CASE REPORT

Listeria endocarditis with Acute Thoracoabdominal Aortic Dissection

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Abstract

The patient was 71-year-old male under treatment at a clinic for hypertension, aortic regurgitation, alcoholic hepatitis and dental treatment. He mainly complained fever and anorexia. Since blood culture examination revealed Listeria monocytogenes and echocardiography exhibited vegetation at the mitral leaflet, the patient was diagnosed as infective endocarditis. Fever and inflammatory reaction were improved after penicillin administration; however, he had fever on the 24th hospital day. CT revealed type IIIb acute thoracoabdominal aortic dissection which was not observed on admission. The blood pressure was controlled with antihypertensive agents. He could leave the hospital on the 61st day.

Key words: listeria endocarditis, acute thoracoabdominal aortic dissection, renal dysfunction, esophageal cancer, alcoholic hepatitis

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Introduction

Listeria monocytogenes is a small gram-positive bacterium widely distributed in nature. Infection with L. monocytogenes usually causes meningoencephalitis and severe sepsis (1) in immunocompromised persons, particularly in newborns, pregnant, and the elderly (2, 3). According to a recent review of the world literature, 68 cases of endocarditis due to L. monocytogenes have been reported in the West (4). The only previously documented case of endocarditis due to L. monocytogenes in Japan was reported by Ono et al 2005 (5).

Case Report

A 71-year-old Japanese man was admitted to our hospital for fever and anorexia. He also presented with a moderate aortic regurgitation. His recent medical history included hypertension, alcoholic hepatitis, and renal dysfunction as well as dental treatment a year before his admission. Neither hypercholesterolemia nor diabetes mellitus was observed. He smoked two or three packages daily for 30 years. The etiology of renal dysfunction was not clarified because the patient refused to take an examination. One month before admission he noticed fever, anorexia, and weight loss. Findings on the initial physical examination exhibited a height of 160 cm, body weight of 58 kg, heart rate of 76 beats/min, blood pressure of 128/56 mmHg, and body temperature of 37.8°C. His skin was dry and his eyes were anemic. A diastolic murmur was detected at the left sternal border. His lung sound was clear on auscultation. No pretibial edema was observed. Laboratory studies on admission revealed inflammation (white blood cell count (WBC) 11,800/μl, C-reactive protein (CRP) 14.1 mg/dl), liver dysfunction (aspartate aminotransferase (AST) 48 IU/l, alanine aminotransferase (ALT) 38 IU/l, and γ-glutamyltranspeptidase (γ-GTP) 616 IU/l), and renal dysfunction (serum creatinine (Cre), 2.50 mg/ml, serum urea nitrogen (BUN) 42.1 mg/ml, Cre clearance 33.3 ml/min). The blood gas analysis showed respiratory alkalosis according to a decrease of carbon dioxide concentration in the blood due to hyperventilation (Table 1). Calcification of the aorta was observed in chest X-ray, abdominal X-ray, and computed-tomography (CT; Fig. 1). The intima-media thicknesses (IMT) of the right and left carotid arteries were 0.8 mm and 0.9 mm in Doppler echocardiography. The patient suffered from severe atherosclerosis. The same L. monocytogenes was observed in one bottle of atrial
Table 1.  On Admission, Blood Test

<table>
<thead>
<tr>
<th>(Blood gas analysis)</th>
<th>(Biochemistry)</th>
</tr>
</thead>
<tbody>
<tr>
<td>pH (7.35-7.45)</td>
<td>TP 7.3 g/dl</td>
</tr>
<tr>
<td>PaO₂ (80-100) mmHg</td>
<td>Alb 3.7 g/dl</td>
</tr>
<tr>
<td>PaCO₂ (35-45) mmHg</td>
<td>Tbil 1.2 mg/dl</td>
</tr>
<tr>
<td>HCO₃ (22-28) mEq/l</td>
<td>AST 48 IU/l</td>
</tr>
<tr>
<td>BE (±2.3-0.4 mEq/l)</td>
<td>ALT 38 IU/l</td>
</tr>
<tr>
<td>SpO₂ (94-99) %</td>
<td>LDH 188 IU/l</td>
</tr>
<tr>
<td>(Hematology)</td>
<td>ALP 726 IU/l</td>
</tr>
<tr>
<td>WBC 11.8 10³ /μl</td>
<td>γ-GTP 616 IU/l</td>
</tr>
<tr>
<td>RBC 3.19 10³ /μl</td>
<td>(Serology)</td>
</tr>
<tr>
<td>Hb 10.9 g/dl</td>
<td>CRP 14.1 mg/dl</td>
</tr>
<tr>
<td>Hct 32.1 %</td>
<td>BNP (&lt;18.4) 176 pg/ml</td>
</tr>
<tr>
<td>Plt 22.9 10³ /μl</td>
<td>(Endocrine)</td>
</tr>
</tbody>
</table>

Figure 1. On admission, X-ray and CT showed calcification of aorta.

blood culture and two bottles of venous blood culture. Transthoracic and transesophageal echocardiography depicted 5.8 × 5.7 mm vegetation at the anterior leaflet of the mitral valve (Fig. 2). Moderate aortic regurgitation and mitral regurgitation were also recognized, and the patient was diagnosed as infective *L. monocytogenes* endocarditis according to the Duke Criteria. Intravenous penicillin was administered for four weeks, at
Figure 2. Transesophageal echocardiography showed a vegetation of 5.8 by 5.7 mm in size on the anterior leaflet of mitral valve and moderate aortic regurgitation.

Figure 3. On the 24th hospital day, chest X-ray showed the dilatation of aortic arch and pleural effusion on left side. CT showed thoracoabdominal aortic dissection and pleural effusion on left side.

a relatively low dose of $1,200 \times 10^4$ units/day, as deemed appropriate for a patient with renal dysfunction and a high risk of drug sensitivity. The WBC and CRP levels were decreased 19 days after penicillin administration, and no growth of the *L. monocytogenes* was observed in the three blood culture bottles 8 days after the antibiotic treatment. Transthoracic echocardiogram revealed a complete clearance of the vegetation 12 days after admission.

The patient’s fever resumed on the 24th hospital day without any pain in the back or chest. No growth of the bacteria was observed in the blood, urine and sputum. A chest X-ray revealed a dilatation of the aortic arch and pleural effusion on the left side. The patient was diagnosed as acute type IIIb thoracoabdominal aortic dissection according to the DeBakey classification, based on CT findings (Fig. 3). The dissection originated from the left subclavian artery to the renal artery. All visceral arteries branched from the true lumen. The maximum diameter of the abdominal aneurysm was $5.0 \times 4.8$ cm. Systolic blood pressure was over 140 mmHg before the onset of acute aortic dissection. Systolic blood pressure was controlled under 120 mmHg with antihypertensive agents. CT revealed no further dilation of the aortic dissection after administration of antihypertensive agents. A suspected esophageal cancer was discovered by endoscopy on the 9th hospital day and diagnosed according to the result of biopsy examination after discharge.

**Discussion**

*L. monocytogenes* is a small gram-positive bacterium
widely distributed in nature. According to a recent review of the world literature, there have been 68 reported cases of endocarditis due to *L. monocytogenes* (4). Clinically, it appears as a subacute disease chiefly manifesting symptoms of fever, weakness, dyspnea, and cardiac murmur (6). About 50% of the patients with endocarditis due to *L. monocytogenes* have a history of rheumatic heart disease, prosthetic valve endocarditis, or mitral valve prolapse. The final diagnosis is confirmed by a moderately positive blood culture. Most patients also present with an underlying immunocompromised condition such as cancer, lymphoma, prolonged corticosteroid therapy, kidney transplantation, chronic hemodialysis, Human Immunodeficiency Virus (HIV) infection, diabetes mellitus, chronic alcoholism, or pregnancy (6-9).

The onset mechanism of infective endocarditis in this case was attributed to the following two factors: ① the patient was alcoholic, ② esophageal cancer had compromised his immune functions during his aortic regurgitations and during the dental treatment he had received one year earlier. The patient’s survival can also be attributed to several factors. The antibiotic treatment was effective against infective endocarditis, though the patient’s renal dysfunction compelled us to reduce the penicillin dosage by half. We observed no signs of thrombosis or valvular lesions, a trigger of heart failure. The onset of the acute aortic dissection is severe atherosclerosis and hypertension. Until now, there has been no case report which describes a patient with *Listeria endocarditis* and acute thoracoabdominal aortic dissection. We attribute the patient’s survival to the stable blood pressure under antihypertensive management which prevented any dilation of the dissection, and no aneurysm observed in the cerebral blood vessels.

### Conclusion

We reported our encounter with an alcoholic patient with aortic regurgitation and a complication of infective endocarditis. A suspected esophageal cancer discovered during endoscopy was diagnosed from a biopsy specimen after discharge. According to literature reviews, infective endocarditis due to *L. monocytogenes* under an immunocompromised condition is very rare and often mortal. Our patient survived from an acute aortic dissection in addition to acute infective endocarditis during his hospital stay.

### References