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Abstract

Lilies are considered nephrotoxic only to domestic cats belonging to the family Felidae of the suborder Feliformia. However, a 7-month-old female meerkat belonging to the family Herpestidae of the suborder Feliformia ingested lilies and presented with oliguria, seizure, tachypnoea, self-biting, and nystagmus. The meerkat died approximately 40 h after lily ingestion. Gross and histopathologic lesions consistent with acute renal failure were conspicuous in the animal. Renal lesions were acute tubular necrosis, corresponding to typical pathological changes of lily toxicosis in cats. In addition, massive hepatocyte necrosis and pulmonary congestion/oedema were observed with focal haemorrhage. These findings suggest that lily toxicosis in meerkats is characterized by severe pulmonary and hepatic failure, in addition to the renal failure experienced by domestic cats.

Keywords	Meerkat, lily, acute tubular necrosis, acute hepatocyte necrosis
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Lily toxicosis in a meerkat (*Suricata suricatta*): A case report

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Summary

Lilies are considered nephrotoxic only to domestic cats belonging to the family Felidae of the suborder Feliformia. However, a 7-month-old female meerkat belonging to the family Herpestidae of the suborder Feliformia ingested lilies and presented with oliguria, seizure, tachypnoea, self-biting, and nystagmus. The meerkat died approximately 40 h after lily ingestion. Gross and histopathologic lesions consistent with acute renal failure were conspicuous in the animal. Renal lesions were acute tubular necrosis, corresponding to typical pathological changes of lily toxicosis in cats. In addition, massive hepatocyte necrosis and pulmonary congestion/oedema were observed with focal haemorrhage. These findings suggest that lily toxicosis in meerkats is characterized by severe pulmonary and hepatic failure, in addition to the renal failure experienced by domestic cats.

Keywords: Meerkat, lily, acute tubular necrosis, acute hepatocyte necrosis

32 The groups belonging to the genera *Lilium* and *Hemerocallis* (Easter lily, Tiger lily,
33 Rubrum, Japanese Show Lily, Stargazer Lily, Oriental Lily, and Day Lily) are
34 considered potentially nephrotoxic to cats (Bennett and Reineke, 2013; Berg *et al.*,
35 2007; Berny *et al.*, 2010; Brady and Janovitz, 2000; Cortinovis and Caloni, 2013;
36 Fitzgerald, 2010; Hadley *et al.*, 2003; Langston, 2002; Mahdi and Van der Merwe,
37 2013; Rumbeiha *et al.*, 2004; Slater and Gwaltney-Brant, 2011). Studies have shown
38 that the ingestion of the whole plant, or just one or two leaves, can be fatal for cats
39 (Fitzgerald, 2010; Rumbeiha *et al.*, 2004). However, nephrotoxic damage after the
40 ingestion of lilies cannot be observed in rodents or rabbits. In dogs, only vomiting and
41 other gastrointestinal signs can be observed after lily ingestion (Fitzgerald, 2010).
42 Further, lily toxicosis has only been reported in cats belonging to the family Felidae of
43 the suborder Feliformia. Here, we present a case of lily poisoning in a meerkat
44 belonging to the family Herpestidae of the suborder Feliformia. To our knowledge, this
45 is the first study describing the clinical and pathological findings of lily poisoning in a
46 meerkat.
47 A 7-month-old female meerkat (*Suricata suricatta*) was observed eating flowers and
48 buds of a *Lilium* 'Casa Blanca' or Oriental Lily. The meerkat presented with clinical

49 signs of vomiting, hypothermia, and tachypnoea, and the owner brought her to a
50 veterinary hospital. At the time of admission, the meerkat presented with oliguria,
51 seizures, tachypnoea, self-biting, and nystagmus. The animal was hospitalized and
52 given oxygen inhalation therapy and transfused by intravenous fluid injection. High
53 concentrations of blood urea nitrogen (BUN) (68.0 mg/dL), creatinine (2.0 mg/dL),
54 glutamic-pyruvate transaminase (GPT) (981.0 mg/dL), and creatine phosphokinase
55 (CPK) (2,036.0 mg/dL) were detected. Upon ultrasound and computerized axial
56 tomography (CAT) examination, both the kidneys and liver were larger than normal.
57 From midnight to morning, the meerkat vomited blood and presented loss of
58 consciousness. Approximately 40 h after lily ingestion, the meerkat died. During
59 necropsy, all tissues and organs were collected and fixed in 10% neutral buffered
60 formalin, embedded in paraffin wax, sectioned at 4 μm, and stained with haematoxylin
61 and eosin (HE).
62 Grossly, renal congestion and perirenal oedema were found. Pulmonary congestion,
63 liver congestion, and partial paleness were observed. Histopathologically, widespread
64 tubular degeneration and necrosis were observed in the proximal tubules of the entire
65 renal cortex (Fig. 1). Proximal convoluted tubules revealed marked granular

66 degeneration and necrosis with loss of nuclei (Fig. 2). Severe congestion was detected
67 from the deep cortex to the outer medulla (Fig. 1). However, no haemorrhage, tubule
68 regeneration, or inflammatory cell infiltration were observed. In the liver, massive
69 hepatocyte necrosis with congestion was detected (Fig. 3). Survival and necrotic
70 hepatocytes included small- to large-sized lipid droplets (Fig. 3). Neither reactive
71 inflammatory cell infiltration nor regenerative hepatocytes were present in the liver.
72 Pulmonary congestion and oedema were observed with focal haemorrhage (Fig. 4). In
73 the cerebral cortex and hippocampus, neuronal degeneration and necrosis were not
74 detected. Glial cell infiltration was also not observed.
75 In the present case, a diagnosis of lily toxicosis was made because of the direct visual
76 observation of lily ingestion, acute renal failure after lily ingestion, and acute tubular
77 necrosis corresponding to typical pathological changes of lily toxicosis in cats.
78 Cats are known to be sensitive to lily ingestion, but there is no age, sex, or breed
79 predilection (Fitzgerald, 2010). Among cats, the mortality rate from Easter Lily
80 toxicosis has been reported to be as high as 50% to 100%, depending on the initiation
81 time of symptomatic treatment. Specifically, high mortality rates are reported if
82 treatment is not initiated before the onset of acute renal failure, which occurs 18–24 h

after lily exposure (Rumbeiha *et al.*, 2004). Lily ingestion severely injures the kidney, leading initially to polyuric kidney failure, which can then lead to extreme dehydration, anuric renal failure, and eventually death (Fitzgerald, 2010). In the present case, massive hepatocyte necrosis, pulmonary congestion and oedema, and acute tubular necrosis were detected, which are compatible with the pathological findings of lily toxicosis. In addition, the meerkat also presented with seizures, which have been observed in previous feline cases of lily poisoning (Fitzgerald, 2010). Disorientation, ataxia, and head pressing have also been observed among cases of lily poisoning, but less frequently (Berg *et al.*, 2007). However, previous studies have not shown neuronal degeneration from lily toxicosis in cats. In the present case, neuronal degeneration and necrosis could not be detected in the neurons. Changes of hippocampal neurons may reflect neurologic disorders as a result of lily poisoning; however, neuronal degeneration and loss from artefact changes could not be easily distinguished in this fatality case. Therefore, the relationship between changes in the central nervous system and signs of central nerve disorders induced by lily toxicosis require further investigation to elucidate the underlying mechanisms of these neuronal disorders.

Acute tubular necrosis is not a specific diagnosis and can result from various nephrotoxins, such as ethylene glycol, boric acid, pharmaceutical drugs, or metals (Cianciolo and Mohr, 2016). Over time, acute tubular necrosis can lead to tubular regeneration, inflammatory cell infiltration, urinary cast, and other tubular degenerations, with the pathological lesions changing from an acute phase to a regenerative or chronic phase (Cianciolo and Mohr, 2016; Terayama *et al.*, 2017). Since in the present case, we only observed tubular necrosis, renal change was comparable to the acute phase. Thus, the observed renal change was undoubtedly caused by lily ingestion.

Pulmonary congestion and oedema along with coinciding lipidosis and hypertrophy of hepatocytes has been previously observed in lily toxicosis of cats (Fitzgerald, 2010). However, massive hepatocyte necrosis and focal haemorrhage of the lung have not been consistently reported in cases of lily toxicosis

Massive hepatocytic necrosis without any cellular reaction, such as inflammatory cell infiltration, suggests that these changes are consistent with the lesions at an acute stage. Therefore, in the present case, the meerkat presented with severe pulmonary and hepatic failure in addition to the renal failure typically experienced by domestic cats. The exact

mode of action and exact toxic substance of lily poisoning remain unidentified. The rapid onset of clinical signs after lily ingestion indicates fast absorption and action of the poison. The metabolism of drugs in cats could be different from that in other species, such as dogs, mice, rats, and rabbits; thus, a feline-specific toxic metabolite may produce different effects (Fitzgerald, 2010). The aqueous extracts of lily leaves and flowers have been shown to be nephrotoxic, with the aqueous floral extracts containing most of toxic compound (Rumbeiha *et al.*, 2004). In the present case, since the meerkat ingested the flower buds containing high levels of the toxic compound, the rapid progression of the clinical symptoms and eventual death are likely attributed to the ingestion of the lily.

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Conflict of Interest Statement

The authors declare no conflicts of interest with respect to the publication of this manuscript.

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170

171 **Figure legends**

172 **Figure 1**. Kidney. Severe tubular degeneration and necrosis are presented in in the renal

173 cortex. Severe congestion is shown in the deep cortex to the outer medulla.

174 Haematoxylin and eosin (HE). Bar, 500 μm.

175 **Figure 2**. Kidney. Proximal convoluted tubules reveal marked granular degeneration

176 and necrosis with lost nuclei. HE. Bar, 100 μm.

177 **Figure 3**. Liver. Severe hepatocyte necrosis is seen with congestion. Hepatocytes

178 include small- to large-sized lipid droplets. HE. Bar, 10 μm.

179 **Figure 4**. Lung. Pulmonary congestion and oedema are observed with focal

180 haemorrhage. HE. Bar, 10 μm.

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