Pyrethroid Toxicity in Cats

Despite the valiant efforts of all the major manufactures of pyrethroid based flea products for dogs to label them "DO NOT USE ON CATS" we see several dozen cases a year of owners that have applied such a product to their cat. This article is intended to be a brief overview of the clinical signs and treatment of this common toxicity. The results presented here are based on clinical experience and not the result of careful and controlled study. However, the principles involved are sound and we have had good success with the treatment plans discussed.

In general the history for cats presented with pyrethroid toxicosis is straightforward. The client will usually volunteer, sometimes with a little direct questioning, that they have applied a flea product to the cat within the last 5 days. Depending on the type of product used and the amount applied clinical signs can appear 6 hours to 3-4 days post application. Many times the client will remember the name of the product or bring the package with them. We have had several clients bring in the package after trying in vain to tear off or otherwise obliterate the warnings against using the product on cats.

The most common clinical signs that we see are generalized uncoordinated whole body muscle tremor and twitching. The cat is conscious and aware but may be hyperreflexic. These signs are distinct from a seizure, which involves loss of consciousness and a more coordinated pattern of muscular activity (i.e. all four legs extended or paddling). Other signs that may occur are drooling (particularly if the exposure is oral), vomiting, ataxia and depression. Both hyperthermia due to muscular activity and hypothermia can be observed. The severity of these signs ranges from mild hypersalivation and ear tremors to a patient that is spastic and impossible to restrain. This collection of clinical signs can be difficult to differentiate from organophosphate (OP) toxicity. A blood cholinesterase level (decreased in OP toxicity) is the best test but availability and turnaround time may make this impractical. Based on the products available to the public we have seen the incidence of OP toxicity rapidly decline as the incidence of pyrethroid toxicity increases. This change in what is available to the public coupled with the owner usually being able to provide the name of the product will rule out the possibility of OP toxicity in most cases.

Treatment is directed at preventing further absorption of the product and alleviating the affects of the toxin. There is currently no way to enhance elimination of the product from the body or block its mechanism. Most of the exposures that we see are dermal and if the initial contact is by phone the client is instructed to bathe the cat at home in a mild shampoo (Dawn dish detergent works well) and then dry the pet thoroughly to prevent chilling. Bathing at the hospital can be at presentation or after sedation depending on how severely the cat is affected. If the exposure was oral activated charcoal should be given. The patient should then have and IV catheter placed and fluids (LRS/Saline) started for supportive care.
Drug therapy is dependent on the severity of the clinical signs. Valium has been widely reported in the literature but is ineffective in many cases and can have unpredictable effects. We currently do not use Valium in the management of these cases. An easily available and more effective drug for management of the mild/moderately affected cat is Methocarbamol (Robaxin). Initially an IV bolus of 55-220 mg/kg is administered by bolusing 1/2 of the dose, waiting 1-2 minutes, then administering the remainder slow IV to effect. The half life of IV Robaxin is 1-2 hours and the drug can be given as needed every 6-8 hours as needed to control twitching with an upper limit of 330mg/kg/day. We have seen immediate and marked therapeutic effects with this protocol. The cat can be switched to oral tablets at 62-132 mg/kg/day divided BID-TID at discharge if mild tremors are still present. Fluid support is continued until vomiting has subsided and preferably until the cat is eating and drinking. Also, the patient should be watched closely for hypothermia, which can cause secondary problems and delay the excretion of the toxin.

For the more severely affected patient or when Robaxin is ineffective we have been using a Propofol drip. The patients’ fluids are run through an administration set that includes a burette. Dilute the Propofol 50/50 in the burette and give a fast drip to effect (sedated with a slight amount of twitching, not anesthesia). Start the cat at a drip rate of one drop every 2-3 seconds initially. Adjust the rate as needed so that the patient always has some twitching present. Decrease the amount of Propofol given slowly over the next 12-18 hours by decreasing the drip rate/increasing the dilution of the Propofol. Close monitoring during the entire time the patient is on Propofol is essential. As time passes and the toxin is cleared less Propofol will be needed; the key is to keep the patient twitching. Mild muscle tremor is our best benchmark for level of sedation, other parameters to monitor are body temperature, MM color, femoral pulse strength and respiratory rate. If the patient becomes hypothermic and the cardiovascular system is compromised at the level of Propofol needed to stop severe twitching, small amounts of IV Robaxin can be used concurrently. Patients that are not twitching are receiving too much Propofol and will die with little warning.

Propofol therapy, while effective, can be fatal if not monitored closely and should not be attempted without the ability to watch the patient closely and advising the client of the risks. In general most patients on a Propofol drip are weaned off in 12-18 hours. Our experience has been that mortality from pyrethroid toxicity is uncommon and that most patients recover within 24 hours.

Due to the wide availability of these products and the mistakes of the public, pyrethroid toxicity will continue to be common in small animal practices. With Robaxin and Propofol being readily available, and the only essential equipment being a burette and a trained eye, any private practitioner can effectively treat these cases.