CASE REPORT

Various Cardiac Abnormalities Caused by Bacterial Myocarditis

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Summary
A 69-year-old woman without any past disease history was hospitalized for heart failure. After hospitalization, she showed myocardial infarction, atrioventricular dissociation, and cardiac dysfunction, and finally she passed away despite intensive care. Autopsy revealed that the cardiac abnormalities were due to bacterial myocarditis possibly resulting from urinary tract infection by E. coli. Although bacterial myocarditis is rare in developed countries, we should consider its possibility when patients show various cardiac abnormalities with bacterial infection.

Key words: Heart failure, Myocardial infarction, Atrioventricular dissociation

Myocarditis induces various cardiac abnormalities including systolic dysfunction and arrhythmia. The most common cause of myocarditis in developed countries is the infection of viruses such as coxsackievirus, adenovirus, enterovirus, parvovirus B-19, parainfluenza viruses and human herpesvirus-6. Although bacterial myocarditis was has been very rare in developed countries, the number of patients with bacterial myocarditis has recently been increasing because of an increase in immunocompromised hosts. In most cases, bacterial myocarditis is suspected based on clinical test results, but few cases have been diagnosed while alive. We here present a patient who suddenly developed myocardial infarction, atrioventricular dissociation, and heart failure without any past disease history. Morbid anatomy revealed that the diagnosis was bacterial myocarditis. Here we describe the various clinical and pathological findings of this case and with some also discussions about bacterial myocarditis in general.

Case Report
A 69-year-old woman visited our hospital because of death of her brother’s death. While she was plunged in grief, she suddenly felt dyspnea. Approximately 2 weeks before, she had visited a local doctor because of wheezing and insomnia. Pleural effusion had been found by chest X-ray examination, and a diuretic had been prescribed. In our hospital, she had slight tachycardia (106/minute) but other physical findings, including blood pressure (118/64 mmHg) and body temperature (35.5°C), were normal (35.5°C). X-ray examination revealed bilateral pleural effusion and pulmonary congestion, and the electrocardiogram showed a negative T wave in I, aVL, and V3-6 leads. Routine blood examination revealed the elevation of C-reactive protein levels (4.8 mg 8 mg/dL) and white blood cell counts (13.9 × 10^3/μL). Ultrasound echocardiography showed dilatation of the left ventricle (left ventricular dimension diastolic/systolic 60/51 mm), systolic dysfunction (ejection fraction 31%), mild aortic regurgitation, and moderate mitral regurgitation. The wall motion was diffusely reduced, and in particular, the wall motions of the inferior apex wall and the anterolateral wall were severely impaired. We diagnosed her as acute decompensated heart failure with unknown etiology and administered furosemide, carperitide and dobutamine. Since she had no fever and no symptoms of pneumonia or urinary tract infection, we did not follow up with no antibiotics. On the 5th hospital day, she experienced chest pain and showed worsening of heart failure without significant change in electrocardiogram. On the 10th hospital day, she experienced chest pain again and the systolic blood pressure declined to 60 mmHg, leading to disturbance of her consciousness. There was ST elevation in V1-3 leads of the electrocardiogram, but there was no significant change in the wall motion as shown by echocardiography. Coronary angiography revealed that there were 90% stenosis in the left main trunk and the ostium of the right coronary artery (Figure 1). We performed percutaneous coronary intervention to the left main trunk and started intra aortic balloon pumping. We treated her with catecholamine in the intensive care unit. She had fever and many increased white blood cell levels were recognized observed in her urine. Since Escherichia coli (E. coli) was found in the urine and blood culture, we started ceftriaxone sodium to which the E. coli was sensitive. We also performed percutaneous coronary intervention to the ostium of the right coronary artery on the 11th hospital...
day. Although her cardiac function did not improve after PCI, the blood pressure was elevated and her condition was stabilized. There were some changes in the ECG: QS patterns in V1-4 before PCI and only in V1 after PCI. On the 14th hospital day, however, her respiratory condition got worsened, and the amount of urine dropped. Her pulmonary arterial pressure was increased and her cardiac index was decreased. The electrocardiogram showed atrioventricular dissociation and a left bundle branch block (Figure 2) without an increase in cardiac enzyme levels. Despite intensive treatment with percutaneous cardiopulmonary support and intra aortic balloon pumping, her condition went worsened further and she eventually and passed away. Morbid anatomy showed that there were remarkably numerous infiltration of many neutrophils in the myocardium, intramyocardial micro abscesses in the left ventricular anterior and posterior walls, and abscesses adjacent to the atrioventricular node. In addition, there were vasculitis with giant cells directly under the epicardium of left ventricular posteroseptal walls and lots of cell debris with neutrophils in endocardium were observed. There was no vegetation in the valves and the bacterial staining (Goodpasture’s stain) was negative. There was no stenosis in coronary arteries including the stent portion of the left main trunk and the osmium of right coronary artery, indicating that supporting the diagnosis of bacterial myocarditis (Figure 3). Since there were a lot of thrombi on the endocardium, we thought that the thrombi caused embolism at the inlet potion of the coronary arteries. We could not rule out the possibility of the infectious endocarditis, however, the cardiac valves were intact and there was no vegetation on valves, suggesting that endocarditis might not have been the cause of the symptoms.

Discussion

Although viral infections are the most common cause of myocarditis in developed countries, bacterial and mycotic myocarditis are increasing also in developed countries because of increases in patients treated by cardiac
open surgery, and anti-cancer and immunosuppressive drugs, and patients with acquired immune deficiency syndrome. Bacterial myocarditis can be caused by many kinds of bacteria such as staphylococcus aureus, streptococcus, pneumococcus, and neisseria meningitidis. Although there are a few reports of urinary tract infections as the cause of bacterial myocarditis, we suggest that urinary tract infection by E. coli might have caused myocarditis in this case, because E. coli was positive in both blood and urine cultures were positive for E. coli. The definitive diagnosis of bacterial myocarditis requires morphologically proven active myocarditis with evidence of bacterial invasion or positive tissue cultures. Although endo-myocardial biopsy is highly specific for the diagnosis of myocarditis, its sensitivity is low and thus it is rare for bacterial myocarditis to be diagnosed while the patient is alive. Non-invasive imaging techniques for the diagnosis of bacterial myocarditis have been investigated, however their usefulness in clinical settings is uncertain. Magnetic resonance imaging or indium-anti-myosin antibody scintigraphy may help to localize the area of inflammation, which would be useful for endomyocardial biopsy, and might be useful to follow disease activity. The underlying mechanisms of the development of bacterial myocarditis are not yet clear. However, several possibilities have been proposed including direct bacterial invasion, bacterial toxins, and immune responses. Bacterial myocarditis needs total management such as treatment of

Figure 3. Observations of morbid anatomy. A: Infiltration of neutrophils in the myocardium. B: High magnification of figure A. C: Intramyocardial micro abscesses. D: Organization around atrioventricular node shown by hematoxylin and eosin staining. E: Fibrosis at atrioventricular node and adjacent abscesses shown by hematoxylin and eosin staining. F: Fibrosis at atrioventricular node and adjacent abscesses showing by Masson-trichrome staining.
the infectious process and heart failure management.8) The prognosis of bacterial myocarditis in the acute stage depends on several factors; 1) the cause of bacterial myocarditis, 2) the extent of the disease, 3) complications such as arrhythmias or tamponade, 4) the degree of circulatory failure, 5) sepsis severity, and 6) response to therapy. Long-term prognosis of bacterial myocarditis might depend on the degree of residual ventricular function and ventricular remodeling.8) In this case, ejection fraction was low and the responses to therapy for sepsis and heart failure were bad. In addition, the patient showed circulatory failure and arrhythmias, suggesting poor prognosis of this case. In conclusion, when a patient of with a bacterial infection shows a variety of cardiac abnormalities, we should consider the possibility of bacterial myocarditis should be considered.

Disclosures

Conflicts of interest: The authors declare that they have no conflict of interest.

References