

MEGAOESOPHAGUS DUE TO MYASTHENIA GRAVIS IN A DOG

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Abstract: A 7-year-old, 32 kg, male golden retriever presented in Madras veterinary College, Teaching veterinary clinical complex, vepery with a history of regurgitation and ptyalism and it developed a progressively reduced exercise tolerance. Results from a complete blood cell count, serum biochemical profile, and urinalysis did not indicate any metabolic abnormalities. Thoracic radiographs revealed a normal distal cervical and thoracic megaesophagus, Haematological examination, blood glucose, serum calcium, sodium, and potassium were all within normal limits. An electrocardiograph was normal. Treated with Tablet. Pyridostigmine - 64 mg orally every 8 to 12 hours for two weeks with Antacids. Pan 40 mg before food for two weeks to minimize stomach acid damage to the esophagus when food is regurgitated from the stomach and advised with elevated feeding and Cisapride is given up to three times daily. The Bailey Chair was used by owner. This case report suggests that patients with megaesophagus due to myasthenia gravis and animal recovered with that oral medication along with advised feeding and managemental practice.

Keywords: Megaesophagus, Myasthenia gravis, Acetylcholine esterase inhibitors, Exercise intolerance.

INTRODUCTION

Megaesophagus (ME) is a disorder characterized by decreased or absent esophageal motility that results in a diffuse dilation of the oesophagus (Washabau,2003) which causes the accumulation of ingesta, dilatation of the oesophageal lumen, food regurgitation and weight loss as the main clinical signs. Megaesophagus may be primary Megaesophagus, which is idiopathic, or secondary Megaesophagus, which occurs in conjunction with other diseases including myasthenia gravis, hypoadrenocorticism, dysautonomia, polyradiculoneuritis, hypothyroidism, polymyopathies and esophageal cancer (Wray J. D and Sparkes A. H, 2006). Canine Megaesophagus was idiopathic (IME) (76%), and the rest were secondary Megaesophagus (24%), mainly involving myasthenia gravis [Manning K et al., 2016].

Myasthenia Gravis (MG) is a disorder of neuromuscular transmission in which autoantibodies against nicotinic acetylcholine receptors (AChRs) at the neuromuscular junction results in reduction of AChRs and muscle weakness (Lindstrom et al., 1987). The disease is characterized by muscle weakness and fatigue observed between the ages of one and eight years (Hopkins, 1992). German shepherd and Labrador/Golden retriever breeds are

the most commonly diagnosed with this disease (Lee et al., 2005). This case report suggests that patients with secondary megaesophagus associated with myasthenia gravis.

HISTORY AND CLINICAL SIGNS

The owner reported vomiting and regurgitation of undigested food a few minutes after eating. Mild dyspnea was observed after light exercise. Physical examination revealed hypersalivation and regurgitation. Lateral thoracic radiograph revealed megaesophagus . There was no visible muscle wasting in the limbs, and resistance to passive movement felt normal. The dog primarily regurgitated solid food with occasional regurgitation of water he dog was bright, alert, and responsive, with a normal rectal temperature, heart rate and respiratory rate. The results of a neurological examination were also normal. Haematological examination, blood glucose, serum calcium, sodium, and potassium were all within normal limits.

TREATMENT

Clinical findings were evaluated and, on the first day treated with Injection. Ringerlactate 300 ml I/V, Injection. Vomidone – 1ml I/V, Injection. Pantaprazole -30mg I/V after radiography suggestive of Megaoesophagus treated with Tablet. Pyridostigmine 64 mg orally every 8 to 12 hours for two weeks with Antacids advised Tablet. Pan 40 mg before food for two weeks to minimize stomach acid damage to the esophagus when food is regurgitated from the stomach along with that Elevated feeding and Cisapride is given up to three times daily. The Bailey chair feeding advised and followed.

DISCUSSION

The most common findings of Megaoesophagus are reported to be walking disorders following exercise such symptoms are also seen in peripheral neuropathy and polymyositis. Cholinesterase inhibitor drugs are the principal agents used in the management of canine Megaoesophagus (Hopkins, 1992). The myasthenia crisis and signs of the Megaoesophagus during treatment can quickly be cured by the use of corticosteroids cannot be recommended in all cases of MG (Cuddon, 1989). In this case dog owner was maintained with elevated feeding schedule and the Bailey Chair for dog and treated with Tablet pyridostigmine for three weeks animal recovered and no regurgitation. Owners can feed their pet in an elevated position on stairs or on a raised platform

Acetylcholinesterase (AChE) inhibitors have been the foundation of therapy for acquired MG and are often the first line of therapy. The mechanism of action of AChE inhibitors is to

inhibit hydrolysis of acetylcholine at the neuromuscular junction prolonging the action of acetylcholine (Gomez et al., 2010)

Pyridostigmine bromide is preferred in most clinical situations because of its longer duration of action and fewer side effects (Shelton,2002).

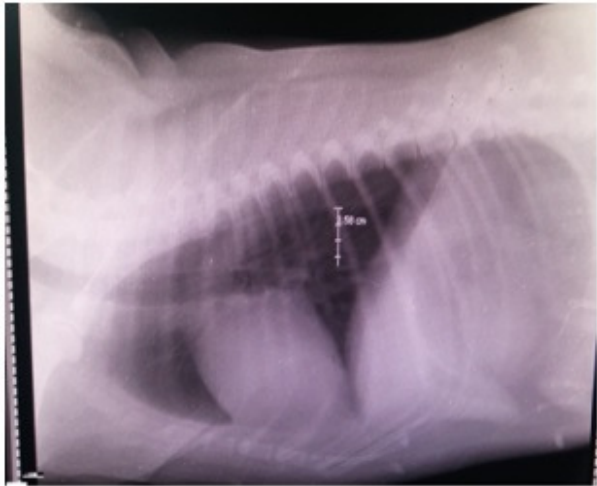
Both pyridostigmine bromide and neostigmine bromide inhibit the hydrolysis of ACh by directly competing with ACh for attachment to AChE. Adverse effects occur because muscarinic receptors located on the exocrine glands increase gastric acid secretion, salivation, and lacrimation. Bradycardia can be seen due to excessive vagal activity. Side effects seen in people are similar in dogs (Punga,2008)

REFERENCES

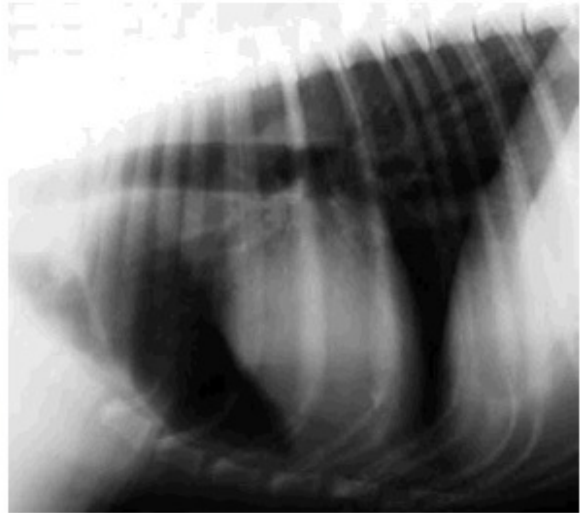
- [1] Cuddon P.A., 1989, Acquired immune-mediated myasthenia gravis in a cat, *Journal of Small Animal Practice*, 30: 511-516
- [2] Gomez AM, Van Den Broeck J, Vrolix K, *et al.* Antibody effector mechanisms in myasthenia gravis – pathogenesis at the neuromuscular junction. *Autoimmunity* 2010; **43** (5–6):353–370.
- [3] Hopkins A.I., 1992, Canine myasthenia gravis, *Journal of Small Animal Practice*, 33: 477-484
- [4] Lee S., Woo-Pil, H., Youn-Ju K., and Tae-wan, K., 2005, A case of acquired myasthenia gravis in German shepherd dog, *Journal of Veterinary Clinics*, 22(4): 392-395
- [5] Manning K., Birkenheuer A. J., Briley J., Montgomery S. A., Harris J., Vanone S. L., Gookin J. L. 2016. Intermittent At-Home Suctioning of Esophageal Content for Prevention of Recurrent Aspiration Pneumonia in 4 Dogs with Megaoesophagus. *J. Vet. Intern. Med.* 30: 1715–1719.
- [6] Punga AR, Sawada M, Stålberg EV. Electrophysiological signs and the prevalence of adverse effects of acetylcholinesterase inhibitors in patients with myasthenia gravis. *Muscle Nerve* 2008; **37**(3):300–307.
- [7] Shelton G.D., 1995, Canine myasthenia gravis, In: R.W. Kirk: *Current Veterinary Therapy-11th ed.* (Publ.) W.B. Saunders Comp., Philadelphia. pp. 1039-1042
- [8] Shelton GD. Myasthenia gravis and disorders of neuromuscular transmission. *Vet Clin North Am* 2002; **32**:189–206.
- [9] Washabau R. J. 2003. Gastrointestinal motility disorders and gastrointestinal prokinetic therapy. *Vet. Clin. North Am. Small Anim. Pract.* 33: 1007–1028, vi. doi: 10.1016/S0195-5616(03)00076-7

[10] Willard M. D. 2003. Disorders of the oral cavity, pharynx, and esophagus. pp.410–412. *In: Small Animal Internal Medicine*. 3rd ed., Mosby, St. Louis.

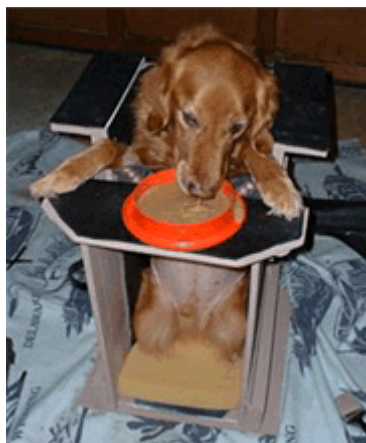
[11] Wray J. D., Sparkes A. H. 2006. Use of radiographic measurements in distinguishing myasthenia gravis from other causes of canine megaesophagus. *J. Small Anim. Pract.* 47: 256–263.



Normal Canine chest Radiography



Radiography of Megaesophagus in dog



Dog fed with Bailey Chair for megaesophagus