Improvements in Pneumatosis Cystoides Intestinalis and Hepatic Portal Venous Gas with Conservative Therapy in a Patient on Maintenance Dialysis

Kenta Torigoe, Hideyuki Arai, Ayuko Yamashita, Yoshiaki Muraya, Yoko Obata and Tomoya Nishino

Abstract

A 77-year-old man on maintenance dialysis developed hypotension, nausea and abdominal pain one hour after beginning to undergo hemodialysis. Abdominal computed tomography (CT) showed gas shadows in the intrahepatic portal vein and the small intestinal wall, but no signs indicating intestinal necrosis. Three days later, the gas shadows on abdominal CT disappeared by conservative therapy. In cases with both pneumatosis cystoides intestinalis and hepatic portal venous gas, intestinal necrosis should therefore be suspected and surgical therapy should also be considered, particularly in hemodialysis patients with a risk of intestinal ischemia. However, conservative therapy may be an option in cases with no intestinal necrosis.

Key words: pneumatosis cystoides intestinalis, hepatic portal venous gas, hemodialysis

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Introduction

Pneumatosis cystoides intestinalis (PCI) is a morphological finding wherein multiple small gas cysts are seen in the intestinal wall. It may be accompanied by intestinal necrosis or several other underlying diseases. Hepatic portal venous gas (HPVG) involves the presence of gas in the portal vein and it is an indicator of intestinal necrosis and a poor prognosis. However, recent advances in imaging have made an early diagnosis possible. Cases with a good prognosis that do not have intestinal necrosis have also been reported. Generally, the presence of both PCI and HPVG is considered to be strongly suggestive of intestinal necrosis (1). Hemodialysis patients are at a particular risk of intestinal ischemia due to vascular calcification from either calcium (Ca)/phosphorus (P) metabolism disorders or hypotension during hemodialysis (2, 3). Surgical treatment should be considered as there is a possibility of intestinal necrosis occurring when both PCI and HPVG are present (1). We herein report a patient demonstrating hemodialysis with PCI and HPVG who improved after undergoing conservative therapy.

Case Report

The patient in this case was a 77-year-old man. In December 2009, he was started on maintenance hemodialysis for chronic renal failure caused by chronic glomerulonephritis. He had a history of alcoholic-decompensated cirrhosis. In February 2014, his blood pressure before hemodialysis was 116/54 mmHg, but after one hour, it had declined to 70/38 mmHg. Since the patient developed persistent nausea and abdominal pain, the dialysis was stopped and he was brought to our hospital via emergency transport.

During this transport, his consciousness was clear and the nausea disappeared. His body temperature was 36.4°C, blood pressure 143/52 mmHg, heart rate 78 beats/minute and oxygen saturation of peripheral artery (SpO2) 100% (room air). On physical examination, mild tenderness was seen at the upper abdomen, but there was no muscular guarding or rebound tenderness. Anemia of the palpebral conjunctiva was observed, but there was no jaundice of the
**Table.** Laboratory Findings on Admission.

<table>
<thead>
<tr>
<th></th>
<th>Complete blood count</th>
<th>Blood chemistry</th>
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<tbody>
<tr>
<td>WBC</td>
<td>7,160 /μL</td>
<td>TP 6.7 g/dL</td>
</tr>
<tr>
<td>RBC</td>
<td>242×10⁴ /μL</td>
<td>Cl 98 mEq/L</td>
</tr>
<tr>
<td>Hb</td>
<td>7.6 g/dL</td>
<td>Ca 8.2 mg/dL</td>
</tr>
<tr>
<td>Hct</td>
<td>23.5%</td>
<td>MCV 665 μL</td>
</tr>
<tr>
<td>PLT</td>
<td>15.9×10⁴ /μL</td>
<td>CRP 1.16 mg/dL</td>
</tr>
</tbody>
</table>

**Coagulation**

<table>
<thead>
<tr>
<th></th>
<th>LDH 270 U/L</th>
<th>Arterial blood gas (room air)</th>
</tr>
</thead>
<tbody>
<tr>
<td>PT-INR</td>
<td>1.20</td>
<td>pH 7.40</td>
</tr>
<tr>
<td>APTT</td>
<td>28.7 sec</td>
<td>PCO₂ 32 mmHg</td>
</tr>
<tr>
<td>Fib</td>
<td>418 mg/dL</td>
<td>PO₂ 108 mmHg</td>
</tr>
<tr>
<td>FDP</td>
<td>5.30 mg/dL</td>
<td>HCO₃⁻ 19 mEq/L</td>
</tr>
<tr>
<td>D-dimer</td>
<td>2.08 mg/dL</td>
<td>ABG -3.4 mEq/L</td>
</tr>
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**Figure.** Simple abdominal CT on admission showed gas images in the intrahepatic portal vein (A) and the intestinal wall (B) (arrows). In abdominal contrast CT, edematous thickening of the intestinal wall was observed on the anal side of the small intestine, where gas was observed in the wall (C) (arrows).

bulbar conjunctiva. Respiratory sounds were clear and there was no heart murmur. A blood chemistry examination showed slightly elevated inflammatory reactions with leukocytes 7,130/μL and C-reactive protein (CRP) 1.16 mg/dL. An arterial blood gas analysis showed mild metabolic acido- sis with pH 7.40, carbon dioxide partial pressure (pCO₂) 32 mmHg and bicarbonate ion (HCO₃⁻) 19 mEq/L (Table). Simple abdominal computed tomography (CT) showed gas shadows in the intrahepatic portal vein and the small intestinal wall (Figure A, B). Since intestinal ischemia was suspected, contrast abdominal CT was performed. From its findings, there were no contrast defects in the mesenteric artery or vein, and the contrast effect was maintained in the intestinal wall. However, edematous thickening of the intes-
tinal wall and stenosis of the lumen were observed on the anal side of the small intestine, where gas was also observed in the wall (Figure C). Based on these findings, PCI and HPVG were diagnosed. Conservative therapy was selected because there were no clear signs of intestinal necrosis, the abdominal symptoms decreased spontaneously and the patient’s overall condition was good. The patient was kept in a fasting state and administered sulfactam/cefoperazone (SBT/CPZ) at 2 g/day and high-flow oxygen using a face mask (5 L/minute, five hours a day). After hospitalization, the abdominal pain disappeared. On day 3 of the illness, simple abdominal CT showed that the gas images had disappeared from the intrahepatic portal vein and the small intestinal wall. Temporary increases in leukocytes to 10,040/μL and CRP to 8.40 mg/dL were observed in a blood examination on day 3, but these had improved on day 6 to a leukocyte count of 5,200/μL and CRP of 2.78 mg/dL. The high-flow oxygen administration was stopped on day 4 of the illness. The patient resumed liquids on day 5 and solids on day 6; no abdominal symptoms were observed. The SBT/CPZ was stopped on day 6. The hypotension and abdominal symptoms did not recur during subsequent hemodialysis sessions.

The patient’s general condition improved and he was discharged on day 14.

Discussion

PCI is a morphological finding that involves numerous small gas cysts in the intestinal wall. This condition is idiopathic in 15% of patients; however, in 85% of patients, an underlying condition is present, such as intestinal obstruction, ischemic intestinal disease, inflammatory bowel disease, chronic obstructive pulmonary disease or collagen disease (4). There are several theories regarding its etiology, such as a mechanical theory involving an increased intestinal pressure and a bacterial theory that involves gas-producing microorganisms (5). Emergency surgery is indicated if intestinal necrosis is present, but should also be considered in cases of: 1) patients aged 60 years or older, 2) a leukocyte level of more than 12,000/μL, 3) vomiting, 4) HPVG, 5) septicemia or 6) acidosis (6). HPVG was first reported in 1955 by Wolfe (7); the condition accompanied a case of necrotic enteritis in a newborn. Previously, it was considered that HPVG showed a poor prognosis as a sign of intestinal necrosis. However, recent advances in imaging have allowed an early diagnosis; the number of patients without intestinal necrosis and with a good prognosis has therefore increased (8). Several causes are reported in addition to intestinal necrosis (49%), such as intestinal dilatation (13%), an abscess (12%), ulcerative colitis (4%), a gastric ulcer (4%), endoscope manipulation (4%) and Crohn’s disease (4%) (8). The mechanism of onset may involve: 1) necrotic intestinal lesions or damage to the intestinal mucosa, 2) severe intestinal dilatation or 3) an intra-abdominal abscess caused by sepsis or an infection by gas-producing bacteria (9). In cases of HPVG, intestinal necrosis is related to: 1) hypotension, 2) elevated serum lactate dehydrogenase (LDH) and 3) PCI. Surgical treatment should be considered in the presence of two or more of these signs (10). Furthermore, PCI and HPVG occur together 31% of the time. If intestinal ischemia is the causative factor, this figure rises to 52% (11). Thus, the presence of both signs suggests intestinal ischemia and is considered to be an indication for emergency surgery (1). In such cases, the mortality rate is 56% and the prognosis is poor (12).

However, if there is no intestinal necrosis, conservative therapy may be an option. For example, in HPVG, conservative therapy is considered in cases of: 1) mild abdominal pain and no signs of peritoneal irritation, 2) a low-grade fever, 3) a rapid improvement of abdominal pain, 4) the relief of symptoms by intestinal decompression and 5) localized gas shadows in the intestinal wall (13). The HPVG volume is not associated with the presence of intestinal necrosis (13). In our case, although an abdominal contrast CT scan was performed due to suspected intestinal ischemia or necrosis because of the presence of both PCI and HPVG, the gas shadows in the intestinal wall were localized and the contrast effect of the intestinal wall was maintained. In addition, no fever or signs of peritoneal irritation were present. The abdominal pain and hypotension were transient and improved quickly. The inflammatory reactions were mild, and there was no elevation of serum LDH. Thus, the patient was treated conservatively (with fasting, antibiotics and high-flow oxygen) instead of surgically and his general condition improved.

As both PCI and HPVG were observed in our case, the presence of intestinal ischemia, but not necrosis, was suspected. We surmised that the patient was in a state of non-occlusive mesenteric ischemia (NOMI) because no contrast defects were found in the mesenteric artery or vein. It is well known that NOMI does not present with clear signs of an acute mesenteric arterial occlusive disease, such as thrombosis or embolism. States of a low cardiac output, a reduced circulating plasma volume, dehydration and hypotension can cause the peripheral mesenteric arteries to spasm, leading to noncontinuous segmental intestinal ischemia or necrosis. The risk of NOMI is particularly high in hemodialysis patients because of vascular calcification caused by Ca/P metabolism disorders or hypotension during hemodialysis (2, 3). In this case, hypotension and other symptoms appeared during hemodialysis, which suggests that ultrafiltration may lead to spasms of the mesenteric artery, resulting in NOMI. An elevated intestinal pressure due to luminal stenosis from intestinal edema likely contributed to the PCI and HPVG. A rapid improvement of intestinal circulation due to the reversal of hypotension likely explains why intestinal necrosis failed to develop in this patient.

Furthermore, in this case, high-flow oxygen was administered as a supplemental therapy. High-flow oxygen has been reported to be effective in cases of symptomatic PCI (14). The therapeutic mechanisms underlying high-flow oxygen therapy include the following: 1) it suppresses the growth of
anaerobic intestinal bacteria that contribute to intestinal pneumatosis and 2) it increases the oxygen partial pressure in the veins, which reduces the partial pressure of non-oxygen (the components of intestinal pneumatosis are non-oxygen: nitrogen, hydrogen, etc.), which causes the pneumatosis to disappear via a concentration gradient (15). Therefore, in cases of symptomatic PCI, high-flow oxygen is therefore considered to be an optional therapy.

In summary, there is a high possibility of intestinal necrosis when both PCI and HPVG are present, and since intestinal ischemia can easily occur in hemodialysis patients, surgical treatment should therefore still be considered. However, if there is no intestinal necrosis, then conservative therapy may also be an option. The decision to operate should be made based on the patient’s overall condition and the examination findings.

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

References

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