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CASE REPORT
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Myasthenia Gravis in an Exotic Shorthair Cat

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ABSTRACT

***Background*:** Myasthenia gravis is a neuromuscular transmission disorder resulting from the deficiency or functional disturbance of the acetylcholine receptors at the neuromuscular junction with consequent impairment of action potential transmission from nerve to muscle and it can be either congenital or acquired. Acquired myasthenia gravis has been described less frenquently in cats. The purpose of this paper is to describe a case of acquired myasthenia gravis in a 6-month-old male Exotic Shorthair cat.

***Case*:** A 6-month-old male Exotic Shorthair cat was referred to the Veterinary Clinic with a 2-day history of flaccid paralysis of the four limbs. The cat was feeding well and defecating normally. Physical examination was unremarkable. On neurological examination the patient was bright, alert and responsive. Flaccid paralysis of the four limbs was evident with loss of muscular tonus and presence of superficial and deep nociception. All spinal reflexes were absent. Hematologic analysis was within normal parameters. Thoracic radiographs were taken and showed no abnormalities. Creatine kinase activity was elevated. Myasthenia gravis was suspected and the cat was submitted to an neostigmine methylsulfate injection and further pyridostigmine bromide administration. Within 15 days there was clinical improvement with muscular tonus and spinal reflexes recovery. The treatment was continued for 3 months. On follow ups the cat showed improvement in clinical signs. After the medication withdrawn, the cat didn't show any of the disease`s symptoms.

***Discussion*:** Myasthenia gravis (MG) is characterized by muscle weakness, explained by the presence of T cell-dependent antibodies to the nicotinic acetylcholine receptors (AChRs) of the postsynaptic membrane in the neuromuscular junction. The nicotinic AChRs play a fundamental role on the neuromuscular transmission and any disturbance on these structures can interfere on muscle contracture and cause weakness due to the impairment of action potential transmission from nerve to muscle. Based on clinical finds and the satisfactory response to neostigmine metilsulfate and pyridostigmine bromide, presumptive diagnostic of MG was confirmed. Autoimmune MG can occur spontaneously in dogs and has been extensively studied and classified as analogous to the human form; although it is also spontaneous in cats, it is a rare condition and few reports are available. The treatment with pyridostigmine bromide was effective and helped on the diagnosis. Anticholinesterase drugs are the treatment of choice for MG because they act prolonging the action of acetylcholine at the neuromuscular junction. The occurrence of megaesophagus in cats is lower than in dogs and the manifestation of MG with a mediastinal mass is higher in cats. No mediastinal masses were present in this case. The cat was an Exotic Shorthair. There is a known relative risk for acquired MG in Abyssinian and Somali cats and, it has been reported in Persians, the closest breed to Exotics. The differential diagnoses to be considered are other disorders of neuromuscular transmission such as congenital MG, botulism, polymyositis, polyneuropathy, organophosphate toxicity, hypoadrenocorticism and hypothyroidism. Autoimmune MG is an important neuromuscular dysfunction that should be considered as differential diagnosis for cats with muscle weakness. Early diagnosis and treatment are decisive and should be used for a better prognosis.

Keywords: flaccid paralysis, acetylcholine, neuromuscular disorder, feline.

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INTRODUCTION

Myasthenia gravis (MG) is a neuromuscular transmission disorder that results from the deficiency or functional disturbance of the acetylcholine receptors (AchR) at the neuromuscular junction with impairment of action potential transmission from nerve to muscle as consequence [11]. MG was first reported in dogs in 1961 [9] and it can be either congenital or acquired [2]. The acquired, or autoimmune form is usually described in adult dogs whereas the congenital form is associated with young animals [9]. The congenital form is due to either a deficiency or functional disorder of the AChR and the acquired form is an immune-mediated disease [2].

Acquired MG has been described less frenquently in cats. Generally, they show muscular weakness, ventroflexion of the neck and palpebral reflexes deficits [4,5,10]. Less commonly, it is reported regurgitation, changes in vocalization and mediastinal neoplasias. The predisposed cat breeds associated are Abyssinians and related Somali cats [10]. The purpose of this paper is to describe a case of acquired MG in a 6-month-old male Exotic Shorthair cat.

CASE

A 6-month-old male Exotic Shorthair cat was referred to the Veterinary Clinic with a 2-day history of flaccid paralysis of the four limbs. The cat was feeding well and defecating normally. Physical examination was unremarkable. On neurological examination the patient was bright, alert and responsive. Flaccid paralysis of the four limbs was evident with loss of muscular tonus and presence of superficial and deep nociception. All spinal reflexes were absent. Hematologic analysis was within normal parameters. Thoracic radiographs were taken and showed no abnormalities. Creatine kinase activity was 1200 U/L (7.2 - 28.2 U/L - reference value for the species).

MG had been suspected and the cat was submitted to an intravenous (IV) neostigmine metilsufate injection[1] (2 mg/kg) and further pyridostigmine bromide[2] administration (2 mg/kg PO BID). Within 15 days there was clinical improvement with muscular tonus and spinal reflexes recovery. The treatment was continued for 3 months. On follow ups the cat showed improvement in clinical signs. After the medication withdrawn the cat didn't show any of the MG symptoms.

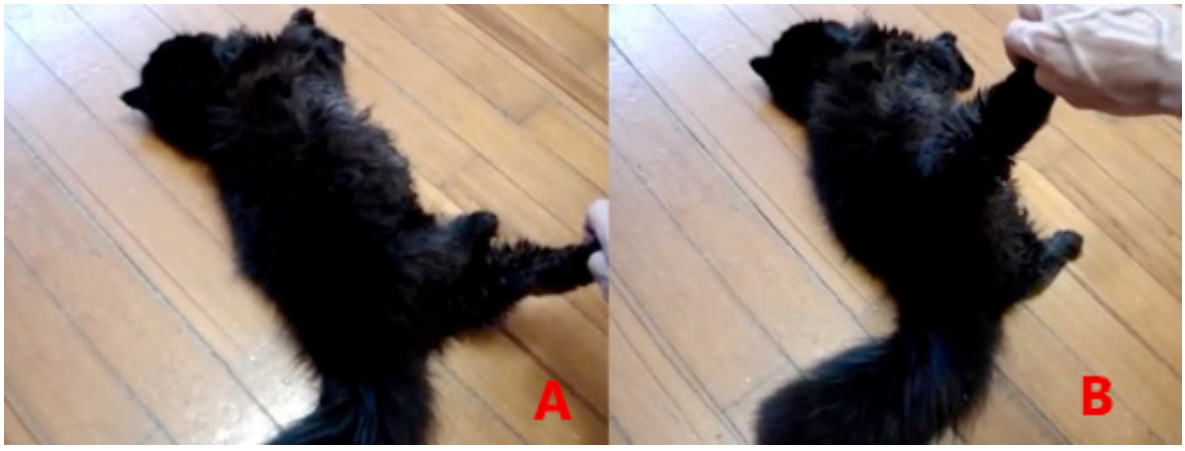

Figure 1. A 6-month-old Exotic Shorthair cat with acquired MG with flaccid paralysis of four limbs. Presence of superficial and deep nociception on left hinflimb (A). Presence of superficial and deep nociception on right hindlimb (B).

DISCUSSION

MG is characterized by muscle weakness, explained by the presence of T cell-dependent antibodies to the nicotinic acetylcholine receptors (AChRs) of the postsynaptic membrane in the neuromuscular junction [11]. The nicotinic AChRs play a fundamental role on the neuromuscular transmission and any disturbance on these structures can interfere on muscle contracture and cause weakness due to the impairment of action potential transmission from nerve to muscle [9,11]. Not only limb muscles can be affected but also ocular, facial, oropharyngeal and esophageal musculature [9]. Based on clinical finds and the satisfactory response to neostigmine metilsulfate and pyridostigmine bromide, presumptive diagnostic of MG was confirmed [6]. Diagnosis can also be made based on the response to edrophonium chloride administration that usually produces a rapid but transient improvement on clinical

signs although this test is neither sensitive nor specific [11]. Specific and sensitive diagnostic tools are available such as the acetylcholine receptor antibodies test by immunoprecipitation radioimmunoassay [8], immunocytochemical identification of antibodies boundig at the neuromuscular junction [2], demonstration of decrement in the compound muscle action potential during nerve stimulation by electromyography [11] and enzyme-linked immunosorbent assay [12], but these tests were not available for this case.

Autoimmune MG can occur spontaneously in dogs [1] and has been extensively studied and classified as analogous to the human form [8]; although it is also spontaneous in cats, it is a rare condition and few reports are available [4,6,8,10]. In a study conducted by Shelton and colleagues [10], 105 felines were diagnosed with MG based on AchR antibodies titers. The observed clinical findings were generalized weakness associated or not with megaesophagus (28.6%), and generalized weakness associated with thymoma (25.7%). Dysphagia and megaesophagus were also reported (14.3%) as well as the occurrence of MG on 5 cats with hyperthyroidism that were treated with methimazole. It was concluded that the occurrence of megaesophagus in cats is lower than in dogs and the manifestation of MG with a mediastinal mass is higher in cats. In another study, it was noted a propensity for hindlimb weakness in dogs with MG [2]. The cat on this report presented hindlimb paralysis but there are no studies showing if there is a tendency for a specific limb weakness on felines. Other species have been used to develop experimental models of acquired MG (rats, mice, guinea pigs and rabbits), but in these animals the immune response is not naturally self-perpetuating [8].

The treatment with pyridostigmine bromide was effective and helped on the diagnosis. Anticholinesterase drugs are the treatment of choice for MG because they act prolonging the action of acetylcholine at the neuromuscular junction [9]. Corticosteroids can also be used [2] but have potential side effects [7]. No mediastinal masses were found in this case. Thoracic radiographs are important to evaluate the presence of such neoplasms [9]. Cyclosporine has also been reported to treat MG with success in dogs [1]. Creatine kinase activity was elevated, similar to another report [4].

The cat was an Exotic Shorthair. There is a known relative risk for acquired MG in Abyssinian and Somali cats and, it has been reported in Persians, the closest breed to Exotics [10]. The differential diagnoses to be considered are other disorders of neuromuscular transmission such as congenital MG, botulism, polymyositis, polyneuropathy, organophosphate toxicity, hypodrenocorticism, hypothyroidism [7]. There is a report of a third degree antrioventricular block in dogs with MG [3] but this condition has not been described in cats.

Autoimmune MG is an important neuromuscular dysfunction that should be considered as differential diagnosis for cats with muscle weakness. Early diagnosis and treatment should be used for a better prognosis.

SOURCES AND MANUFACTURERS

[1]Prostigmine®, Roche, São Paulo, SP, Brazil

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Declaration of interest. The authors report no conflicts of interest. The authors alone are responsible for the content and writing of the paper.

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