Clinical, Radiographic and Pathological Features of Persistent Gastroesophageal Intussusception in an Adult Dog: A Case Report

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Abstract

The aim of this report is to describe the clinical, radiographic and pathological appearances of a persistent gastroesophageal intussusception in association with idiopathic megaesophagus in an adult dog. A 6-years old, male Golden Retriever was referred for evaluation of chronic regurgitation and vomiting. Radiographic diagnosis of persistent gastroesophageal intussusception (GEI) with megaesophagus was made. Survey and contrast radiography evidenced a distension of the esophageal medial and caudal portions due to a dense soft-tissue mass and the barium cloud not pass to abdominal portion, where outlined the striations of muscle of the distal esophagus around the mass. Necropsy findings revealed a completed gastroesophageal intussusception with megaesophagus. Strangulation and incarceration of the whole stomach and intussusceptum resulted in detrimental effects on the cardiopulmonary system with vascular compromise of the intussuscepted organs. Histopathological results revealed tissue injury, circulatory disturbance and inflammatory reaction at dilated esophagus, affected lung and intussuscepted organs.

Keywords: diagnosis, dog, gastroesophagus, intussusception

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Introduction

Gastroesophageal intussusception (GEI) is a rare disease characterized by the retrograde invagination of the stomach into the esophagus without displacement of the gastroesophageal junction. The other abdominal structures including spleen, pancreas, proximal duodenum, omentum or any combination can be occasionally involved in the intussusceptum. Clinical signs associated with GEI are usually characterized by acute regurgitation or vomiting, dyspnea, hematemesis and abdominal discomfort or pain. GEI usually affects young dogs (less than 1 year old) with particular high prevalence in the German Shepherd and male gender (Cornell and Selcer, 2002). The underlying etiology of GEI is not well understood. The preexisting esophageal diseases including megaesophagus, abnormal esophageal motility and abnormal anatomical abnormalities of the esophageal hiatus, may be the risk factors of GEI (Graham et al., 1998; Gowen et al., 1999). The purpose of this study was to report a rare persistent case of GEI in an adult dog and demonstrate the relation between clinical signs, radiographic analysis and the pathological findings. The possible cause of GEI in this case was also discussed.

Case history

Physical examination: An 18-kg, 6-years old, male Golden Retriever was referred to Small Animal Hospital, Chulalongkorn University due to chronic onset of regurgitation and vomiting for three years. The frequency of signs, initially rare, had increased over a period of years. Vomitus contained indigested foods, mucous exudates and sometimes blood content. The other clinical signs were anorexia, weight loss, depression, and lateral recumbency. On physical examination, the dog had poor body condition score and severe dehydration. Normal lung sounds and capillary refill time with strong femoral pulse were observed. The remainder of the physical examination was unremarkable. Clinical laboratory data before death including a complete blood cell count and a serum biochemical panel showed leukocytosis (35,800 cells/µl, normal: 6,000-17,000 cells/µl) characterized by neutrophilia with the left shift (segmented neutrophil 32,986 cells/µl, normal: 3,000-11,400 cells/µl; band neutrophil 2,148 cells/µl, normal: 0-300 cells/µl) and slight increase of serum alkaline phosphatase (106 IU/l, normal: 10.1-106 IU/l). These conditions might be consistent with an inflammatory condition or a stress response. Slight elevations in packed-cell volume (57%, normal: 37-55%) were
consistent with dehydration. Serum electrolyte abnormalities included slight hypokalemia (3.42 mmol/l, normal: 3.8-5.6 mmol/l) and metabolic alkalosis (pH 7.451, normal: 7.35-7.45; pCO₂ 36.3 mmHg, normal: 35-45 mmHg; HCO₃ 24.7 mmol/l, normal: 24.5 mmol/l), probable consequences of the protracted vomiting.

Figure 1. Left-right lateral survey thoracic radiograph. Well marginated, oval soft tissue opacity (arrowheads) is seen in the caudodorsal part of the thorax causing ventral displacement of the heart and trachea.

Radiographic examination: Survey and contrast radiography of the period of three years earlier showed abnormalities of the esophagus. The proximal part of the esophagus was dilated with gas. A tentative radiographic diagnosis was megaesophagus. Three years later, thoracic film of the last time at presentation revealed a distension of the esophageal caudal portion due to a soft-tissue dense mass. The opacity mass located from mid to caudal aspects of the thoracic cavity. The left diaphragmatic lung lobes were compressed by the caudal portion of this mass. The cranial portion of the mass had distinct rounded margins that were contiguous with gas in a dilated esophagus. The trachea and heart were remarkably displaced to a ventral direction (Figure 1). In the abdominal cavity, the stomach was not found. Differential diagnosis of these changes included esophageal hiatal hernia, foreign body, tumor masses in the mediastinal or pulmonary parenchyma and gastroesophageal intussusception. Contrast radiography evidenced the distension of the medial and caudal parts of the esophagus using amount of barium in the thoracic caudal portion. Barium could not pass to abdominal portion, where outlined the striations of muscle of the distal esophagus around the mass. These contrast radiography confirmed the diagnosis of a gastroesophageal intussusception (Figure 2). The corrective surgery for GEI was decided to perform. However, the dog died from shock during the anesthesia. Necropsy was done and all the affected organs were collected in 10% buffered formalin for routine pathological evaluations.

Macrosopic findings: At necropsy, the dog was cachexia and exhibited minimal fat stores. The mucous membranes and subcutis was severe pale. All parts of esophagus were dilated (10 cm in diameter) with a completed gastroesophageal intussusception at the caudal portion of esophagus (Figure 3). The entire stomach was found into the distal esophagus; some proximal parts of the duodenum and pancreas were involved (Figure 4). The stomach could be reduced into its normal position with difficulty owing to the tight stricture of distal esophageal lumen, where was completely occluded by the intussuscepted stomach. The longitudinal sections of intussuscepted gastric segment revealed erythematous, eroded gastric mucosa and submucosal edema (Figure 5). Trachea filled with mucous content and barium. The lungs had patchy, red to purple atelectasis affecting 60% of the total lung volume. The whole heart was mildly dilated and myocardial hypertrophy. The pancreas was white and moderately hard consistency and the texture was white in color. The adipose tissue around the pancreas and duodenum contained multiple chalky white appearances. Spleen was shrinkage and pale in color consistent to hypovolumic shock. Liver was moderate congested with bile precipitation. The small intestines were enlarged and filled with yellowish catarrhal mucous content. The mucosal surface of intestines was slightly thickened. Both kidneys were severe congested with multifocal mild hemorrhages distributed from the cortico-medullary junction to the medulla.
Grossly, marked dilation of esophagus and GEI is observed with lung atelectasis and emphysema. E: esophagus; H: heart; L: lungs; IS: intussuscepted stomach.

There is a completed gastroesophageal intussusception at the caudal portion of esophagus. E: esophagus; IS: intussuscepted stomach.

Cross section of intussuscepted gastric segment, gastric mucosa and serosa reveal erythema and erosion with severe submucosal hemorrhage (arrows).

Microscopic examination revealed lymphohistiocytic cell infiltration in mucosal and submucosal areas throughout the esophagus with esophageal gland hyperplasia. Hemorrhage with thrombosis in submucosa and moderately Zenker degeneration of esophageal muscles was also found. Severe mucosal and submucosal hemorrhage with thrombosis was observed in the stomach (Figure 6). Pancreas revealed severe chronic pancreatitis with fat necrosis. Heart showed Zenker degeneration of myocardium with hypertrophic vasculopathy. Chondroid metaplasia of mitral valve with focal necrosis was also observed. Severe multifocal hemorrhages were found at the renal cortico-medullary junction with moderate tubular degeneration. Lungs showed severe atelectasis with emphysema. Chronic lymphadenitis characterized by infiltration of mononuclear cells, mainly macrophages and plasma cells was observed in affected lymph nodes.

Microscopically, severe hemorrhage with thrombosis is visible in submucosa of the incarcerated stomach. Note thrombosis in blood vessels (arrows). HE staining, Bar: 700 μm.

The diagnosis of this case was persistent gastroesophageal intussusception in association with acquired megaesophagus.

Discussion

There are two forms of gastroesophageal intussusception (GEI) that have been reported (Werthern et al., 1996; Cornell and Selcer, 2002). A chronically recurrent form of GEI causes intermittent gastrointestinal signs including chronic regurgitation or vomiting. The prognosis of this recurrent type is good when the corrected surgery for intussusception is performed. The other type of GEI is persistent condition that may be acute in onset, resulting in acute esophageal obstruction and severe respiratory distress. The stomach and other abdominal organs herniate and become entrapped within the caudal thoracic lumen in which the compression of caudal parts of lung can produce respiratory distress. The prognosis for this persistent form is poor. This form of GEI has been rarely reported in the dog. The GEI in this case was early suspected in the clinical assessment and recognized after thoracic survey and contrast radiographic examination. However, the dog died before surgical treatment due to the severity of the clinical signs. According to necropsy perform; pathological findings could indicate a persistent form of gastroesophageal intussusception. Herniation of whole stomach with some parts of proximal duodenum and pancreas into the caudal esophagus.
compressed and impaired the expansion of caudal parts of lung lobes resulting in respiratory distress due to lung atelectasis and emphysema. Strangulation and incarceration of stomach and intussusception resulted in rapid deterioration of the dog’s condition, hypovolemic shock and subsequent death. Local circulatory obstruction characterized by severe submucosal hemorrhage and thrombosis was observed in incarcerated stomach and caudal esophagus. The strangulation of the affected organs lead to stasis of blood flow and induced endothelial injury of the vessels resulting in hemorrhage and thrombosis. Some parts of incarcerated pancreas showed pancreatic damage and injury.

In the present case, megaesophagus had been found and diagnosed before the occurrence of GEI. It may be suggested that the prior presence of a megaesophagus may predisposed this dog to the intussusception. Chronic regurgitation and vomiting signs of megaesophagus may play an important role to initiate the active and retrograde esophageal peristalsis leading to the invagination of the stomach into the esophagus. Megaesophagus or esophageal achalasia is a condition in which the esophagus is dilated and peristalsis fails to occur properly. Animals with this condition may have signs of regurgitation, vomiting, malnutrition and often in association with aspiration pneumonia. Megaesophagus interferes with effective peristalsis propulsion of the food bolus passing down into the stomach. Retention of some ingesta in esophagus may lead to esophagitis and putrefaction in dependent parts (Jones et al., 1997). In this case, diffuse chronic esophagitis was observed throughout the mucosal and submucosal areas. Megaesophagus can be congenital or acquired. Congenital idiopathic megaesophagus is an inherited disorder that relatively common in dogs (Jones et al., 1997; Martinez et al., 2001; Pietra et al., 2003). Acquired megaesophagus occurs in adult dogs secondary to any disorder that disturbs neural system of swallowing reflex or esophageal muscle function. Various disorders associated with the development of acquired megaesophagus include the dysfunction due to foreign bodies, tumors or strictures of esophagus, neurologic disorders such as canine distemper, autoimmune diseases such as myasthenia gravis, and intoxication such as lead poisoning and tetanus (Brown et al., 2007). The megaesophagus in this case might be acquired condition because age of the dog, the duration of sign and clinical history. Unfortunately, we could not clarify the underlying causes of megaesophagus in this case.

Gastroesophageal intussusception (GEI) is a life-threatening condition that requires an accurate, early recognition and prompt diagnosis with urgent surgical approach. Rapid deterioration of the animal’s condition can progress to hypovolemic shock and definitely death because of strangulation and obstruction of the intussusception with vascular compromise of the invaginated organs (Jergens, 1997). Survey or contrast radiography, esophagram and endoscopy should be done to confirm the intussusception. Differential diagnosis of GEI includes a sliding or paraesophageal hiatal hernia, foreign body and masses in mediastinal area or lung (Van Camp et al., 1998; Van Geffen et al., 2006; McGill et al., 2009).

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References