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CASE REPORT

Suspect severe gastroenteritis associated with ingestion of caterpillar setae fragments in a cat

K Stone,[a]* L Sullivan[b] and R Korman[a]

A 6-month-old male neutered Domestic Shorthair cat was assessed for a 24-h history of anorexia, lethargy, depressed mentation, mild abdominal pain and persistent bradycardia. Abdominal ultrasound revealed marked thickening of the gastric wall, gastric distension, free abdominal fluid, distension and corrugation of the small intestine. Full-thickness histopathological biopsy of the gastric wall demonstrated intralesional chitinous structures whose morphology (size and presence of obvious barbs) supports these structures being urticating hairs (setae). A processionary caterpillar is considered most likely as these are the most common urticating caterpillars in Australia. This is the first case of suspected severe gastroenteritis associated with the ingestion of caterpillar setae fragments in a cat.

Keywords cat; gastroenteritis; peritonitis; processionary caterpillar; setae; urticating

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Processionary caterpillars are members of the Lepidopteron family Notodontidae, subfamily Thaumetopoeidae.[1] Incidences of Australian processionary caterpillar causing adverse effects to humans and livestock have been reported since the early 1900s.[2] The most commonly recognized veterinary conditions associated with Processionary Caterpillar Setae migration is Equine Amnionitis and Fetal Loss syndrome.

Ochrogaster lunifer (Lepidoptera: Notodontidae) is found throughout the coastal and inland Australia where its larvae feed on *Acacia*, *Eucalyptus* and *Corymbia* spp. trees.[2] When pregnant mares ingest *O. lunifer* larvae or exuviae (the cast or sloughed skin of larvae), setae penetrate the gut and migrate throughout the body, ultimately causing a range of outcomes including focal mucoid placentitis, compromised foals, abortion and perinatal death.[2] In these mares, setal fragments were found at all tissue levels in the gastrointestinal tract from the mucosa to the serosa[1] and within lesions of hyperplastic serositis of the small and large intestines and within lymph nodes, livers and uteri.[1]

The morphology of processionary caterpillars' setae allows penetration of the skin or mucous membranes of animals and migration through tissues. As they migrate, setae carry bacteria into internal organs.[3] Processionary caterpillar setae are composed of chitin. Chitin degradation in mammalian tissues is primarily by chitotriosidase, a chitinase-like protein present in macrophages.[1] Presence and breakdown products of chitin stimulate a proinflammatory response in humans. Macrophage stimulation may be responsible for the mononuclear and granulomatous reactions focused around setal fragments.[1]

In dogs, clinical signs after ingestion of processionary caterpillars include tongue lesions leading to tongue necrosis, ptyalism and vomiting. Systemic signs are reported in up to 55% of affected animals, ranging from vomiting and dyspnoea to hypovolaemia and death.[3]

Clinical signs in cats appear similar to other species, with 91% of cats presenting with tongue lesions; however, a previous publication reported symptoms to be less severe with an excellent prognosis.[4]

This case report aims to describe severe gastroenteritis in a cat suspected secondary to ingestion of caterpillar setae. In this case, intralesional chitinous structures were detected on histopathology of full-thickness gastric biopsies, and the morphology, including the size and presence of obvious barbs, supports these structures being urticating hairs (setae). Processionary caterpillars are the most common urticating caterpillars in Australia. This is the first case reported in a cat causing severe systemic clinical signs and death.

Clinical features

A 6-month-old, 3.5 kg, male neuter Domestic Shorthair cat, presented to an afterhours emergency Centre with a history of anorexia and lethargy for 24 h. The cat was housed indoors with access to a large outdoor enclosure and was commonly seen to eat small lizards and geckos. Within the enclosure, there was access to non-toxic herbs and grasses. The cat was fed a commercial brand wet and dry cat food, was fully vaccinated for feline calicivirus, feline herpes virus 1 and feline panleukopenia virus. Fluralaner and moxidectin (Bravecto plus™) were used for endoparasitic and ectoparasitic prophylaxis. The cat received no other medication and no previous illnesses reported. The cat had been in the owner's possession since a kitten and lived with a litter mate that was asymptomatic.

Initial physical examination by an emergency veterinary revealed depressed mentation, mild abdominal pain and 5%–7% dehydration. A persistent bradycardia of 108 bpm (beats per minute) was present with normal cardiovascular rhythm and pulse pressure. Respiratory rate and effort were normal with no adventitious breath sounds. Venous blood gas analyses (Table 1) demonstrated mild respiratory alkalosis and mild metabolic acidosis; haematology identified a

*Corresponding author.
[a]Veterinary Specialist Services, The Centre @ Springwood, Underwood, Queensland 4119, Australia; kstone@vss.net.au
[b]QML Vetnostics, Murarrie, Queensland 4172, Australia

Table 1. Biochemistry

Biochemical parameter	Value	Reference interval
Urea, mmol/L	5.83	5.36–11.42
Creatinine, µmol/L	84	71–159
Urea/creatinine	0.069	
Ionised phosphorus, mmol/L	1.43	0.84–1.94
Calcium, mmol/L	2.19	2.20–3.00
Total protein, g/L	70	60–80
Albumin, g/L	29	23–35
Globulin, g/L	41	28–48
Alb glob ratio	0.7	
Glucose, mmol/L	7.00	3.9–7.2
TCHO, mmol/L	2.46	1.81–5.17
ALT, U/L	58	0–100
AST, U/L	44	0–50
ALP, U/L	23	0–90
GGT, U/L	<10	0–10
Total bilirubin, µmol/L	<1	0–9
Amylase, U/L	903	100–500
Lipase, U/L	20	0–60
Creatinine kinase, U/L	205	50–310
Magnesium, mmol/L	1.04	0.74–1.11
Triglycerides, mmol/L	0.46	0.17–1.19
Sodium (Na), mmol/L	149	147–156
Potassium (K), mmol/L	3.8	3.4–5.3
Chloride (Cl), mmol/L	112	107–125
Na:K ratio	39	
Estimated osmolality mOsm/kg	299 mOsm/kg	

Heska Element DC5X Chemistry analyser.

monocytopenia (0.05×10^9/L [reference interval {RI} 0.07–1.36×10^9/L]) and eosinopenia (0.02×10^9/L [RI 0.06–1.93×10^9/L]). Multiple biochemical analyses (Table 1) demonstrated a mild hypocalcaemia (2.19 mmol/L [RI 2–2.3 mmol/L]). Three-view thoracic radiographs were unremarkable and two-view abdominal radiographs (Figure 1) demonstrated gaseous distension of the small intestine and stomach. Abdominal (AFAST) and thoracic (TFAST) sonography for trauma, triage and tracking were performed. The AFAST had negative fluid scores in all four quadrants (diaphragmatic-hepatic, spleno-renal, cysto-colic and hepato-renal); TFAST was also normal; (left side and right side – wet dry/third space). Retrovirus testing for feline leukemia virus (FeLV) and feline immunodeficiency virus (FIV) was negative (Anigen Rapid [BioNote] kit).

Initial treatment by the emergency veterinarian was provided to correct hydration status; intravenous fluid therapy was commenced with Hartmann's 10 mL/kg IV bolus followed by fluid rate of 7.4 mL/kg/h for 6 h and 4.7 mL/kg/h for the next 16 h. Antinausea and analgesia were administrated (maropitant 1 mg/kg intravenously every 24 h and methadone 0.2 mg/kg intravenously every 4 h).

The cat was transferred to a specialist veterinary hospital in Underwood, Queensland, Australia for further assessment, diagnostics and treatment.

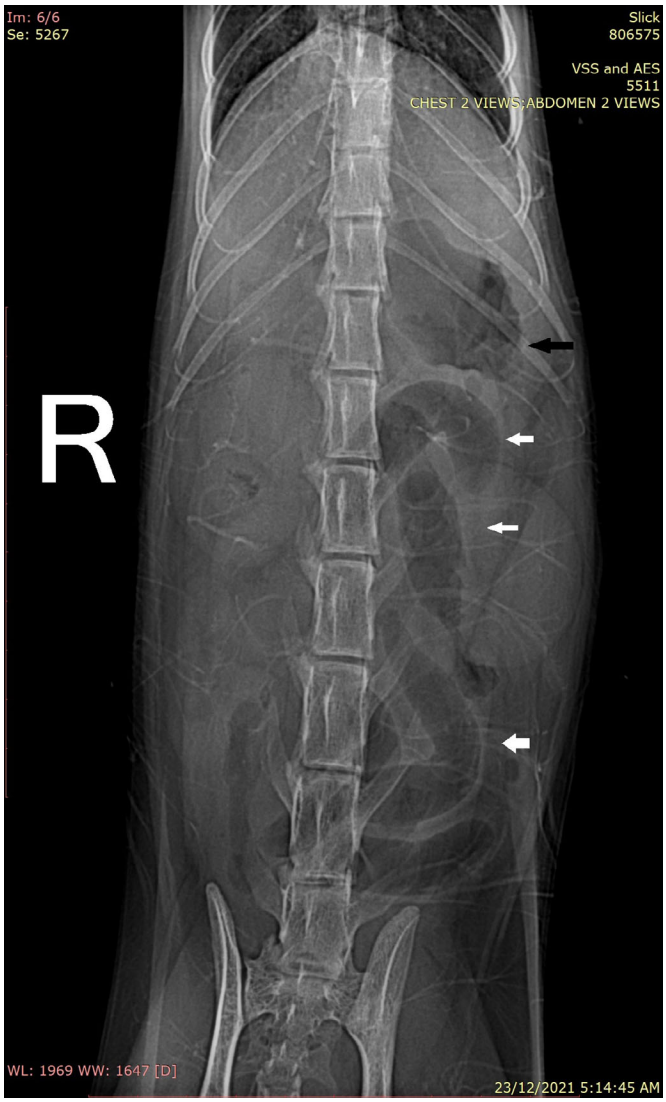

Figure 1. Dorsoventral abdominal radiographs demonstrating gastric (black arrow) and intestinal gaseous distension (white arrows).

On repeat physical examination, the cat remained bradycardic (range 108–120 bpm). An electrocardiogram (ECG) and echocardiography confirmed normal cardiac structure and function. The ECG revealed a sinus bradycardia (89 bpm), which was atropine responsive and considered due to elevated vagal tone secondary to gastrointestinal disease.

Abdominal ultrasound (Figure 2) revealed trivial abdominal fluid cranial to the urinary bladder. The gastric wall was markedly oedematous in appearance and measured 4.9 mm in diameter with mixed echogenic fluid within the gastric lumen and mild gastric distension. There was no evidence of a foreign body or pyloric outflow obstruction. The proximal duodenum had mild distension with gas drop out. Small intestinal loop diameter ranged from 6 to 9.1 mm and were distended with fluid. Intestinal plication and corrugation of the mucosa and muscularis layer were present.

Over the next 12 h, despite supportive treatment, the cat's demeanour deteriorated and he developed marked depression and projectile

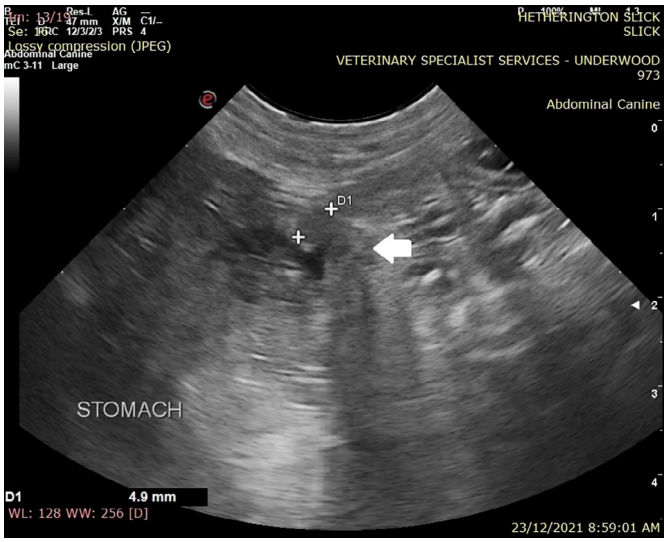

Figure 2. **Abdominal ultrasound through the stomach demonstrating marked thickening of the gastric wall (white arrow).**

bilious vomiting. Repeat abdominal sonography demonstrated marked gastric fluid accumulation and distension. An exploratory laparotomy was performed to assess for an unidentified foreign body.

A midline laparotomy was performed by a specialist surgeon and a thorough exploration of the abdominal cavity did not identify a gastrointestinal foreign body or obstruction. Markedly enlarged mesenteric and gastric lymph nodes, diffuse thickening of the gastric wall and free abdominal fluid were confirmed. Biopsy was performed of the stomach and mesenteric and gastric lymph nodes and these tissues submitted for histopathology. Lymph node tissue was submitted for bacterial and fungal culture (Table 2). Abdominal fluid was submitted for cytology (Table 3) and bacterial culture (Table 4).

Differential diagnoses for diffuse thickening of gastric wall included gastritis, chronic hyperplastic gastropathy, neoplasia (adenocarcinoma, leiomyoma/leiomyosarcoma, lymphoma) and pyogranulomatous gastritis amongst others. Differential diagnoses for abdominal lymphadenopathy were reactive hyperplasia, atypical infection (*Toxoplasma gondii*, *Mycobacterium* species, fungal disease, *Salmonella* and *Listeria species* infection), inflammatory bowel disease and lymphoma.

Postoperative management included intravenous fluid therapy with potassium supplementation, esomeprazole (1 mg/kg IV every 12 h), maropitant (1 mg/kg IV every 24 h), metoclopramide constant rate infusion (1–2 mg/kg/24 h) and metronidazole (10 mg/kg IV every 12 h). A fentanyl constant rate infusion (2–4 μg/kg/h) was provided for analgesia and a 12 μg fentanyl patch was placed after the patient was normothermic.

Peritoneal fluid analysis (Table 3) identified a neutrophilic exudate with mildly elevated numbers of inflammatory cells and low numbers of erythrocytes in a lightly proteinaceous background. A differential cell count consisting of 72% non-degenerate neutrophils, 27% macrophages and 1% small lymphocytes was present. Occasional clusters of reactive mesothelial cells were observed. No overt infectious agents or neoplastic cells were identified. Bacterial culture was negative (Table 4).

On histopathological evaluation of haematoxylin and eosin-stained sections of stomach, the submucosa was markedly oedematous. Rare golden setal fragments were identified within the lamina propria and submucosa, surrounded by an infiltrate of macrophages, neutrophils and eosinophils (Figures 3 and 4). One of these structures was transversely sectioned with barbs evident. Histopathological evaluation of

Table 2. **Microbiological examination of abdominal lymph node**

Microbiological examination	Specimen: Abdominal lymph node tissue
Gram stain	Numerous leucocytes seen No microorganisms seen
Bacterial culture	Organism: *Escherichia coli* scant
Susceptibility	
Ampicillin/Amoxycillin	Sensitive
Amoxycillin + clavulanic acid	Sensitive
Cephalexin	Sensitive
Cephalothin	Sensitive
Enrofloxacin	Sensitive
Gentamicin	Sensitive
Doxycycline	Sensitive
Trimethoprim + sulfa	Sensitive
Cefovecin	Sensitive

Performed at QML Vetnostics, 11 Riverview Place, Metroplex on Gateway, Murarrie QLD 4172 Australia.

Table 3. **Peritoneal fluid analysis**

Peritoneal fluid analysis	
White cells	5.6×10^9/L
Red cells	0.014×10^{12}/L
Protein	11.9 g/L
Colour	Slightly turbid pink fluid
Microscopic evaluation	Mildly elevated numbers of inflammatory cells and low numbers of erythrocytes in a lightly proteinaceous background. A differential cell count consists of 72% non-degenerate neutrophils, 27% macrophages and 1% small lymphocytes. Occasional clusters of reactive mesothelial cells are also observed. No overt infectious agents or neoplastic cells are identified.
Interpretation	Neutrophilic exudate
Comments	No bacterial or other infectious agents are identified cytologically, but this does not eliminate the possibility of an infectious cause.

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Table 4. Peritoneal fluid microbiological examination

Microbiological examination	Specimen: Abdominal fluid
Gram stain	A few leucocytes seen No microorganisms seen
Bacterial culture	No growth after extended incubation

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gastric and mesenteric (not further specified) lymph nodes revealed nonspecific reactive lymphoid hyperplasia, and low numbers of neutrophils and eosinophils were also identified within the nodal sinuses.

Faecal cytology (Table 5) was negative for caterpillar setae and no other gastrointestinal parasites were identified.

The diagnosis based on clinical signs and histopathology was severe gastric submucosal oedema with rare, embedded setae and associated pyogranulomatous to eosinophilic gastritis secondary to ingestion of caterpillar setae.

Postoperatively the cat had intermittent bradycardia (120–180 bpm), mild pyrexia 39.5C°, anorexia and episodes of ptyalism, nausea and regurgitation when offered food. The cat also developed marked tenesmus with mucus over the following 24 h. Repeat abdominal ultrasound demonstrated ongoing gastric ileus (thickened gastric wall [3.2 mm] with normal layering; ongoing moderate gastric distension up to 2.5 cm with anechoic luminal contents). Small intestinal layering was normal with normal wall diameter. The colon had normal wall layering and wall width of 2 mm. Within the lumen of the colon, there was heterogenous fluid and luminal distension up to 9 mm. Free abdominal fluid was absent.

A literature review of caterpillar setae migration in other species demonstrated that ingestion or contact with setae causes intense irritation and a marked inflammatory reaction occurs.[4] An anti-

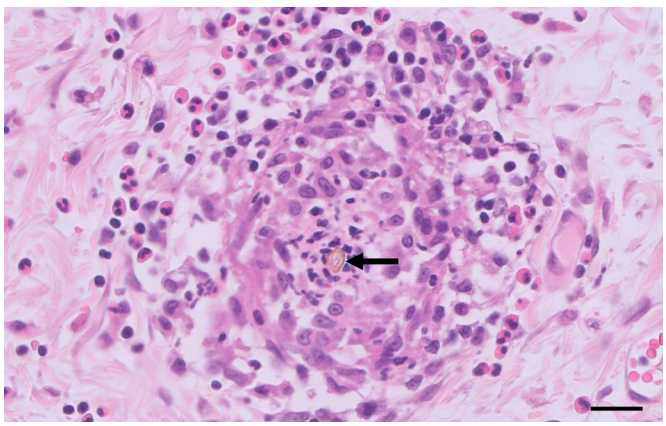

Figure 3. Histopathological findings. Rare golden and refractile setal fragments (arrow) were detected within the mucosa and submucosa, often surrounded by an inflammatory infiltrate composed predominately of macrophages, neutrophils and eosinophils. Haematoxylin and eosin, bar = 20 µm.

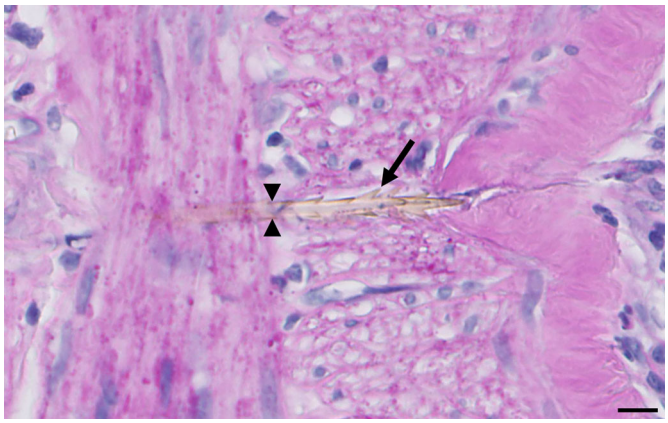

Figure 4. In some sections, the embedded setal fragments (this one delineated between the arrowheads) could be seen to have a sharp tapered end and short angled barbs (arrow) along the shaft. Haematoxylin and eosin, bar = 10 µm.

inflammatory dose of 0.06 mg/kg dexamethasone was administrated intravenously. The cat improved clinically, ate and was discharged with oral gabapentin 7 mg/kg every 12 h, maropitant 2 mg/kg every 24 h and ondansetron 0.5 mg/kg every 8–12 h.

Initially, the cat did well; however, 48 h after discharge, the cat developed lethargy and diarrhoea with tenesmus. No further vomiting was noted. Repeat abdominal ultrasound demonstrated a normal gastric wall with no luminal distension. There was hyperechoic fat surrounding the stomach and the duodenum was corrugated. The colon had a thickened wall with loss of normal layering and liquid material within the lumen (overall colonic wall diameter 5.8 mm). The mesenteric lymph nodes had an overall width of 7.8 mm with a heterogenous appearance. Treatment was commenced with amoxicillin clavulanic acid 15 mg/kg every 12 h per os for 10 days for possible antibacterial translocation across the abnormal colonic wall. It was expected that the diarrhoea would continue given the ultrasonographic appearance of the colon. The cat was discharged.

Table 5. Faecal cytology

Faecal cytology	
Microscopic evaluation	The unstained slide is examined. Parasites are not identified.
Microscopic evaluation	A diff-Quik stained faecal impression smear is examined and reveals numerous mixed but primarily rod-shaped bacterial forms and a large amount of finely granular refractile crystalline material. PAS positive structures are not identified within a PAS stained impression smear.
Comment	PAS positive material is not evident. Significant cytologic abnormalities are not identified. Finely granular refractile crystalline material is presumably of cat litter origin.

Performed at QML Vetnostics, 11 Riverview Place, Metroplex on Gateway, Murarrie QLD 4172 Australia.

Ten days later; the cat presented for acute vomiting and recurrence of diarrhoea. The cat had lost 1 kg in body weight. Physical examination revealed approximately 8% dehydration and thickened, irregular intestines on abdominal palpation. Abdominal ultrasound demonstrated flocculent free abdominal fluid with multiple prominent enlarged mesenteric lymph nodes. These were homogenous in appearance except for one that appeared cystic. Three separate regions of intestine demonstrated complete loss of layering and marked corrugation. Multiple adhesions were suspected, although an atypical intussusception was not excluded.

Repeat exploratory laparotomy was declined on financial grounds and supportive care was attempted. Over the following 24 h, the cat developed large-volume haematemesis and symptoms of cardiovascular compromise. Due to severe blood loss and lack of immediately available, appropriately typed feline blood, a xeno-transfusion of canine blood was given. The cat continued to have further haematemesis episodes and continued to deteriorate clinically and his owners elected to euthanase. Inflammation secondary to the caterpillar setae resulting in erosion through a large gastric vessel was suspected as the cause of the large-volume haematemesis and sudden severe deterioration. Postmortem examination was declined by the owners.

Discussion

To the best of our knowledge, this is the first reported case of ingestion and migration of caterpillar setae fragments in a cat reported in Australia and the first case of severe gastric oedema and irregular gastric inflammation associated with ingestion of caterpillar setal fragments resulting in severe clinical illness including gastric haemorrhage and death in a cat.

The only other study of processionary caterpillar ingestion in cats is from Europe by Pouzot-Nevoret et al in 2018. This study retrospectively reported 11 cats envenomated with the European pine processionary caterpillar *Thaumetopoea pityocampa*, a close relative to the processionary caterpillar found in Australia, Asia and Africa.[4] Tongue lesions were identified in 91% of cats. Labial, gingival, facial or cutaneous oedema in 6% and vomiting in 36% of cats. Ptyalism was identified in 91% of the reported cases.[4] Two cats in this study did not require hospitalization. Nine cats required hospitalization for 12–48 h only. One cat in the study was anorexic secondary to severe tongue necrosis. None of the 11 cats in this study required consultation after discharge.[4] The study reported an overall survival rate of 100% with follow-up ranging from 4 months to 12 years.[4]

In this case, diagnosis was based on clinical signs and histopathology of pyogranulomatous to eosinophilic gastritis secondary to ingestion of caterpillar setae, which were identified in histopathological sections at the sites of gastric inflammation.

It is postulated that the streamline shape of the setae with sharply pointed ends and angled barbs facilitate mechanical penetration through tissues as the means of setae dissemination.[5] This cat did not demonstrate tongue necrosis or ptyalism as reported in other cases and no oesophageal injury was suspected clinically. Caterpillars may curl up into a ball position to defend themselves from predators, so when ingested this may have protected the oropharynx and oesophagus from inflammation.

In the horse, peristaltic movements of the gastrointestinal tract and myometrial contractions of the gravid uterus are likely to facilitate penetration into tissues.[5]

In the gastrointestinal tract of mares, setal penetration evoked diffuse mucosal inflammation resulting in acute gastritis, duodenitis, jejunitis, ileitis, typhlitis and colitis characterized by variable numbers of neutrophils in the superficial mucosa.[5] In horses, an influx of neutrophils into the mucosal surfaces occurs most likely due to disruption of mucosal barrier from diffuse and repeated mechanical penetration of setal fragments. This then allows increased exposure of the mucosa to gut microflora and stimulation of inflammatory mediators.[5] In this case, consideration could have been given to starting antibiosis sooner given the risk of bacterial penetration by the setae and the presence of neutrophils and eosinophils within the infiltrate, rather than solely macrophages. In the study by Pouzot-Nevoret et al, 91% of cats received intravenous steroids at anti-inflammatory doses in their therapeutic plans.[4] This study had an overall survival rate of 100% and therefore consideration of the use of corticosteroids earlier given the marked inflammation of the gastric mucosa.

Information in the literature on treatment of mammalian species including humans is sparse. In people the treatment of choice for processionary caterpillar exposure is a moderately strong topical corticosteroid and oral antihistamine. For severe symptoms, systemic corticosteroids may be given. Corticosteroids, beta-2-mimetics and/or parasympatholytic sprays are indicated in patients with mucosal involvement.[6]

Most documented reactions in humans have been caused by airborne setae.[7] Understanding the distribution of airborne urticating agents such as true setae is important for managing their impact on communities. In Australia, the distribution of processionary caterpillars occurs along coastal Australia, ranging from temperate to tropical regions.[7] When processionary caterpillars leave their nest to pupate in the soil, the nest disintegrates and either the nest and/or its contents fall on to the pastures below or at the base of the tree. The shed exoskeleton of the processionary caterpillar is very light and is easily spread across paddocks by wind. It has been shown that once in the air, caterpillar setae can travel a number of kilometres from their source.[8] The wind speed and how high the caterpillar activity is in a host tree also greatly affect the setae dispersion. Modelling has shown that the highest concentration of true setae dispersing from caterpillar activity five metres high in their host tree in a slight breeze (typical night-time condition) occurs approximately 50–100 m from the source.[7]

Control measures used against processionary caterpillar may be physical, chemical or biological. Physical control can only be used successfully against the larva while it is still in the nest. The nests and the branches to which they are attached are carefully cut from the tree and burnt. Chemical control is expensive and timely involving the use of residual insecticides in infested areas. Chemical control can be effected by climatic factors, thus reducing the efficiency of the control programme. Biological controls have been used in France

and Italy and involve the use of selective insect pathogens such as *Bacilus thuringiensis* with average mortality rates of 81% and between 62.5% and 99.8% respectively.[9] An understanding of the interaction between host-plant quality and host-plant apparency on insect egg-laying behaviour can be used effectively in an effort to help with strategies for managing natural habitats and plantations for conservation and pest control. In order to reduce the destabilizing effects of high insect population growth rates, mixed planting can disrupt the host-searching efficiency of potential insect pests.[8]

More research is required to investigate the most effective method of control of the processionary caterpillar. Exposure to individual cats can be reduced by keeping cats indoors, monitoring the cat's local environment and removing/treating the nests of processionary caterpillars.

Conclusion

The incidence of caterpillar setae migration causing disease in Australian cats maybe under-recognised and underdiagnosed. This case report aims to alert clinicians to consider processionary caterpillar setae as a potential cause of clinically severe gastrointestinal illness in cats as well as other clinical signs reported in the study by Pouzot-Nevoret et al. We suggest questioning owners about caterpillar contact in cases with acute gastrointestinal illness and cats with tongue lesions, labial, gingival, facial, and cutaneous oedema and ptyalism.

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Conflicts of interest or sources of funding

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