Original Article

Journal of Feline Medicine and Surgery
2018, Vol. 20(8) 685–689
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DOI: 10.1177/1098612X17723776
journals.sagepub.com/home/jfm

This paper was handled and processed by the European Editorial Office (ISFM) for publication in *JFMS*

Pine processionary caterpillar *Thaumetopoea pityocampa* envenomation in 11 cats: a retrospective study

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Abstract

Objectives The aim of this study was to describe the clinical manifestations in cats of contact with caterpillars of the pine processionary moth.

Methods Data were retrospectively obtained from the medical records (2004–2016) of cats that had been in contact with caterpillars of the pine processionary moth.

Results Eleven cats were included in the study. The prevalence of lepidopterism was 0.13%. Tongue lesions and ptyalism were both present in 10/11 (91%) cats. Systemic signs consisted exclusively of vomiting and were encountered in 4/11 (36%) cats. The survival rate was 100%. Long-term follow-up data were available for 7/11 cats, and none of the cats showed impaired quality of life or definitive sequelae.

Conclusions and relevance The clinical presentation of lepidopterism in cats appears to be similar to that in other animals; however, the clinical signs are less severe than those previously reported, mainly owing to the cautious behaviour of this species. Moreover, the prognosis is excellent, the length of hospitalisation is short (maximum 48 h) and our study showed the absence of any long-term disability after hospital discharge.

Accepted: 6 July 2017

Introduction

Caterpillars of processionary moths are the larvae of the genus *Thaumetopoea*, and are found in Europe; close relatives are found in Australia, Asia and Africa.[1] The caterpillar of the pine (*Thaumetopoea pityocampa*) and the oak (*Thaumetopoea processionea*) processionary moths seem to be most relevant sensitisers in Europe.[1]

The caterpillar of the pine processionary moth (CPPM; Figure 1) may affect human and animal health, as even a single contact is known to provoke strong inflammatory and allergic reactions.[2] The CPPM is a widely and frequently seen species that can be found in rural and urban areas in Central and Southern Europe.[3] The natural distribution of these caterpillars has recently expanded in latitude and longitude as a result of of climate change and global warming.[4] To protect themselves against natural enemies, caterpillars of the processionary moths have evolved an urticating apparatus made by thousands of removable urticarial setae disposed dorsally and medially on the abdominal larva segments.[5] Those setae contain at least seven irritant allergens, of which thaumetopoein is the most important.[1] Urticarial hairs penetrate through prey skin by means of the proximal extremity, and the tip containing the venom breaks off and causes a cutaneous reaction in the victim, leading to envenomation.[6] Envenomation may occur when larvae

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Figure 1 Caterpillars of the pine processionary moth *Thaumetopoea pityocampa* and visualisation of their setae

leave trees in head-to-tail processions to search for pupation sites in soil.

Compared with the literature on human medicine, the veterinary literature contains few studies on lepidopterism.[2,7,8] In dogs, the most recent available CPPM study, performed by the authors of this study, describes the main clinical signs as tongue lesions that can lead to tongue necrosis, ptyalism and vomiting.[9] To our knowledge, no study describing these lesions is available in cats. However, outdoor cats may be exposed to CPPMs, and envenomation may occur.

The aim of our study was to describe, for the first time, the clinical manifestations of *T pityocampa* exposure in cats, including the time from exposure to admission, clinical signs, treatments, sequelae, outcome and long-term follow-up.

Materials and methods

Cats admitted to the intensive care unit of a veterinary hospital (SIAMU, VetAgro Sup, Campus Vétérinaire de Lyon, France) between 2004 and 2016 were retrospectively enrolled in this study. A computer-based search of the university database was conducted using the terms 'pine processionary caterpillar envenomation', 'envenomation', 'pine processionary caterpillar intoxication', 'pine processionary caterpillar', 'stomatitis' and 'glossitis'. All medical records that reported any contact with the CPPM and included clinical signs, as well as history consistent with lepidopterism, were selected. Cats were excluded if the clinical signs were not consistent with caterpillar envenomation or in cases with incomplete medical records. The recorded data included the signalment, month of presentation, time from CPPM exposure to presentation of the clinical sign, length of hospitalisation, physical examination with the description of local lesions and systemic signs or complications, laboratory results, therapeutic measures, outcome and presence of permanent injuries.

Long-term outcomes were assessed based on interviews with the owners at the time of study enrolment. The owners were asked the following questions: (1) whether necrotic lesions or changes in tongue colour remained or appeared after discharge; (2) whether the consequences of CPPM envenomation led to consultation with another veterinarian; (3) whether the cat's appetite was present despite tongue injuries; and (4) whether their cat suffered sequelae or disabilities as a result of the event.

All descriptive data are presented as median (range) and expressed as the percentage of total cats.

Results

Case selection

The medical record review initially identified 30 eligible cats admitted between 2004 and 2016. Nineteen cats were excluded because their final diagnosis was inconsistent with lepidopterism, thus leaving a total of 11 cats available for the study. During the same period, 8590 individual cats were brought to our facility. This amount equated to a prevalence of 0.13%. Seventy-two percent of these cats were male; 28% were female. Among the males, 50% were castrated; among the females, 66% were spayed. The median age at presentation was 3 years (range 5 months to 8 years), with a median weight of 3.5 kg (range 2.2–4.9 kg). Ten cats (91%) were domestic shorthairs, and the remaining animal was a Siamese. The months of presentation were April (n = 6), March (n = 2), May (n = 2) and November (n = 1). The elapsed time between the exposure and first presentation was <2 h for five cats (45%) and >24 h for the remaining six cats (55%).

Clinical signs and blood analysis

Clinical signs at admission are summarised in Table 1. The median body temperature was 39.4°C (range 38.1–40.5°C). Biochemistry panels were performed in five cats, and the results were unremarkable for all cats.

Treatment

All cats received an oral flushing at admission. Eight cats (73%) received a solution of sodium bicarbonate diluted in an isotonic sterile saline solution (ratio 1:1), and the remaining three (27%) cats received an isotonic saline solution. Hydropulsion with an eye-cleaning solution (Ocryl; TVM) was performed in all cats for removal of potentially present setae in the eyes. Ocular lesions were assessed by a fluorescein test (Fluorescein 0.5% collyre; TVM). No ocular lesions were observed. Based on pain scores, analgesia was individually managed with opiates and administered in 4/11 (36%) cats. Corticosteroids were administered in 10 cats (91%) with dexamethasone (Dexadreson; Intervet) at 0.1 mg/kg IV (8/11; 73%) or methylprednisolone

Table 1 Clinical signs at admission for the 11 cats

Clinical signs	n (%)
Tongue lesions	10 (91)
Oedema	10 (91)
Ulcerations	4 (36)
Vesicular lesions	3 (27)
Change in colour	1 (9)
Necrosis	1 (9)
Other	
Labial oedema	1 (9)
Gingival oedema	3 (27)
Facial oedema	1 (9)
Cutaneous erythema	1 (9)
Ocular lesions	0 (0)
Systemic signs	
Ptyalism	10 (91)
Vomiting	4 (36)

(Solumedrol; Pfizer) at 0.5 mg/kg IV (2/11; 18%), once daily for both. Broad-spectrum antibiotics were added to the therapeutic plan in six cats (55%). Amoxicillin-clavulanic acid (Augmentin; GlaxoSmithKline) was used in five cats (83%) and metronidazole (Flagyl; Sanofi-Aventis) in one (17%). One cat received low-molecular-weight heparin therapy (nadroparin calcium [Fraxiparine; Aspen France]).

Hospitalisation data

After the initial evaluation, two (18%) cats were discharged immediately after admission because of financial concerns. Nine cats (82%) required hospitalisation ranging from 12–48 h. All hospitalised cats (9/11) received intravenous fluid therapy. During hospitalisation, information regarding food intake was available for six cats. Anorexia was reported for only one cat, which presented 96 h after CPPM contact and required assisted nutrition (oesophagostomy feeding tube) owing to the presence of painful tongue necrosis.

Outcome

The overall survival rate was 100%. The response rate for the survey was 64% (7/11). At the time of inclusion, the long-term follow-up period varied from 4 months to 12 years. Only one cat (1/7; 14%) was described with persistent tongue necrosis; this was the only cat that presented with necrotic lesions and required assisted nutrition during hospitalisation. According to the owner's statement, the lesions did not impair the cat's ability to eat or drink at home. None of the cats required further consultation after discharge.

Discussion

To the best of our knowledge, this study is the first to report on CPPM exposure in cats. Commonly described as less curious and exploring their environment in a manner different from that of dogs, outdoor cats may also be exposed to CPPMs. The incidence of CPPM contact in cats was 0.13% in this study. In comparison, the prevalence of CPPM contact in dogs in the same institution was 0.57%. Compared with the number of dogs presented for CPPM exposure, cats were presented infrequently. A possible explanation for this is that the CPPM envenomations are less likely to be witnessed as cats often travel outside without supervision.[10] No available study has reported the prevalence of CPPM contact in dogs or cats in the literature.

Pine processionary moth envenomation affects humans and animals, and across species, the young are particularly vulnerable because of their natural curiosity and playfulness.[2,11] In cats, the age range seems to be wider than that in other species, and affected cats seem to be older than affected dogs.[2,9] Males seem to be over-represented in our study. This over-representation of males was not found in dogs for CPPM contact,[9] or in other envenomation in cats,[8,12] but the small sample size in this study should not be over-interpreted.

The highest risks of CPPM exposure occur from the end of winter to spring, when mature larvae form processions on the ground to search for food and pupation sites in the soil. As previously described in dogs,[1,6,7] most of the cats in the present study were presented in March and April. Only one cat presented clinical signs in November, and two cats showed clinical signs in May. The presence of different species of processionary moths in our area may explain the presentation of those cats for exposure to CPPMs out of the classical range. In the region local to this study, *T pityocampa* is the main species of processionary moth (Figure 1), and forms processions from November to March.[11] However, *T processionea*, commonly known as the oak processionary caterpillar, is also present in this area and forms processions from May to July.[13] This expanded time frame of presentation was already found in a similar study published in dogs by the same authors.[9] In this context, CPPM contact should not be excluded in cats if the signs are consistent with such exposure, even if the clinical signs develop in a period of the year when exposure is not common.

In our study, only one cat presented signs of anorexia secondary to tongue necrosis and required an oesophagostomy tube; all other cats managed to eat normally during and after hospitalisation. Available data in dogs suggest that tongue necrosis is more frequent in dogs than in cats.[9] The authors of the present study previously showed in dogs that elapsed time between CPPM contact and first oral flushing was a key determinant for the progression of necrotic lesions, and the best results were observed when flushing occurred within 6 h of contact.[9] Dogs with delayed oral flushing had significantly more tongue necrosis at admission and developed more tongue necrosis during hospitalisation than other dogs.[9] In the present study, 55%

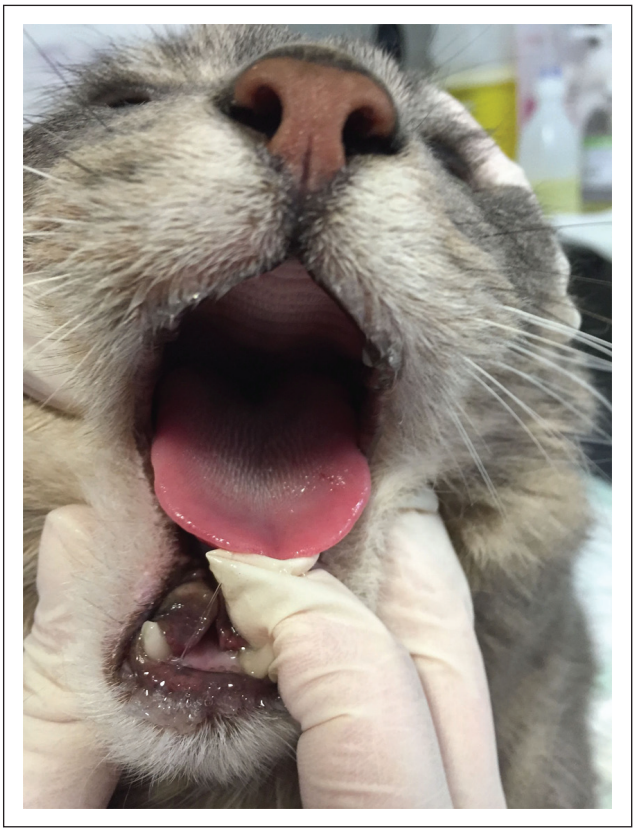

Figure 2 Tongue oedema and ptyalism in a cat at admission 1 h after exposure to the caterpillar of the pine processionary moth

of the cats were presented more than 24 h after CPPM contact, and only one cat had tongue necrosis. This is expected based on differences in behaviour: a cat tends to play or antagonise its prey, whereas a dog is more likely to explore with its nose and tongue.[10]

In our study, oral lesions, such as lingual and gingival oedema (Figure 2), ulcerations and vesicular abrasions were the main clinical features reported at admission following CPPM exposure. The clinical signs of CPPM envenomation result from mechanical irritation induced by active contact through sniffing, licking or eating caterpillars and the subsequent release of thaumetopoein from the removable setae, the specialised device from which caterpillar venoms is injected.[1] Reported in 91% of cases, ptyalism might be the result of the reaction to irritant allergens, the consequence of lingual oedema and/or of pain. While direct contact is a common form of exposure, hairs may also be windblown and lead to passive exposure of the eyes and skin.[3] Indeed, keratitis with corneal cellular infiltrates, anterior uveitis, conjunctival hyperaemia and corneal ulcers have been previously reported in dogs.[7] The absence of ocular lesions may be related to our standard admission protocol, including eye flushing and care that may have reduced the onset of delayed lesions. This result could also be related to the different behaviour of cats and dogs. For example, cats are more fastidious than dogs in terms of grooming and therefore buccal exposures are more likely than ocular exposure in this species. The last explanation could be the difference in cat's eye anatomy and the presence of the protective mobile third eyelid. This difference of lesion localisation between dogs and cats has already been observed in other envenomations.[10,12] While no ocular lesions were reported in the present study, the authors advise conducting a full ophthalmological examination at admission, based on available data in humans and dogs.

In this study, only a few systemic signs were reported, and they were not severe (vomiting [36%] and ptyalism [91%]). In dogs, systemic signs have been reported in up to 55% of affected animals, and these signs range from vomiting and dyspnoea to hypovolaemia and death.[2,8,9] Even if severe systemic signs were not described in cats in our study, clinicians should be aware of all possible systemic complications when managing these patients. Once again, this difference could be a result of the cautious behaviour of cats and to the small number of patients in this study. Another explanation is that cats will often hide when injured, so the most affected cats may not return home for the owners to obtain treatment options.[10]

The management of cats after CPPM contact is entirely symptomatic and supportive and involves elimination of the urticating hairs and allergic reaction control. The clinical relevance of mucosal and skin flushing has been noted in previous reports and appears to be the cornerstone to preventing necrosis, especially in the tongue.[2,9] In the present study, 91% of cats received intravenous steroids at anti-inflammatory doses in their therapeutic plans. While some of our patients received dexamethasone and others received methylprednisolone, no study has been able to demonstrate any superiority of one treatment over the other in the management of CPPM envenomation. While additional studies are required, steroid administration may be considered if needed to decrease inflammation.[1] In our study, antihistaminic drugs were not administered in any cat, but antihistamines can be useful in reducing thaumetopoein-induced mast-cell degranulation.[3] As no guidelines exist for the use of antihistaminic drugs, this result is more a clinician-related decision in our institution, but antihistaminic drugs could be useful in addition to corticosteroids therapy.

In our population, all cats received pain medication, as it has been established that pain may slow healing and limit recovery and food intake.[8] In this study, the administration of broad-spectrum antibiotics was performed at the clinician's discretion. In fact, there are no data on whether antimicrobial treatment should be systematically used. Indeed, in the context of

antimicrobial stewardship, use of antibiotics should not be a part of the usual treatment protocol. Once the diagnosis of CPPM envenomation is clear, use of antibiotics may only be necessary in special cases, as in the presence of systemic signs, such as hyperthermia, haemodynamic impairments, and/or extensive or severe local lesions.

In the present study, the overall survival rate was 100%. Our long-term follow-up assessment confirmed that no cat developed sequelae or disability as a result of this event. This study is the first to report the outcomes of patients several months after hospitalisation. Based on these findings, the authors consider the prognosis of CPPM exposure to be excellent in cats, which is similar to that reports in other species.[2,9]

Some limitations are inherent to this study, including its retrospective nature. Although this is the first report of CPPM envenomation in cats, the included cohort was small, which limited this review to a descriptive analysis. Finally, considering the length of the study period, our management procedures may have evolved over the years.

Conclusions

The clinical features documented in this first report of CPPM envenomation in cats have similarities with those found in humans and dogs, with tongue lesions and ptyalism. However, tongue necrosis and systemic signs appear to be less frequent and pronounced in cats than in dogs, which is likely mainly owing to the cautious behaviour of cats. The prognosis of affected cats appeared excellent, with few disabilities remaining from the exposure. Further studies are needed to confirm these preliminary results in cats.

Conflict of interest The authors declared no potential conflicts of interest with respect to the research, authorship, and/or publication of this article.

Funding The authors received no financial support for the research, authorship, and/or publication of this article.

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