A 2-year-old castrated male shorthaired Miniature Dachshund was referred to the Veterinary Teaching Hospital of Nippon Veterinary and Animal Science University with fever, frequent vomiting, and watery diarrhea. Rectal temperature occasionally exceeded 40°C (104°F), and the vomitus was a yellow liquid flecked with blood.

Diagnostic tests on day 1 included a fecal examination, CBC, serum biochemical analyses, and radiographic examinations including a barium study. Abnormal findings included severe mature neutrophilia (43.6 × 10^9 neutrophils/L; reference range, 3.0 to 11.5 × 10^9 neutrophils/L), monocytosis (6.8 × 10^9 monocytes/L; reference range, 0.15 to 1.4 × 10^9 monocytes/L), and slightly high serum amylase activity (2,450 U/L; reference range, 1,300 to 2,300 U/L), with low serum lipase activity (301 U/L; reference range, 500 to 1,500 U/L). Radiographic examination revealed increased opacity in abdominal adipose tissue. This change was more pronounced in the cranial abdominal area, so that outlines of the liver and spleen and the position of the intestines were obscured (Fig 1). In addition, slightly delayed gastric emptying time was revealed by the barium study. The dog was treated for 7 days with fluid therapy, famotidine (10 mg, SC, q 24 h), metoclopramide (0.2 mg/kg [0.09 mg/lb], IM, q 12 h), and loperamide hydrochloride (0.08 mg/kg [0.036 mg/lb], PO, q 8 h). However, vomiting and diarrhea continued. On day 7, the dog regurgitated a large quantity of coffee-ground texture vomitus. A CBC, serum biochemical analyses, ultrasonography, and endoscopic examination were performed on that day. Abnormalities included low RBC count (4.23 × 10^6/µL; reference range, 5.5 to 8.5 × 10^6/µL), low PCV (30%; reference range, 37 to 55%), low hemoglobin concentration (9.5 g/dL; reference range, 12 to 18 g/dL), and mature neutrophilia (35.3 × 10^9 neutrophils/L). Ultrasonography revealed a mass between the stomach and spleen and around the small intestine. The gastric wall seemed to be irregularly thickened, and there was little evidence of gastric motility. Endoscopy revealed blood clots at the fundus of the stomach and many petechial hemorrhages on the gastric mucosa. A stomach ulcer that was approximately 1.5 cm in diameter was also observed at the lesser curvature; the gastric ulcer was not perforated.
Surgery was performed to determine a definitive diagnosis and excise the gastric ulcer. During laparotomy, the omentum was seen to be slightly yellow but not icteric, irregularly thickened, and fragile; the omentum covered the surface of the abdominal organs (Fig 2), but there was no evidence of hydroperitoneum. A mass that involved the gastrosplenic ligament and the splenic artery was adhered to the greater curvature of the stomach. A large mesenteric mass, formed around the root of the mesentery, was connected to the mass that involved the gastrosplenic ligament (Fig 3). Portions of the small intestine were adhered to this mesenteric mass, and a decline in intestinal motility was suspected. There were no abnormalities of other organs in the abdominal cavity including the pancreas, liver, kidney, spleen, and bladder. We excised the gastric ulcer and omentum and, since we could not remove the entire mesenteric mass, biopsied the mesenteric mass. Results of aerobic bacteriologic culture of a sample from the omentum were negative. No fungal culture was performed.

Histologic examination of the biopsy specimen from the mesenteric mass revealed granulomatous inflammation with a central necrotic area and extensive fibrosis in adipose tissue. The inflammatory cells were predominantly foamy macrophages, and lymphocytes and plasma cells had also infiltrated the lesion (Fig 4). There were no mycotic organisms or acid-fast bacteria in the tissues, and no vasculitis was found. Histologic examination of the omentum revealed granulomatous inflammation similar to the mesenteric mass. There was no evidence of mycotic organisms or acid-fast bacteria in the tissues. The gastric tissues adjacent to the ulcer were heavily infiltrated with inflammatory cells, predominantly neutrophils, and areas of fibrosis and necrosis were also present. These changes appeared to extend into the mesenteric attachment to the greater curvature of the stomach.

We diagnosed steatitis of the adipose tissue of the mesentery and omentum, and also recognized that the lesions closely resembled those of a human disease called mesenteric panniculitis. One week after surgery, ultrasonographic examination revealed increased gastrointestinal motility, but the mass around the intestine remained. Treatment was begun by use of the treatment protocols for human mesenteric panniculitis and canine subcutaneous nodular panniculitis. Prednisolone was administered for 2 days (4 mg/kg [1.8 mg/lb], SC, q 24 h) and the dosage was then reduced (2 mg/kg [0.9 mg/lb], SC, q 24 h) and continued for 7 days. For approximately 1 month thereafter, we continued medication with prednisolone (1 mg/kg [0.45 mg/lb], PO, q 24 h). Although the dog subsequently remained in good condition, to avoid the adverse effects of prednisolone we reduced the frequency of administration (1 mg/kg, PO, q 3 d) in combination with cyclosporin (5 mg/kg [2.3 mg/lb], PO, 5 d/wk). After 1.5 years, the mass in the abdominal cavity was indiscernible via radiography or ultrasonography, and the dog was in good health. Maintenance therapy with prednisolone and cyclosporin was continued for 2.5 years. The dog then suddenly died because of a perforation of the ileum caused by ingestion of a foreign body (a toy). Necropsy revealed that parts of
Mesentery were atrophied or cicated, and parts of the intestine were affected by adhesions or stenosis.

Steatitis, an inflammatory condition involving adipose tissue, occasionally develops in the abdominal adipose tissue of humans. The mesentery is more frequently affected than other abdominal adipose tissues, but the process can also involve the omentum, retroperitoneal structures, and the vena cava. In humans, this condition is called mesenteric panniculitis when the mesentery is mainly affected. Typical appearance includes thickening of the mesentery or development of l or more hard, rubbery, nodular masses. Although the disease is considered benign, the prognosis may be influenced by the stage or recurrence of the disease. The etiology of mesenteric panniculitis remains unknown, but predisposing factors such as infection, recent surgery, prior abdominal trauma, ulcerative disease, autoimmune disease, drugs, and retained suture material have been implicated. Subcutaneous nodular panniculitis is often diagnosed in dogs. This is a skin disorder in which subcutaneous adipose tissue is primarily affected. Predisposing factors may include physical (mechanical, thermal, or chemical) damage within the subcutaneous fat, immune-mediated drug reaction, infection, autoimmune disease, pancreatic diseases, and postvaccinal inflammation. In the dog reported here, none of the possible etiologies of inflammation of abdominal adipose tissue could be ruled out entirely except surgery and retained suture material. Thus, it was impossible to determine the cause of disease in this dog. Bacterial infection was probably not a factor based on the negative results of bacteriologic cultures and the histopathologic findings. The dog had no history of recent abdominal damage or medication with any drugs. In addition, serum amylase activity was slightly high on day 1, but serum lipase activity was not increased and activities of these enzymes were not increased subsequently, suggesting that pancreatitis was not present. In addition, at laparotomy, the pancreas was normal.

In this dog, inflammation of the abdominal adipose tissue was suspected from the results of radiographic examination and ultrasonography. Steatitis was definitively diagnosed by use of biopsy and histologic examination. Interpretation of clinical signs of this disease in dogs is difficult, so recognition may be delayed and dogs may have end-stage steatitis by the time a diagnosis is made. For this reason, surgical treatment may be needed before any medications are administered.

In the dog reported here, the mesenteric and omental masses appeared to suppress gastric motility. When gastric motility is reduced, stomach contents accumulate and constantly stimulate the gastric mucosa, and the risk of gastric ulceration increases. The immediate cause of gastric ulceration in this dog was not clear, but decreased motility may have been a factor.

Although the intestine was in close approximation to the mesenteric mass with no evidence of adherence to any other organs at the time of surgery, necropsy 2.5 years later revealed severe adhesions between sections of intestine and cication of the mesentery. These changes suggest that even if a treated animal’s general condition appears to be good and no intra-abdominal masses are found by ultrasonography, inflammation of the adipose tissue can progress to necrosis, fibrosis, and cicatrization. These changes may lead to adhesion, contraction, and impaired functioning of the intestine. Therefore, we believe that a barium study must be performed to examine the route of the intestine during the course of treatment. If adhesion, contraction, or impaired functioning of the intestine is suspected, surgery should be performed before clinical signs appear.

The prevalence and etiology of steatitis in dogs are not known, but the disease clearly involves sterile inflammation of adipose tissue in the abdominal cavity that results in adhesions, hypomotility of abdominal organs, or both. This disease should be considered in the diagnosis of abdominal masses.

References