocardial disease should be considered in the patient who presents with anasarca and an elevated central venous pressure.

2. Cardiac tamponade, a rare cause of anasarca, when seen, should suggest concomitant visceral pericardial constriction.

3. The absence of reduction in central venous pressure with drainage of the pericardial effusion suggests visceral pericardial constriction.

4. Pleurodesis for persistent pleural effusions should be reserved for idiopathic, large, chronic pleural effusions.

5. Treatment for effusive-constrictive pericarditis requires extensive debridement of the visceral and parietal pericardium to ensure adequate cardiac filling and output.

6. Hospitalists and intensivists caring for patients with complicated cardiac hemodynamics should manage patients in conjunction with a cardiologist.

**Suggested Reading**


Wachter RM, Goldman L. The hospitalist movement 5 years later. JAMA 2002; 287:487–494