Suspected lily toxicosis in a meerkat (Suricata suricatta): A case report

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Abstract

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

Keywords: acute hepatocyte necrosis, acute tubular necrosis, lily, meerkat
The groups belonging to the genera *Lilium* and *Hemerocallis* (Easter lily, Tiger lily, Rubrum, Japanese Show Lily, Stargazer Lily, Oriental Lily, and Day Lily) are considered potentially nephrotoxic to cats [1-4, 6-12]. Studies have shown that the ingestion of the whole plant, or just one or two leaves, can be fatal for cats [7, 11]. However, nephrotoxic damage after the ingestion of lilies is not observed in rodents or rabbits. In dogs, only vomiting and other gastrointestinal signs can be observed after lily ingestion [7]. Furthermore, lily toxicosis has only been reported in “domestic cats,” belonging to the family Felidae of the suborder Feliformia. Here, we present a case of suspected lily poisoning in a meerkat (*Suricata suricatta*), belonging to the family Herpestidae of the suborder Feliformia. To our knowledge, this is the first study describing the clinical and pathological findings of lily poisoning in an animal of a taxon different from the domestic cat.

A 7-month-old female meerkat was observed eating the flowers and buds of *Lilium ‘Casa Blanca’* or Oriental Lily. The meerkat was presented to our veterinary hospital by the owner with vomiting within a few hours after eating, hypothermia after three episodes of vomiting, and tachypnea within 12 hours after vomiting. At the time of admission, the meerkat presented with oliguria, seizures, tachypnea, self-biting, and nystagmus. The animal was hospitalized and administered oxygen inhalation therapy and transfused by intravenous fluid injection. High concentrations of blood urea nitrogen (68.0 mg/dl), creatinine (2.0 mg/dl), glutamic-pyruvate transaminase (981.0 mg/dl), and creatine phosphokinase (2,036.0 mg/dl) were detected. Upon ultrasonography and computerized axial tomography examination, both the kidneys and liver were larger than normal. From midnight to morning, the meerkat vomited blood and presented with loss of consciousness. Approximately 40 hr after lily ingestion, the meerkat died. During necropsy, all tissues and organs were collected and fixed in 10%
neutral buffered formalin, embedded in paraffin wax, sectioned at 4 µm, and stained with hematoxylin and eosin (HE).

Grossly, renal congestion and perirenal edema were found. Pulmonary congestion, liver congestion, and partial paleness were observed. Histopathologically, widespread tubular degeneration and necrosis were observed in the proximal tubules of the entire renal cortex (Fig. 1). Proximal convoluted tubules revealed marked granular degeneration and necrosis with the loss of nuclei (Fig. 2). Severe congestion was detected from the deep cortex to the outer medulla (Fig. 1). However, no hemorrhage, tubule regeneration, or inflammatory cell infiltration were observed. In the liver, massive hepatocyte necrosis with congestion was detected (Fig. 3). Survival and necrotic hepatocytes included small- to large-sized lipid droplets (Fig. 3). Neither reactive inflammatory cell infiltration nor regenerative hepatocytes were present in the liver. Pulmonary congestion and edema were observed with focal haemorrhage (Fig. 4). No significant change was seen in other organs. Gut content analysis was not performed.

In the present case, a diagnosis of suspected lily toxicosis was made because of the direct visual observation of lily ingestion, acute renal failure after lily ingestion, and acute tubular necrosis corresponding to the typical pathological changes associated with lily toxicosis in cats.

Cats are known to be sensitive to lily ingestion, but there is no age, sex, or breed predilection [7]. Among cats, the mortality rate from Easter Lily toxicosis has been reported to be as high as 50–100%, depending on the initiation time of symptomatic treatment. Specifically, high mortality rates are reported if treatment is not initiated before the onset of acute renal failure, which occurs 18–24 hr after lily exposure [11]. Lily ingestion severely injures the kidney, initially leading to polyuric kidney failure, which can then lead to extreme dehydration, anuric renal failure, and eventually, death.
In the present case, massive hepatocyte necrosis, pulmonary congestion and edema, 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 [7]. Disorientation, ataxia, and head pressing have also been observed among cases of lily poisoning, but less frequently [2]. However, previous studies have not shown neuronal degeneration from lily toxicosis in cats.

Acute tubular necrosis is not a specific diagnosis and can result from various nephrotoxins, such as ethylene glycol, boric acid, pharmaceutical drugs, or metals [5]. Over time, acute tubular necrosis can lead to tubular regeneration, inflammatory cell infiltration, urinary casts, and other types of tubular degeneration, with the pathological lesions changing from acute to regenerative or chronic [5, 13]. Since in the present case, we only observed tubular necrosis, renal change was comparable to the acute phase. Thus, the observed renal change was probably caused by lily ingestion.

Pulmonary congestion and edema, along with coinciding lipidosis and hypertrophy of hepatocytes, has been observed in lily toxicosis in cats [7]. However, massive hepatocyte necrosis and focal hemorrhage 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 [7]. The aqueous extracts of lily leaves and flowers have been
shown to be nephrotoxic, with the aqueous floral extracts containing most of the toxic
compounds [11]. In the present case, since the meerkat ingested lily flower buds
containing high levels of the toxic compound, the rapid progression of the clinical
symptoms and eventual death are likely attributed to the flower ingestion.

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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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Figure 1. Kidney. Severe tubular degeneration and necrosis are presented in the renal cortex. Severe congestion is shown in the deep cortex to the outer medulla. Hematoxylin and eosin (HE). Bar, 500 µm.
Figure 2. Kidney. Proximal convoluted tubules reveal marked granular degeneration and necrosis with lost nuclei. HE. Bar, 100 µm.
Figure 3. Liver. Severe hepatocyte necrosis is seen with congestion. Hepatocytes include small- to large-sized lipid droplets. HE. Bar, 10 µm.
Figure 4. Lung. Pulmonary congestion and edema are observed with focal hemorrhage.

HE. Bar, 10 µm.