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Current State of Knowledge

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CANINE IDIOPATHIC MEGAESOPHAGUS

Etiology - Most cases of adult-onset megaesophagus have no known etiology and are referred to as acquired idiopathic megaesophagus. The syndrome occurs spontaneously in adult dogs between 7 to 15 years of age without sex or breed predilection. The disorder has been compared erroneously to esophageal achalasia in humans. Achalasia is a failure of relaxation of the lower esophageal sphincter and ineffective peristalsis of the esophageal body. A similar disorder has never been rigorously documented in the dog. Several important differences between idiopathic megaesophagus in the dog and achalasia in humans have been documented. Although the etiology(ies) has not been identified, some studies have suggested a defect in the afferent neural response to esophageal distension similar to what has been reported in congenital megaesophagus.

Clinical Examination

Routine hematology, serum biochemistry, and urinalysis should be performed in all cases to investigate possible secondary causes of megaesophagus (e.g. hypoadrenocorticism). Survey radiographs will be diagnostic for most cases of megaesophagus. Contrast radiographs may be necessary in some cases to confirm the diagnosis, evaluate motility, and exclude foreign bodies or obstruction as the cause of the megaesophagus. Endoscopy will confirm the diagnosis and may further reveal esophagitis, a frequent finding in canine idiopathic megaesophagus.

If acquired secondary megaesophagus is suspected, additional diagnostic tests should be considered, for example: serology for nicotinic acetylcholine receptor antibody, ACTH stimulation, serology for antinuclear antibody, serum creatine phosphokinase activity, electromyography and nerve conduction velocity, and muscle and nerve biopsy. Additional medical investigation will be dependent upon the individual case presentation.

Hypothyroidism has been cited as an important cause of idiopathic megaesophagus in the dog, although risk factor analysis has not revealed a clear association. Thyroid function testing (e.g., TSH assay, TSH stimulation, free and total thyroid hormones) should be performed in individual suspicious cases.

Treatment

Animals with secondary acquired megaesophagus should be appropriately differentiated from other esophageal disorders and treated. Dogs affected with myasthenia gravis should be treated with pyridostigmine (1.0-3.0 mg/kg PO BID) and/or corticosteroids (prednisone 1.0-2.0 mg/kg PO or SQ BID), dogs affected with hypothyroidism should be treated with levothyroxine (22 mg/kg PO BID), and dogs affected with polymyositis should be treated with prednisone (1.0-2.0 mg/kg PO BID). If secondary disease can be excluded, therapy for the congenital or acquired idiopathic megaesophagus patient should be directed at nutritional management and treatment of aspiration pneumonia. Affected animals should be fed a high-calorie diet, in small frequent feedings, from an elevated or upright position to take advantage of gravity drainage through a non-peristaltic esophagus.

Dietary consistency should be formulated to produce the fewest clinical signs. Some animals handle liquid diets quite well, while others do better with solid meals. Animals that cannot maintain adequate nutritional balance with oral intake should be fed by temporary or permanent tube gastrostomy. Gastrostomy tubes can be placed surgically or percutaneously with endoscopic guidance.

Smooth muscle prokinetic (e.g., metoclopramide or cisapride) therapy has been advocated for stimulating esophageal peristalsis in affected animals, however metoclopramide and cisapride will not likely have much of an effect on the striated muscle of the canine esophageal body. Bethanechol has been shown to stimulate esophageal propagating contractions in some affected dogs and is therefore a more appropriate prokinetic agent for the therapy of this disorder. Because of the high incidence of esophagitis in canine idiopathic megaesophagus, affected animals should also be medicated with oral sucralfate suspensions (1 g q8h for large dogs 0.5 g q 8h for smaller dogs 0.25 to 0.5 g q8h to q12h for cats).

GASTRIC EMPTYING DISORDERS

Gastric emptying disorders are fairly common in dogs and cats. They result from disease processes that alter normal
gastric functions, i.e. storage of ingesta, mixing and dispersion of food particles, and timely emptying of gastric contents into the small intestine. Disorders of gastric emptying arise from mechanical obstruction, or from defective propulsion. Anatomic lesions (e.g. malignancy, hyperplasia, foreign bodies) cause delayed gastric emptying because of mechanical obstruction.

Diagnosis and management of mechanical obstruction is usually straight-forward. Disorders of defective propulsion, on the other hand, cause delayed gastric emptying because of abnormalities in myenteric neuronal or gastric smooth muscle function, or because of abnormalities in antropyloroduodenal coordination. A number of primary conditions have been associated with these functional disorders, including infectious or inflammatory disease, ulcer, and post-surgical gastroparesis. Delayed gastric emptying has also been associated with a number of secondary conditions, including electrolyte disturbances, metabolic disorders, concurrent drug usage (cholinergic antagonists, adrenergic agonists, opioid agonists), acute stress, and acute abdominal inflammation. Recovery from gastric dilation/volvulus is almost always associated with significant myoelectrical and motor abnormalities in the dog. Diagnosis and management of the delayed gastric emptying disorders may not be so straightforward. Nutritional and medical management, including smooth muscle prokinetic agents (e.g., cisapride, erythromycin, and ranitidine), are important components of therapy.

**SMALL INTESTINAL TRANSIT DISORDERS**

A number of small intestinal transit disorders have been described in dogs and cats, including enteritis, post-surgical pseudo-obstruction, nematode infection, intestinal sclerosis, and radiation enteritis. Vomiting and diarrhea are the most important clinical signs associated with these disorders. Overgrowth of small intestinal bacteria, a common sequela to disordered motility, contributes to these clinical signs. Transit disorders associated with mechanical obstruction should always be differentiated and treated appropriately. Delayed transit associated with functional disorders should be managed with dietary modification (low fat diets) and prokinetic agents (cisapride, tegaserod, or metoclopramide). Tegaserod, a new 5-HT3 partial agonist, has recently been reported to normalize intestinal transit in opioid-induced bowel dysfunction in dogs.

**COLONIC MOTILITY DISORDERS**

**History**

Constipation, obstipation, and megacolon may be observed in cats of any age, sex, or breed, however, most cases are observed in middle aged (mean = 5.8 years), male cats (70% male, 30% female) of Domestic Shorthair (46%), Domestic Longhair (15%), or Siamese (12%) breeding.

**Physical Examination**

Colonic impaction is a consistent physical examination finding in affected cats. Other findings will depend upon the severity and pathogenesis of constipation. Dehydration, weight loss, debilitation, abdominal pain, and mild to moderate mesenteric lymphadenopathy may be observed in cats with severe idiopathic megacolon. Colonic impaction may be so severe in such cases as to render it difficult to differentiate impaction from colonic, mesenteric, or other abdominal neoplasia.

Cats with constipation due to dysautonomia may have other signs of autonomic nervous system failure, such as urinary and fecal incontinence, regurgitation due to megaesophagus, mydriasis, decreased lacrimation, prolapse of the nictitating membrane, and bradycardia. Digital rectal examination should be carefully performed with sedation or anesthesia especially in those cats with recurring bouts of constipation.

Pelvic fracture malunion may be detected on rectal examination in cats with pelvic trauma. Rectal examination might also identify other unusual causes of constipation, such as foreign bodies, rectal diverticula, stricture, inflammation, or neoplasia.

**Differential Diagnoses**

Several authors have emphasized the importance of considering an extensive list of differential diagnoses (e.g., neuromuscular, mechanical, inflammatory, metabolic/endocrine, pharmacologic, environmental, and behavioral causes) for the obstipated cat.

A review of published cases, however, suggests that 96% of cases of obstipation are accounted for by idiopathic megacolon (62%), pelvic canal stenosis (23%), nerve injury (6%), or Manx sacral spinal cord deformity (5%). A smaller number of cases are accounted for by complications of colopexy (1%) and colonic neoplasia (1%); colonic hypo- or aganglionosis was suspected, but not proved, in another 2% of cases. Inflammatory, pharmacologic, and environmental/causal behavioral causes were not cited as predisposing factors in any of the original case reports. Endocrine factors (obesity, n=5; hypothyroidism, n=1) were cited in several cases, but were not necessarily impugned as part of the pathogenesis of megacolon.

**Therapeutic Plan**

The specific therapeutic plan will depend upon the severity of constipation and the underlying cause. Medical therapy may not be necessary with first episodes of constipation. First episodes are often transient and resolve without therapy. Affected animals should always be re-hydrated if dehydration has contributed to the onset of clinical signs. Mild to moderate or recurrent episodes of constipation usually require some medical intervention. These cases may be managed, often on an outpatient basis, with dietary modification, water enemas, oral or suppository laxatives, and/or colonic prokinetic agents.
Severe cases of constipation usually require brief periods of hospitalization to correct metabolic abnormalities and to evacuate impacted feces using water enemas, manual extraction of retained feces, or both. Follow-up therapy in such cases is directed at correcting predisposing factors and preventing recurrence. Subtotal colectomy will become necessary in cats suffering from obstruction or idiopathic dilated megacolon.

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