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Intravenous Lipid Emulsion for the Treatment of Ivermectin Toxicity in a Cat: A Case Report

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Authors' contributions

This work was carried out in collaboration among all authors. All authors read and approved the final manuscript.

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Case Report

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ABSTRACT

A two-years-old male cat weighing three kg was presented to the Referral Veterinary Polyclinic of ICAR – Indian Veterinary Research Institute (IVRI), Izatnagar with history of anorexia, no water intake, ataxia, head tilting downward, sudden recumbency and loss of vision. While taking history the owner declared about the use of off-label oral ivermectin preparation @ 2.5 mg/kg total dose (5 times the recommended dose) once only. Cat had high rectal temperature (105° F), normal mucus membrane, tachypnoea (70/min), tachycardia(145/min). At the beginning, the patient received 20% lipid emulsion (Intralipid) at 1.5 mL/kg body weight, Diazepam 0.5 mg/kg b. wt. IV once,

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dexamethasone 0.5 mg/kg b. wt., and 25 mg/kg b. wt. bid of cefotaxime. The patient also received constant rate infusion therapy at 0.25 ml/kg/h, OD, and isotonic crystalloid solution (0.9% NS) at 30 ml/kg body weight for a total of 10 minutes.
After three days, the cat's clinical condition returned to normal state. This case reports helps to readers in managing an emergency condition of ivermectin toxicity.

Keywords: Ivermectin; mydriasis; lipid emulsion therapy; veterinary toxicology.

1. INTRODUCTION

Ivermectin is the 22, 23-dihydro derivative of avermectin B_1, a macrocyclic lactone produced by an actinomycete, *Streptomyces avermitilis*. It is active at extremely low dosage against a wide variety of nematode and arthropod parasites, apparently by virtue of its action on the mediation of neurotransmission by γ-aminobutyric acid [1]. In mammals, these drugs have a wide margin of safety when the blood-brain barrier is intact or when an appropriate dose is used [2]. In both nematodes and insects, ivermectin functions as a positive allosteric modulator of glutamate-gated chloride channels. It binds to receptors on these channels, opening them to an influx of chloride ions that causes flaccid paralysis [3]. Ivermectin and its analogue also modulate other ion channels and have effects on the mammalian host brain when the blood–brain barrier is impaired [4]. Ivermectin intoxication is quite well documented in dogs [5] and only a few reports of ivermectin intoxication in cats have been reported [6] ivermectin toxicity can occur when excessive doses are administered (above 500 µg/kg in cats). Symptoms of ivermectin toxicosis are mainly neurological and include mydriasis, blindness, ataxia, tremors, disorientation, and mentation changes ranging from depression to coma [7].

2. HISTORY AND OBSERVATION

A two-year-old male cat weighing three kg was presented to the Referral Veterinary Polyclinic of ICAR – Indian Veterinary Research Institute (IVRI), Izatnagar with history of anorexia, no water intake, ataxia, head tilting downword, sudden recumbency (Fig. 1) and loss of vision. While taking history owner declared about the use of off-label oral ivermectin preparation @ 2.5 mg/kg total dose (5 times the recommended dose) once only. It had a high rectal temperature (105° F), normal mucus membrane, tachypnoea (70/min), tachycardia (145/min), mydriasis, no menace reflex and no pupillary light reflex (Fig. 2), according to the clinical examination.

Fig. 1. Cat in lateral recumbency

Fig. 2. Absence of menace reflex and PLR

Table 1. Hematological parameter

Parameter	Before treatment	After treatment
Hb gm%	11.3	10.4
PCV %	35.3	32.7
TEC 106/ul	4.3	4.9
Total leucocyte count 10^3/ul	**47.8**	**11.2**
Neutrophil %	**82**	**60**
Lymphocyte %	11	25
Platelets 10^3/ul	520	457

3. RESULTS

Cat treated with normal saline, Inj. 20% lipid emulsion (intralipid Fig. 3) @1.5 ml/kg b.wt over 10min followed by CRI @ .25ml/kg for 30 min OD for 3 days, Inj. meloxicam 0.2 ml through IV, Inj. Cefotaxime @25 mg/kg b.wt through Intravenous route twice in a day for 5 days, administration of vitamin c, B_{12}, folic acid and

nicotinamide 0.5ml via Intravenous route for 5 days, Inj. Dexamethasone 0.5mg/kg Intravenously b.wt for 3 days, Inj. Diazepam 0.5 mg/kg b. wt through intramuscular route once. At re-evaluation on next day, the kitten's rectal temperature was 101.6 ˚F, heart rate was 110 bpm, and respiration rate was normal. The owner reported that the animal was eating and walking normally. Mild ataxia was observed during the clinical assessment (less than the previous day). Nonetheless, there was positive PLR in addition to the threat reaction. Parenteral administration of isotonic crystalloid solution (0.9% normal saline) was continued for two days without altering the course of treatment due to the notable improvement in the clinical symptoms. The cat was acting normally on the third day following the presentation; it was lively and playful, responding to outside stimuli, and there had been no additional reports of ataxia or blindness.

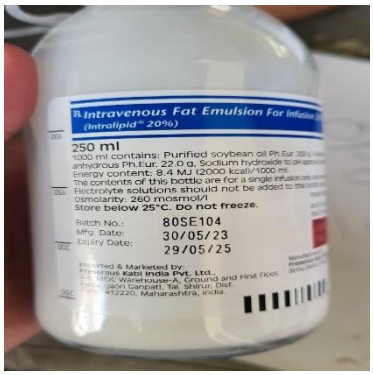

Fig. 3. Intralipid

4. DISCUSSION

There is no specific treatment for this type of toxicosis. Hence, intravenous administration of lipids is a relatively new approach in managing toxicity from lipophilic compounds. Intra Lipid Emulsion (ILE) is increasingly being used in both human and veterinary toxicology as a treatment for intoxication with lipid-soluble drugs. There are two main theories regarding the possible mechanisms of action of Intra lipid emulsion. The first theory suggests that the lipid, in the form of ILE, serves as an energy substrate for the heart. In instances of cardiovascular collapse due to substances like local anaesthetic agents, the enhanced supply of myocardial energy substrate can improve cardiac performance. The second, and more widely accepted theory, is known as the “lipid sink” theory. According to this theory, when supraphysiologic doses of ILE are administered, the drug is partitioned into a lipid compartment in the bloodstream based on its lipid solubility [8]. Genetic predisposition, particularly MDR1 gene mutations, which can increase susceptibility to ivermectin toxicity. Avermectins, particularly ivermectin and abamectin, have been the subject of numerous investigations that have demonstrated their ability to cause nephrotoxicity in a variety of animals, including mice, rats, rabbits, and bats [9,10,11]. The primary molecular mechanism by which avermectins cause kidney damage is lipid peroxidation, which is brought on by reactive oxygen species [12]. The liver is the main organ that catabolizes and neutralizes most toxins and drugs present in the body. These drugs or toxins may induce hepatic injury, which can escalate into complete hepatic failure and even death of the animal may also occur [13]. As discussed earlier, avermectins induce neurotoxicity by damaging the brain, which is responsible for production of reproductive hormones; therefore, they indirectly affect the reproductive system of animals as well [14].

5. CONCLUSION

As with most toxicities, prevention is the key. The recommended dose of ivermectin in cats is 200 µg/kg (less than 500 µg/kg) [15,16]. This case reinforces the need for careful consideration of drug selection and dosing in feline patients to prevent such adverse outcomes. Further researches will be needed to investigate the mode of action and the individual variation in pets animals in general will be needed.

DISCLAIMER (ARTIFICIAL INTELLIGENCE)

No AI technologies has been used during the writing or editing of this manuscript.

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COMPETING INTERESTS

Authors have declared that no competing interests exist.

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