Group B Streptococcal Myocarditis

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Abstract

Bacterial myocarditis is an uncommon form of infectious myocarditis. The definitive diagnosis requires histopathology with evidence of bacterial invasion. We report a case of group B streptococcal myocarditis secondary to septicaemia with complete atrioventricular (AV) block and new left bundle branch block. The histopathology revealed patchy small foci of myocyte necrosis. The necrotic areas contained mixed inflammatory cell infiltration with a predominance of neutrophils. Necrosis in AV node was also observed. Gram stain in the necrotic area showed clusters of gram positive cocci in agreement with the results of haemoculture. Bacterial myocarditis is a devastating complication of bacteraemia. This case highlights the ability of group B streptococci to cause life-threatening infections in adults without clear predisposing factors to serious infection. Bacterial myocarditis may progress quickly and be associated with a fatal outcome.

Keywords: Bacterial myocarditis; Group B Streptococcus; Electrocardiography; Atrioventricular

Introduction

The prevalence of bacterial myocarditis is not well established. The few published studies describe a post-mortem prevalence ranging from 0.2% to 1.5% [1]. The most common bacterial cause of myocarditis is Staphylococcus aureus, although infections with a broad range of bacterial pathogens have been described [1]. The definitive diagnosis requires histopathology-proven active myocarditis with evidence of bacterial invasion or positive tissue cultures. The management of bacterial myocarditis consists of aggressive and early antibiotic treatment, appropriate haemodynamic support, and treatment of the arrhythmias or complications.

Case Report

A 73-year-old-woman presented with fever and back pain. The patient reportedly felt feverish and took over-the-counter medications for a few days, but the symptoms did not improve. She had a history of type 2 diabetes, hypertension, and dyslipidemia. Graves disease 10 years earlier had been treated with I-131 ablation. Medications included enalapril, metformin, simvastatin, levothyroxine, and omeprazole. The patient reportedly felt feverish and took over-the-counter medications for a few days, but the symptoms did not improve. She had a history of type 2 diabetes, hypertension, and dyslipidemia. Graves disease 10 years earlier had been treated with I-131 ablation. Medications included enalapril, metformin, simvastatin, levothyroxine, and omeprazole.

An initial electrocardiography revealed normal sinus rhythm with incomplete right bundle branch block (Figure 1A). Repetitively, haemoglobin was 10.7 g/dl, total white cell count was 3,140 per cubic millimetre with 75% of neutrophils, and the platelet count was 84,000 per cubic millimetre. Serum creatinine was 3.1 mg/dl and potassium was 3.8 mmol/l. The creatine kinase level was 2,217 U/l and troponin T level was 1,392 ng/l. Blood cultures were drawn and an empiric treatment with ceftriaxone and intravenous fluid was started. Two hours later, she developed complete atrioventricular block and left bundle branch block with a heart rate of 50. Figure 1B: Two-hours later, there was complete atrioventricular block and left bundle branch block with a heart rate of 50.

Group B streptococci were found in the blood cultures taken on admission. Postmortem examination of the heart showed variable stenosis of the coronary arteries due to intimal fibrosis. The stenosis varied from 30-70% of the luminal area. However, no thrombosis

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Bacterial myocarditis is an uncommon form of infectious myocarditis. It is usually seen in the context of overwhelming sepsis or infection of the adjacent organs. Infective endocarditis is a common underlying cause of bacterial myocarditis as well. Predisposing factors for bacterial myocarditis described in early reports included bacteraemia, neutropaenia, myocardial infarction, osteomyelitis, and recent surgical procedures [1]. The potential pathogens include Staphylococci, Streptococci, Proteus, Klebsiella, Escherichia coli, Listeria monocytogenes, Clostridium perfringens, and Corynebacterium diptheriae [1,2].

Clinical presentation of patients with bacterial myocarditis is dominated by sepsis or cardiac involvement including myocardial infarction, pericarditis, heart failure, atrioventricular block, ventricular tachycardia, or sudden death. Circulatory failure and shock can also be a prominent feature of acute bacterial myocarditis [8]. Differentiating sepsis-induced myocardial depression from bacterial myocarditis may be challenging. Both conditions share common features such as ventricular dysfunction and elevated serum troponin. A novel biomarker, ST2 (suppression of tumorigenicity 2) is a blood protein confirmed to act as a decoy receptor for interleukin-33. ST2 seems to be markedly induced in mechanically overloaded cardiac myocytes and potentially attenuate the extent of cardiac damage, inflammatory cardiac activation, adverse myocardial remodelling [9]. Nevertheless, ST2 was also found to be increased in patients suffering from pulmonary disease, systemic infection, or inflammation. Thus, it may increase in the patients with sepsis. Endomyocardial biopsy is obtained in only a minority of patients with sepsis and ventricular dysfunction. Moreover, endomyocardial biopsy is highly specific for the diagnosis of myocarditis, but its sensitivity is low [10]. Factors such as disease distribution, stage of the disease process and sampling error influence the sensitivity of endomyocardial biopsy, bacterial myocarditis may be underdiagnosed in sepsis. Conduction-system disturbance is a valuable clue suggesting myocardial involvement. Advanced atrioventricular block has been reported in myocarditis associated with endocarditis, diphtheritic toxin, Meningococci, or Listeria monocytogenes [1]. To our knowledge, the current case is the first report of group B streptococcal myocarditis in association with complete atrioventricular block.

Conclusions

The diagnosis of bacterial myocarditis is challenging particularly in patients with sepsis. Endomyocardial biopsy is obtained in only a minority of patients with sepsis and ventricular dysfunction. Conduction-system disturbance is a valuable clue suggesting myocardial involvement. We present a clinical case of a devastating complication of bacteraemia in order to increase recognition of bacterial myocarditis.
References


