pISSN 1598-298X / eISSN 2384-0749
J Vet Clin 33(4) : 210-213 (2016)
http://dx.doi.org/10.17555/jvc.2016.08.33.4.210

Journal of Veterinary Clinics
www.ksvc.or.kr

Chronic Intestinal Pseudo-Obstruction Caused by Intestinal Adenocarcinoma in a Mixed-Breed Dog

Heesup Moon*, Subin Hong*, Sangyeoun Kim*, Sungguon Park*, Taehwan Kim*, Nahyun Kim and Jae-Hoon Lee*[1]**

**Institute of Animal Medicine, College of Veterinary Medicine, Gyeongsang National University, Jinju 52828, Korea*
***Korea Institute of Toxicology, Jinju 52900, Korea*

(Received: July 13, 2016 / Accepted: August 16, 2016)

Abstract : A 9-year-old, mixed-breed, castrated male dog presented with a one-week history of severe vomiting. Abdominal radiography showed a dilated small intestine loop. Abdominal ultrasonography showed small-intestine distension up to 5 cm in diameter without mechanical obstruction. During exploratory laparotomy, an aperistaltic ileal segment was resected. There was no narrowed cavity in the resected plane. Histopathologic and immunohistochemical findings confirmed visceral myopathy due to intestinal adenocarcinoma in the ileum and revealed partial destruction of the longitudinal and circular muscles with fibrosis. Chronic intestinal pseudo-obstruction caused by adenocarcinoma was diagnosed after considering the above investigative results.

Key words **:** chronic intestinal pseudo-obstruction, intestinal adenocarcinoma, visceral myopathy, dog.

Introduction

Chronic intestinal pseudo-obstruction (CIPO) is defined as the presence of chronic intestinal dilatation and dysmotility in the absence of mechanical obstruction (7). CIPO is rarely diagnosed in small animals, but is well documented in human medicine (4,5), where intestinal idiopathic pseudo-obstruction is classified as acute or chronic according to duration of the disease (13). Acute intestinal pseudo-obstruction can result from intestinal injury, intestinal surgery, sepsis, or infectious disease (1,4). The two main classifications of primary CIPO in human medicine are visceral myopathy and visceral neuropathy, and the main cause of secondary one is progressive systemic sclerosis (13). In veterinary medicine, only visceral myopathy has been reported. Other potential secondary causes are immune-mediated disease, endocrine disease, and pharmacologic factors (4).

CIPO is characterized by clinical signs of mechanical obstruction, intermittent gastrointestinal dysfunction, and weight loss, but without actual mechanical obstruction, namely, functional ileus (4,7,13). Only 10 documented cases of CIPO have been reported in the veterinary literature (seven cases in dogs, two in cats, and one in a horse (4,6,8,10)). This report describes a dog with CIPO showing non-typical visceral myopathy caused by intestinal adenocarcinoma.

Case

A nine-year-old, mixed-breed, castrated male dog (weight, 8.4 kg) was referred to the Gyeongsang National University Animal Medical Center with a one-week history of severe vomiting, loss of appetite, anorexia, and weight loss. The amount of feces had decreased in the 2 weeks prior to referral. The intermittent vomiting had persisted for one month. A radiographic contrast study prior to referral had not identified intestinal obstruction. Treatment with antiemetics failed to resolve the vomiting, so the dog was referred to our center.

A grade 5 systolic murmur was heard on clinical examination. The body condition score was 2/5 with depressed. The dog defecated during palpation of the abdomen. Hematologic analysis revealed granulocytosis (12.7×10^9/L, reference range 3-11.8×10^9/L). Although the erythrocyte count was within normal limits, the red cell distribution width-corpuscular volume was slightly high (17.9%; reference range, 12%-16%). Serum biochemistry indicated slightly low total protein (5.2 g/dL; reference range, 5.4-8.2 g/dL) with normal globulin (2.7 g/dL; reference range, 2.3-5.2 g/dL) and albumin (2.5 g/dL; reference range, 2.5-4.4 g/dL). Potassium was within normal limits (3.9 mmol/L; reference range, 3.7-5.8 mmol/L), but mild hypocalcemia (8.3 mg/dL; reference range, 8.6-11.8 mg/dL) and moderate hyponatremia (134 mmol/L; reference range, 138-160 mmol/L) were observed.

Thoracic radiography was unremarkable. Abdominal radiography showed multifocal irregular-shaped mineral opacities, likely caused by residual contrast medium admitted in the local animal hospital. Dilation in overall small intestine was identified, suggesting small bowel obstruction. Abdominal ultrasonography revealed a small intestine filled with echogenic fluid and distended up to 5 cm, but without visible obstruction by a mass or foreign body. Anechoic fluid was detected in the abdominal cavity and attributed to ascites. In the absence of visible mechanical obstruction, a tentative diagnosis of CIPO was made.

[1]Corresponding author.
E-mail : jh1000@gnu.ac.kr

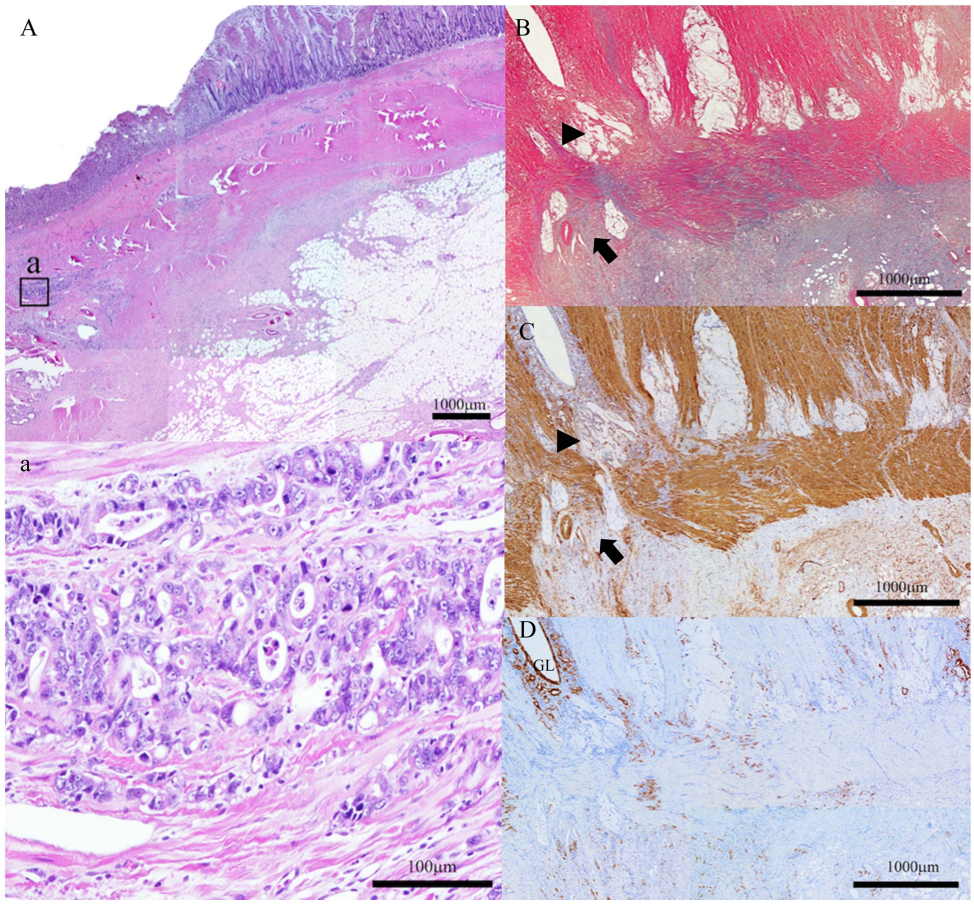

Fig 1. (A) Hyperemic intestine with severe distension up to 5 cm in diameter. (B) Narrowed portion at the end of an aperistaltic segment and a mass (arrow). (C) Mass (arrow) observed in the resected malfunctioning segment. (D) Transverse section of resected segment. (D-a) Dilated portion. (D-b, c) Portion narrowed by the mass. (D-d) Normal portion. There was no visible obstruction in the resected portion.

Other potential causes of enteropathy, including parvovirus infection, parasitic infection, and pancreatitis, were ruled out by the Canine Parvovirus Antigen Test Kit, the Canine Pancreatic Lipase Test Kit, and the Canine Heartworm Antigen-Anaplasma Phagocytophilum-Platys-Borrelia Burgdorferi-Ehrlichia Canis-Ewingii Antibody Test Kit.

Exploratory laparotomy was performed to rule out complete obstruction. Cefazolin 25 mg/kg was administered intravenously (IV) for antibacterial prophylaxis during the procedure. Atropine (0.04 mg/kg, subcutaneously (1)), butorphanol (0.2 mg/kg, SC), and diazepam (0.2 mg/kg, IV) were used as preanesthetic drugs prior to surgical treatment. Following induction with etomidate (2 mg/kg, IV), anesthesia was maintained with inhaled isoflurane. The patient was positioned ventrodorsally and the abdominal area was prepared in aseptic fashion. A medial skin incision was made using a No. 15 blade. As soon as the abdomen was incised, a severely distended, hyperemic ileum was observed (Fig 1A). No peristalsis was noted, and functional ileus was identified. The ingesta could be moved manually through the malfunctioning portions of the small intestine and the narrowed portion. Malfunctioning loops were identified during laparotomy but with no mechanical obstruction. A mass was recognized at the end of an aperistaltic segment (Fig 1B, 1C), which was surgically resected. No obstruction or narrowed cavity was observed in the resected plane (Fig 1D).

Hartman/Dextrose was administered postoperatively at a maintenance dosage. Metronidazole (30 mg/kg, infused IV at a constant rate), cefazolin (25 mg/kg, IV, every 12 h), vitamin K1 (1 mg/kg, SC, every 24 h), and famotidine (0.5 mg/kg, IV, every 12 h) were administered for one day following surgery. A gas bubble, loss of serosal margin, and anechoic fluid were identified on postoperative radiography. The findings on abdominal ultrasonography were consistent with those of radiography. Serum biochemistry demonstrated low total protein (3.6 mg/dL) with low albumin (1.5 mg/dL) and low globulin (2 mg/dL). Partial parenteral nutrition was provided at a maintenance dosage postoperatively. There was mild hypocalcemia (8.3 mg/dL; reference range, 8.6-11.8 mg/dL) and hyperphosphatemia (8.4 mg/dL; reference range, 2.9-6.6 mg/dL). Hematology demonstrated severe leukocytopenia (2.2×10^9/L, reference range 6-17 $\times 10^9$/L). The dog died on the first postoperative day because of peritonitis.

Histopathologic and immunohistochemical findings in the small intestine of the dog showed muscular, serosal hyperplasia, lymphatic infiltration (Fig 2A). Partial destruction of the longitudinal (arrow, Fig 2B, 2C) and circular (arrowhead, Fig 2B and 2C) muscles was also observed. Glandular hyperplasia seen in the muscular layer (Fig 2D) was indicative of adenocarcinoma.

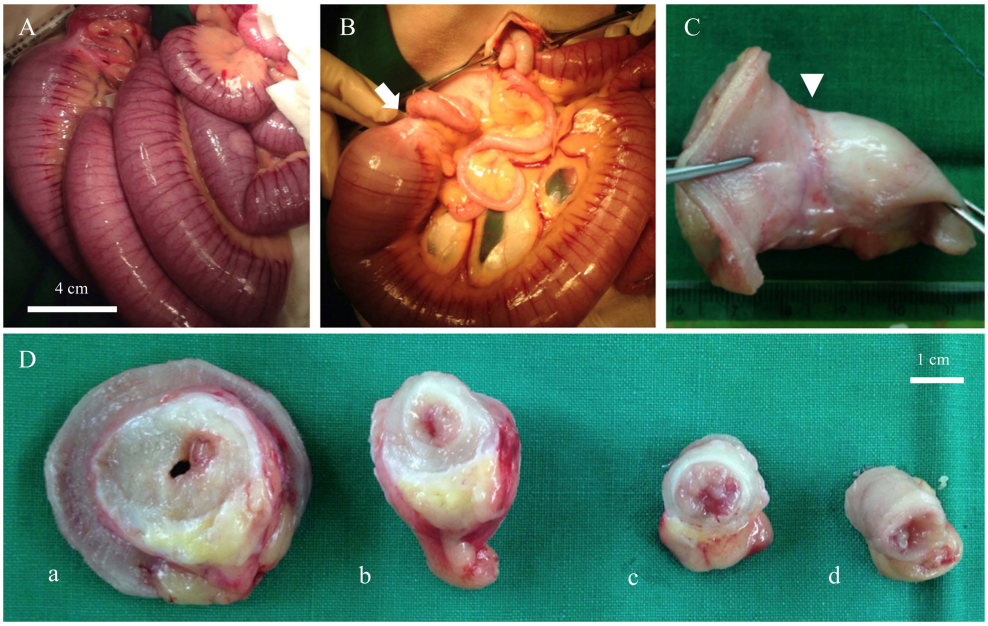

Fig 2. Histological section of the narrowed segment. (A) Hematoxylin & eosin staining. Muscular, serosal hyperplasia, with lymphatic infiltration (a) Hematoxylin & eosin staining. Magnified picture of box. Partial destruction of longitudinal muscle (arrow) and circular muscle (arrow head) [(B) Masson trichrome staining, (C) Horseradish perixodase immunohistochemical examination] (D) Pan-cytokeratin staining. Adenocarcinoma with glandular hyperplasia (GL) in the muscular layer.

Discussion

In the present case, CIPO was tentatively suspected in view of the lack of evidence of mechanical obstruction on diagnostic images. Functional ileus and a mass that was not obstructing the intestinal cavity were identified during exploratory laparotomy. The diagnosis of CIPO was made on the basis of histopathologic findings indicating damage to the muscular layer.

According to the criteria used in human medicine, there was no evidence of an acute etiologic cause for the chronic pseudo-obstruction found in the present case. In small animal practice, there have been no reports of neuropathy, as indicated by dysphagia, esophageal symptoms, and achalasia, which are commonly associated with disorders of the nervous system (4,13). Further, systemic sclerosis, characterized by thick fibrotic skin and esophageal involvement, has not been reported in small animal practice (3,11). Our patient did not present with clinical features of neuropathy or systemic sclerosis.

The typical histopathologic features of visceral myopathy described in human medicine are vacuolar degeneration, muscle thinning and fibrosis, of the longitudinal layer in particular (12). The present dog had no thinning but did have thickening of the longitudinal and circular muscles with fibrosis. No vacuolar degeneration were identified. Among the reported cases from small animal practice, only one cat reported in 2005 had true visceral myopathy (6). The other cases were reported to have CIPO, but showed histopathologic features different from those reported in human medicine. Four dogs reported in veterinary medicine had atrophy, fibrosis, and mononuclear infiltration of the muscularis externa (2,13). Two dogs had atrophy of the muscularis externa without fibrosis, either with or without mononuclear cell infiltration. One dog had hyperplasia of the circular muscle without fibrosis but did have inflammation. Another dog showed myocytic vacuolar degeneration (6). Only two dogs had circular muscle involvement. None of the animals with CIPO had damaged longitudinal muscle, which is reported to be the most common site of injury in humans, except for a cat described in 2005. One of the two cats was diagnosed with diffuse intestinal lymphosarcoma but no histopathologic details were given. The present case was found to have visceral myopathy caused by intestinal adenocarcinoma, with partial destruction of the longitudinal and circular muscles, fibrosis and lymphatic infiltration.

Various medical treatments, including metoclopramide, erythromycin, and cisapride, have been tried for CIPO in humans, but have not been successful, presumably because the there is no response to these agents in the intestines (6). Lack of response to these drugs is the hallmark of CIPO (4). In the present case, the referring veterinarian documented complete unresponsiveness to antiemetics. There is no optimal treatment for CIPO in veterinary medicine (9). All cases were euthanized, except for one cat treated by surgical resection of the most severely affected portion of jejunum in 2005 (14). Unsuccessful abdominal surgeries were performed in three dogs (4). We performed abdominal surgery in the present dog, but the dog died on the day after surgery.

To our best knowledge, this is the first report in veterinary literature of the clinical findings in a dog diagnosed with CIPO caused by intestinal adenocarcinoma.

References

1. Bueno L, Fioramonti J, Ruckebusch Y. Postoperative intestinal motility in dogs and sheep. Am J Dig Dis 1978; 23: 682-689.
2. Burns G, Karcher L, Cummings J. Equine myenteric

ganglionitis: a case of chronic intestinal pseudo-obstruction. Cornell Vet 1990; 80: 53-63.
3. D'Angelo WA, Fries JF, Masi AT, Shulman LE. Pathologic observations in systemic sclerosis (scleroderma): a study of fifty-eight autopsy cases and fifty-eight matched controls. Am J Med 1969; 46: 428-440.
4. Dvir E, Leisewitz A, Van der Lugt J. Chronic idiopathic intestinal pseudo-obstruction in an English bulldog. J Small Anim Pract 2001; 42: 243-247.
5. Faure C, Goulet O, Ategbo S, Breton A, Tounian P, Ginies J-L, Roquelaure B, Despres C, Scaillon M, Maurage C. Chronic intestinal pseudoobstruction syndrome clinical analysis, outcome, and prognosis in 105 children. Dig Dis Sci 1999; 44: 953-959.
6. Harvey AM, Hall EJ, Day MJ, Moore AH, Battersby IA, Tasker S. Chronic Intestinal Pseudo-Obstruction in a Cat Caused by Visceral Myopathy. J Vet Med 2005; 19: 111-114.
7. Hirano I, Pandolfino J. Chronic intestinal pseudo-obstruction. Dig Dis 2000; 18: 83-92.
8. Lamb WA, France MP. Chronic intestinal pseudo-obstruction in a dog. Aust Vet J 1994; 71: 84-86.
9. Mathias JR, Clench MH. Neuromuscular diseases of the gastrointestinal tract. Specific disorders that often get a non-specific diagnosis. Postgrad Med 1995; 97: 95-98, 101-102, 105-108.
10. Moore R, Carpenter J. Intestinal sclerosis with pseudo-obstruction in three dogs. J Am Vet Med Assoc 1984; 184: 830-833.
11. Perez MI, Kohn SR. Systemic sclerosis. J Am Acd Dermatol 1993; 28: 525-547.
12. Rohrmann Jr C, Ricci M, Krishnamurthy S, Schuffler M. Radiologic and histologic differentiation of neuromuscular disorders of the gastrointestinal tract: visceral myopathies, visceral neuropathies, and progressive systemic sclerosis. Am J Roentgenol 1984; 143: 933-941.
13. Schuffler MD, Rohrmann CA, Chaffee RG, Brand DL, Delaney JH, Young JH. Chronic Intestinal Pseudo-Obstruction: A report of 27 cases and review of the literature. Medicine 1981; 60: 173-196.
14. Swayne D, Tyler D, Farrell R, Cornelius L. Sclerosing enteropathy in a dog. Vet Pathol Online 1986; 23: 641-643.