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A Rare report of ocular myasthenia gravis in male Chippiparai dog

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Abstract
A two years old male Chippiparai Dog was presented to the Veterinary Clinical Complex, Veterinary College and Research Institute, Orathanadu with a history of progressive weakness in their appendicular muscles, along with an acute onset of retching and regurgitation. On clinical observation, animal was emaciated with uncoordinated gait but the reflexes were found to be normal and exhibited progressive weakness upon exercise. On neurological examination, absence of withdrawal and deep pain reflex along with ptosis of both the eyelids, sluggish menace reflex and extra ocular palsies were noticed. Contrast radiography revealed megaesophagus. On biochemical analysis hyperproteinemia and hyperglobulinemia were observed. Based on these clinical observations and radiography, the case was confirmed as Ocular form of Myasthenia Gravis. The animal was treated with Inj. Neostigmine @ 0.04mg/kg SC at 6 hourly interval and Tab. Micronized Progesterone @0.2 mg/kg PO SID for 2 weeks. After 14 days of therapy the dog showed mild improvement in gait with improved appetite and no emesis were observed.

Keywords: Dog, ptosis, ocular form, megaesophagus, progesterone

Introduction
Myasthenia gravis is a disease caused by an abnormality in the acetylcholine receptor (AChR), which receives messages from the nerve and signals the muscles to contract. There are two forms of Myasthenia Gravis is congenital (inherited) and acquired. In the congenital form, animals are born with decreased numbers of receptors. In the acquired form, there is an autoimmune disorder on the acetylcholine receptor, rendering it dysfunctional. Ocular form of myasthenia gravis (OMG) can mimic isolated cranial nerve palsies, gaze palsies, internuclear ophthalmoplegia, blepharospasm, or a stroke. The common symptoms are progressive weakness, stiffness of limb muscles and quidding of food and water which is due to weakness of the esophagus. Due to paralysis, aspiration of food and water occurs which leads to pneumonia and result in cough and respiratory distress [1]. The first case report on ocular myasthenia gravis was reported in 9-year-old male neutered Border collie dog [2].
Myasthenia Gravis occurs more commonly in dogs than cats. The incidence in dogs is bimodal, affecting animals of any age predominantly young adult (2-4 years) and geriatric (9-13 years) patients. Breed that are predisposed are Golden Retrievers, German Shepherd Dogs, Akitas, German Short Hair Pointers, Terriers, Chihuahuas, Great Danes and Newfoundlands appear predisposed. The focal form of Myasthenia Gravis, consisting solely of weakness of the facial, pharyngeal, laryngeal or oesophageal muscles, occurs in 36– 43% of dogs. 57-64% of dogs show generalized weakness and 90% of dogs show concurrent megaesophagus. Paraneoplastic disease can occur, most commonly associated with thymomas. The latter appears to be more common in cats with studies reporting a cranial mediastinal mass in cats (26%) compared to dogs (3%) [3].

Materials and Methods
A Chippiparai dog aged two years was presented to VCC, VCRI, and Orathanadu with history of inappetence, progressive weakness in their appendicular muscles, along with an acute onset of retching, regurgitation and ataxia for the past one month. On clinical examination, all the vital parameters were normal. Animal was emaciated (Fig 1) with uncoordinated gait but the reflexes were found to be normal and exhibited progressive weakness upon exercise, resembling conditions like polymyositis. On neurological examination absence of withdrawal and deep pain reflex along with ptosis of both the eyelids and sluggish menace response were noticed.

On ocular examination of eyes, extraocular paralysis and sluggish menace response of the left eye and pupillary light reflex of both eyes were present. On administration of tropicamide mydriasis and ptosis of eyelid were noticed (Fig 2). Contrast radiography showed the presence of mega oesophagus. The animal was treated with micronized progesterone @ 0.2 mg/kg PO SID and Inj. Neostigmine @ 0.04mg/kg and supportive therapy were provided. The dog treated for 14 days and mild improvement in gait was noticed. The dog shown uneventful recovery after two weeks of treatment.

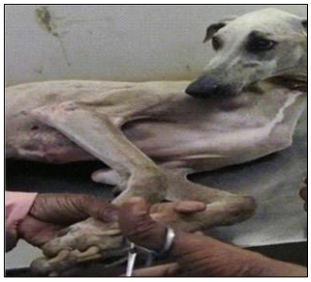

Fig 1: Emaciated and no spinal reflexes

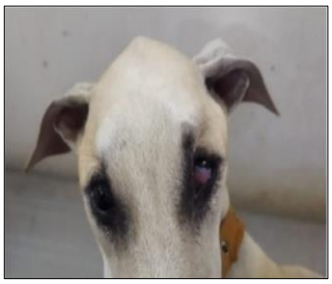

Fig 2: Ptosis of eyelid (left)

Results and Discussion

Ocular examination of dog showed absence of menace reflex in left eye and pupillary light reflex present in both the eyes and mydriasis noticed in both the eyes after administration of tropicamide (table 1). Serum biochemical analysis revealed increased total protein and globulin levels (table 2) as albumin/globulin ratio will go down in auto-immune conditions [5]. Electrocardiographical examination revealed low amplitude QRS complex (Fig.3) indicative of pulmonary infiltration. Contrast radiography showed the presence of megaesophagus (Fig 4)

Table 1: Ocular Examination of Dog

Parameters	Oculus Dexter (OD) (Right Eye)	Oculus Sinister (OS) (Left Eye)
Menace Reflex	Present	Absent
Pupillary Light Reflex	Present	Present
Tropicamide Administration	Mydriasis	Mydriasis

Table 2: Hemato-biochemical values of dog affected with ocular myasthenia

Parameters	Value	Reference Values
Hemoglobin (g/dl)	11.2	12-19
PCV (%)	34	37-57
RBC (*10^{6})	6.1	5-9
WBC (*10^{3})	7.5	5-15
Albumin (g/dl)	4.3	2.3-3.1
Globulin (g/dl)	5.8	2.7-4.4
Total Protein (g/dl)	10.1	5.4-7.5

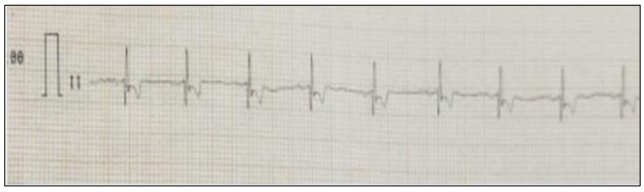

Fig 3: Electrocardiograpghy- Lead II- Low voltage QRS complex (10 mv/25 mm)

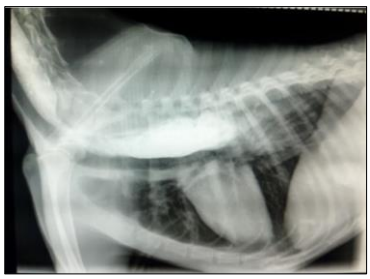

Fig 5: Contrast radiography - Lateral thorax - Megaesophagus

The signs of ptosis and third eyelid prolapse are classical signs of human ocular form of Myasthenia Gravis whereas progressive skeletal muscle weakness, regurgitation and multiple cranial nerve deficits were the clinical signs commonly noticed in dogs but rarely dog also showed similar clinical signs that exhibited in humans which in accordance with previous report [2]. The above clinical signs should be differentiate from the other neuromuscular diseases. Regurgitation causes should be differentiated from oesophageal affections like oesophagitis, foreign body obstructions and motility disorders which could be confirmed by thoracic radiography. The muscle weakness with no improvement should be differentiated from myopathies that could be supported by electromyography, biochemical analysis of creatinine kinase and basal cortisol levels. The cranial nerve defects ruled out from cranial myo-neuropathies and brain stem lesions. The ataxia was differentiated from hypoglycaemia, anaemia, dehydration, musculoskeletal problem and neurological problems. The confirmatory test is tensilon test that indicate elevated Acetylcholine auto antibodies.

The treatment was provided to the dog for 14 days by Micronized progesterone which cause remyelination of nerve [4] and Neostigmine which acts as a parasympathomimetic drug [5]. Dog showed improvement in the gait. As it is presented with mega-esophagus feeding tube technique followed for administration of medicaments and food [6]. The dog showed tremendous recovery after two weeks of treatment.

Summary and Conclusion

Better management practices would help in faster recovery of animals. The novel therapy using combination of micronized progesterone and neostigmine helps in faster recovery of dog and better improvement noticed. But further studies required in this field.

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