Journal of Clinical Case Reports
ISSN: 2165-7920

Roongsangmanoon and Raveesunthornkiat, J Clin Case Rep 2015, 5:9
DOI: 10.4172/2165-7920.1000593

Case Report Open Access

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. Grave's disease 10 years earlier had been treated with I-131 ablation. Medications included enalapril, metformin, simvastatin, levothyroxine, and omeprazole. The patient was ill looking, afebrile with a pulse rate of 65 beats per minute, a blood pressure of 100/60 mmHg, and a respiratory rate 20 breaths per minute. The first and second heart sounds were normal, without murmurs or rubs. There was no tenderness along the length of the spine and no tenderness at the costovertebral angle bilaterally. Systemic examination failed to reveal a focus of infection elsewhere and there was no neurological deficit.

An initial electrocardiography revealed normal sinus rhythm with incomplete right bundle branch block (Figure 1A). Reportedly, haemoglobin was 10.7 g/dl, total white blood 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), severe acidosis, and hypoglycaemia. There was no urine output. Haemodialysis was commenced to correct the acidosis. During the subsequent hours her condition deteriorated rapidly. She died within 24 hours of admission in spite of cardiopulmonary resuscitation and high doses of vasopressors and inotropes.

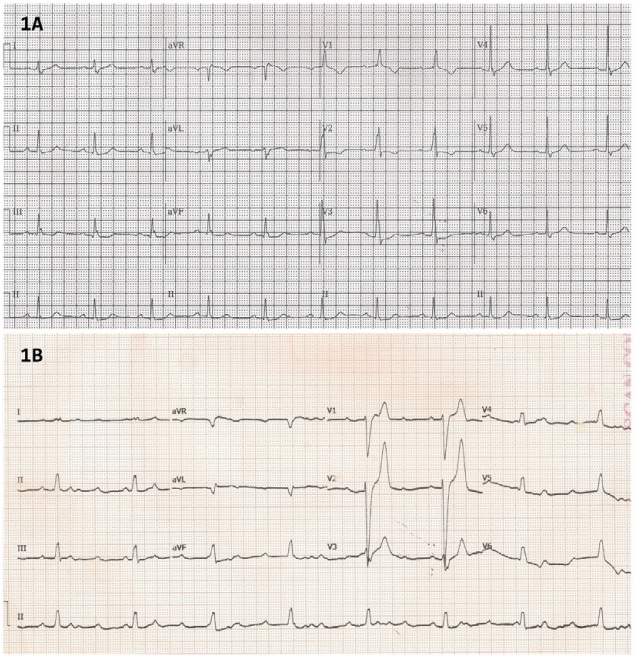

Figure 1A: The initial electrocardiography revealed normal sinus rhythm with incomplete right bundle branch block.
B: Two-hours later, there was complete atrioventricular block and left bundle branch block with a heart rate of 50.

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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Received August 17, 2015; **Accepted** September 10, 2015; **Published** September 17, 2015

Citation: Roongsangmanoon W, Raveesunthornkiat M (2015) Group B Streptococcal Myocarditis. J Clin Case Rep 5: 593. doi:10.4172/2165-7920.1000593

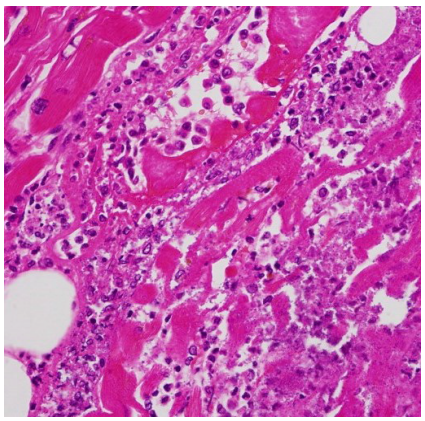

Figure 2: The necrotic areas contained mixed inflammatory cell infiltration with a predominance of neutrophils. Necrosis in the AV node was observed.

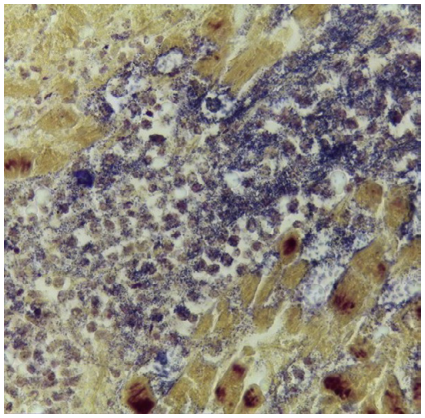

Figure 3: Gram staining in the necrotic area showing clusters of Gram positive cocci corresponding to the result of haemoculture.

was identified. Serial sectioning of the heart showed mild concentric ventricular hypertrophy. All valve leaflets showed smooth surfaces. Microscopically, the myocardium contained patchy small foci of myocyte necrosis. These necrotic cardiac myocytes were fragmented and had loss their nuclei. The necrotic areas contained mixed inflammatory cell infiltration with a predominance of neutrophils. Necrosis in AV node was observed (Figure 2). One medium-sized vessel in the myocardium showed intense active inflammation in the vessel wall. The inflammation also extended to the adjacent myocardium. Gram staining in the necrotic area showed clusters of gram positive cocci corresponding to the results of haemoculture (Figure 3).

Discussion

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, Meningococci, Proteus, Klebsiella, Escherichia coli, Listeria monocytogenes, Clostridium perfringens, and Corynebacterium diphtheriae [1,2].

The most common group B streptococcal infections in adults are bacteraemia, pneumonia, skin and soft tissue infections, bone and joint infections which usually occur in association with ageing, diabetes mellitus, malignancy, and HIV infection [3]. Cardiac infection due to Group B streptococci usually manifests as endocarditis, most commonly in immunocompromised patients [4]. Primary myocarditis associated with Group B streptococci is extremely rare. The usual pathological findings of streptococcal myocarditis are neutrophilic collections and microabscess in myocardium [5]. Other histopathological patterns reported in group B streptococcal myocarditis include diffuse myocarditis with suppurative pericarditis in which the myocardial infiltration consists mainly of lymphocytes and plasma cells with a small amount of neutrophils [6].

In this case, there were multiple microscopic foci of neutrophilic infiltration accompanied with necrosis of myocytes. The inflammation was randomly distributed and included atrioventricular node. Generally, neutrophilic infiltration with necrosis is also present in acute myocardial infarction. Moderate stenosis of coronary arteries was also identified in the patient. However, lack of thrombus or association with any vascular territories exclude an acute myocardial infarction diagnosis. In addition, collections of gram-positive cocci, intense active inflammation in the vessel wall without evidence of endocarditis suggest myocarditis caused by bacteraemia [7].

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.

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