

Case 5-2005: A 53-year-old man with Depression and Sudden Shortness of Breath (Volume 352; 7)

抑うつと突然の息切れをきたした53歳男性

**Clinical course after the diagnostic procedure**

Bedside TTU showed AR. TEU ([omitted](#)) showed an aortic-root abscess, a large area consistent with vegetation on the right aortic cusp and a leaflet fenestration (with moderate-to-severe AR), an area of vegetation on the base of the anterior mitral-valve leaflet, and mild mitral regurgitation. Left ventricular systolic function was mildly impaired. There was a small pericardial effusion.

A transvenous pacemaker was inserted, and the pt was admitted to the ICU. Nafcillin, vancomycin, and gentamicin were administered. The next morning he was taken to the OR. The aortic valve had been largely destroyed, with pus emanating from the intraventricular septum. There were fenestrations in both the right and noncoronary cusps. Purulent material extended laterally from the intraventricular septum, past the commissure of the right and left coronary cusps onto the anterior leaflet of the mitral valve, where there was another abscess, 1 cm in diameter. This was resected along with the majority of the anterior leaflet of the mitral valve. A homograft was used to replace the aortic valve, the left ventricular outflow tract, and the anterior leaflet of the mitral valve. A CABG was performed. The results of cultures of blood, abscess contents, and the resected tissue were negative.

The patient's postoperative course was complicated by acute renal and hepatic failure and candida sepsis, and he died after one month of hospitalization. Permission for an autopsy was declined.

経胸壁心エコーでは大動脈逆流が見られ、経食道エコーでは、大動脈基部の膿瘍、右冠尖の疣贅と弁穿孔、僧帽弁前尖の基部の疣贅、軽度僧帽弁逆流症が見られた。左室収縮能は軽度障害されていた。心内膜滲出液が少量見られた。

頸静脈ペースメーカーが挿入され、患者はICUに入院した。Nafcillin, vancomycin, and gentamicinが投与された。翌日手術が行われた。大動脈弁は大幅に破壊され、膿が心室中隔から出ていた。右冠尖、無冠状動脈弁には穿孔がみられた。膿性物質は、心室中隔から側方に進み、右冠尖と左冠尖の交連を超え、僧帽弁前尖へ進んでいた。僧帽弁前尖には、もう一つ直径1 cmの膿瘍が見られ、僧帽弁前尖の大部分とともに切除された。ホモグラフトで大動脈弁、左室流出路、僧帽弁前尖は置換された。CABGが行われた。血液培養、膿瘍、切除組織培養は陰性だった。

術後患者は急性腎不全、急性肝不全、カンジダ敗血症をおこし、1ヶ月の入院後死去した。剖検は断られた。

**Differential Diagnosis (D/D)**

The initial CXR ([Figure 2A](#)) reveals asymmetric airspace disease, worse on the R side than on the L, a small right pleural effusion, and pulmonary vascular redistribution consistent with pulmonary venous hypertension. The findings are suggestive of **congestive heart failure** with asymmetric

pulmonary edema, but they could represent a **right-sided pneumonia**. A CXR obtained after intubation ([Figure 2B](#)) shows the development of diffuse pulmonary edema.

The TEU shows a multilobed area of vegetation, 2 cm by 1 cm by 0.75 cm, on the base of the anterior mitral leaflet. There is mild MR. The aortic root is thickened, suggesting an aortic-root abscess, and an area of echolucency on the right coronary leaflet indicates perforation. On color Doppler imaging, there is a jet of AR directed posteriorly across the left ventricular outflow tract, striking the anterior leaflet of the mitral valve. The AR causes pan-diastolic flow reversal on spectral Doppler imaging of the distal ascending aorta.

This case presents two common challenges that we face in the ED: **1.the request for medical clearance of a patient with psychiatric symptoms** and **2.the need simultaneously to treat and determine the cause of acute, severe shortness of breath**.

### #1.Medical Clearance of Psychiatric Patients

A dx of major depressive disorder was made, and medical clearance was requested. “**Medical clearance**” is a term used in EDs for the medical evaluation of patients who are to be admitted with psychiatric symptoms. It is a request with two goals: **to rule out organic disease as a cause of the patient’s behavioral changes** and **to screen for an acute illness that might be more than an inpatient psychiatric facility can handle**, whether or not the illness is the cause of the behavioral changes.

#### Problems with “medical clearance” .

This label may impede further medical care, by implying that the patient has no physical health problems.

The evaluations performed for medical clearance may be inconsistent from one institution or practitioner to another, inadequate, or both.

#### Then what should you do?

1.Careful questioning to detect metabolic, neurologic, endocrine-related, or infectious causes of behavioral change.

2.A thorough review of all medications and any history of illicit substance use.

3.Some clues in the patient's history can help distinguish between organic (or medical) and functional (or psychiatric) causes of alterations in behavior ([Table 2](#)).



Screening laboratory tests have a low yield for pts with a known psychiatric disorder and a normal hx and PE. However, in pts without a hx of psychiatric disease, the odds of identifying a metabolic cause are higher.

**The pt had symptoms of a major depressive disorder but had no hx of psychiatric illness. For this reason, a thorough evaluation was both indicated and performed and led to the first clues to his underlying illness: the abnormal results of CXR and the elevated white-cell count.**

### #2.Acute Shortness of Breath

While he was still in the ED, the pt became suddenly and profoundly SOB. A very different challenge: He now needed simultaneous diagnosis and management of this life-threatening symptom.

The pt had rales to the apexes of both lungs, distended neck veins, and a CXR that confirmed the signs of **pulmonary edema**. The ECG showed a new left bundle-branch block and intermittent third-degree heart block.

Our d/d included **acute myocardial infarction, a pulmonary embolus or pericardial effusion with tamponade, and valvular dysfunction**.

Two problems for the cardiologist: **1. the d/d of acute (or so-called flash) pulmonary edema** and **2.the management of invasive endocarditis**.

**1,Flash pulmonary edema** is distinguished by rapid onset — appearing over a period of minutes — as we saw in this pt. Most cases of pulmonary edema that are described as acute are really subacute — arising over hours or even days. Recognizing this distinction can help to narrow the list of possible causes. In this case of flash pulmonary edema, there were three possible cardiac causes: **A.left ventricular dysfunction, B.valvular dysfunction, and C.arrhythmia.**

**A.Acute left ventricular dysfunction** can be either systolic or diastolic.

The most common mechanism of acute left ventricular **systolic** dysfunction is **acute myocardial infarction or ischemia**. In this pt, there was no antecedent CP to suggest ischemia or infarction; instead, the CP appeared with the onset of his dyspnea.

**Acute myocarditis**, a common cause of subacute pulmonary edema, can occasionally cause flash pulmonary edema, as can the **apical ballooning syndrome**. In cases of the apical ballooning syndrome, there is acute dilatation and akinesis of the midventricular and apical left ventricular walls, resulting in markedly impaired systolic function. The syndrome appears to be precipitated by acute emotional or physiological stress, by way of unknown mechanisms; an echocardiogram is required to make the diagnosis. In this pt, the initial PE and ECG showed **no evidence of myocarditis.**

**Diastolic** dysfunction typically occurs in pts with long-standing hypertension and left ventricular hypertrophy and is often precipitated by acute or accelerated hypertension. However, since this pt had neither a hx of HT nor HT on admission, diastolic dysfunction would be unlikely.

**B.**In pts with **mitral or aortic stenosis**, acute pulmonary edema can be precipitated by either volume overload or an arrhythmia. This pt did not have a hx of valvular stenosis, and a murmur was not heard on the initial PE. A large **left atrial myxoma** can produce acute MS and pulmonary edema, although this is rare.

**Valvular regurgitation** is more common than stenosis as a cause of flash pulmonary edema. In a pt with **preexisting mitral-valve prolapse**, MR can worsen abruptly when there is spontaneous rupture of the chordae tendineae. This pt had no hx of mitral-valve prolapse and no evidence of an evolving myocardial infarction. **Acute severe aortic regurgitation** can also result from a proximal (type A) aortic dissection, and this can cause CP that occurs simultaneously with the onset of dyspnea, as in this patient.

**C.Tachyarrhythmias** may precipitate pulmonary edema in the setting of underlying left ventricular dysfunction or valvular dysfunction — particularly valvular stenosis — but rarely cause pulmonary edema in a pt with a structurally normal heart. In this pt, there was no hx of preexisting cardiac disease.

**2.Finally, infective endocarditis(C128)** can cause flash pulmonary edema. **Mitral-valve endocarditis** may cause regurgitation due to rupture of the chordae tendineae, abnormal leaflet coaptation, leaflet destruction producing perforation or tears, or invasion of mitral-annular tissue that produces a fistula between the left atrium and ventricle. **Aortic-valve endocarditis** may cause regurgitation due to cusp prolapse, valve destruction, or the formation of a fistula between the aorta and the left ventricular outflow tract or other cardiac chambers.

This pt did not have f/c, but he did have night sweats, a cough, anorexia, and leukocytosis; thus, although his symptoms are far from classic for endocarditis, they are suggestive enough that endocarditis should be considered.

Even after the occurrence of flash pulmonary edema, no murmur was detected. Nevertheless, it is important to recognize that when examining an ill patient with acute respiratory distress in a noisy ED, it may be difficult to hear any but the loudest murmur. Moreover, the murmurs associated with acute severe AR & MR are often much more subtle than one might expect. Thus, in this setting, **the absence of a murmur does not rule out the presence of severe regurgitation.** Finally, the pattern of progressive conduction abnormalities on the ECG — presentation with marked first-degree atrioventricular block, development of a left bundle-branch block, and then complete heart block — is consistent with rapidly progressive invasive endocarditis.

The ECG that was obtained when the pt's breathing suddenly became labored showed normal left ventricular systolic function with no sign of MI or the apical ballooning syndrome. A jet of AR was present, and although it did not appear initially to be severe, in the context of flash pulmonary edema one should suspect that the jet is in fact larger than it appears. Indeed, the

TEU confirmed that the AR was severe, and it showed that the regurgitation was the result of endocarditis and not aortic dissection.

### Discussion of Management

This is not a typical case of invasive infective endocarditis. "Invasive" refers to extension of the infection from the valve into the adjacent cardiac tissues. **The possibility of invasive endocarditis should be considered when fever persists or recurs despite appropriate ax therapy, or when new or evolving cardiac-conduction abnormalities appear**, as they did in this pt. First-degree AV block is the most common cardiac-conduction abnormality, but only a minority of cases progress to complete heart block. About 50 percent of cases of invasive endocarditis are caused by *Staphylococcus aureus*. *Streptococcus viridans* species and *Staphylococcus epidermidis* are also relatively common pathogens, particularly on prosthetic heart valves. In some cases, even with invasive disease, no organism is identified — this is so-called **culture-negative endocarditis**.

As soon as the diagnosis of endocarditis is suspected, **blood cultures(bcx) should be performed**, and **ax therapy administered**.. Surgery to repair the damaged cardiac structures and to try to eradicate the infection is the treatment of choice unless there is a clear contraindication.

### Final Diagnosis

Invasive endocarditis, possibly due to bartonella.

After the pt died, a report from the Centers for Disease Control and Prevention noted that serologic tests had been positive for bartonella (*Bartonella henselae* IgG, 1:128; *B. quintana* IgG, 1:512). Bartonella has been recognized with increasing frequency over the past decade as a cause of culture-negative endocarditis and may cause as many as 3 percent of all cases of endocarditis.

Typically, bartonella causes endocarditis in pts who are homeless, who have alcoholism, or who have been exposed to cats