The Journal of Veterinary Medical Science

NOTE

Internal Medicine

Pneumatosis coli after partial ligation of congenital portosystemic shunt in a dog

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J. Vet. Med. Sci.
80(10): 1549–1552, 2018
doi: 10.1292/jvms.18-0229

Received: 24 April 2018
Accepted: 8 August 2018
Published online in J-STAGE: 17 August 2018

ABSTRACT. Pneumatosis coli is a rare intestinal disorder in dogs that is characterized by submucosal or subserosal emphysema of the colon. An 8-year-old castrated male Shih Tzu developed anorexia and hematochezia after undergoing surgery for a congenital splenophrenic shunt. Abdominal radiographic examination revealed linear radiolucency throughout the wall of the large intestine. Results of abdominal computed tomography revealed intramural gas tracking along the colon and rectum. Based on these findings, a diagnosis of pneumatosis coli was made. The dog was treated with antibiotics, but the general condition gradually deteriorated, and the dog died 6 days after the day of diagnosis. It was suspected that portal hypertension following partial ligation of congenital portosystemic shunt may have been associated with pneumatosis coli in this case.

KEY WORDS: canine, colitis, intramural gas

An 8-year-old castrated male Shih Tzu was presented to a primary care hospital with 1-week history of anorexia. Results of blood tests revealed an elevation of liver enzyme, and the dog was administered supportive therapy. The dog's clinical signs did not improve, and it was referred to the Veterinary Medical Center of the University of Tokyo. At presentation, no abnormality was detected on physical examination. Results of complete blood cell count (CBC) were within reference ranges. Abnormalities in the results of blood biochemical tests included elevation of serum alanine aminotransferase (ALT) (397 U/*l*; reference intervals [RI], 17–78 U/*l*) and serum alkaline phosphatase (ALP) (2,335 U/*l*; RI, 47–254 U/*l*), hyperammonemia (299 μg/d*l*; RI, 16–75 μg/d*l*), and mild hypoalbuminemia (2.0 g/d*l*; RI, 2.6–4.0 g/d*l*). Increased total serum bile acid concentrations were observed (preprandial—53.6 μmol/*l*; RI, <5 μmol/*l* and postprandial—146.6 μmol/*l*; RI, <20 μmol/*l*). Results of the coagulation tests revealed slight elevation of prothrombin time (PT) but activated partial thromboplastin time (aPTT), thrombin-antithrombin complex level (TAT), and fibrinogen level were within the reference ranges as follows: PT, 9.4 sec (RI, 6.8–8.6 sec); aPTT, 17.9 sec (RI, 13.1–26.9 sec); TAT, 0.115 *n*g/m*l* (RI, <0.2 *n*g/m*l*); and fibrinogen level, 284 mg/d*l* (RI, 88–336 mg/d*l*). Results of an examination using computed tomography (CT) revealed a congenital splenophrenic shunt, enlargement of the right adrenal gland, and a splenic mass. The dog was administered oral lactulose (0.5 m*l*/kg every 12 hr, Monilac, Chugai Pharma, Tokyo, Japan), and the clinical signs improved. The dog underwent laparotomy and ligation of the splenophrenic shunt and liver biopsy 14 days after diagnosis of congenital splenophrenic shunt. Because the tentative ligation of the shunt during the operation induced an increase in the blood pressure of the mesenteric vein from 9 to 18 mmHg, partial ligation using sutures and biopsy of the liver were performed. The histopathological examination of the liver revealed fibrosis and microvascular dysplasia. The dog developed anorexia and depression after the operation, and small amounts of bloody mucous stool were frequently observed from 5 days after the operation. Abdominal radiographic examination revealed linear radiolucency throughout the wall of the large intestine (Fig. 1). Ultrasonographic examination of the abdomen showed bright echoes within the layers of the colonic wall (Fig. 2), suggesting an accumulation of intramural gas; ascites was not observed. Abdominal contrast-enhanced CT examination without anesthesia for detecting the underlying disease which cause accumulation of gas in the large intestine revealed extraluminal gas tracking along the colon but underlying disorder was not observed (Fig. 3). Based on these findings, a diagnosis of pneumatosis coli was made. The dog was administered oral metronidazole (16 mg/kg every 12 hr, Flagyl, Shionogi Pharma, Osaka, Japan), and oral lactulose was discontinued. However, the dog's general condition worsened. Two days after the diagnosis, the linear radiolucency visible on abdominal radiographic examination had not improved, and endoscopic examination was conducted without anesthesia.

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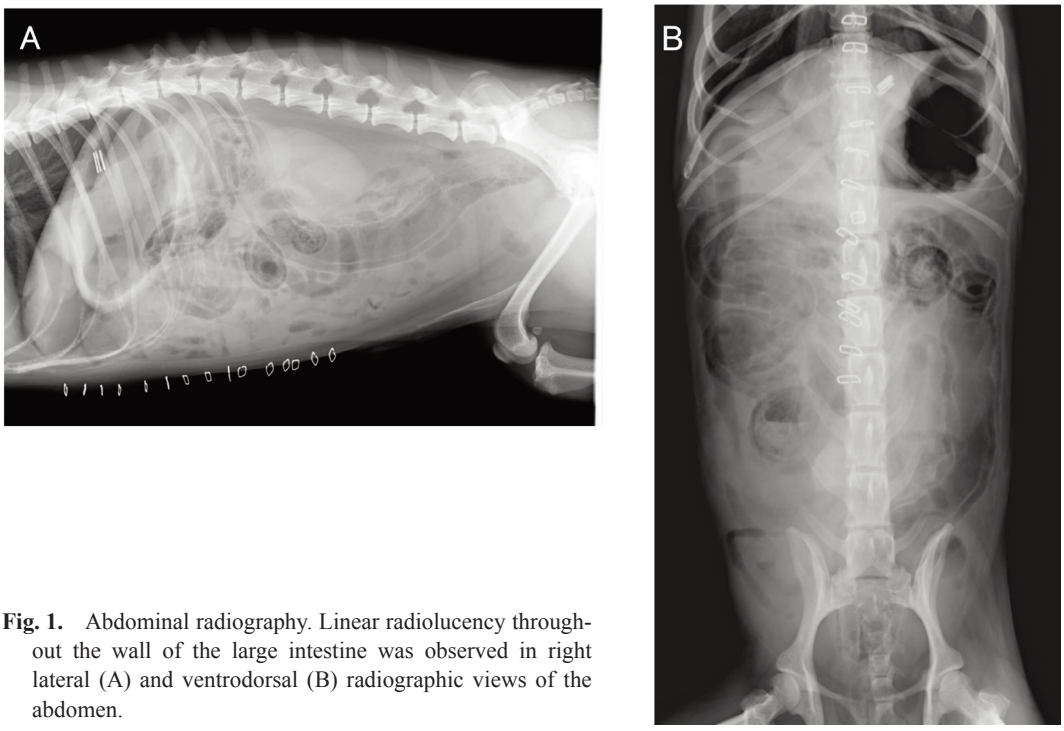

Fig. 1. Abdominal radiography. Linear radiolucency throughout the wall of the large intestine was observed in right lateral (A) and ventrodorsal (B) radiographic views of the abdomen.

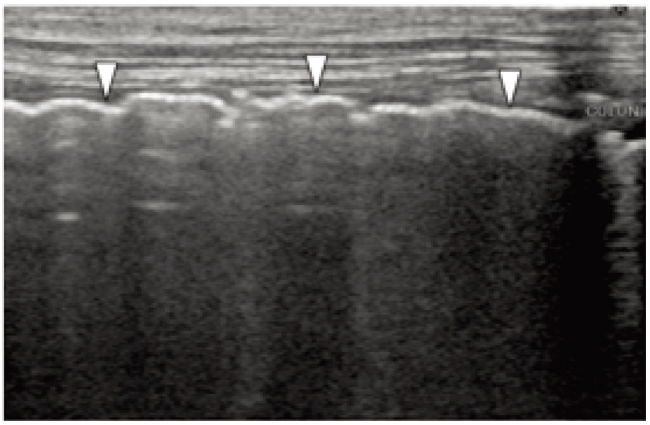

Fig. 2. Ultrasonography of the abdomen. Bright echoes (arrowheads) within the layers of the colon wall confirming the accumulation of intramural gas was observed in abdominal ultrasonography.

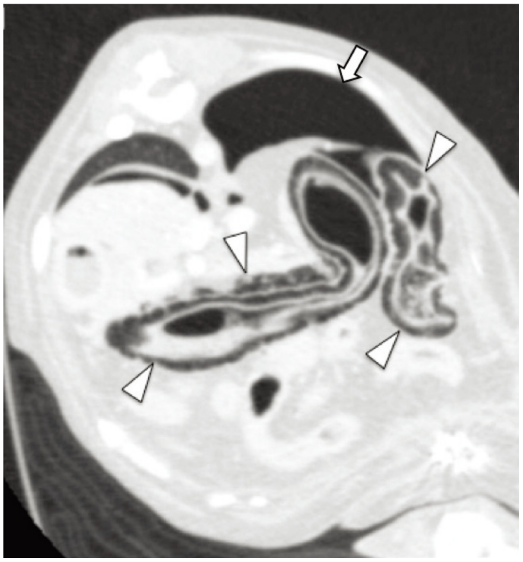

Fig. 3. Abdominal computed tomography. Intramural gas (arrowheads) tracking along the colon was observed. Postoperative pneumoperitoneum (arrow) was observed.

Endoscopic examination of the colon revealed marked erosions, edema, and hyperemia of the mucosa (Fig. 4). Despite the continued treatment, the general status of the dog gradually deteriorated, and the dog died 6 days after the day of diagnosis of pneumatosis coli. The colonic mucosa was collected via endoscopic forceps during a postmortem examination (2 hr after death), and histological examination showed atrophy of the intestinal villi due to degeneration, necrosis of the epithelial cells, and many vacuoles in the submucosa (Fig. 5).

Pneumatosis intestinalis is a rare intestinal disorder characterized by submucosal or subserosal emphysema of the intestine in humans. This gas accumulation may occur in the stomach, small bowel, or large bowel. When gas is present in the large bowel, it

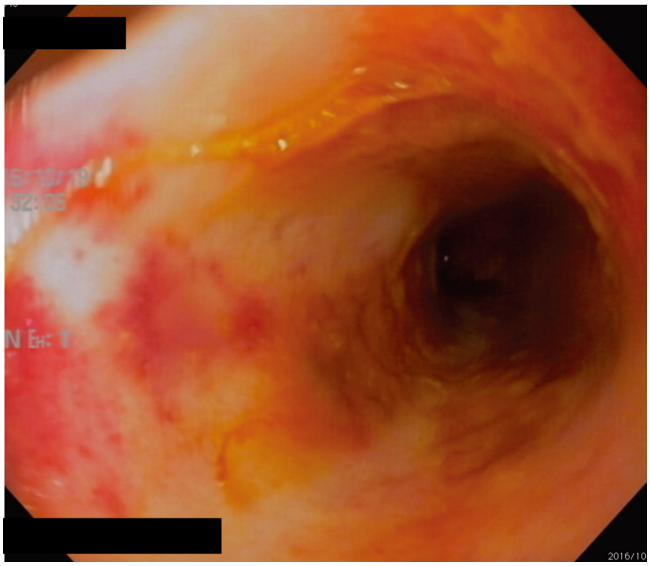

Fig. 4. Endoscopic examination of the colon. Marked erosions, edema, and hyperemia of the mucosa was observed.

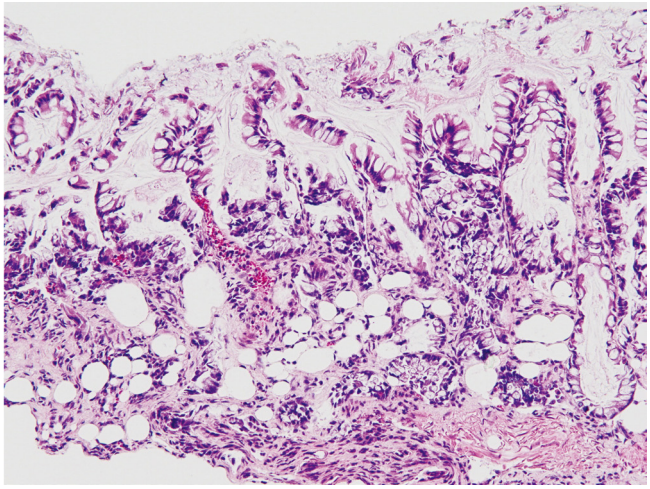

Fig. 5. Histological images of biopsy of the colon. Atrophy of the intestinal villi due to degeneration and necrosis of the epithelial cells, and lots of vacuoles in the submucosa were observed (hematoxylin and eosin stain × 100).

is called pneumatosis coli [10, 16, 17]. Pneumatosis intestinalis has also been reported in dogs (5 cases in the colon and 1 case in the small intestine) [1, 3, 9, 13, 17, 18] and two cats [7, 12].

In the 5 dogs with pneumatosis coli, the clinical signs were diarrhea (5/5), tenesmus (4/5), hematochezia (3/5), and inappetence (2/5). The clinical signs appeared abruptly without obvious underlying causes in 4 cases. In 1 case, the clinical signs began 2 days after an emergency laparotomy due to complications of a cesarean delivery in which a segment of the ascending colon was ablated [1]. A portal-azygos shunt was detected in 1 case although its relationship with pneumatosis coli was unclear [13]. The diagnosis was made by radiographic (5/5), ultrasonographic (3/5), or CT (1/5) imaging. Histological examination of colonic biopsies was conducted in 1 case, and it showed mild chronic colitis with mild eosinophilic infiltration [3]. The pathogenesis of canine pneumatosis coli is still unclear because of a small number of canine cases of pneumatosis coli. Enhanced intestinal permeability to gas secondary to mucosal injury provoked by previous partial colectomy was considered the most likely underlying mechanism of pneumatosis coli in 1 dog [1].

The cause of pneumatosis intestinalis is also unknown in human medicine; the two main theories hypothesizing on the pathogenesis of pneumatosis intestinalis suggest either a mechanical or bacterial etiology [16]. The mechanical theory proposes there is an increase in the intraluminal pressure caused by mechanisms such as bowel obstruction or emphysema, resulting in the dissection of gas into the bowel wall from either the lungs via the mediastinum or the intestinal lumen. Alternatively, the bacterial theory suggests gas-producing bacilli enter the submucosa through areas of increased mucosal permeability or mucosal breaks, causing the formation of gas confined within the bowel wall. In the present study, fecal culture was not performed and its relationship with pneumatosis coli was unclear, which is one of the limitation of this study.

Although the cause of intestinal necrosis in this case was not clear, several mechanisms are proposed. First, the increase in the blood pressure of the mesenteric vein due to the ligation of the splenophrenic shunt may have induced the colonic mucosal injury. The hepatic fibrosis that was observed in the liver may have worsened the increase in the blood pressure of the mesenteric vein due to the ligation. The second possible cause is microthrombus. Although a thrombus was not detected by contrast-enhanced CT examination in the present case, we could not exclude the possibility of a microthrombus.

Lactulose is metabolized by bacterial enzymes into carbon dioxide and hydrogen gas, and it increases the amount of gas in the colon [2, 4]. Although the adverse effects of lactulose are usually minimal and rarely happen, there have been a few case reports suggesting lactulose can cause pneumatosis intestinalis in humans [5, 10]. In these cases, pneumatosis coli was encountered in a patient who had undergone liver transplantation [11] and a patient with cirrhosis [6]. In addition to the accumulation of gas caused by lactulose, these underlying problems may be interwoven to induce pneumatosis coli. In the present case, the increased blood pressure of the mesenteric vein caused by the partial ligation of the splenophrenic shunt and the use of lactulose may have been related to the development of pneumatosis coli.

Regarding prognosis, there are both mild/nonfatal and clinically worrisome cases of pneumatosis intestinalis in humans, which depend on the severity of underlying desease. Surgical intervention such as colectomy is needed for the latter cases with intestinal necrosis, whereas the patients with benign pneumatosis intestinalis without necrosis improved with medical treatment [8, 14, 15]. However, it is difficult to differentiate between benign and clinically worrisome pneumatosis intestinalis without performing laparotomy [19]. In the previously reported 5 dogs with pneumatosis coli [1, 3, 9, 13, 17], 4 cases were treated with antibiotics and/or food therapy (highly digestible low-residue diet) and 1 case was not given any specific treatment, and all cases improved. In the

present case, the prognosis was poor despite the antibiotic therapy, and histological examination of the colon revealed degeneration and necrosis of the mucosal epithelial cells, indicating that it may have corresponded to the clinically worrisome pneumatosis intestinalis in humans. And pneumatosis coli occurred immediately after the ligation of the shunt vessel in this case, which suggested that the increase of the pressure of mesenteric vein due to the ligation of the shunt correlated with pneumatosis coli.

In conclusion, pneumatosis coli was diagnosed in a dog that had undergone surgery for a splenophrenic shunt, and the surgery may have been associated with pneumatosis coli in this case. Pneumatosis coli is rare disease, but it should be considered in dogs that develop congestion of the intestinal vessels as a result of changes in the blood flow and hydrostatic pressures. Furthermore, the prognosis of pneumatosis coli is not always good in dogs, and surgical treatment should be considered when the response to supportive therapy is poor.

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