Culture-negative Peritonitis Caused by Splenic Infarction in a Continuous Ambulatory Peritoneal Dialysis Patient

Hye Eun Yoon, Il Kim, Young Wook Kim, Hyun Wha Chung and Seok Joon Shin

Abstract

A 43-year-old diabetic woman on peritoneal dialysis, developed left upper abdominal pain and culture-negative cloudy peritoneal dialysate. The dialysate had WBC counts of 1,532/μL with 90% polymorphonuclear cells. The patient did not respond well to anti-bacterial therapy. Abdominal CT scan revealed diffuse atherosclerosis in the abdominal vessels and wedge-shaped splenic infarction. Anticoagulation therapy was initiated and an improvement in peritonitis was observed without peritoneal catheter removal. Thus, in peritoneal dialysis patients with diffuse atherosclerosis or the risk of systemic embolization, symptoms of unexplained left upper quadrant pain and culture-negative peritonitis should be evaluated to rule out splenic infarction.

Key words: continuous ambulatory peritoneal dialysis, peritonitis, splenic infarction

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Introduction

Peritonitis is a major complication in peritoneal dialysis (PD) patients and is an important cause of mortality. PD-related infectious peritonitis is usually treated using adequate anti-bacterial therapies. However, in up to 22% of all cases, PD effluent cultures prove to be negative, and thus the dilemma of appropriate diagnosis and management arises in patients with culture-negative cloudy dialysate (1). Splenic infarction due to thromboembolism, atherosclerosis or hematologic disorders is an unusual cause of acute culture-negative peritonitis with increased polymorphonuclear (PMN) leukocytes. Splenic infarction is often difficult to diagnose clinically, so the fact that it is a cause of culture-negative peritonitis in PD patients is frequently overlooked. In this report, we present a case of culture-negative peritonitis caused by splenic infarction (CNPSI) in a continuous ambulatory peritoneal dialysis (CAPD) patient, with a review of the relevant literature.

Case Report

A 43-year-old diabetic woman on CAPD for the past 5 years was admitted to the hospital due to left upper abdominal pain radiating towards the left shoulder and cloudy PD effluent. She had a pertinent history of dilated cardiomyopathy with chronic atrial fibrillation and was on regular digoxin and warfarin treatment for 4 years. Three years prior to admission, the patient had undergone cardiac catheterization, which showed diffuse three-vessel coronary artery disease. The progressive left upper abdominal pain was described to be constant and colicky with radiation towards the left shoulder and it was aggravated by respiratory inspiration. There was no recent history of trauma. On examination, she was afebrile, with a blood pressure of 150/90 mmHg. The patient’s turbid effluent was inoculated (approximately 10 mL) in two blood-culture bottles. Then, 50 mL of dialysate was collected and centrifuged. The sediment after centrifugation was processed for further culture procedures. The patient’s initial dialysate WBC count was 1,532/μL with 90% polymorphonuclear cells, and RBC count was 40/μL. Gram stain of dialysate fluid was clear with no growth on subsequent culture. Under suspicion of peritonitis, she received empirical intraperitoneal cefazolin and gentamycin. Her liver function tests were within normal limits. Laboratory investigations including repeated peritoneal effluent bacterial, fungal and acid-fast bacillus cultures were...
negative. Abdominal CT scan showed diffuse atherosclerotic changes with calcified plaques in the abdominal aorta and splenic vessels. There was also a partial and wedge-shaped changes with calcified plaques in the abdominal aorta and splenic vessels (arrow). (C) Follow up CT scan shows regression of the infract area of spleen (arrow).

Discussion

Culture-negative or sterile peritonitis is a common problem in PD patients. Possible causes for negative cultures include small volume samples, inappropriate microbiological culture techniques or bacterial infection when the patient is previously treated with antibiotics (1, 2). The 2010 International Society for Peritoneal Dialysis (ISPD) Guideline suggests that an optimal culture technique is the combination of sediment culturing of 50 mL effluent and bedside inoculation of 5-10 mL effluent in two blood culture bottles. This method could lower culture negative rate to less than 5% (3). The management of CAPD patients with culture-negative peritonitis should be customized according to their underlying etiology. Cloudy dialysate of culture-negative peritonitis may be due to pathologic increases of either cellular or noncellular constituents of peritoneal fluid. In cellular causes of cloudy dialysate, elevation in the levels of PMN leukocytes, eosinophils, erythrocytes or monocytes may produce turbid peritoneal dialysate. The pathologic conditions leading to cellular culture-negative peritonitis include inflammation of intraperitoneal or juxtaperitoneal viscera, atypical infections, drug-induced chemical peritonitis, and dialysate endotoxin infection (4). In the current case, the patient presented PMN leukocytes predominant cellular culture-negative peritonitis. Atypical infection such as fungus or mycobacteria was not identified despite an optimal culture technique, and an abdominal CT scan showed splenic infarction. With due consideration of all the results, the peritonitis was found to be compatible with CNPSI.

Previously, there have been only a few cases of CNPSI and the clinical characteristics of those reports are described in Table 1. Common symptoms in previous CNPSI reports were left upper abdominal pain frequently radiating towards left shoulder area and cloudy dialysate. The patients were usually afebrile. Rarely, splenic infarction can progress to splenic abscess following secondary infection or splenic rupture, and in turn, results in relapsing peritonitis or massive hemoperitoneum, respectively (5, 6). Dialysate of CNPSI...
has a wide range of WBC counts and accompanies increased levels of PMN leukocytes. Splenic infarction results in localized edema of the spleen and stretching of the capsule. This can presumably cause peritoneal inflammation with the influx of PMN leukocytes in the dialysate. Sometimes, dialysate of CNPSI can be presented as bloody, accompanying an increased RBC count. It is suggested that leaking capillaries related to inflammation may cause the transient hemoperitoneum (7).

There are a number of reports on causes of splenic infarction in CAPD patients due to extensive atheromatous disease, peripheral embolism with hematologic abnormalities and isolated thromboembolism of splenic artery without any systemic features (2, 5, 7). There is a possibility that the cause of splenic infarction in the present case was embolization of a cardiac origin, because the patient had chronic atrial fibrillation and was on oral anticoagulant therapy. However, on echocardiogram, we could not find any evidence of thromboembolism from a cardiac source. In addition, the patients in the previous reports of CNPSI as well as the present case had diabetes in common (2, 5, 7). It is unclear that diabetes per se is the cause of sterile peritonitis in CAPD patients. On the other hand, it is well known that severe vascular disease, extensive atherosclerosis and thromboembolic events are common in advanced diabetic patients. Therefore, in the present case, the longstanding diabetic condition may have contributed to make extensive atherosclerotic lesion in the vessels including splenic artery, which might have caused splenic infarction and resulted in culture-negative peritonitis. There are some similarities between the case of Blake et al. and the present case with respect to the presence of extensive atheromatous disease as a cause of splenic infarction (5). They reported that the patient had diffuse atheroma in the splenic arterial tree with evidence of recent thrombotic occlusion, and the splenic infarction progressed to splenic abscess and relapsing peritonitis. Previously, atherosclerotic disease of the aorta has been reported as a source of emboli leading to splenic infarction (8). Likewise, the present patient showed extensive atherosclerotic lesions and calcified plaques in abdominal aorta and splenic vessels without any thromboembolic sources or hematologic disorders. As far as we know, this case is the first reported case of atherosclerotic splenic infarction causing CNPSI without any other complications in a CAPD patient.

The 2010 ISPD Guideline suggests that if a culture-negative peritonitis has no clinical improvement by 5 days, despite empirical anti-bacterial therapy, peritoneal catheter removal should be considered (3). In this aspect, CNPSI treatment can be a management dilemma. Diagnosis of splenic infarction in CAPD patients may be more delayed than non-PD patients, due to empirical antibiotic treatment (2). In addition, if higher dialysate WBC count is maintained as in the present case, it is difficult to make a decision with respect to change in antibiotics or removing peritoneal catheter. In previous reports as described in Table 1, all the patients of CNPSI were treated with empirical anti-bacterial therapy at the beginning of peritonitis. However, after the diagnosis of splenic infarction, anticoagulation therapy was started and clinical improvement was achieved. Long-term anticoagulation therapy in CNPSI should be considered for high risk patients as with any systemic thromboembolism. The clinical course of a CNPSI is gradual resolution of pain and decrease of dialysate WBC counts without any clinical complications. But, if it progresses to relapsing peritonitis due to splenic abscess or massive hemoperitoneum secondary to splenic rupture, catheter removal or splenectomy might be necessary (2). In conclusion, the possibility of a CNPSI should be kept in mind while treating patients with left upper abdominal pain and with culture-negative cloudy dialysates, who do not respond to antibiotic therapy, especially in the presence of a predisposing cause.

### Table 1. Clinical Features of Peritonitis Associated with Splenic Infarction in Continuous Ambulatory Peritoneal Dialysis Patients

<table>
<thead>
<tr>
<th>References</th>
<th>Age/Sex</th>
<th>Clinical presentation</th>
<th>Dialysate WBC count (% PMN)</th>
<th>Cause of Splenic infarction</th>
<th>Treatment</th>
</tr>
</thead>
<tbody>
<tr>
<td>Blake et al. (1989)(5)</td>
<td>26/F</td>
<td>Splenic abscess</td>
<td>Not described</td>
<td>Atherosclerosis</td>
<td>Splenectomy</td>
</tr>
<tr>
<td></td>
<td></td>
<td>Relapsing peritonitis</td>
<td></td>
<td>Polycythemia Thromboembolism</td>
<td>Anticoagulation</td>
</tr>
<tr>
<td>Syed et al. (1993)(7)</td>
<td>32/F</td>
<td>CNPSI</td>
<td>760 (90)</td>
<td>Dysfibrinogenemia Thromboembolism</td>
<td>Anticoagulation</td>
</tr>
<tr>
<td>Kanagasundaram et al. (1998)(6)</td>
<td>68/M</td>
<td>Spleenic rupture</td>
<td>244 (70)</td>
<td>Apical thrombus Embolism</td>
<td>Splenectomy</td>
</tr>
<tr>
<td>Yang et al. (2007)(2)</td>
<td>57/M</td>
<td>CNPSI</td>
<td>9,089 (98)</td>
<td>Isolated splenic thromboembolism</td>
<td>Anticoagulation</td>
</tr>
<tr>
<td>Present case</td>
<td>43/F</td>
<td>CNPSI</td>
<td>1,532 (90)</td>
<td>Atherosclerosis</td>
<td>Anticoagulation</td>
</tr>
</tbody>
</table>

CNPSI: culture-negative peritonitis caused by splenic infarction
The authors state that they have no Conflict of Interest (COI).

References