MEGAESOPHAGUS AND ITS MANAGEMENT IN THREE DIFFERENT LARGE BREED ADULT BITCHES

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Acquired Secondary Megaesophagus in adult dogs can result from numerous neuromuscular, endocrine or inflammatory disorders as well as from obstructive lesions (Stronmbeck and Guilford, 1990; Twedt, 1995), Gastroesophageal sphincter (GES) achalasia during secondary peristalsis (Boria et al., 2003), hyper and hypoadrenocorticism, esophageal dismotility, paraesophageal hiatal hernia, delayed transit disorders or hypomotility, hypertrophic osteopathy, tigersnake envenomation, dysautonomia, acquired myasthenia gravis and canine distemper (Mossallanejad et al., 2010). This report puts on record the successful surgical and non-surgical management of megaesophagus in three adult large breed bitches.

Case History and Observations

Three bitches – one Labrador Retriever, 4 years of age, 24 kg body wt., whelped once, one GSD, 5 years of age, 17 kg of body wt., whelped 5 pups once and one Golden Retriever, 11 years of age, respectively were presented to the University Clinics with a history of chronic regurgitation having no prodormal phase of vomiting, dysphasia, repeated swallowing attempts, ptysialism, halitosis and were maintained by life-saving fluid infusion and supportive therapy since last 15 days to 1.5 months. Regurgitated materials were not bile-stained. All three bitches were refractory to medicinal treatments done by referring veterinarians. On physical examination the bitches were 2-3 out of 5 body condition score and cachectic but bright, alert and responsive with normal rectal temperature, heart and respiratory rate without any respiratory tract problem as all the cases were receiving courses of antibiotic therapy. The average result of biochemical parameters like blood glucose fasting (78 mg/dl), Serum bilirubin (total 0.56 mg/dl, conjugated – 0.20 mg/dl, Unconjugated 0.36 mg/dl), Protein (total – 6.3 gm/dl, albumin – 2.5 gm/dl, Globulin – 3.8 gm/dl), Alkaline phosphatase – (146 n.μ/ml), SGPT (24 μ/l), SGOT (36 μ/l), BUN – (11mg/dl) and Serum Creatinine (0.84 mg/dl) were within normal ranges including T₄(21.0 n mol/l), TSH (0.09 ng/ml) and Na⁺ : K⁺ (140/5 or 28). The 1st bitch was examined through barium esophagogram after administration of 50ml of microbar (Barium Sulphate Paste) and 30 ml of distilled water under 4 ml of I/V diazepam. The second bitch received lesser dose for this examination. In both the cases dilated thoracic esophagus with stasis of barium sulphate suspension and normal thickness of GES with failure to open for transport were noticed (Fig.1 & Fig.2). In 3rd bitch dilated esophagus was sufficient for diagnosis of megaesophagus in digital survey radiography (Fig.3). X-ray upto 2nd day post-barium administration revealed no other abnormality of G.I. tract. By laboratory findings, radiography and physical examination hypothyroid-related neuropathy, hypoadrenocorticism or myasthenia gravis or presence of obstructive lesions were eliminated and

Treatment and Discussion

Lower sphincter achalasia was treated surgically in first case (Fig.4). Distal esophagomyotomy (Modified Heller’s method) was performed through midventral incision at supine position under
general anaesthesia. The abdominal incision was from umbilicus to xiphoid process. The stomach was retracted by Babcock forceps. The distal part of the esophagus, the lower esophageal sphincter region and the cardia was visualized. A longitudinal incision was made on ventral aspect of gastro esophageal junction through tunica serosa and tunica muscularis extending from distal 2 cm of the esophagus to proximal 2 cm of stomach. The esophageal and gastric mucosa bulged through the complete incision. Routine closure and standard follow up were made (Fig.5). The owner of the 2nd and third bitches afraid of the operation but all the owners strictly followed for elevated feeding at 45° on Baily chair and walking at 90° on hind limb for 10 min. after food. On the basis of telephone message and visit of the local veterinarian quick result was achieved with the first bitch as after two years it became 32 kg of body wt. from that of 24 kg pre-operation and needs no elevated feeding or extra-care. One and half years after post-management the G.S.D. bitch also gained body wt. being 30 kg from emaciated pre-treatment 17kg as did well the golden retriever with comparatively prolonged elevated feeding.
Congenital idiopathic megaesophagus in pups were reported by Gahlot et al. (2003) and Punnuswamy et al. (2008). Cases of Acquired megaesophagus have been reported by Boria et al. (2003), Anil Kumar et al. (2009) and Massallanejad et al. (2010). Since 1970’s a lot of works has been made in this field. Surgical attempt was made here following Kipperman and Straw (1988) and Boria et al. (2003). The diazepam was given to stop the regurgitation during contrast radiography. Unfortunately the definitive cause for the last two dogs’s acquired megaesophagus was not determined. Cricopharyngeal achalasia (upper achalasia) is a congenital condition in dogs. From clinical signs, age of onset, biochemical and radiographic evaluation and complete response to esophago-myotomy it can be concluded that the first dog in this report had acquired lower esophageal achalasia.

Summary

Three adult bitches—one Labrador Retriever, one GSD and one Golden Retriever with the history of chronic regurgitation were radiologically evaluated for Megaesophagus. From biochemical parameters, clinical symptoms and history it was diagnosed as Gastro esophageal Sphincter achalasia. In first bitch distal esophagomyotomy (modified Heller’s method) through midventral incision was done. All the bitches were kept on elevated feeding at 45 degree on Baily Chair and walking at 90 degree on hind limb for 10 minutes. All did well up to one and half years post operation.

Reference:


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