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Scientific Report

The possible relationship of megaesophagus and *canine distemper* in two German shepherd dogs

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Summary

Megaesophagus (esophageal dilatation) is a neuromuscular disease. In this disorder, the esophagus is abnormally stretched and air is collected with food in it. Two male dogs aged 4 and 6-year-old, German shepherd breeds and weighing 18.4 and 23.2 kg were presented to the Veterinary Hospital of Shahid Chamran University with complaints of regurgitation, dehydration, anorexia, depression, dysphagia, myoclonus, hypersalivation, gurgling sound during swallowing and fatigue in the past month. They were not vaccinated against distemper and other viral diseases. Physical examination revealed a relatively thin body condition. A plain radiograph of the chest revealed advanced megaesophagus with aspiration pneumonia. Rapid antigenic test (immunochromatography assay) was positive for *canine distemper*. CBC was abnormal (leucopenia, lymphopenia and neutropenia). Supportive treatments were administered for them. We advised the dogs be placed in a vertical position for 15 min after the meal. Only one dog was sent back home in good health four weeks later and the disease did not relapse for more than six months. The second dog had a symptom of megaesophagus and did not respond to supportive treatments completely. A possible relationship between megaesophagus and *canine distemper* infection has been reported in the present survey.

Key words: Megaesophagus, *Canine distemper*, Dog, Ahvaz

Introduction

Megaesophagus is a disorder characterized by diffuse esophageal dilatation. This syndrome may occur as a congenital idiopathic disorder or it may manifest in adult animals as an idiopathic or acquired lesion. The underlying pathophysiologic mechanism for acquired idiopathic megaesophagus is unknown. Acquired secondary megaesophagus may result from many disorders, especially diseases causing diffuse neuromuscular dysfunction such as hypoadrenocorticism, lead poisioning, chushing syndrome, hypothyroidism, tetanus, glycogen storage diseases, systemic lupus erythmatosis (SLE), myasthenia gravis, dysautonomia, dermatomyositis and *canine distemper*. Other causes of segmental or diffuse esophageal hypomotility include foreign bodies, stricture, vascular ring anomalies and esophagitis (Jergens, 2005). Megaesophagus is the most common cause for regurgitation in the dog. Considerable variability exists in the frequency and timing of regurgitation episodes after meal ingestion. Weight loss and emaciation occur secondary to malnutrition in animals having long-standing disease. Respiratory distress and fever indicate aspiration pneumonia. In this condition, they are unable to move food actively toward the stomach and are unable to empty themselves of the ingesta (ingested food and liquid). In order to treat, or at least manage the megaesophagus adequately, it is very important to determine the underlying cause. Promotility drugs are currently of unproven benefit in the management of idiopathic megaesophagus in dogs. Both metoclopramide and cisapride are smooth muscle prokinetic agents that have no effect

on the striated muscle of the esophageal body (Boria *et al*., 2003; Jergens, 2005). Rapid diagnosis of viral infections is especially important in order to isolate infected dogs and prevent secondary infections of susceptible animals. The immunochromatography assay is the most rapid field diagnostic method used in clinical practice, because the test procedure is simple and can be performed by veterinarians (Esfandiari and Klingeborn, 2000). The present report describes two cases of megaesophagus in dogs infected with *canine distemper* and a possible relationship between them.

Materials and Methods

Two dogs, aged 4- and 6-year-old, male, German shepherd breeds and weighing 18.4 and 23.2 kg were referred to the Veterinary Hospital of Shahid Chamran University with complaints of regurgitation, dehydration, anorexia, depression, dysphagia, myoclonus, hypersalivation, gurgling sound with swallowing and fatigue for the past month. They had not been vaccinated against *canine distemper* and other viral diseases. Ketamine (15 mg/kg) and acepromazine (0.15 mg/kg) were injected for sedative effects. With a history of dysphagia and pytalism, radiographs of the oral cavity, pharynx, neck, thorax and abdomen were obtained to evaluate foreign bodies, masses, abnormal anatomic structures and dental or temporomandibular joint diseases.

Rapid antigenic test (immuno-chromatography assay) was carried out for identification of *canine distemper*. The kit was a commercial rapid CDV Ag test kit (Manufactured by Anigen, Animal genetics, Inc., Korea, 2008), and the manufacturer's instructions were followed. The samples were provided by a swab from the conjunctiva, then they were inputted and mixed into the assay diluents. The presence of two color bands (T and C) within the result window indicated a positive result (Esfandiari and Klingeborn, 2000).

Blood samples (2 ml) were collected from the cephalic vein of the dogs to determine complete blood count (CBC). An acid-base analysis, electrolyte panel, blood urea nitrogen (BUN), glucose estimations and an electrocardiogram were measured in the affected dogs. The esophagus was evaluated by endoscopy also. Echocardiography was not performed, because the electrocardiogram was normal and there was no radiographic evidence of cardiomegaly. Supportive treatments (particularly broad-spectrum antibiotics) were administered for them. We advised that the studied dogs be placed in a vertical position for 15 min after the meal. Nutritional management included small frequent feedings of a canned food diet.

Results

A plain radiograph of the chest revealed advanced megaesophagus with aspiration pneumonia in the affected dogs. Endoscopic studies revealed severe dilatation of the esophagus. No inflammatory response or esophagitis was seen in the esophagus wall. Rapid antigenic test (immuno-chromatography assay) was positive for *canine distemper*. Complete blood count indicated leucopenia, lymphopenia and neutropenia. Only one dog was sent back home in good health four weeks later and the disease did not relapse for more than six months. The second dog had a symptom of megaesophagus and did not respond to supportive treatments completely. The initial diagnostic tests, consisting of an acid-base analysis, an electrolyte panel, blood urea nitrogen, glucose estimations and an electrocardiogram were normal. No other abnormalities were noted. The megaesophagus and alveolar pattern (aspiration pneumonia) are seen in Fig. 1. Figure 2 shows the thorax in the dog that showed an improvement in megaesophagus.

Discussion

Megaesophagus was confirmed by a plain radiograph of the chest in the affected dogs. A possible relationship was seen between megaesophagus and *canine distemper* in our cases. We suspected the megaesophagus disorder based on clinical signs, particularly regurgitation. Radiographs of the cervical neck and thorax

revealed the presence of air with food in the esophagus. Pulmonary alveolar opacities were seen, so it was indicative of aspiration pneumonia. Routine hematology, serum biochemistry and urinalysis were performed to screen for acquired causes of megaesophagus. In our study, endoscopy was used for the differential diagnosis of other diseases such as foreign bodies and esophagitis. Animals that have advanced aspiration pneumonia should have a gastrostomy tube placed for enteral nutritional support. Dogs with acquired megaesophagus should be evaluated for esophagitis, peripheral neuropathies, laryngeal paralysis and chronic or recurrent gastric dilatation with or without volvulus (Jergens, 2005).

In the present survey, broad-spectrum antibiotics were administered for the treatment of aspiration pneumonia. The vertical position is the best feeding position for megaesophagus. *Canine distemper* virus may involve a defect in vagal afferent innervation of the esophagus. It is characterized as selective vagal afferent dysfunction in dogs with idiopathic megaoesophagus. Similar processes may be active in disorders of visceral organ systems subserved by vagal afferents (Holland *et al*., 2002).

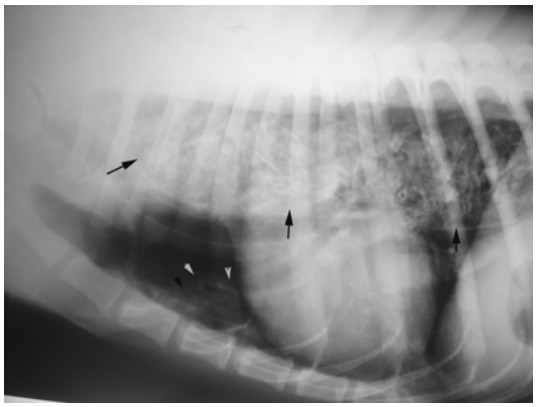

Fig. 1: Left lateral radiograph of thorax of the affected dog with megaesophagus, black arrows: ventral border of esophagus and white arrow heads: alveolar pattern (aspiration pneumonia) dorsal to the third and forth sternebrea

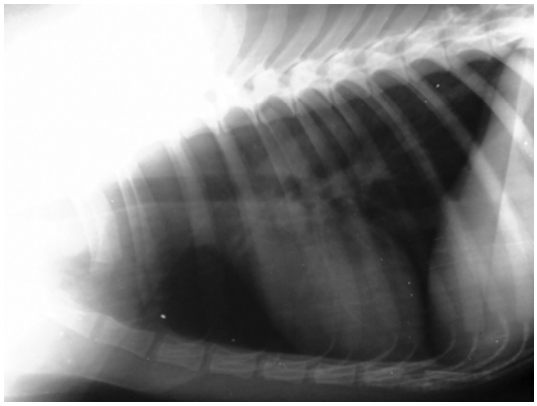

Fig. 2: Right lateral radiograph of thorax in the improved dog of megaesophagus after six months

The affected dogs were re-evaluated for 6 months in order to monitor the disease progression. Thoracic radiographs were repeated to assess esophageal dilatation and aspiration pneumonia. The affected dogs were middle-age and German shepherd breed. Familial predisposition has been reported in this breed (Jergens, 2005).

Some animals succumb to repeated episodes of aspiration pneumonia or are euthanized because of their irreversible disease. In our study, megaesophagus was resolved in only one dog, after treatment of concurrent *canine distemper*. The second dog had a symptom of megaesophagus and did not respond to supportive treatments completely. Kamrani *et al*. (2002) made the first report of feline megaesophagus in Iran. In their report, a kitten with a history of depression, labored breathing and poor appetite was referred to the Veterinary Hospital of Tehran University. Plain and contrast radiography revealed advanced megaesophagus. After two months, the kitten showed remarkable improvement, clinically and radiography.

Acquired secondary megaesophagus may result from many disorders, especially diseases causing diffuse neuromuscular dysfunction such as concurrent presence with hyperadrenocorticism (Burgener *et al*., 2007), hypoadrenocorticism (Melian *et al*., 1999), esophageal dysmotility (Bexfield *et al*., 2006), paraoesophageal hiatal hernia (Kirkby *et al*., 2005), delayed transit disorders or hypomotility (Washabau, 2003), esophageal achalasia and failure of the lower esophageal sphincter to open (Boria *et al*., 2003), hypertrophic osteopathy (Watrous and Blumenfeld, 2002), double aortic arch and concomitantly dilatation of the cranial oesophagus (Ferrigno *et al*., 2001), tiger

snake envenomation in four cases (Hopper *et al*., 2001), hypothyroidism (Huber *et al*., 2001), dysautonomia (Detweiler *et al*., 2001) and acquired myasthenia gravis (Gaynor *et al*., 1997).

Our study showed the concurrent presence of *canine distemper* infection and secondary megaesophagus in the affected dogs. There is a possible relationship between them. Affected animals should be re-evaluated at 1 to 2-month intervals to monitor disease progression. Some animals with idiopathic megaesophagus may improve over time (months) with diligent supportive care. The prognosis with acquired idiopathic megaesophagus is generally poor.

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References

Bexfield, NH; Watson, PJ and Herrtage, ME (2006). Esophageal dysmotility in young dogs. J. Vet. Intern. Med., 20: 1314-1318.

Boria, PA; Webster, CR and Berg, J (2003). Esophageal achalasia and secondary megaesophagus in a dog. Can. Vet. J., 44: 232-234.

Burgener, IA; Gerold, A; Tomek, A and Konar, M (2007). Empty sella syndrome, hyperadrenocorticism and megaoesophagus in a dachshund. J. Small Anim. Pract., 48: 584-587.

Detweiler, DA; Biller, DS; Hoskinson, JJ and Harkin, KR (2001). Radiographic findings of *canine dysautonomia* in twenty-four dogs. Vet. Radiol. Ultrasound., 42: 108-112.

Esfandiari, J and Klingeborn, B (2000). A comparative study of a new rapid and one-step test for the detection of parvovirus in faeces from dogs, cats and mink. J. Vet. Med. B Infect. Dis. Vet. Public Health. 47: 145-153.

Ferrigno, CR; Ribeiro, AA; Rahal, SC; Orsi, AM; Fioreto, ET; Castro, MF; Mchado, MR and Singaretti, F (2001). Double aortic arch in a dog (*Canis familiaris*): a case report. Anat. Histol. Embryol., 30: 379-381.

Gaynor, AR; Shofer, FS and Washabau, RJ (1997). Risk factors for acquired megaesophagus in dogs. J. Am. Vet. Med. Assoc., 211: 1406-1412.

Holland, CT; Satchell, PM and Farrow, BR (2002). Selective vagal afferent dysfunction in dogs with congenital idiopathic megaesophagus. Auton. Neurosci., 99: 18-23.

Hopper, K; Beck, C and Slocombe, RF (2001). Megaoesophagus in adult dogs secondary to Australian tiger snake envenomation. Aust. Vet. J., 79: 672-675.

Huber, E; Armbrust, W; Forster, JL; Ribiere, T and Grosclaude, P (2001). Resolution of megaesophagus after treatment of concurrent hypothyroidism in a dog. Schweiz. Arch. Tierheilkd., 143: 512-514.

Jergens, AE (2005). Diseases of the esophagus. In: Ettinger, SJ and Feldman, EC (Eds.), *Ettinger's textbook of veterinary internal medicine*. (6th Edn.), Vol. 2, Philadelphia, PA, Elsevier Saunders. PP: 1298-1310.

Kamrani, AR; Masoudifard, M and Vajhi, AR (2002). A case report of feline megaesophagus in Iran. J. Fac. of Vet. Med., University of Tehran. 57: 49-50.

Kirkby, KA; Bright, RM and Owen, HD (2005). Paraoesophageal hiatal hernia and megaoesophagus in a three-week-old Alaskan malamute. J. Small Anim. Pract., 46: 402-405.

Melian, C; Stefanacci, J; Peterson, ME and Kintzer, PP (1999). Radiographic findings in dogs with naturally-occurring primary hypoadrenocorticism. J. Am. Anim. Hosp. Assoc., 35: 208-212.

Washabau, RJ (2003). Gastrointestinal motility disorders and gastrointestinal prokinetic therapy. Vet. Clin. North Am. Small Anim. Pract., 33: 1007-1028.

Watrous, BJ and Blumenfeld, B (2002). Congenital megaesophagus with hypertrophic osteopathy in a 6-year-old dog. Vet. Radiol. Ultrasound., 43: 545-549.