A 66-Year-Old Man With Fever, Hypotension, and Complete Heart Block*

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(CHEST 2001; 120:2053–2056)

A 66-year-old man presented to the emergency department with a history of increasing confusion for 8 days, intermittent headaches and abdominal pain for the last 1 week, and history of a fall at home on the day of hospital admission. There was no history of loss of consciousness. No fever was documented at home. Medical history was significant only for long-term alcohol use. The patient was not receiving any medication at home.

Physical Examination

Examination showed an agitated, confused gentleman with a BP of 90/60 mm Hg and a pulse rate of 31 beats/min. The patient had a right frontal abrasion. The neck was supple. Lungs were bilaterally clear to auscultation. Cardiac auscultation revealed 3/6 systolic murmur at left sternal base. There was diffuse tenderness on abdominal examination, but no guarding or rebound tenderness was present. Bowel sounds were heard normally. No focal neurologic deficit was found. There was no lymphadenopathy or rash. The remainder of the physical examination was normal. His temperature reached 40°C in the emergency department.

Investigations

Admission data showed a total WBC count of 22,000/µL, with polymorphonuclear cells of 85%, bands of 10%, and hematocrit of 36%. The serum bicarbonate level was 15 mEq/L; BUN, 61 mg/dL; creatinine, 2.4 mg/dL; lactate dehydrogenase, 418 IU/L; amylase, 224 IU/L; and lipase 289 IU/L. Serum aspartate and alanine aminotransferase levels were 100 IU/L and 55 IU/L, respectively, with a total bilirubin level of 4.3 mg/dL and alkaline phosphatase level of 294 IU/L. Coagulation profile showed prothrombin time of 19 s and activated partial prothrombin time of 42 s. Results of the first set of cardiac enzymes were normal.

Arterial blood gas analysis done on room air showed pH of 7.27, PaCO₂, 27 mm Hg; and PaO₂, 99 mm Hg. Serum and urine toxicology screen results were negative. Urinalysis findings were significant for a small amount of blood and 5 to 10 WBCs per high-power field. Chest radiographic findings were normal. The patient’s ECG is shown in Figure 1.

What is the expected diagnosis?

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Manuscript received May 8, 2001; revision accepted June 29, 2001.
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Diagnosis: Infective endocarditis presenting with complete heart block

Discussion

The differential diagnosis of new complete heart block in otherwise healthy people should include myocardial infarction, drug toxicity, and myocarditis. A patient with recent onset of fever and new heart block should alert the clinician to consider infective endocarditis with perivalvular extension of infection (PVEI) or myocardial abscess. The presence of a new systolic murmur should strengthen the clinical suspicion of infective endocarditis.

Conduction abnormalities that develop in patients with active infective endocarditis may represent extension of infection from the valve leaflets into the surrounding myocardium. Extension of infection from the aortic valve into the septum can lead to significant conduction abnormalities, with bundle-branch blocks or complete heart block. Extension from the mitral valve is less common, and the conduction abnormalities resulting from such extension include low-grade atrio-ventricular blocks or supraventricular arrhythmias. A study of cardiac anatomy makes it clear why aortic valve endocarditis more commonly causes significant conduction problems than mitral valve endocarditis. The conduction system, particularly the right and left bundles, is closely related anatomically to the aortic valve. The atrioventricular node, though adjacent to the mitral valve, is not as close to this valve as the aortic valve is to the bundle branches. Therefore, mitral valve endocarditis that spreads to perivalvular tissue usually causes first-degree or second-degree heart block. Third-degree heart block is unusual; if present, it is accompanied by a narrow complex QRS. Aortic valve endocarditis can result in first-degree or second-degree heart blocks in addition to bundle-branch blocks, hemiblocks, and complete heart block. These latter complications are not uncommon, especially if noncoronary and right cusps of the valve are involved. Perivalvular extension of infection is a serious complication of bacterial endocarditis. The most common type of extension of the infection involves the cardiac tissue immediately adjacent to the valve ring (also referred to as perivalvular abscess). Other types include the development of aneurysms, intracardiac fistulas, and valve dehiscence. Aortic valve infection most commonly leads to perivalvular extension.

The exact incidence of PVEI is difficult to determine for two reasons: the different definitions used by different investigators, and varying demographic and clinical characteristics of the patients in different series. Two postmortem series of patients with native valve endocarditis overall showed a 30% incidence of PVEI. Among the left-sided valves, the aortic valve was most likely to develop perivalvular abscesses. For prosthetic valves, the incidence of PVEI has been reported to be from 54 to 100% in different necropsy and surgical series.

The ECG is the easiest diagnostic modality to use for evaluation of the possibility of a PVEI. In a series of 211 episodes of native valve endocarditis, 20 patients (9.5%) had unstable conduction abnormalities. Pathologic information was available for 12 of these cases, 8 of which had extension of infection beyond the valve leaflets. However, only 8 of 29 patients with pathologically confirmed extension of infection had conduction abnormalities. Thus, the presence of conduction abnormalities on ECG is relatively specific (89%) but very less sensitive (28%) for PVEI. The positive predictive value of unstable conduction abnormalities on the ECG had positive predictive value of 66% for any type of PVEI. The results from two smaller series have demonstrated similar findings.

The echocardiogram has been used extensively to evaluate patients with endocarditis. The usefulness of all echocardiographic techniques is dependent on a large degree on the skill of the technician performing the test. Two-dimensional (2-D) transthoracic echocardiography has potential utility in diagnosis of perivalvular abscess. Most of the published literature on role of 2-D echocardiography in diagnosing PVEI is retrospective and suffers from selection bias. In a blinded prospective study of the 2-D echocardiogram, reported only in 3 of 17 patients with PVEI, a diagnosis was made preoperatively. The study reported missing 71% of aortic root abscess and 62% of intraventricular abscesses. Transesophageal echocardiography has been shown to have 76% sensitivity and 95% specificity in diagnosing PVEI. Cardiac catheterization with angiography is the most invasive test used to diagnose PVEI. Its utility lies in its ability to detect aneurysms, primarily of the sinus of Valsalva, valve dehiscence, and annular/perianular abscesses. This method is not as useful in detecting septal or other myocardial abscesses.

Development of conduction abnormalities during native valve endocarditis, particularly when persistent, is a poor prognostic sign and is often associated with invasive infection and hemodynamic deterioration due to valve dysfunction. In one large series,
persistent conduction abnormalities were associated with a higher mortality rate than the conduction defects, which resolved during the antibiotic treatment. No patient with persistent heart block survived 1 year without valve replacement. The onset of heart failure is the most widely accepted indication for surgery during active infective endocarditis. Since perioperative mortality correlates with hemodynamic instability at the time of valve replacement, surgical intervention is advocated before severe heart failure supervenes. Though there is no consensus, some experts recommend valve replacement in patients with native valve endocarditis, especially if the aortic valve involvement produces unstable conduction abnormalities persisting for more than a week despite optimal medical therapy.

The present patient underwent placement of a temporary transvenous pacemaker for management of the third-degree heart block shown on his ECG. The patient was evaluated for possibility of meningitis, sepsis with possible intra-abdominal source, or infective endocarditis. Initial bedside echocardiography in the emergency department revealed normal left ventricular systolic function, concentric left ventricular hypertrophy, and trace tricuspid regurgitation. The patient was intubated and treatment with dopamine was initiated because of hemodynamic instability and admission to the medical ICU. The patient was initially administered ceftriaxone, metronidazole, and ampicillin. On the second hospital day, blood culture findings were positive for Gram-positive cocci in both aerobic and anaerobic bottles. Antibiotic treatment was changed from ampicillin to vancomycin. The patient continued to require hemodynamic support. Repeat echocardiography showed vegetation on the aortic valve and a mass in the right atrium consistent with vegetation (Fig 2). There was evidence of aorto-right atrium fistula. The Gram-positive cocci were identified as *Streptococcus viridans* on the third hospital day. Immediate transfer to a cardiac surgery service was arranged. However, the patient died on the fourth hospital day before surgery could be performed.

**Clinical Pearls**

1. Infective endocarditis should be a major differential diagnosis in patients presenting with recent onset of fever and new conduction abnormalities. A presence of new systolic murmur should strengthen the suspicion for infective endocarditis.
2. New unstable conduction abnormalities in patients with confirmed or clinically suspected infective endocarditis mandates aggressive search for perivalvular extension of infection.
3. Surgical consultation is advisable in case of persistent conduction abnormalities even after adequate antimicrobial therapy for infective endocarditis.
SUGGESTED READING


