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Case Report

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Extraocular myositis in a female puppy

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Abstract
Extraocular myositis (EOM) is not commonly encountered in dogs. It is generally diagnosed based on clinical features of exophthalmos without third eyelid protrusion, pain or vision loss. The traditional treatment of choice is prednisolone. This report describes a case of a mixed-breed puppy with clinical signs consistent with EOM, the use of ascorbic acid as an adjuvant to traditional corticosteroid therapy and rapid resolution of the condition without recurrence. It also shows that prolapse of the third eyelid and ptosis of the lower eyelids are possible signs of EOM during recovery. This is the first report of this sort from Africa and therefore the report is of epidemiological significance.
Keywords: Exophthalmos, Extraocular myositis, Myositis, Nictating membrane, Steroid.

Introduction

The occurrence of extraocular myositis (EOM) is unusual both in veterinary practice and in the scientific literature. Most veterinarians may never encounter the disease. The earliest report describing this condition was by Carpenter and his colleagues in 1989. They reported a polymyositis in extraocular muscles in two unrelated dogs (Carpenter *et al*., 1989).Dinae Shelton's group reviewed 200 cases of canine myositis, in which only two cases were EOM (Evans *et al*., 2004). The rarity of the condition and good response to oral steroid treatment seem to have discouraged a thorough investigation into the pathogenesis of this condition (Williams, 2008).

In most reports, larger percentage of the affected breeds are Golden Retrievers and usually younger dogs are involved, mostly females (Ramsey *et al*., 1995; Evans *et al*., 2004; Williams, 2008). The condition is usually characterized by a painless bilateral exophthalmos without involvement of the nictating membrane. EOM responds well to corticosteroid therapy. Although the pathogenesis is unknown, it is difficult to draw a parallel between the pathogenesis of EOM and the so-called similar conditions in humans (Williams, 2008).The majority, if not all, of the published reports on EOM are from America and Europe and the condition has not to date been reported in Africa.

This, then, is the first case report of clinically diagnosed canine EOM in Africa and the youngest dog to be reported with the condition so far. This indicates that the disease is not limited to continents of Europe and America. Also, there has been no known report of bilateral eyelid ptosis during recovery as seen in this case.

Case history

A 15 weeks old entire mixed breed bitch weighing 8kg, was presented with sudden onset of bilateral exophthalmos (Fig. 1), impaired vision, as seen by infrequent instances of bumping into obstacles, poor ability to focus with jerky head movement in an attempt to focus, increased sclera show and 'startled' expression (Fig. 1). Prior to presentation, the puppy had been treated by a livestock assistant (who estimated the weight of the dog to be 10kg), with parenteral oxytetracycline 1 mg/kg and dexaphenylarthrite (a drug containing phenylbutazone and dexamethasone) at a dose of 0.0012 mg/kg phenylbutazone and 0.023 mg/kg dexamethasone for 3 consecutive days without any remission. It was confirmed that the animal exhibited no sign of jaw pain and the appetite was not depressed prior to treatment by the livestock assistant. Erroneously assuming that the dog was infected with canine distemper and was a hopeless case, the owner had initially requested that the dog be euthanized on initial presentation. The dog had been maintained on commercial dog food alone. It was vaccinated against rabies and dewormed about two weeks before onset of the condition.

Physical examination revealed no abnormality except for the ocular symptoms. There were no lesions on the eyeballs and ocular movements were normal. The nictitating membranes were normal and the animal was not in any form of ocular or muscular pain. There

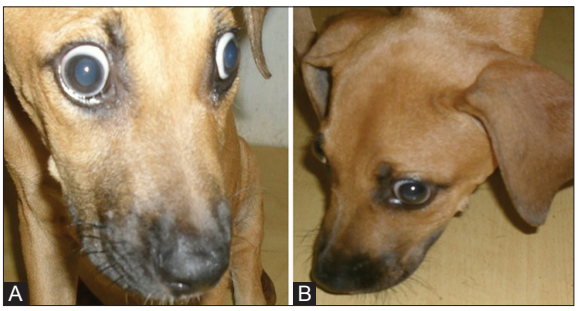

Fig. 1. (A and B) Marked sclera show, 'startled' appearance, exotropia and exophthalmus.

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was minimal resistance to retropulsion of the globe into the orbit with eyelids closed. Hematology, rectal temperature, heart and respiratory rates were normal. The animal was otherwise in good health with very good appetite and responded when called by her name. Pupillary light reflexes were normal. There were no clinical signs of hyperthyroidism. Clinical signs were limited to ocular signs only and the masticatory muscles were not affected. The puppy had no difficulty opening it mouth neither did it show any sign of pain in the masticatory muscles during physical examination. Masticatory muscles myositis was thus ruled out.

A diagnosis of EOM was made, based on the history, clinical examination and classical presenting signs.

A steroid regimen was commenced; 1.25 mg/kg Prednisolone (Prednisolon Ratiopharm, Ratiopharm, The Netherlands) tablets with 12.5 mg/kg Ascorbic Acid (LETAP pharmaceuticals Limited, Accra, Ghana) daily for two weeks. Marked response to treatment was observed after the first week, as seen by marked reduction in exophthalmos. At the third week, a tapering dose of prednisolone was administered. On the first day of the third week (day 15 of treatment), 10 mg of prednisolone was administered and the dose was then tapered with 7.5 mg, 5 mg, 2.5 mg, 1.25 mg, 1 mg given on days 16, 17, 18, 19 and 20 of treatment respectively. Steroid administration was terminated after day 20 while maintaining the dosage of ascorbic acid throughout the 3weeks of treatment. Ocular signs completely resolved at the end of the third week, without any serious side effects.

At the end of two weeks the dog was weighing 12kg. During treatment, there was bilateral lower eyelid ptosis and mild protrusion of the third eyelid. These resolved in less than a month post treatment. The bitch was monitored for ten months, no recurrence was observed.

Discussion

David Williams published the largest EOM case series so far. In this series, 37 cases were reported. The common features among these cases were bilateral exophthalmos without protrusion of the third eyelid (Williams, 2008). Although EOM is generally characterized by exophthalmos (Azoulay and Jongh, 2011) as seen in this case report, cases of enophthalmos and extreme ventral, ventromedial or medial strabismus have been reported (Allgoewer *et al*., 2000). The pathogenesis of this condition is not fully understood, but the one dominant histopathologic picture of the condition is that of mononuclear cellular infiltration (Carpenter *et al*., 1989; Ramsey *et al*., 1995; Evans *et al*., 2004). This has led some authors to postulate that the condition is likely due to an immune mediated mechanism. One report has linked the pathogenesis to an infectious agent (Pumorola *et al*., 2004) although it should not be presumed that such occurrence is relatively common (Williams, 2008). Diagnosis of EOM is usually based on pathognomonic clinical signs, which was evident enough in this case to warrant such diagnosis. The use of MRI, CT scan, and biopsy of affected muscle (which is difficult to access) has been said to be, not only an unnecessary exercise but for the latter diagnostic step of biopsy an unethical one in most cases (Williams, 2008).

One similar condition that may present bilateral exophthalmos and could have been confused with EOM in this case was eosinophilic Masticatory Muscle Myositis (MMM) (Evans *et al*., 2004). Although both are focal myositis, myositis in MMM basically involve the muscles innervated by the Trigeminal nerve, i.e., the Masseter muscle, Temporalis muscle, Pterygoids, Tensor Tympani and Tensor Palatine. These muscles contain the 2M fibers against which the auto-antibodies are directed. This generally is not the case with EOM (Evans *et al*., 2004). In acute MMM there is exophthalmos due to the forward displacement of the eyeball by the inflamed masticatory muscles whereas in the chronic phase, atrophy of the affected muscle result in enophthalmos. One major clinically distinguishing feature is the absence of pain in EOM (as seen in this case report) unlike MMM (Williams, 2008). In MMM, there is typically protrusion of the nictating membrane which may be accompanied by episcleral congestion and possible exposure keratitis depending on the chronicity. Pyrexia and anorexia should be expected in MMM, as pain accompanies any attempt to open the jaw (Campbell *et al*., 1979; Evans *et al*., 2004; Mitchell, 2008; Williams, 2008). Protrusion of the third eyelid, although rare, has been reported in EOM (Williams, 2008). In this case report, protrusion of the third eyelids did not occur until treatment commenced (Fig. 2). Hence, this shows that it is difficult to make a sweeping assertion that protrusion of the third eyelid is limited to MMM. The young bitch in this case report was otherwise healthy, although vision was impaired,

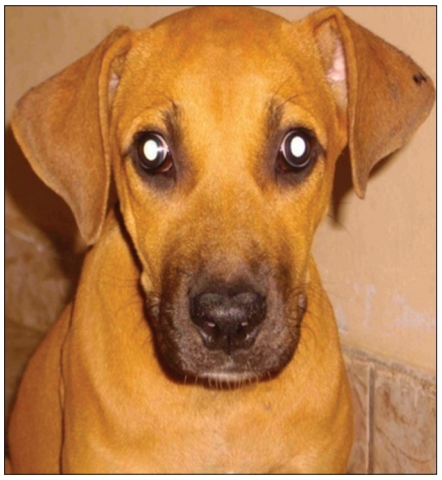

Fig. 2. Absence of startled appearance, protrusion of the third eyelid and weight gain (About 2 weeks of treatment).

probably due to the compression of the optic nerves by markedly enlarged extraocular muscles in the posterior orbit (Carpenter *et al*., 1989).

Mild bilateral ptosis of the lower eyelids was observed towards the end of treatment, there is no known report of a similar case. It could be associated with previous excessive distention of the lower eyelid by the enlarged globe and rapid reduction in size of the proptosed globe during treatment, causing an unequal rate of retraction between the globe and the elastic eyelids, this leaves the eyelids saggy. This resolved without treatment. Also, the increase in appetite and weight are due to the side effect of the steroid. The use of corticosteroid in the treatment of EOM is popular (Shelton *et al*., 1985; Williams, 2008). Although there have been report of relapses (Carpenter *et al*., 1989; Williams, 2008), prednisolone seems to be the drug of choice. Prolonged usage can result in undesirable effects. Azathioprine has been used as substitutes in cases where there are undesirable side effects of steroids (Mitchell, 2008; Williams, 2008). In this case, the use of prednisolone did not result in any severe side effects. Tapering the dosage towards the end of treatment period reduced the chances of any serious side effects (Fraser, 1986; Williams, 2008). Alongside the usual therapy for treatment of EOM, ascorbic acid was administered during treatment since it possesses good anti-inflammatory properties (Wannamethee *et al*., 2006; Mikorova *et al*., 2012). Recovery was without recurrence. The use of ascorbic acid as an adjuvant medication in the treatment of myopathies or EOM in particular (considering its established anti-inflammatory properties) may be a topic requiring further research.

The actual cause of the condition could not be ascertained, as with most cases of EOM. Histopathologic diagnosis and CT scan or MRI was not done, not only because of cost and ethics but also due to inaccessibility of such facilities for veterinary use in Gambia. It is noteworthy that almost two weeks before presentation, the animal was vaccinated against rabies. Since EOM is thought to be an immune-mediated condition, the rabies vaccination could have been a trigger factor in the pathogenesis of EOM in this young bitch. Vaccination has been associated with several autoimmune reactions; EOM in this case could have been triggered by vaccination. This although is subject to further research.

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