Oleander intoxication in New World camelids: 12 cases (1995–2006)

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Objective—To characterize the clinical and clinicopathologic effects and evaluate outcome associated with oleander toxicosis in New World camelids.

Design—Retrospective case series.

Animals—11 llamas and 1 alpaca.

Procedures—Medical records from a veterinary medical teaching hospital from January 1, 1995, to December 31, 2006, were reviewed. Records of all New World camelids that had detectable amounts of oleandrin in samples of serum, urine, or gastrointestinal fluid were included in the study. Descriptive statistics were used to evaluate the history, physical examination findings, clinicopathologic data, and outcome of affected camelids.

Results—11 llamas and 1 alpaca met the inclusion criteria of the study. Either oleander plants were present where the camelids resided (n = 7) or oleander plant material was identified in the hay fed to the camelids (5). One llama was dead on arrival at the hospital, and another was euthanized upon admission because of financial concerns. Of the 10 treated camelids, 9 had evidence of acute renal failure, 7 had gastrointestinal signs, and 4 had cardiac dysrhythmias on initial evaluation. The overall mortality rate was 25%, but the mortality rate for the 10 camelids that were medically treated was 10%.

Conclusions and Clinical Relevance—In New World camelids, oleander intoxication was associated with a triad of clinical effects (ie, renal, gastrointestinal, and cardiovascular dysfunction). Oleander intoxication often represented a herd problem but carried a fair to good prognosis if treated promptly. Oleander toxicosis should be considered a differential diagnosis in sick camelids. (J Am Vet Med Assoc 2009;235:305–310)

Oleander is an evergreen shrub or small tree in the dogbane family Apocynaceae. There are 2 genera of oleander—*Nerium* and *Thevetia* spp. The former has only 1 polymorphic species, called *Nerium oleander* (> 400 pink, white, purple, and yellow cultivars), and the latter has 8 species, including *Thevetia peruviana* (yellow oleander). *Nerium oleander* is native to the Mediterranean region and western China, whereas *T peruviana* is native to tropical America and to the West Indies. Both of these species are drought tolerant and have been exported and planted on road sides, pastures, and property borders in the United States, Mexico, and Australia; in these locations, livestock often have easy access to the plants. Although oleander is an attractive, fast-growing plant, which makes it suitable for use along fence lines, it is highly toxic to most mammalian species. Oleander contains 2 main classes of toxins—cardenolides and triterpenoids. The cardenolides, or cardiac glycosides, and their aglycone metabolites are considered the primary toxins of oleander. The cardiac toxic effects of these compounds are primarily a result of Na⁺,K⁺-ATPase pump inhibition, which effectively increases intracellular calcium concentrations and ultimately triggers positive inotropic effects. The triterpenoids in oleander are considered irritants that directly affect the digestive tract, but there are very limited data to support this. All parts of the plant, including the leaves, seeds, fruit, and root, are toxic whether fresh or dried.1

The toxic effects of oleander in many species, including horses, cattle, sheep, goats, dogs, cats, birds, and humans, are well described.2–6 As few as 10 medium-sized leaves of oleander can cause clinical signs leading to death in cattle and horses within 2 to 8 hours of consumption.7 In livestock, oleander poisoning often results from accidental ingestion of plant clippings, plant material that is baled with hay, or plant material that is chopped into silage. Sublethal oleander exposures often result in nonspecific clinical signs similar to those expected from overdose of most cardiac glycosides. To our knowledge, there are no published reports of the clinical course or appropriate treatment of oleander toxicosis in New World camelids. Research on the effects of oleander in New World camelids is needed because this plant appears to be a common cause of intoxication in these animals. We have unpublished data, for example, that indicate that oleander intoxication was the leading cause of death in llamas and alpacas submitted for necropsy to the California Animal Health and Food Safety laboratory in 2005. Thus, the purpose of the study reported here was to characterize the clinical and clinicopathologic effects and evaluate outcome associated with oleander toxicosis in New World camelids.

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**Materials and Methods**

**Criteria for case selection**—The medical records of animals examined at the Veterinary Medical Teaching Hospital of the University of California, Davis, from January 1, 1995, to December 31, 2006, were searched to identify those relating to New World camelids that were treated for oleander intoxication. A camelid was included in the study if oleandrin, the primary cardiac glycoside of oleander, was detected in samples of urine, serum, or gastrointestinal contents from the individual camelid or affected herd members. Oleandrin concentration in samples was quantified by use of a previously published liquid chromatography–mass spectrometry–mass spectrometry method.²

**Medical records review**—Information collected from the medical records included signalment, history, clinical signs at the initial evaluation, clinicopathologic data, treatments, outcome, and necropsy findings. Long-term follow-up information for camelids that survived to discharge from the hospital was collected via telephone conversations with the owners; these conversations occurred between 2 years and 3 months and 5 years and 9 months after discharge from the hospital.

**Statistical analysis**—Descriptive statistics including mean, median, and SD values were used to describe the data.

**Results**

The medical record search revealed 29 camelids that had a clinical diagnosis of oleander toxicity, and 1 additional animal in which oleander intoxication was suspected. Seventeen of these camelids were excluded because of the presumptive nature of the diagnosis or lack of confirmation of the presence of oleandrin in samples of urine, serum, or gastrointestinal contents collected from those camelids or affected herd members. Eleven llamas and 1 alpaca met the criteria for inclusion in the study. The alpaca was an 8-month-old female. The 11 llamas included 5 sexually intact males (age range, 7 months to 6 years), 2 castrated males (ages, 4 and 11 years), and 4 females (age range, 2 to 8 years). The mean ± SD age of the 12 camelids was 4 ± 2.4 years (median age, 3 years). The mean ± SD weight of the camelids was 118 ± 34 kg (259.6 ± 74.8 lb; median, 128.3 kg [282.5 lb]). Only 1 llama was examined as an individual case; in all other instances, the alpaca was an 8-month-old female. The llama was 3 years and 9 months after discharge from the hospital.

**Table 1**—Clinicopathologic abnormalities detected at the time of hospital admission in 10 New World camelids that underwent treatment for oleander toxicity.

<table>
<thead>
<tr>
<th>Variable</th>
<th>Mean ± SD</th>
<th>Median</th>
<th>Range</th>
<th>Reference range</th>
</tr>
</thead>
<tbody>
<tr>
<td>RBC count (× 10³ cells/µL)</td>
<td>12.3 ± 3.46</td>
<td>11.96</td>
<td>7.61–17.37</td>
<td>10.5–17.2</td>
</tr>
<tr>
<td>WBC count (cells/µL)</td>
<td>12,062 ± 6,048</td>
<td>12,164</td>
<td>4,700–24,410</td>
<td>8,000–21,400</td>
</tr>
<tr>
<td>Band cell count (cells/µL)</td>
<td>800.8 ± 1,220</td>
<td>376</td>
<td>0–3,798</td>
<td>0–147</td>
</tr>
<tr>
<td>Monocyte count (cells/µL)</td>
<td>937.1 ± 456</td>
<td>873</td>
<td>153–1,591</td>
<td>0–1,009</td>
</tr>
<tr>
<td>Blood glucose (mg/dL)</td>
<td>212 ± 67.4</td>
<td>200</td>
<td>150–347</td>
<td>86–168</td>
</tr>
<tr>
<td>BUN (mg/dL)</td>
<td>83 ± 41.2</td>
<td>89.5</td>
<td>23–141</td>
<td>9–34</td>
</tr>
<tr>
<td>Serum creatinine (mg/dL)</td>
<td>7.2 ± 3.2</td>
<td>7.8</td>
<td>1.5–11.8</td>
<td>1.1–3.2</td>
</tr>
</tbody>
</table>

Of the 12 camelids, 1 was dead on arrival at the hospital, and 1 was euthanized at admission because of financial reasons. Physical examination findings were recorded in the 10 camelids for which treatment was attempted. The remarkable physical examination abnormalities included dysrhythmia (n = 4), lethargy (7), and decreased to absent auscultable gastrointestinal tract motility (7). The cardiac abnormalities were characterized as bradycardia in all 4 affected camelids (heart rate range, 36 to 56 beats/min).² Electrophysiography was performed in 3 camelids; detected abnormalities included second-degree atrioventricular block (n = 1) and irregular or variable R-R intervals (sinus arrhythmia [2]). At the initial evaluation, 7 camelids had diarrhea or soft feces; 1 camelid did not defecate for the first 24 hours after admission and then developed diarrhea the next day. Fecal output on the day of admission was not recorded for 2 camelids.

At the time of admission, a CBC and serum biochemical analyses were performed for all 10 treated camelids (Table 1). Mild anemia was detected in 3 of the 10 camelids. White blood cell counts varied; 3 camelids had leukopenia, 1 camelid had leukocytosis, and 6 camelids had counts that were within reference limits. Band cytophilia was evident in 6 of the 10 camelids; 4 had monocytosis.²

Serum biochemical abnormalities detected at the time of admission included hyperglycemia (6/10 camelids) and azotemia (9/10 camelids; Table 1). Azotemia resolved prior to discharge in all surviving camelids. The mean duration of azotemia was 4.1 ± 2.9 days (median, 4.0 days). Blood lactate concentration was measured in 9 camelids within 4 hours of initiating fluid therapy on the day of admission. Mean blood lactate concentration was 1.19 ± 0.55 mmol/L (median, 1.15 mmol/L; range, 0.5 to 1.9 mmol/L).

Nine camelids received IV fluid therapy: 8 were administered a balanced electrolyte solution,¹ and 1 was administered physiologic saline (0.9% NaCl) solution. Nutritional support was provided for 6 camelids; 4 were given partial parental nutrition¹⁰ IV at a rate of 1.2 kcal/kg/h (0.55 kcal/lb/h), and 2 were treated IV with a 1.25% dextrose solution at a rate of 0.13 to 0.26 kcal/kg/h.
(0.6 to 0.8 mg/kg/min [0.06 to 0.12 kcal/lb/h] [0.27 to 0.36 mg/lb/min]). Three camelids were treated with ultralente insulin1 (0.1 to 0.4 U/kg [0.05 to 0.18 U/lb], SC, q 12 to 24 h). Adsorbents were administered to 6 of the 10 camelids. Four camelids were treated with activated charcoal via orogastric intubation: 1 received 5 doses (1.67 g/kg [0.76 g/lb] each), 1 received 4 doses (1.5 g/kg [0.68 g/lb] each), and 2 received 1 dose (doses not recorded). Two camelids were administered di-tri-octahedral smectite (100 mg/kg [43.5 mg/lb]) orally once. Ceftiofur1 was administered to 7 camelids (2.2 mg/kg [1.0 mg/lb], IV, q 12 h). Three camelids were given bolus doses of furosemide (0.5 to 1 mg/kg [0.23 to 0.45 mg/lb], IV). Two camelids were treated with anthelmintics (fenbendazole [20 mg/kg [9.1 mg/lb]], PO, q 24 h for 5 days), and 1 camelid received fenbendazole and sulfadimethoxine (doses not recorded) for 5 days because nematodes and coccidia were evident in feces microscopically. One camelid was treated with vitamin E (10 U/kg [4.5 U/lb], PO, once daily for 3 weeks) and selenium (0.02 mg/kg [0.01 mg/lb], SC, once). Duration of hospitalization ranged from 6 to 11 days.

Of the 10 camelids that were admitted and underwent treatment, 9 survived to discharge from the hospital, and 1 died shortly after admission (approx 40 to 42 hours after exposure to oleander). The interval from exposure to initiation of treatment for the other camelids was not specifically recorded. The 5 camelids that belonged to the same herd were admitted to the hospital approximately 24 to 72 hours after the owner first noticed clinical signs of lethargy and loose feces. The overall mortality rate among the 12 camelids with oleander intoxicosis was 23%; however, for the 10 camelids that underwent treatment, the mortality rate was only 10%.

Long-term follow-up information was available for all camelids that survived to discharge from the hospital. The alpaca failed to thrive following discharge and was euthanized more than 1 year later; however, it had been successfully bred and delivered a healthy cria subsequent to the episode of oleander intoxicosis. Prior to death, the alpaca developed marked uremia, which may have been related to the prior oleander poisoning. The remaining 8 llamas were alive at the time of follow-up (2 to 5 years after discharge from the hospital). According to the owners, 4 of these 8 camelids did not have any health problems. The other 4 camelids lived at the same farm and were alive at 2.8 years after discharge from the hospital. The owner reported that one of those camelids developed a heart murmur, but weight loss was not evident, and the animal appeared clinically normal. Another of those camelids was overtly healthy, but was perceived to have undergone behavioral changes (ie, increased avoidance of people). A third camelid was perceived to have increased susceptibility to heat and cold stress. The fourth camelid at that farm was initially considered to have stunted growth; however, its dam gave birth to another cria that was also of small stature, making it unlikely that the small physical size was related to the previous oleander intoxicosis. It is unknown whether the oleander intoxicosis had any relationship to the reported heart murmur, behavioral change, and owner perception of increased susceptibility to environmental temperature extremes in 3 camelids.

All 3 camelids that died or were euthanized at the hospital underwent necropsy. One camelid had mild multifocal renal hemorrhage and severe, segmentally extensive mucosal and submucosal hemorrhage of the jejunum. Only cardiac abnormalities, which included mild, multifocal myocardial degeneration and necrosis, were detected in the second camelid. The third llama had pathologic changes in renal, cardiac, and gastrointestinal tissues. The findings included diffuse acute tubular intraepithelial vacuolar degeneration and intraductular mineralization of the renal cortex and medulla, rare intrasarclemmal vacuolar degeneration, and moderate diffuse nephrophilic, eosinophilic, and lymphocytic enteritis with hemorrhage and submucosal edema in the small intestine.

Discussion

To our knowledge, there are no published reports that describe the treatment of oleander intoxication in New World camelids and provide long-term follow-up information regarding affected animals. The data obtained in the study of this report suggest that oleander toxicity is a clinically important problem in New World camelids. Seven of the 12 camelids included in the study ingested oleander that was growing on or near the farm property. None of the affected animals were malnourished at the time of ingestion. Given these findings, it is likely that the affected animals were curious or did not find the plant entirely unpalatable. The remaining 5 camelids ingested oleander that was baled with hay, which highlights the importance of investigating feed sources as a potential source of exposure.

As the findings of the present study highlight, cardiac glycoside toxicosis is an important differential diagnosis for lethargy, anorexia, bradyarrhythmias, azotemia, or diarrhea in camelids. Considering the common and nonspecific clinical signs of lethargy and anorexia in sick New World camelids, the possibility of oleander exposure should be discussed with owners of animals with such signs; samples of serum, gastric contents, and urine should be submitted for detection of oleandrin if any possibility of exposure exists. In the present study, physical examination, clinicopathologic, and necropsy findings indicated that pathologic changes in the cardiac, gastrointestinal, and renal systems are commonly associated with oleander intoxication in New World camelids. Cardiac disease was evidenced by dysrhythmias and bradycardia and histopathologic changes within the myocardium. Gastrointestinal tract disease was also prominent; of the 12 camelids included in the study, 6 had a history of anorexia, and 7 had a history of diarrhea or constipation. All 3 camelids that underwent necropsy had evidence of gastrointestinal tract insult.

In the present study, acute renal failure was a prominent feature of oleander intoxication. Most of the camelids for which treatment was initiated (9/10) were azotemic at the time of initial evaluation. In those camelids, the degree of azotemia and the duration (mean, 4.1 ± 2.9 days) despite fluid therapy suggested that prerenal azotemia was unlikely. Additionally, the pathologic renal changes identified via necropsy support the suggestion that toxins of oleander affect the kidneys of New World camelids. Results of a prospective study2 in sheep indicated that renal disease was a consistent component of oleander intoxication; in a group of 5 sheep given lethal doses of oleander, all developed renal congestion and widespread tubular necrosis. Another prospective study3 in sheep to evaluate the effects of daily
low-dose oleander ingestion also revealed renal changes associated with oral administration of dried leaves of the plant. It appears that acute renal failure is also a feature of oleander intoxication in goats and equids.\(^{1,11,12}\) In contrast, renal disease does not appear to be a consistent complication of oleander intoxication in humans. To our knowledge, only a single case series report\(^{10}\) describes jaundice and renal failure that resulted from ingestion of yellow oleander in 7 humans. In a case series of 351 humans with yellow oleander poisoning (53 of which had full serum biochemical profiles performed), only 1 elderly person had high serum creatinine concentration; that individual, as well as 3 others, had high BUN concentration. The effects of cardiac glycosides, including those of oleandrin and ouabain, have also been studied extensively in rodents. Like humans, rodents do not appear to commonly develop acute renal failure as a result of oleander intoxication. Instead, rodents are unique in their clinical response to oleander exposure in that they develop CNS signs.\(^{13-17}\) The reasons for these species' differences are not known and may represent differences in absorption of, metabolism of, or receptor interactions with cardiac glycosides.

The pathogenesis of renal failure as a result of oleander intoxication is unknown. It is feasible that inhibition of the Na\(^+\)K\(^+\)-ATPase pump in the renal tubules results in direct nephrotoxic effects. Sodium reabsorption in the tubules is Na\(^+\)K\(^+\)-ATPase dependent and is consequently a target of oleandrins toxins. In porcine proximal renal tubular cell cultures, binding of cardiac glycosides to the proximal tubular cells results in internalization of the Na\(^+\)K\(^+\)-ATPase pump with a consequent decrease in sodium reabsorption.\(^{18}\) It is also possible that renal failure is a result of hypoperfusion, which may develop secondary to the effects of oleander toxins on the cardiovascular system. Oleandrin-induced injury to the gastrointestinal tract, which leads to decreased absorption of water and third-space losses of fluids, may contribute to dehydration and hypovolemia. Ultimately, hypovolemia can lead to decreased renal blood flow, renal tubular necrosis, and renal failure.\(^3\)

With prompt treatment, the prognosis for humans with toxicity appears to be good to excellent. In 1 report,\(^9\) administration of syrup of ipecac in children with oleander intoxication within hours of ingestion resulted in a 100% survival rate. The camels included in the present study were treated orally with adsorbents; the goal was to bind toxins within the gastrointestinal tract. There is some evidence that the use of multiple doses of activated charcoal may be effective in treating humans with yellow oleander intoxication. In a study\(^{20}\) of 402 adults with oleander intoxication in Sri Lanka, all patients were treated with gastric lavage, oral administration of a dose (50 g) of charcoal once, and administration with atropine, after which 201 patients received multiple doses of charcoal and 201 patients received a placebo. The mortality rate in the group treated with placebo was 8%, whereas the mortality rate in the group treated with multiple doses of charcoal was 3%.\(^5\) In that study,\(^20\) the affected individuals received prompt treatment (within 24 hours after poisoning), which is likely to have contributed to the low mortality rate. However, the ability of the gastrointestinal tract decontamination to influence the overall clinical outcome of oleander poisoning in other animals, and especially in New World camels, has not been evaluated. A considerable amount of oleander toxins (approx 60%) is excreted via biliary excretion through feces, whereas urinary excretion is not considered a major route of elimination (removing < 10% of the dose).\(^{21}\) In addition, cardiac glycosides undergo enterohepatic circulation.\(^{1,12}\) Thus, repeated administration of activated charcoal may be an effective treatment for oleander intoxication in New World camels.

Recently, an in vitro study conducted by one of our group revealed that products containing activated charcoal are more effective as adsorbents for binding oleander toxins than are products containing di-tri- and octahedral smectites.\(^{22}\) However, the ability of these adsorbents to alter the clinical outcome in oleander-poisoned camels remains to be evaluated.

For the camels included in the present study, an additional treatment for oleander toxicosis that may have been helpful includes the administration of polyclonal anti-digoxin Fab fragments. These are antibodies that bind cardiac glycosides, including digoxin and oleandrin.\(^{14,24-26}\) Such Fab fragments have been used in small animals affected by oleander poisoning with good success.\(^20\) However, none of the camels in the present study were administered digoxin Fab fragments; Fab fragments are expensive, and treatment of a large animal such as a camelid would be cost prohibitive. It is important to consider the potential for impaired renal function following administration of digoxin Fab fragments because the antibodies bind cardiac glycosides and the resulting complexes are renally excreted. If the patient is anuric or has decreased renal function, the complexes will not be excreted and can disassociate, thereby releasing the cardiac glycoside back into the circulation.\(^{27,28}\) Therefore, even if digoxin Fab fragments are available, renal function should be assessed prior to administration. Another new approach for the treatment of oleander intoxication in humans is a combination of Fab fragments and hemofiltration, whereby oleander toxins are removed by use of filter devices (without dependency on normal renal function).\(^{27,28}\) The authors are not aware of studies evaluating the efficacy of Fab fragments or hemofiltration in oleander-intoxicated New World camels or other large animals.

The treatment of oleander intoxication in the camels of the present study was centered on supportive care and the administration of adsorbents. The supportive care consisted of IV fluid therapy, parenteral nutrition, and administration of anti-inflammatory drugs and antimicrobials. One camelid received antioxidants in the form of vitamin E and selenium. General recommendations for treatment of camelids with oleander intoxication can be made based on the results of the present study and from those made for affected humans. These treatment recommendations include the use of activated charcoal, IV fluid therapy, parenteral nutrition, and antiarrhythmic drugs as necessary for dysrhythmias. Administration of digoxin Fab fragments should be considered where finances permit, and the effects of such treatment in oleander-intoxicated camelids warrants investigation.

The necropsy results in the present study differ somewhat from those reported in a previous study\(^7\) of oleander-poisoned camels (among other livestock) in California in which the primary lesions included pulmonary edema and congestion, gastritis, and myocardial degeneration and necrosis. Findings of that study did not include pathologic changes in the kidneys. In the 3 camels that underwent necropsies in the present study, pulmonary congestion or edema was not detected; however, renal damage was identified in 2 of the 3 animals. The reasons for these differences are not clear, but 1 hypothesis is that the differences in necro
ropysy findings may have been attributable to the interval between exposure and death. Camelds that ingest high doses of oleander and that die quickly may not develop histologic lesions, but are likely to develop agonal pulmonary edema. It is possible that renal lesions develop at a later time point, compared with development of cardiac dysrhythmias or myocardial changes. The 2 necropsied camels in the present study that had evidence of renal damage had been exposed to oleander approximately 36 hours or approximately 40 to 42 hours earlier. It is likely that many of the camels included in the previous study were sentinel animals that were exposed to a large dose of oleander and died suddenly from the cardiac effects. Another factor that may have influenced the findings of that study and the study of this report was differences in necropsy techniques of the pathologists. In the present study, the 2 camels in which renal changes were detected were admitted to the hospital alive, and clinicopathologic analyses were performed ante-mortem. The knowledge of existing azotemia may have directed pathologists to focus on changes in the kidneys.

Eleven of the 12 camels with oleander toxicosis in the present study represented herd poisonings. Only 1 camel was evaluated as an individual case, without other herd members being affected. It appears that oleander intoxication commonly occurs as a herd problem among New World camels, which is not surprising considering the management practices of most cameld farms. The llama that was evaluated as an individual case had been exposed to oleander when it had been displayed at a child’s birthday party. This finding is in contrast to data reported in another study of livestock oleander intoxications in California, in which oleander poisoning in only 1 of 6 camels from different farms represented a herd problem. The llamas in that previous report died and were submitted for necropsy only; thus, it is possible that they may have represented sentinel animals and that other camels from the same herds were also affected. Because detailed histories of those camels were not available, it is impossible to identify the reasons for the difference in herd-wide versus individual animal intoxication identified in that study and the study of this report. It is plausible, however, that only a small percentage of livestock with oleander toxicosis in California are admitted to our facilities for treatment or necropsy. Thus, the data included in the present and previous study may not be representative of the occurrences of oleander intoxication among camels in California. This should also be considered when evaluating the apparent sex predilection of oleander toxicosis in our study. In both the present and previous studies, a sex predilection appears to be present; in the 2 studies combined, 12 of 17 camels for which sex was recorded were male. It is likely that the apparent predilection is biased because of the low number of cases in each study. However, it is not possible to rule out a true sex predilection, which may be a result of male cameld behavior that leads to ingestion of greater amounts of oleander. It is also possible that male camels are more susceptible to oleander toxins or that owners were more willing to pursue treatment or permit necropsy of male camels. Further investigation of this possible sex predilection is needed.

The results of the present study indicated that oleander intoxication in New World camels carries a fair to good prognosis with treatment. The overall mortality rate in the present study was 25%, but only 10% in camels for which treatment was attempted (ie, excluding those that were dead on arrival at the hospital or euthanized prior to treatment because of financial reasons). The mortality rate may have been influenced by the presence of sentinel animals in the herd. A common history included recent deaths of 1 or more herdmates; it is possible that these deaths had alerted owners to a herd problem, which caused them to seek earlier medical care for the camels. Despite this apparent good long-term prognosis, 1 camel was euthanized because of renal failure greater than 1 year after oleander intoxication; this highlights the importance of monitoring renal function in affected animals in the months following exposure.

As the data in the present study have indicated, oleander intoxication should be considered a differential diagnosis for lethargy and anorexia, which are fairly nonspecific signs, in New World camels. In affected camels, the renal, gastrointestinal, and cardiovascular systems are typically affected. Oleander toxicosis may occur as a herd problem; therefore, if 1 animal is known to have been poisoned, owners and veterinarians should consider that other animals may have been exposed. A definitive diagnosis of oleander intoxication is established via detection of oleandrins in biological specimens collected from an affected animal. The prognosis for camels with oleander intoxication is fair to good if prompt treatment is provided.

References

Effect of firocoxib or flunixin meglumine on recovery of ischemic-injured equine jejunum
Vanessa L. Cook et al

Objective—To determine whether treatment of horses with firocoxib affects recovery of ischemic-injured jejunum, while providing effective analgesia.

Animals—18 horses.

Procedures—Horses received saline (0.9% NaCl) solution (1 mL/50 kg, IV), flunixin meglumine (1.1 mg/kg, IV, q 12 h), or firocoxib (0.09 mg/kg, IV, q 24 h; n = 6 horses/group) before 2 hours of jejunal ischemia. Horses were monitored via pain scores and received butorphanol for analgesia. After 18 hours, ischemic-injured and control mucosa were placed in Ussing chambers for measurement of transepithelial resistance and permeability to lipopolysaccharide. Histomorphometry was used to determine denuded villus surface area. Western blots for cyclooxygenase (COX)-1 and COX-2 were performed. Plasma thromboxane B₂ and prostaglandin E₂ metabolite (PGEM) concentrations were determined.

Results—Pain scores did not significantly increase after surgery in horses receiving flunixin meglumine or firocoxib. Transepithelial resistance of ischemic-injured jejunum from horses treated with flunixin meglumine was significantly lower than in saline- or firocoxib-treated horses. Lipopolysaccharide permeability across ischemic-injured mucosa was significantly increased in horses treated with flunixin meglumine. Treatment did not affect epithelial restitution. Cyclooxygenase-1 was constitutively expressed and COX-2 was upregulated after 2 hours of ischemia. Thromboxane B₂ concentration decreased with flunixin meglumine treatment but increased with firocoxib or saline treatment. Flunixin meglumine and firocoxib prevented an increase in PGEM concentration after surgery.

Conclusions and Clinical Relevance—Flunixin meglumine retarded mucosal recovery in ischemic-injured jejunum, whereas firocoxib did not. Flunixin meglumine and firocoxib were effective visceral analgesics. Firocoxib may be advantageous in horses recovering from ischemic intestinal injury. (Am J Vet Res 2009;70:982–1000)