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Fundoscopic findings in a cat with acute ivermectin toxicity: A case report

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Abstract

Ivermectin is a commonly used antiparasitic drug; however, inappropriate administration may lead to toxicity in small animals. The present report describes the neuro-ophthalmic findings in a cat with suspected ivermectin toxicosis. A one-and-a-half-year-old female non-descript cat weighing 4 kg was presented with a history of sudden onset tremors, obtundation, blindness, and dilated pupils. According to the owner, ivermectin had been administered orally and by injection one day prior to presentation for suspected mite infestation. Clinical examination revealed a heart rate of 103 beats/min, respiratory rate of 26 breaths/min, and rectal temperature of 37.2 °C. Neurological examination showed mental dullness, rotatory nystagmus, hindlimb proprioceptive deficits, and generalized tremors. Ophthalmic examination revealed bilaterally absent menace response, dazzle reflex, and pupillary light reflex with fixed mydriasis. Schirmer tear test and intraocular pressure values were within normal physiological limits. Fundoscopic examination demonstrated a hyperreflective tapetum with normal retinal vasculature and no visible abnormalities of the optic disc. The findings suggest that visual impairment associated with ivermectin toxicosis may occur without obvious structural changes in the retina. Early recognition of neurological and ophthalmic signs is important for the diagnosis of ivermectin toxicity in cats.

Keywords: Ivermectin toxicosis, cat, blindness, funduscopy, neuro-ophthalmic examination

Introduction

Ivermectin is a semi-synthetic macrocyclic lactone commonly used as an antiparasitic drug. Toxicity develops when the drug penetrates the blood-brain barrier and enhances the activity of gamma-aminobutyric acid (GABA), leading to neurological and ocular manifestations such as hypersalivation, tremors, ataxia, dilated pupils with reduced pupillary response, blindness, and in severe cases, death (Merola *et al.*, 2012) ^[1]. Most reported cases of ivermectin toxicosis involve animals that were accidentally or deliberately exposed to formulations not intended for their species (Muhammad *et al.*, 2004, Kenny *et al.*, 2008) ^[2, 3]. The risk of toxicosis is especially high when high-concentration ivermectin formulations intended for large animals are administered to small animal species.

Mechanisms underlying blindness in animals with ivermectin toxicosis are unknown. Previous studies have suggested that both intracranial (Plummer *et al.*, 2006) ^[4] and retinal (Kenny *et al.*, 2008) ^[3] mechanisms may contribute to the clinical manifestations of ivermectin toxicity. Gamma-aminobutyric acid (GABA), the principal inhibitory neurotransmitter in the mammalian retina, plays a critical role in the modulation and processing of visual signals. Therefore, disruption of GABA-mediated neurotransmission may contribute to the visual disturbances observed in affected animals. Ophthalmic examination may reveal multifocal areas of retinal oedema, retinal folds, and mild separation of the retina (Epstein *et al.*, 2013; Merola *et al.*, 2009) ^[5, 6]. Electroretinography (ERG) commonly demonstrates decreased amplitudes of both a- and b-waves, indicating impaired retinal function. However, visual deficits observed in affected animals may also be associated with involvement of the central nervous system (Pollio *et al.*, 2018) ^[7]. Intravenous lipid emulsion (ILE) therapy has been advocated in the management of lipophilic toxicants, including ivermectin. Its therapeutic effect is believed to occur through the “lipid sink” phenomenon, whereby the lipid phase

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sequesters circulating toxin molecules, ultimately lowering their bioavailability and limiting distribution to target tissues (Epstein *et al.*, 2013)^[5].

This report aims to describe the fundoscopic findings in a cat with suspected ivermectin toxicity. Although intravenous lipid emulsion (ILE) therapy was advised, the animal was not presented for subsequent treatment or follow-up assessment. Furthermore, due to the emergency nature of the case, comprehensive hematological and biochemical analyses were not available.

Materials and Methods

A one-and-a-half-year-old female non-descript intact cat weighing 4 kg was presented to the Department of Veterinary Surgery and Radiology, MR College Of Veterinary Science and Research Centre, Jhajjar, Haryana, India with the complaint of sudden onset of tremors, obtundation, blindness, and dilated pupils. The cat had no relevant medical history. One day before presentation, the owner reportedly administered ivermectin, both orally and via injection, in an attempt to treat a suspected mite infestation. Neuro-ophthalmic test (Menace response, PLR, Palpebral reflex, Corneal reflex), Aqueous tear tests (Schirmer’s strip test reading in mm), intraocular pressure and fundoscopic examination was done.

Rebound tonometer (icare Tonovet plus) was used to measure intraocular pressure (IOP). A magnetised pin, which had a plastic tip at its end, was placed in the instrument. After selecting the canine option, proper holding of the instrument was detected by the light source present on the instrument (it should be parallel to the ground). The instrument takes six readings and averages them. Fundoscopic examination was conducted without pharmacological mydriasis, as the pupils were already adequately dilated, allowing satisfactory visualization of the ocular fundus (Fig. 1). Monocular indirect ophthalmoscopy was performed in the cat using a 20-diopter indirect condensing lens (Volk) and a Finoff transilluminator (Heine). The animal was restrained in sternal recumbency in a darkened room, with assistance provided to gently maintain eyelid opening. The light source was positioned at the examiner’s temple and directed toward the patient’s eye. After identification of the tapetal reflex (Fig. 1), the condensing lens was held in front of the eye to facilitate examination of the ocular fundus. Fundus images were captured using the iPhone 13 camera.



Fig 1: Dilated pupil (mydriasis) showing a prominent tapetal reflex in the eye of the cat.

Results and Discussion

A one-and-a-half-year-old, female non-descript intact cat weighing 4 kg was presented with the complaint of sudden

onset of tremors, obtundation, blindness, and dilated pupils. On presentation, physical examination revealed a heart rate of 103 beats per minute, a respiratory rate of 26 breaths per minute, and a rectal temperature of 37.2 °C. No other abnormalities were detected. Neurological examination revealed mental dullness, rotatory nystagmus, proprioceptive deficits in the hindlimbs, and generalized tremors. Ophthalmic examination demonstrated bilaterally absent menace responses, dazzle reflexes, and pupillary light reflexes (PLRs) (Table 1), along with fixed mydriatic pupils. Intraocular pressure (IOP) and Schirmer tear test (STT) values were within normal physiological limits.

Table 1: Detailed ocular examination (neuro-ophthalmic, STT and IOP tests) conducted in cat; STT: Schirmer tear test; IOP: Intraocular Pressure; OD: Oculus dexter; OS: Oculus sinister

Neuro-ophthalmic examination								STT (mm)		IOP (mmHg)	
Menace		PLR		Palpebral		Corneal					
OD	OS	OD	OS	OD	OS	OD	OS	OD	OS	OD	OS
-	-	-	-	+	+	+	+	20	22	15	20

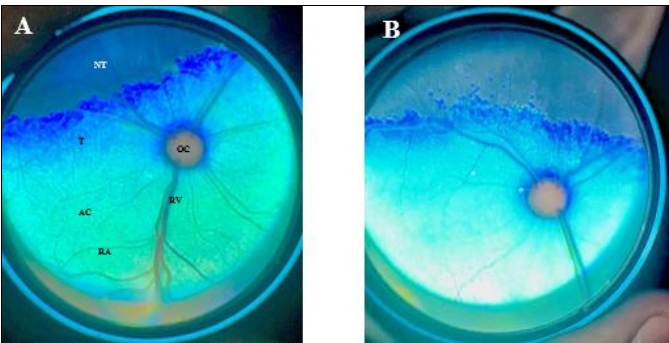


Fig 2: Fundoscopic image (A&B) showing hyper-reflective tapetum. “T” Tapetum; “NT” Non-Tapetum; “RV” Retinal Vein; “RA” Retinal Arteries; “OC” Optic Cup; “AC” Area Centralis

Fundoscopic examination of the cat showed a hyperreflective tapetum with normal retinal vasculature and no visible abnormality of the optic disc (Fig. 2). In contrast, more pronounced retinal changes have been described in other reports of ivermectin toxicosis. Two affected dogs presented with blindness and mild obtundation, and ophthalmic examination revealed retinal edema and retinal folds (Kenney *et al.*, 2008)^[3]. Fundoscopy in those cases demonstrated multifocal fold-like grey opacities in the non-tapetal retina and linear dark lesions within the tapetal region. Small multifocal bullous retinal detachments were also noted dorsolateral to each optic disc, while the optic nerve heads and retinal vasculature remained normal (Arad *et al.*, 2025)^[8]. Parenteral fluid therapy with an isotonic crystalloid solution (0.9% normal saline) was administered for three consecutive days. Cefotaxime was given at a dose of 25 mg/kg body weight intravenously twice daily for five days. Diazepam was administered intramuscularly at 0.5 mg/kg body weight as a single dose. Dexamethasone was given intravenously at 0.5 mg/kg body weight for three days. In addition, vitamin C, vitamin B12, folic acid, and nicotinamide were administered intravenously at a dose of 0.5 ml for five days. Intravenous lipid emulsion (20%) therapy was advised as part of the treatment protocol at a dose of 1.5 ml/kg body weight administered over 10 minutes, followed by a continuous rate infusion (CRI) of 0.25 ml/kg for 30 minutes once daily for three days, as previously described for the management of ivermectin toxicosis in cats (Rajpurohit *et al.*, 2024)^[9]. However, the owner did not present the animal for further

treatment, and therefore lipid emulsion therapy and follow-up evaluation could not be performed.

Conclusion

This case highlights the occurrence of acute neurological and ophthalmic manifestations associated with suspected ivermectin toxicosis in a young cat. The clinical findings of tremors, obtundation, blindness, and fixed mydriasis with absent menace, dazzle, and pupillary light reflexes were consistent with neurotoxic effects. Despite these functional deficits, fundoscopic examination revealed no structural abnormalities of the retina or optic disc. Early recognition of these clinical signs is important for the diagnosis and management of ivermectin toxicosis in cats.

Conflict of Interest

Not available

Financial Support

Not available

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