INTERNAL MEDICINE

□ CASE REPORT □

Bacterial Peritonitis due to Duodenal Perforation by a Fish Bone in an Elderly Peritoneal Dialysis Patient

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Abstract

The patient, a 77-year-old-man, began peritoneal dialysis (PD) in August 2005. In January 2009, he developed lower abdominal pain and cloudy PD effluent. A diagnosis of peritonitis was made and *Escherichia coli* was detected in cultures of the PD effluent. An abdominal computed tomography scan showed a fish bone in the duodenal wall. An upper gastrointestinal endoscopy was performed, and a 3-cm fish bone was removed. We thus recommend careful investigation with the possibility of enteric peritonitis from the intestinal tract when *E. coli* is detected in effluent cultures during PD.

Key words: peritoneal dialysis, bacterial peritonitis, fish bone, perforation

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Introduction

Compared with hemodialysis, peritoneal dialysis (PD) has many merits, including: 1) the ability to preserve residual renal function, 2) less disturbance of hemodynamics, 3) arteriovenous access and skin needling are unnecessary, 4) little restriction on diet, and 5) frequent hospital visits are unnecessary. Although the incidence rate of PD-related bacterial peritonitis has been reduced due to recent improvements in connect systems, this complication remains the biggest reason for patients to discontinue PD (1). Thus, the treatment and prevention of bacterial peritonitis is essential to ensure the feasibility of long-term PD treatment and the maintenance of good peritoneal function.

Most cases of bacterial peritonitis occur because of touch contamination, exit-site infection, or tunnel infection. However, enteric peritonitis due to intraperitoneal organ diseases such as intestinal perforation have been reported to make up only 6% of the total cases of bacterial peritonitis in PD patients (2). It has also been reported that the mortality rate of PD patients who contract bacterial peritonitis due to gastrointestinal perforation is high (about 40%) (3). However, it is difficult to diagnose enteric peritonitis in PD patients because their acute abdominal symptoms can be masked by the peritoneal lavages in the PD therapy. It is reported that a delayed diagnosis of enteric peritonitis leads to increased severity of the disease and a high mortality rate of 40% (3). In the present case, since the bacterial peritonitis was due to *Escherichia coli*, a computed tomography (CT) scan of the abdomen was performed to search for an intraperitoneal cause, which found to be a linear fish bone perforating an ascending portion of the duodenum. An upper gastrointestinal endoscopy was performed, and a fish bone was found and removed from the site. It was surmised that the perforation by the fish bone caused *E. coli* to leak into the abdominal cavity and led to the bacterial peritonitis. To our knowledge, this is the first reported case of bacterial peritonitis caused by a fish bone perforating the duodenal wall in a PD patient.

Case Report

The patient in this case report was a 77-year-old man. In August 2005, the patient began PD due to renal failure caused by chronic glomerulonephritis. From January 2009,

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Table 1. Laboratory Data in Onset of Peritonitis

<Peripheral blood>			<blood chemistry>					
WBC	4,700	/mm^3	TP	5.0	g/dL	AST	10	IU/L
Seg.	64	%	ALB	2.0	g/dL	ALT	10	IU/L
Lymph	28	%	BUN	47	mg/dL	LDH	148	IU/L
Mono	4	%	Cr	6.82	mg/dL	Amy	1	IU/L
Baso	0	%	UA	5.0	mg/dL	FBS	88	mg/dL
Eosion	4	%	Na	138	mEq/L			
RBC	281	×10^4/mm^3	K	3.3	mEq/L	<Immunological>		
Hb	8.7	g/dL	Cl	97	mEq/L	CRP	5.5	mg/dL
Hct	26.7	%	Ca	8.4	mg/dL			
Plt	14.1	×10^4/mm^3	IP	2.7	mg/dL	< PD effluent >		
			TC	121	mg/dL	WBC	1,590	/mm^3
			TG	57	mg/dL	Seg.	96	%

the volume of his urine decreased and edema appeared in his legs. The dose of furosemide and the frequency of PD fluid exchange were increased, but the symptoms worsened. In mid-June, he was admitted to the hospital because of whole-body edema and bilateral pleural effusion with dyspnea. His history included a cerebral infarction at the age of 62 years, and this caused left-sided partial hemiplegia. A chest radiograph on admission showed a large, bilateral accumulation of pleural effusion. Following treatment with icodextrin, the patient's symptoms gradually improved after admission. On day 5, he suddenly developed lower abdominal pain. His vital signs were as follows: blood pressure, 164/77 mmHg; heart rate, 80 bpm; and body temperature, 37.1℃. Physical examination revealed severe abdominal tenderness and muscle defense in the lower abdomen. Because turbidity was observed in the PD effluent, bacterial peritonitis was suspected. Clinical laboratory data were as follows: white blood cell count (WBC), 4,700/μL and C-reactive protein (CRP) level, 5.5 mg/dL (Table 1). Further, the WBC count in PD fluid was 1,590/μL (neutrophils, 96%). Gram staining of the PD effluent showed a large number of neutrophils but no clear signs of bacterial phagocytosis. On the same day, he was treated with daily intraperitoneal injections of 1 g cefazolin and 1 g ceftazidime, and his renal replacement therapy was temporarily changed from PD to hemodialysis. Five days after the onset of peritonitis, *E. coli* was detected in a culture of the first cloudy PD effluent. Since we suspected the presence of intraperitoneal inflammation, caused by, for example, diverticulitis or gastrointestinal perforation, an abdominal CT scan was obtained to examine the peritoneal cavity. Numerous air bubbles were found in the peritoneal cavity, and a linear fish bone was seen in the duodenal wall (Fig. 1). An emergency upper gastrointestinal endoscopy was performed (Fig. 2), and we found a 3-cm fish bone perforating the duodenal wall. The bone was removed by forceps, and 3 sites of perforation were clipped using a transendoscopic method. As the patient's records showed that he had eaten grilled fish for dinner 3 days before the onset of peritonitis, a bone from this fish was considered to be the cause of the peritonitis. After endoscopy, the patient was instructed to fast for 3 days. When he began eating again, the abdominal symptoms did not worsen. The abdominal pain disappeared and the PD effluent became clear 1 week after starting antibiotics. The CRP level had risen to 9.78 mg/dL at the peak, but 1 week after onset, it improved to 2.54 mg/dL. Fourteen days after the onset of peritonitis, an upper gastrointestinal endoscopy showed that the perforation had closed. The daily intraperitoneal injections of 1 g cefazolin and 1 g ceftazidime were terminated after 3 weeks. The patient was then switched back to PD and he recovered from the peritonitis.

Discussion

PD-related peritonitis occurs at a rate of 0.23-0.29 times per patient-year (4), and the incidence rate has been decreasing due to factors such as improvements in connect systems (5). However, prevention, early detection, and early treatment of bacterial peritonitis are essential to ensure the feasibility of long-term PD and to maintain healthy peritoneal function. Here, we report the first case of bacterial peritonitis caused by duodenal perforation from a fish bone in a hospitalized PD patient.

Pathogenic microorganisms in PD-related peritonitis consist of about 70% gram-positive and 20-30% gram-negative microbes (5). Of the 210 cases of PD-related peritonitis caused by enterobacteria, 111 were *E. coli* (52.9%), 57 were *Klebsiella* species (27.1%), and 12 were *Enterobacter* species (5.7%) (6). Thus, *E. coli* is the most frequently identified causative microorganism: *E. coli*-related peritonitis is thought to occur from touch contamination or exit-site infection that leads to enteric peritonitis from the intestinal tract, but in many cases the route of bacterial entry is not clear (6). Although previous reports of peritonitis due to intestinal perforation are rare, there have been reports related to perforation by a piece of bamboo (7), peritoneal catheters (8), diverticular perforation (9), colitis (8), and intestinal amyloidosis (10).

In the present case, *E. coli* was detected in a culture of the PD effluent. Some researchers advise that if enterobacteria, especially *E. coli*, are detected in PD effluent, one should be aware of gastrointestinal perforation as a possible differential diagnosis in PD patients presenting with peritonitis (11). Thus, we suspected gastrointestinal perforation as

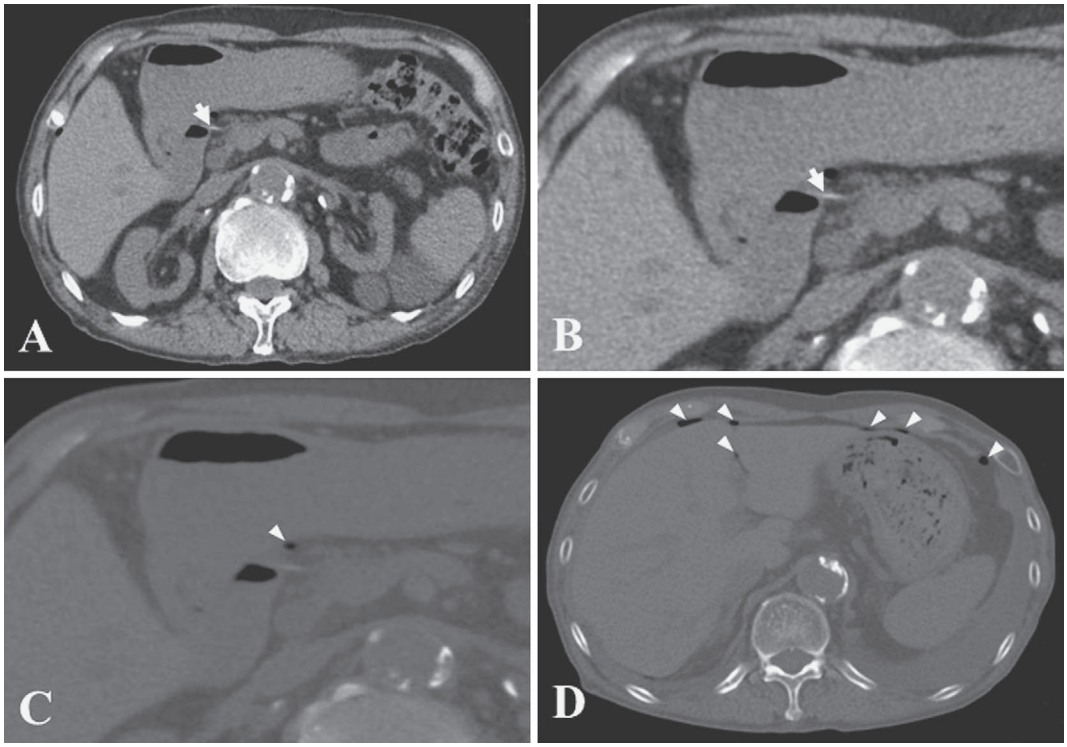

Figure 1. **The findings of abdominal CT scan. An abdominal CT scan showed a linear fish bone in the duodenal wall (1A and 1B), air bubbles around the fish bone (1C), and multiple air bubbles (1D) in the peritoneal cavity.**

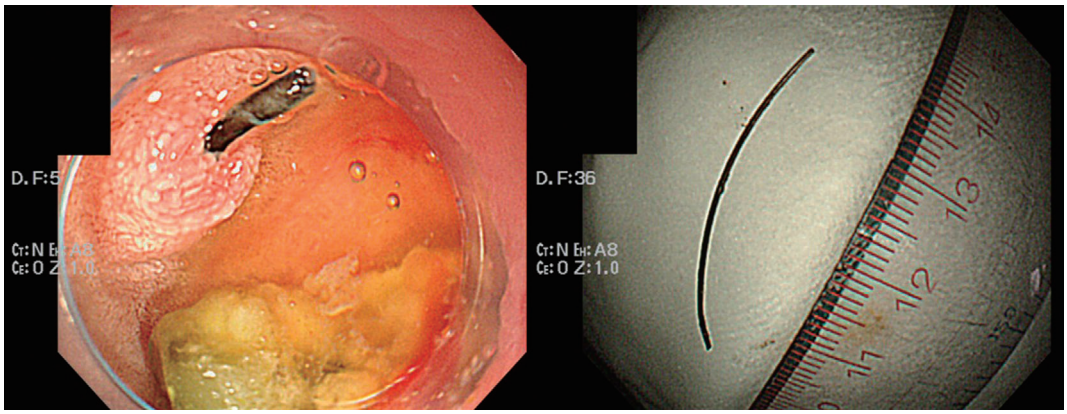

Figure 2. **The findings of upper gastrointestinal endoscopy revealed a 3-cm fish bone which perforated the duodenal wall.**

the most likely possibility during our investigations. An abdominal CT scan to examine the peritoneal cavity found a fish bone in the duodenal wall, as well as the presence of free air. From this finding, we deduced that *E. coli* had leaked into the abdominal cavity, causing the bacterial peritonitis.

When *E. coli* or multiple bacteria are detected in cultures of the PD effluent, complicated causes of peritonitis such as diverticulitis or gastrointestinal perforation should be suspected. However, it takes several days for culture results to be obtained, making early diagnosis difficult. It has been reported that amylase levels of ≥50 IU/L in the PD effluent indicate serious intraperitoneal complications (acute pancreatitis, gastrointestinal perforation, etc.), and the levels are not increased in simple continuous ambulatory peritoneal dialysis peritonitis (12). This benchmark can be useful in making an early diagnosis of complicated peritonitis. In the present case, we suspected that the patient's left-sided partial hemiplegia might have resulted in touch contamination as a cause of peritonitis, so the amylase level in the PD effluent was not measured initially. In future cases where there is a possibility of gastrointestinal perforation, measurement of effluent amylase level could be helpful in early detection.

Since air bubbles can enter the abdominal cavity during PD, it is difficult to make a definitive diagnosis of gastrointestinal perforation based solely on the presence of free air

in the peritoneal cavity, as seen in the CT scan in this case. There are reports, however, that one should suspect gastrointestinal perforation when a large volume of free air is present (13). In the current case, we suspected duodenal perforation by a fish bone because we detected multiple air bubbles —some of which were seen adjacent to the fish bone—and reached a diagnosis after endoscopy.

Perforation in PD patients mostly occurs in the lower gastrointestinal tract, and in approximately 50% of cases, it is caused by colonic diverticula. Such cases generally require surgery, which reportedly leads to PD weaning (14, 15). In this case, perforation was in the upper gastrointestinal tract, which has fewer resident bacteria than the lower gastrointestinal tract (16). Unlike the vulnerable peripheral tissues of the perforated part caused by ulcers, it was thought that PD can be continued after closing the perforation with endoscopic clipping, since it was a small perforation caused by a sharp foreign object (17).

The number of elderly people undergoing dialysis has increased recently. Since many elderly people have complications involving serious declines in cardiac function, PD therapy, which can be undertaken at home and has less effect on hemodynamics, is recommended to maintain quality of life. However, since elderly people also often have impaired chewing and swallowing abilities, they are more likely to aspirate foreign objects. Although aspiration of foreign objects by elderly people is not rare, the frequency of such objects perforating the gastrointestinal tract has been reported to be only about 1% (18). Goh et al. surveyed 62 cases where surgery was necessary because an aspirated foreign object had perforated the gastrointestinal tract. Of the 60 cases where the foreign object could be identified, 55 cases (93% of the total) involved toothpicks, fish bones, or chicken bones (19). The location of the perforation was the anus or rectum in 18 cases, the ilium in 17 cases, the jejunum in 12 cases, the stomach in 6 cases, the large intestine in 4 cases, the duodenum in 2 cases, and Meckel's diverticulum in 1 case. As seen in the present case, many dialysis patients have a history of cerebrovascular disease, and they are more likely than non-dialysis patients to experience gastrointestinal perforation or piercing from aspirating foreign objects (20). Therefore, aspiration should be taken seriously in such patients. Typically, the abdominal symptoms are severe and the patients show muscle defense and abdominal tenderness. However, in PD patients with frequent changes in PD fluid, their acute abdominal symptoms might be attenuated by the peritoneal lavages of PD therapy. Hence, early detection of gastrointestinal perforation-related peritonitis is difficult and there is a greater possibility of delayed diagnosis and serious illness in PD patients (3).

If multiple enteric organisms are grown, particularly in association with anaerobic bacteria, the risk of death is increased and a surgical evaluation should be obtained (1). When *E. coli* or multiple microorganisms, particularly in association with anaerobic bacteria, are detected in the PD fluid of elderly patients, the possibility of foreign object aspiration should be considered. In addition, gastrointestinal perforation should be kept in mind as a possible cause of foreign body-induced perforative peritonitis. The abdominal CT scan should be examined promptly, and the amylase levels in the PD effluent should be evaluated to clarify the cause of peritonitis.

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

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