Infective Endocarditis Presenting as Acute Renal Failure and Unusual Complications

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Abstract

Infective endocarditis can be a diagnostic challenge. Few cases have so far reported infective endocarditis presenting as renal failure. Pseudoaneurysms of the mitral-aortic intervalvular fibrosa and splenic abscess are rare complications of infective endocarditis. We herein report a case of an 80-year-old man admitted due to anorexia, malaise, edema of the legs and renal failure. A progressive degradation of the patient’s renal function was documented and hemodialysis was started. Blood cultures revealed the presence of \textit{Enterococcus faecalis}, and the patient was treated with ampicillin and gentamicin. The transesophageal echocardiogram findings showed pseudoaneurysms of the mitral-aortic intervalvular fibrosa and in the aortic root wall. Due to abdominal pain, an abdominal CT scan was performed and showed a cystic lesion compatible with splenic abscess. The patient received 6 weeks of antibiotic treatment. There was progressive clinical improvement and regression of the splenic abscess, but no recovery of the renal function. This report illustrates an unusual clinical presentation of infective endocarditis with unusual complications that were successfully treated with antibiotics.

Key words: infective endocarditis, renal failure, pseudoaneurysm, splenic abscess

(DOI: 10.2169/internalmedicine.54.3236)

Introduction

The incidence of infective endocarditis is 30 to 100 episodes per million patient-years (1). Despite improvements in diagnostic and therapeutic strategies, the fatality rate has not significantly decreased since the end of the 1970s; more than one-third of the patients will die within the first year of diagnosis (1).

The diagnosis of infective endocarditis remains a challenge. In a recent large prospective study, most patients diagnosed only had a few of the classic clinical hallmarks of infective endocarditis (2). There are few cases in the literature reporting infective endocarditis presenting with renal failure (3). We herein present a patient with infective endocarditis, admitted for an acute kidney injury, who developed unusual complications.

Case Report

An 80-year-old man, ex-smoker, with a history of diabetes mellitus, dyslipidemia, arterial hypertension, valvular heart disease and bladder cancer was admitted to our hospital because of malaise, anorexia, edema of the legs and renal failure. Three years previously, the patient had received a Freedom Solo 23 bioprosthesis for an aortic valve replacement. The patient had been doing well until 3 months before admission when he went to the emergency department because of fever, urinary urgency and incontinence for the last 11 days. His laboratory data is summarized in Table. The patient was discharged with a prescription for amoxicillin clavulanic acid (875 mg bid) and ibuprofen (600 mg bid). Four days later, he returned to the emergency room complaining of severe bilateral lumbar pain. A lumbosacral spine radiograph showed only degenerative changes. Renal
ultrasonography findings revealed kidneys with normal size and good corticomedullary differentiation without obstruction. His laboratory tests showed elevated inflammatory markers (Table) and he was discharged with a prescription for ciprofloxacin (500 mg bid) and nimesulide (100 mg id). However, the next day the patient was admitted in another hospital for a mental status change. Electrocardiography (ECG) showed complete atrioventricular block and atrial flutter. A pacemaker was implanted and he started oral anticoagulation. Two weeks later, he developed dysarthria, diplopia and imbalance and he was admitted due to an acute ischemic stroke. The patient was discharged 3 days later with a mild imbalance. Two months later (7 days before admission in our hospital), he was seen in the emergency room for lumbar pain, macroscopic hematuria and edema of the legs. He was discharged with a prescription for levofloxacin (750 mg id) and furosemide (40 mg id). Seven days later, his cardiologist sent him to the emergency department because of deterioration of his renal function. His analysis showed a creatinine level of 2.90 mg/dL and urea level of 99 mg/dL.

On admission to the emergency room, the patient complained of anorexia and malaise which he had experienced for several weeks. He maintained macroscopic hematuria and edema of the legs. The patient had no other symptoms. He did not have a fever and his blood pressure was 130/110 mmHg and pulse rate was 75/min. Cardiac auscultation revealed a 2/6 aortic systolic murmur which had previously been identified. Pulmonary auscultation demonstrated basal inspiratory crackles. His laboratory data, including complete blood count, biochemistry and blood gas analysis, is summarized in Table. Renal ultrasonography was unremarkable.

The patient was admitted due to acute kidney injury. Routine cultures, including blood and urine, were obtained. A peripheral blood smear showed rare schistocytes and the urinalysis findings detected rare eosinophils. The autoimmune study including antinuclear antibodies, antineutrophil cytoplasmic antibodies (ANCA MPO and ANCA PR3) and anti-glomerular basement membrane antibodies, serum protein electrophoresis, immunofixation electrophoresis and serum immunoglobulins were normal. Hypocomplementemia was documented one week after the appearance of edema of the

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**Table.** Time Course of Patient’s Laboratory Data.

<table>
<thead>
<tr>
<th></th>
<th>90 days before admission</th>
<th>86 days before admission</th>
<th>7 days before admission</th>
<th>On admission</th>
<th>3 days after admission</th>
<th>11 months after discharge</th>
</tr>
</thead>
<tbody>
<tr>
<td><strong>Urea (mg/dL)</strong></td>
<td>53</td>
<td>54</td>
<td>50</td>
<td>123</td>
<td>151</td>
<td>90 (under HD)</td>
</tr>
<tr>
<td><strong>Creatinine (mg/dL)</strong></td>
<td>1.2</td>
<td>1.30</td>
<td>1.4</td>
<td>3.4</td>
<td>4.50</td>
<td>3.50 (under HD)</td>
</tr>
<tr>
<td><strong>Hemoglobin (g/dL)</strong></td>
<td>13.8</td>
<td>13.0</td>
<td>10.7</td>
<td>11.1</td>
<td>10.1</td>
<td>15.5</td>
</tr>
<tr>
<td><strong>White cell count</strong></td>
<td>10.83</td>
<td><strong>14.53</strong></td>
<td>8.38</td>
<td>8.92</td>
<td><strong>9.02</strong></td>
<td>6.52</td>
</tr>
<tr>
<td><strong>Platelets count</strong></td>
<td>162</td>
<td>192</td>
<td><strong>82</strong></td>
<td>74</td>
<td>70</td>
<td>142</td>
</tr>
<tr>
<td><strong>C-reactive protein</strong></td>
<td>93.6</td>
<td><strong>106.2</strong></td>
<td>46.2</td>
<td><strong>107.7</strong></td>
<td>79.3</td>
<td>4.0</td>
</tr>
<tr>
<td><strong>Urine analysis</strong></td>
<td>Mild protein</td>
<td>Mild protein</td>
<td>Protein and erythrocyt (without casts)</td>
<td>Mild protein and erythrocyt (without casts)</td>
<td>Protein and erythrocyt (without casts)</td>
<td>-</td>
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<tr>
<td><strong>pH</strong></td>
<td></td>
<td></td>
<td></td>
<td>7.506</td>
<td></td>
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<tr>
<td><strong>pO2 (mmHg)</strong></td>
<td></td>
<td></td>
<td></td>
<td>87.8</td>
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<tr>
<td><strong>pCO2 (mmHg)</strong></td>
<td></td>
<td></td>
<td></td>
<td>32.3</td>
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<tr>
<td><strong>HCO3- (mmol/L)</strong></td>
<td></td>
<td></td>
<td></td>
<td>25</td>
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<tr>
<td><strong>INR</strong></td>
<td></td>
<td></td>
<td></td>
<td>3.1</td>
<td></td>
<td>89</td>
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<tr>
<td><strong>24 hour urinary sodium (mEq)</strong></td>
<td></td>
<td></td>
<td></td>
<td>29</td>
<td></td>
<td></td>
</tr>
<tr>
<td><strong>24 hour urinary potassium (mEq)</strong></td>
<td></td>
<td></td>
<td></td>
<td>1.86</td>
<td></td>
<td></td>
</tr>
</tbody>
</table>

Note: Values outside the normal range are formatted in bold text.
Legend: protein – proteinuria; erythrocyt – erythrocyturia; HD – hemodialysis
legs [C3: 66 mg/dL (normal range 83-177 mg/dL); C4 and CH50 were within the normal ranges]. Renal scintigraphy did not indicate evidence of a renal embolism. Because of the patient’s recent history of fever and aortic bioprosthesis, stroke, complete atrioventricular block and multiple antibiotic schemes with persistent, elevated inflammatory markers, we suspected infective endocarditis and ordered a transthoracic echocardiogram that revealed no vegetations. A progressive degradation of renal function was observed and hemodialysis was started on sixth day after admission (Fig. 1). At this time, the preliminary blood cultures revealed a Gram-positive coccus in chains. Because infective endocarditis had not been ruled out, a transesophageal echocardiogram was performed (Fig. 2) and revealed a normally functioning aortic bioprosthesis, normal left ventricle systolic function and pseudoaneurysms of the mitral-aortic intervalvular fibrosa (20×6 mm) and in the aortic root wall. There were no periprosthetic leaks or shunts between cardiac cavities. Meanwhile, a definitive blood culture revealed the presence of Enterococcus faecalis and ampicillin plus gentamicin were prescribed. Gentamicin trough levels were within toxic levels (defined as ≥2 μg/mL) in some determinations reaching a maximum level of 4.37 μg/mL in one determination. Cardiac surgery was not considered due to the high risk of the procedure. On the thirtieth day of admission, because of a poorly localized and slight abdominal pain, an abdominal echography was performed and showed a nodular image in the spleen. An abdominal CT scan revealed a cystic lesion, 100×44 mm, with contrast uptake at thickened walls, compatible with splenic abscess (Fig. 3). CT guided percutaneous drainage was proposed, but the patient refused. Control blood cultures were negative.

He had sustained apyrexia and completed 6 weeks of antibiotic treatment. He was stable at discharge with residual diuresis between 350 and 1,800 mL/day on hemodialysis (three times a week). No events were recorded after his discharge. Eleven months after discharge, the analysis showed a normal complete blood count, an albumin level of 37.4 g/L, and a normal level of C-reactive protein (Table). Echocardiograms were similar to the previous examinations. Control abdominal CT scan showed a small lesion in the spleen (24×22 mm). We settled on periodic examinations due to the patient’s high surgical risk and good response to medical
treatment.

**Discussion**

Enterococcal prosthetic valve infective endocarditis is similar to enterococcal native valve infective endocarditis (4). Both occur primarily in older men and are predominantly community-acquired. However, in prosthetic valves there is a higher risk of myocardial abscess formation and fewer valve vegetations.

Acute renal failure is a common complication of infective endocarditis which occurs in approximately one-third of the patients (5). The causes of renal failure in infective endocarditis are multifactorial (6). It may be explained mainly by acute glomerulonephritis due to immune complex deposition; renal infarction; acute interstitial nephritis and acute tubular damage due to nephrotoxic agents (e.g., vancomycin and aminoglycosides); and cortical necrosis due to underperfusion of the kidney. In this case, the renal failure is probably a result of acute glomerulonephritis caused by immune complex deposition because it started before antibiotic treatment for infective endocarditis, with a short period of exposure to nonsteroidal anti-inflammatory drugs, in a hemodynamically stable patient with hematuria, edema of the legs and hypocomplementemia. We also suspected interstitial nephritis because of patient’s deteriorating renal function in relation to the previously prescribed antibiotics; however, the patient did not have a rash, arthralgias or eosinophilia. We did not perform a renal biopsy because the patient’s condition had worsened.

There are few reports in the literature on infective endocarditis presenting with renal failure. Renal failure resulting in hemodialysis treatment is not generally recognized as a complication of infective endocarditis (7). Renal failure in the context of infective endocarditis is typically resolved with successful antibiotic treatment (8). However, irreversible renal failure can occur if the diagnosis and antibiotic treatment are delayed because the severity of glomerulonephritis is related to the duration of the infection (8). In the present case, complete atrioventricular block and a previous history of renal failure had worsened. We did not perform a renal biopsy because the patient’s condition had worsened.

In summary, infective endocarditis should be considered in the differential diagnosis of acute renal failure. If the diagnosis is delayed, renal dysfunction may be irreversible and other complications may arise.

**The authors state that they have no Conflict of Interest (COI).**

**References**

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