

Permethrin is an active ingredient widely available in some over-the-counter insecticide products. Permethrin-based products are often contraindicated in cats due to an increased risk of toxicity (Sutton et al, 2007). An estimated 288 cases of feline permethrin toxicosis are reported per year (Sutton et al, 2007), one fifth of reported cases Malik et al (2010) suggests may result in fatalities. This article provides a patient care report describing the presentation and intensive nursing care provided to a feline patient with permethrin toxicosis following application of a canine permethrin-based spot on flea treatment.

Feline
 Domestic long hair
 2 years
 Female (neutered)
 3.20 kg

Reason for admission

The patient was found collapsed outside the owner's home and was presented to the hospital with cold extremities, profuse diarrhoea and active muscle tremors. The owner had applied a canine permethrin-based spot on flea treatment the previous evening.

Major body systems assessment

Following presentation to the veterinary practice, the major body systems were assessed.

Z the patient had moderate tachycardia (220 beats per minute) and weak peripheral pulses. No pulse deficits, indication of arrhythmia or audible murmurs were detected. Mucous membranes were pale pink, tacky and capillary refill time (CRT) was 2 seconds. The patient's core body temperature was 34.1°C and a subtle loss of skin turgor was determined. These findings were considered consistent with marked hypothermia and a moderate degree of dehydration. The tachycardia with weak peripheral pulses and prolonged CRT observed were likely consistent with hypotension secondary to hypovolaemia (Boag and Hughes, 2005).

Z the patient's respiratory rate was 28 breaths per minute, with no increased effort or abnormalities detected on thoracic auscultation.

Z at presentation the patient demonstrated a decreased level of consciousness and response to stimuli (obtunded). The patient was non-ambulatory and recumbent, deep pain sensation was present and frequent muscle tremors were observed. Pupil size, position, pupillary responses were normal and menace response was present. Interpretation of

- z Hypovolaemia with secondary hypotension
- z Hypothermia
- z Permethrin exposure
- z Muscle tremors
- z Recumbent and non ambulatory.

Aims of intensive nursing care

The aims of intensive nursing care were identified as:

- z Replacement of circulating volume deficit and restoration of systemic blood pressure
- z Normothermia
- z Prevent further permethrin absorption
- z Maintain patient comfort and prevent complications associated with prolonged recumbency.

Initial management/veterinary interventions

A 22 g intravenous catheter (Jelco, Smiths Medical) was placed in the left cephalic vein. Fluid therapy was initiated at a rate of 60 ml/kg for the first hour, using an isotonic crystalloid solution (compound sodium lactate (Aquapharm No11, Animalcare Ltd)). Fluid therapy was administered at incremental doses of 10 ml/kg given over 10 minute intervals. Fluid therapy was administered to clinical effect and evaluated by frequent physical examination to end points of resuscitation, as suggested by Brown and Otto (2008). End points of resuscitation were determined as improvement in mucous membrane colour and capillary refill time, restoration of pulse quality, reduction in heart rate and improvement in patient mentation. Methocarbamol (Robaxin-V; Fort Dodge) 40 mg/kg was crushed with saline and administered rectally, diazepam (Hamelin Pharmaceutical Ltd) 1mg/kg was administered intravenously; both were administered to reduce muscle tremors through musculoskeletal relaxation.

Discussion

Permethrin is a Type 1 pyrethroid, a synthetic derivative of natural pyrethrins. Permethrin is an active ingredient in some over-the-counter insecticide products (Table 1). Permethrin is considered a neurotoxin (Sutton et al, 2007). Following application permethrin binds to sodium channels in the nerve axon, causing these channels to remain open for longer than normal. This results in changes of sodium movement across the membrane producing repetitive discharge of the nerves, which gives rise to the clinical signs of seizures or tremors observed in permethrin toxicosis (Schleier III and Peterson, 2011). Permethrins are lipophilic compounds (Boland and Angles, 2010) which are metabolised by glucouronide conjugation by the liver (Anadon et al, 2009). Dymond and Swift (2008) suggest cats are more likely than dogs to present with permethrin toxicosis because the feline

liver has limited amounts of glucouronide transferase enzyme necessary for permethrin metabolism by glucouronide conjugation. Sutton et al (2007) predicted, based on a 3 month prospective study of 8 Affenpinschers, that cats have limited amounts of glucouronide transferase enzyme necessary for permethrin metabolism by glucouronide conjugation.

Based on the above information, the authors suggest that the following interventions should be considered:

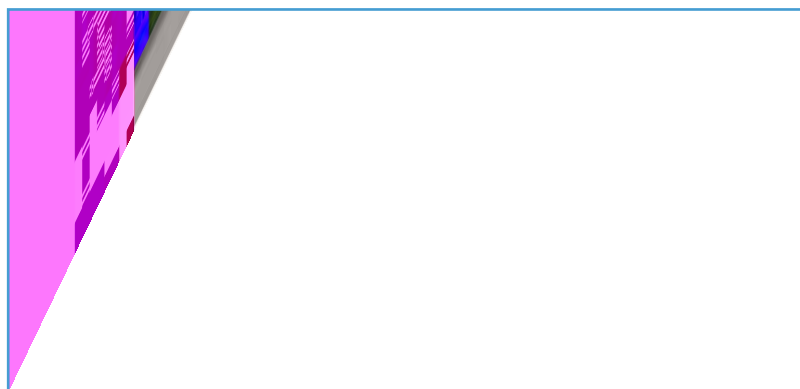
1. Fluid therapy

2. Normothermia

hypothalamus stimulates peripheral vasoconstriction to conserve core body temperature (Oncken et al, 2001) with consequential hypotension and poor peripheral perfusion (Kirby, 2004). Boland and Angles (2010) also suggest that hypothermia could increase permethrin activity at the sodium channels, which may exacerbate the adverse effects of permethrin toxicosis, although an internal body temperature below 25°C may be necessary to produce such effects. Re-warming was initiated using a paediatric incubator set to 32°C and 50–60% humidity, and core body temperature was monitored using an indwelling rectal probe. Armstrong et al (2005) advocate the use of a forced air warming blanket (i.e. Bair Hugger; Advanced Anaesthesia Specialists UK Ltd) to provide active surface warming and minimise heat loss. Although, a Bair Hugger was available it was decided that patient placement in the incubator was preferable. This enabled constant observation of the patient which may have been restricted if the Bair Hugger had been used. Core body temperature monitoring was continued beyond achievement of normothermia as repetitive and vigorous muscular activity due to prolonged or severe muscle tremors, which could also impair adequate ventilation, may predispose the patient to hyperthermia, hypercapnia, hypoxemia and metabolic acidosis (due to lactic acid release) which without intervention could produce central nervous damage (Dymond and Swift, 2008; Anadon et al, 2009).

A number of authors recommend dermal decontamination to minimise further exposure to permethrin (Sutton et al, 2007; Dymond and Swift, 2008; Linnett, 2008; Anadon et al, 2009); it is advocated that luke-warm water is used as hot water could cause dilation of the peripheral vessels and potentially increase permethrin absorption. Anadon et al (2009) and Sutton et al (2007) further recommend the use of a mild detergent to aid dermal decontamination as permethrin is a lipophilic compound insoluble in water. Due to the patient's hypothermic state the patient was bathed only at the site of application using luke-warm water and a mild detergent. The patient was dried thoroughly to prevent further heat loss through evaporation. An Elizabethan collar was placed to prevent self grooming to minimise the risk of secondary exposure through oral absorption.

As the patient was initially non ambulatory and recumbent, padded and absorbent bedding was provided to optimise patient comfort and minimise soiling in the event of urination. The patient was placed on a VetBed (Petlife International Ltd) to ensure provision of padded bedding and enable any urine passed to wick away from the patient. Patient bedding was checked at regular intervals to ensure any urine or faeces passed were removed to prevent urine and faecal scalding and minimise



moisture contact with the patient's skin. The patient was turned every 4

hours

to ensure adequate ventilation and to prevent

Adverse Reaction Surveillance Scheme administrated by the Veterinary Medicines Directorate or Veterinary Poisons Information Service.

Case reflection

Within the author's practice this case represents one of six cases presented within a 4-week period. Five of these cases were related to owner error and one was as a result of ingestion of a permethrin-based cat flea collar. Patient presentation, level of veterinary intervention and intensive nursing care required was variable. One patient exhibited minimal muscle tremors and was treated on an outpatient basis. Three patients, including the case discussed, required intensive nursing care and observation for a 48-hour period. One patient was presented with severe muscle tremors and hyperthermia, sedation using

Recommendations for practice

Despite current cautionary labelling (*Figure 1*) as suggested by Sutton et al (2007) an estimated 288 cases of permethrin toxicosis are reported per year. With one fifth of cases reported resulting in fatalities (Malik et al, 2010). However, these figures are presumptive and the true number of cases presented is likely to exceed this figure. Permethrin-based products are often sold in over-the-counter presentations and as such may lack appropriate professional guidance with regard to correct use and application. The author suggests that there is a need to raise awareness of the potential of life-threatening complications associated with permethrin exposure in cats. In the patient described, and in other cases of feline permethrin toxicosis presented to the author's practice, the suspected adverse reactions observed were not reported. The author considers that in future cases and as a profession, active reporting of suspected adverse reactions to permethrin-based products should be encouraged to enable quantification and evaluation to the true extent of permethrin toxicosis and potentiate intervention as appropriate. Suspected adverse reactions may be reported to the product manufacturer; the Suspected