Intensive nursing care provided to a patient with permethrin toxicosis

Abstract

This article describes and evaluates the intensive nursing care provided to a feline patient with permethrin toxicosis. Permethrin is an active ingredient in some over-the-counter insecticide products. Cats are particularly sensitive to permethrin toxicosis due to a deficiency of glucouronide transferase enzyme which is necessary for permethrin metabolism. Nursing interventions should include provision of supportive care to maintain hydration, normovolaemia, normothermia and patient comfort. Recommendations for future practice include increasing awareness of the potential life-threatening complications of permethrin toxicosis and active reporting of suspected adverse reactions to enable effective monitoring and intervention as appropriate.

Key words: permethrin, cat, toxicosis, nursing care, suspected adverse reactions

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.

Species: Feline
Breed: Domestic long hair
Age: 2 years
Sex: Female (neutered)
Weight: 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.

Emma Tompkins GradDipVN RVN is Veterinary Nurse/ Laboratory Technician, The Wylie Veterinary Centre, Upminster, Essex. UK

Major body systems assessment

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

- **Cardiovascular system:** 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 34.1ºC 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).
- **Respiratory system:** the patient's respiratory rate was 28 breaths per minute, with no increased effort or abnormalities detected on thoracic auscultation.
- **Neurological system:** 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 neurological function should cross reference both assessment of the cardiovascular and respiratory systems as alteration in the patient's mental state could be related to inadequate perfusion of the brain rather than a primary neurological condition (Aldrich, 2007).

Veterinary investigations

A blood sample was obtained to provide an emergency database, which enabled evaluation of the patient's clinical status and provided baseline parameters. This included measurement of haematological and biochemical parameters. The emergency database demonstrated a moderate leukocytosis (white blood cell count 28.48 x 10^9/l (5.0–18.0 x 10^9/l)) with neutrophilia and lymphopenia; results were consistent with a cortisol-mediated stress leukogram (Gilor and Gilor, 2011). All other parameters were within reference range.

Problem list

After initial patient assessment a medical problem list was generated to enable prioritisation of patient care:

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

Aims of intensive nursing care

The aims of intensive nursing care were identified as:

- Replacement of circulating volume deficit and restoration of systemic blood pressure
- Normothermia
- Prevent further permethrin absorption
- 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) 1 mg/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 reported cases of permethrin toxicosis to the Veterinary Poisons Information Service (VPIS) London and assuming that reports remained consistent, that 288 cases of permethrin toxicosis would be reported per annum. These figures are presumptive, and true figures may be under represented. Malik et al (2010) conducted a study of 750 reported cases of permethrin toxicosis to the Australian Pesticides and Veterinary Medicines Authority (APVMA) over 2 years, 22% of which resulted in fatalities. Potential routes of feline exposure include direct application of a permethrin-based product authorised in dogs and ingestion of permethrin-based product, for example contact or grooming of a dog within the household who has had recent application of a permethrin-based product.

When fluid is lost from the extracellular space, for example with gastrointestinal losses (profuse diarrhoea), fluid movement occurs from the intravascular space as a compensatory response to maintain an effective circulating volume (DiBartola, 2012). Further compensatory responses are initiated by stimulation of the sympathetic nervous system including peripheral vasoconstriction and an increase in heart rate to attempt to restore vascular volume and blood pressure therefore improve perfusion (Welsh and Girling, 2004). Dehydration secondary to profuse diarrhoea can cause significant fluid loss from the extracellular space; subsequently hypovolaemia with secondary hypotension may be observed (Brown and Otto, 2008). The therapeutic goal of fluid therapy in this patient was to re-establish normal circulating volume and therefore arterial blood pressure (Boag and Hughes, 2005), rehydrate the patient to restore extravascular volume and correct whole-body fluid homeostasis. Re-establishment of arterial blood volume and homeostasis will correct local and systemic hypoperfusion.

As recommended by Pachtinger and Drobatz (2008) an isotonic crystalloid solution, compound sodium lactate, was administered which provided replacement of the intravascular volume deficit. The initial volume administered was titrated to effect, a bolus of 10 ml/kg was given over a 10 minute interval and the patient was assessed after each bolus to determine clinical improvement. A reduction in heart rate, restoration of normal mucous membrane colour and capillary refill time, improvement in pulse strength and mentation were considered consistent with improvement in circulating volume, although such variables are considered subjective. More objective assessment of parameters including central ve-

Table 1. Examples of permethrin-based products

Product name	Manufacturer	Target species
Activyl Tick Plus Spot-on-solution	MSD Animal Health	Dogs
Advantix Spot-on Solution	Bayer PLC	Dogs
Armitage Pet Care Felt Flea Collar	Armitage Pet Care	Cats
Armitage Pet Care Flea and Tick Drops	Armitage Pet Care	Dogs
Beaphar Dog Flea and Tick Drops	Beaphar UK	Dogs
Beaphar Insecticidal Dog shampoo	Beaphar UK	Dogs
Beaphar Soft Cat Flea Collar	Beaphar UK	Cats
Bob Martin Catwalk Fashion Flea Collar	Bob Martin (UK) Ltd	Cats
Bob Martin Spot on Solution	Bob Martin (UK) Ltd	Dogs
Bob Martin Permethrin 1% w/w Flea Shampoo	Bob Martin (UK) Ltd	Dogs
Canac Soft Flea Collar	Sinclair Animal and Household Care Ltd	Cats
Canovel Insecticidal Flea and Tick 1% w/w Shampoo and Conditioner	Pfizer	Dogs
Canovel Long-Acting Flea and Tick 2% w/w Cutaneous Spray Solution	Pfizer	Dogs
Defendog 2% Cutaneous Spray Solution	Virbac S.A.	Dogs
Duowin, cutaneous Spay, Solution	Virbac S.A.	Dogs
Easi-drop Flea and Tick Drops	Hyperdrug Pharmaceuticals Ltd	Dogs
Johnson's Felt Cat Flea Collar	Johnsons Veterinary Products Ltd	Cats
Johnson's Flea and Tick Drops	Johnson's Veterinary Products Ltd	Dogs
Johnson's Insecticidal Flea and Tick Drops, cutaneous solution	Johnson's Veterinary Products Ltd	Dogs
Johnson's 4Fleas Powder for Cats and Dogs 1.05% w/w Cutaneous Powder	Johnson's Veterinary Products Ltd	Cats, Dogs
Johnson's 4Fleas Shampoo	Johnson's Veterinary Products Ltd	Dogs
Vetzyme JDS 1% w/w Insecticidal Dog Shampoo	Bob Martin (UK) Ltd	Dogs
Wilko Cat Flea Collar	Sinclair Animal and Household Care Ltd	Cats
Wilko Dog Flea Drops	Sinclair Animal and Household Care Ltd	Dogs

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nous pressure (CVP), arterial blood pressure (ABP) and quantification of urine output may have been beneficial (Pachtinger and Drobatz, 2008); however the veterinary surgeon (VS) elected not to monitor these parameters during initial stabilisation. A fluid pump was used to ensure accurate administration of fluids and minimise the risk of volume overload.

After initial stabilisation, fluid therapy was continued to provide maintenance requirements and attenuate on-going losses. Dymond and Swift (2008) and Anadon et al (2009), both advocate provision of fluid therapy to maintain intravascular volume, hydration and to prevent renal tubular damage associated with myoglobin breakdown products, which could be released from damaged muscles due to prolonged or severe muscle tremors.

The patient was presented in a hypothermic state (34.1°C); in response to hypothermia the posterior

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 2 hours to prevent the development of hypostatic pneumonia, atelectasis and decubitus ulcers. Hypostatic pneumonia is caused by reduced pulmonary perfusion, commonly attributed to prolonged pressure as a result of gravity on a dependent side which decreases pulmonary blood flow and can promote atelectasis and possibly pulmonary oedema if the thin-walled pulmonary venous vasculature is compressed (Elphee, 2011). Tefend (2002) and Boland and Angles (2010) both suggest that the patient should be positioned and supported in sternal recumbency, to facilitate respiration and minimise hypostatic congestion, however, with seizure activity and frequent muscle tremors it was difficult to maintain the patient in a sternal position. It was decided that frequent turning and placement of the patient in lateral recumbency with head and neck extended, to maintain airway patency, was preferable. Decubitus ulcers can occur as a consequence of a patient's bodyweight exerting pressure on the skin over a bony prominence resulting in local or regional tissue ischaemia and necrosis (Tefend, 2002). Although, this may be considered of greater relevance in patients with a larger bodyweight than a feline patient it is an important consideration for any patient with prolonged episodes of recumbency. Friction, shearing, moisture and ischaemia may also provide further contributing factors in the formation of decubitus ulcers (Campbell and Parish, 2010). If a patient is immobile for more than 2 hours the patient may be at risk of decubitus ulcer formation (Bansal et al, 2005). Campbell and Parish (2010) consider that the intervention of repositioning a patient every 2 hours is based on anecdotal evidence and suggest further research and evaluation is necessary to provide an evidence base and determine the optimal frequency of repositioning. However, frequent patient repositioning will minimise the potential for complications associated with prolonged recumbency and pressure on a dependent side. An ocular lubricant carbomer (Gel Tears; Chauvin Pharmaceuticals Ltd) was applied every 4 hours to prevent corneal drying and ulceration (Boland and Angles, 2010).

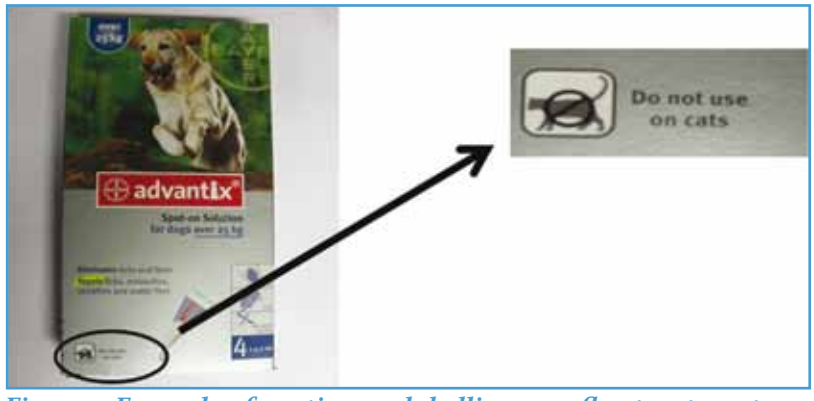

Figure 1. Example of cautionary labelling on a flea treatment.

Key points

- Permethrin-based products are often contraindicated in cats.
- Permethrin sensitivity in cats is thought to be due to a deficiency in the liver enzyme, glucouronide transferase, necessary for permethrin metabolism.
- Nursing intervention may be prolonged, and complications caused by prolonged recumbency and muscle treomors should be considered.
- Supportive care may be required to maintain hydration, normovolaemia, normothermia and patient comfort.
- Veterinary nurses can increase awareness of permethrin toxicosis.

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 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 a constant rate infusion of propofol (Vetofol, Norbrook) was necessary to control muscle tremors and reduce core body temperature, the patient was discharged 4 days after initial presentation. All patients described recovered fully and required no further treatment.

Conclusion

The requirement of intensive nursing care for patients with permethrin toxicosis should prompt veterinary nurses to consider the potential complications of prolonged recumbency and muscle tremors, and ensure provision of supportive care to maintain hydration, normovolaemia, normothermia and patient comfort. Veterinary professionals should encourage an increased public awareness of the potentially life-threatening complications of permethrin toxicosis and actively report suspected adverse reactions to enable effective monitoring and intervention. **VN**

Conflict of interest: none.

References

Aldrich J (2007) Assessment and diagnosis of shock. In: King L, Boag A, eds. *BSAVA Manual of Canine and Feline Emergency and Critical Care,* 2nd edition. British Small Animal Veterinary Association, Gloucester: Ch 3

Anadon A, Martinez-Larranaga MR, Martinez MA (2009) Use and Abuse of Pyrethrins and Synthetic Pyrethroids in Veterinary Medicine. *Vet J* **182**(1): 7–20

Armstrong SR, Roberts BK, Aronsohn M (2005) Perioperative hypothermia. *J Vet Emerg Crit Care* **15**(1): 32–7

Bansal C, Scott R, Stewart D, Cockerell CJ (2005) Decubitus Ulcers: A Review of the Literature. *Int J Dermatol* **44**(10): 805–10

Boag AK, Hughes D (2005) Assessment and Treatment of Perfusion Abnormalities in the Emergency Patient. *Vet Clin North Am Small Anim Pract* **35**: 319–42

Boland LA, Angles JM (2010) Feline Permethrin Toxicity: Retrospective Study of 42 Cases. *J Feline Med Surg* **12**(61): 61–71

Brown AJ, Otto CM (2008) Fluid Therapy in Vomiting and Diarrhea. *Vet Clin North Am Small Anim Pract* **38**(3): 653–75

Campbell C, Parish LC (2010) The decubitus ulcer: Facts and controversies. *Clinics in Dermatology* **28**(5): 527–32

DiBartola SP (2012) *Fluid, Electrolyte and Acid Base Disorders in Small Animal Practice.* 4th ed. Saunders, Missouri

Dymond NL, Swift IM (2008) Permethrin Toxicity in Cats: A Retrospective Study of 20 Cases. *Aust Vet J* **86**(6): 219–23

Elphee P (2011) Nursing of the Spinal Patient in General Practice. *Veterinary Nursing Journal* **26**(9): 305–7

Gilor S, Gilor C (2011) Common Laboratory Artifacts Caused by Inappropriate Sample Collection and Transport: How to Get the Most out of a Sample. *Top Companion Anim Med* **26** (2): 109–118

Kirby, R., 2004. The Cat is not a Small Dog in ICU: Parts I and II. *World Small Animal Veterinary Association Congress 2004.* http://www.vin.com/proceedings/Proceedings.plx?CID=WSAVA2004&PID=pro8592&O=Generic (accessed 13th January, 2013)

Linnett PJ (2008) Permethrin Toxicosis in Cats. *Aust Vet J* **86**(1-2): 32–5

Malik R, Ward MP, Seavers A et al (2010) Permethrin spot-on intoxication of cats: Literature review and survey of veterinary practitioners in Australia. *J Feline Med Surg* **12**(1): 5–14

Oncken AK, Kirby R, Rudloff E (2001) Hypothermia in Critically Ill Dogs and Cats. *Compendium* **23**(6): 506–21

Pachtinger GE, Drobatz K (2008) Assessment and Treatment of Hypovolaemic states. *Vet Clin North Am Small Anim Pract* **38**(3): 629–43

Schleier III JJ, Peterson RKD (2011) Pyrethrin and Pyrethroid Insecticides. In: Lopez O, Ernandez-Bloanos JG, eds. *Green Trends in Insect Control.* Royal Society of Chemistry Publishing, Cambridge: Ch 3

Sutton NM, Bates N, Campbell A (2007) Clinical effects and outcome of feline permethrin spot-on poisonings reported to the Veterinary Poisons Information Service (VPIS), London. *J Feline Med Surg* **9**(4): 335–9

Tefend, M., 2002. Nursing Management of Neurologic Patients. *World Small Animal Veterinary Association Congress 2002.* http://www.vin.com/proceedings/Proceedings.plx?CID=WSAVA2002&Category=&PID=13453&O=Generic (accessed 13th January, 2013)

Veterinary Medicines Directorate (2013) *Product Information Database.* http://www.vmd.defra.gov.uk/ProductInformationDatabase/ (accessed 9th January, 2013)

Welsh E, Girling SJ (2004) Fluid Therapy and Shock. In: D.Lane, B. Cooper & L.Turner, eds. *BSAVA Textbook of Veterinary Nursing, 4th edition*. British Small Animal Veterinary Association, Gloucester: Ch 19