Oleander Poisoning in Cattle in Devghat, Nepal- A Case Report

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ABSTRACT

Oleander poisoning, though widely seen in the Mediterranean region, is rarely seen in Nepal. The poisonous effect of the fodder is due to the accidental ingestion of the different parts of 'oleandrin' which contains cardiac glycosides called cardenolides causing mortality in animals. In our study, 8 lactating cows were affected after oleander leaves were accidentally mixed and chopped with Napier. After the ingestion of the mixed fodder, out of 8 cows, 5 died instantly and the remaining 3 were showing severe clinical signs such as severe depression, anorexia, ruminal atony, diarrhea, serous nasal discharge, tachycardia, and irregular heartbeat. The diagnosis of the poisoning was made by finding the chopped leaves of oleander in the feed and ruminal content. The visual observation of the oleander leaves along with gross lesions specifically in the heart causing coagulative necrosis- as it acts as cardiac glycosides- we came to diagnose the oleandrin poisoning. As there was not the availability of digoxin-specific antibodies for the therapy, symptomatic and supportive treatment was done, and the good prognosis was seen on morbid animals within a week.

***Keywords**: HPLC, Glycosides, NSAID$_S$*

INTRODUCTION

Oleander is the decorative shrub grown in gardens for aesthetic purposes primarily based in the Mediterranean and Asian regions of the tropical and subtropical regions (Mahin et al., 1984, as cited in (Oryan et al., 1996). The oleander leaves, flowers and seeds and all parts, if ingested are poisonous to human, animals and certain insects (Langford & Boor, 1996). The toxic compound present in the shrub having cardiac glycosides activity called cardenolides is detected by HPLC (Us et al., 2012). Karawya et al. (1969) performed an experiment to conclude that seeds and roots contain the highest percentage of total cardenolides, followed by fruits and leaves. Cardenolides act their effect by halting the

Na+-K+-ATPase pump in cardiomyocytes. As a result, the level of intracellular Na level is increased, which in turn aggravates the level of calcium hindering the positive ionotropic effect. The resting membrane is also increased, causing increased excitability and slow atrioventricular conduction due to altered vagal tone (Ceci et al., 2020).

In the present study, the authors describe the occurrence of a cattle herd intoxication in Devghat, Tanahun due to the accidental incorporation of oleander leaves in the Napier fodder. The diagnosis was done by clinical signs, post-mortem and the presence of oleander leaves in the ruminal content and residual fodder in the manger.

CASE HISTORY

A case was reported in the cattle farm located at Devghat, Tanahu, Nepal during the month of September 2022. The farmer was unaware of the accidental incorporation of the oleandrin neither was aware about the poisonous effects oleandrin in animals. In the morning 10 am, the feed was ingested by cattle. The farm owner called a local veterinary technician. The technician administered NSAIDs, but the condition did not get better. Later in the morning, the conditions aggravated with diarrhea, severe depression, and prolonged sternal decubitus. The technician later consulted with the Department of Veterinary Surgery and Pharmacology of the Agriculture and Forestry University. The team from the university went there and examined the remaining 3 animals before starting a therapy.

CASE FINDINGS

After the ingestion of feed, the animal began to show symptoms within 6 hours, that include depression, anorexia, lack of rumination, and pedaling. The case was so acute that, out of 8 animals, 5 animals were found to be dead by the night time.

There were the most common symptoms among the remaining animals and all showed varying degrees of depression and weakness. Out of 3 animals, one was showing nasal discharge and was severely dehydrated with dry mucus membranes and shrunken eyes. All of them were anorectic and had varying degrees of abdominal colic. Yellowish diarrhea was observed with no blood taints. Severe respiratory signs like labored breathing (dyspnea) with respiratory rales (crackle sound) and coughing were observed. The heartbeat was abnormal with tachycardia. During the feed observation, crushed Nerium oleander leaves and flowers were found.

Necropsy was performed on all dead animals and all of them showed similar lesions like congestion on visceral organs like lungs, liver, and kidneys. Myocarditis with both external and internal hemorrhage (endocardium) was seen on the heart. Lungs were severely

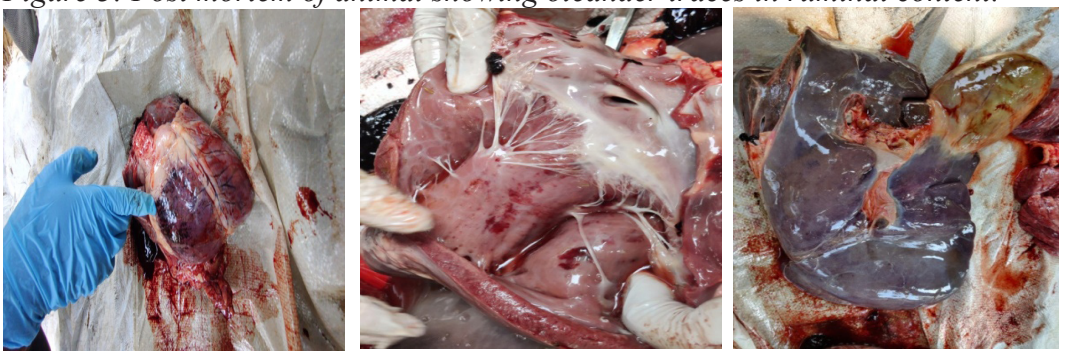
congested with pneumonic changes. The liver was severely congested and hemorrhagic. Diffuse hemorrhage was observed on the gastrointestinal system with unidentified reddish nodules. There was also mild hydrothorax, hydropericardium, and ascites. We suspected the case might be poisoning. The clinical signs of the poisoning are similar with the study of Galey et al. (1996) and with the visual traces of oleander in leaves and ruminal content we came to diagnose the poisoning.

(A) **(B)**

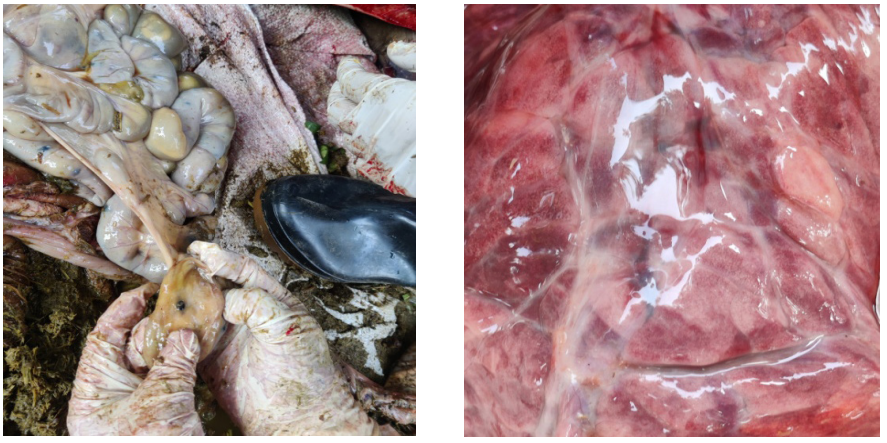
Figure 1
(A) Oleander around the vicinity of the farm; (B) oleander leaves in chopped fodder.

Figure 2: Animal in sternal recumbency pooled in diarrhea with head extended.

Figure 3: Post mortem of animal showing oleander traces in ruminal content.



(A) (B) (C)



(D) (E)

Figure 4: (A)Diffused hemorrhagic myocardium; (B) Hemorrhagic endocardium (C) Congested liver; (D) Congested and hemorrhagic lungs; (E) Unidentified black nodules in intestine

Management

Rajapaksa (2009), in his review, described the treatment of toxicity with supportive care, symptomatic treatment, gastric decontamination, managing cardiac arrhythmia and digoxin-specific antibodies. The treatment in our case was done duly as aforementioned. The digoxin-specific antibody was not used in our therapy due to the inaccessibility of the antibody. After confirming the oleander poisoning, we started to treat the remaining 3 animals and remaining 3 cattle recovered within one week. One pregnant cattle gave birth a healthy calf one month earlier of its term.

Table 1. Drug Regimen for the Therapy of Oleander Poisoning at Devghat.

Medication and Therapy	Dosage	Route	Total Days of Administration
NS + DNS	25 ml/kg, once daily	IV	3
Magnesium Sulfate	250 gm/animal, twice Daily	PO	3
Rumenatas Boli	3 boli/animal, twice daily	PO	5
Atropine Sulfate	0.5 mg/kg, twice daily	IV	2
Furosemide	0.5mg/kg, once daily	IV	2

Note. IV stands for Intra Venous, PO for Per Oral.

DISCUSSION

In our study, we found the most striking gross lesions in the heart, lungs, liver, intestine and rumen. The clinical signs and gross lesions were in line with the experimental study of poisoning in monkeys (Schwartz et al., 1974). The good prognosis of the morbid animals showed that time management and therapy of the animal are crucial. As the blood, serum, heart, liver and milk samples of oleander-poisoned animals show traces of oleandrin (Gosetti et al., 2019; Ceci et al., 2020). So, we advised farmers not to have milk of suspected oleander poisoning.

CONCLUSION

Though poisoning from oleander is not a common toxicological problem in Nepal, their plantation around the vicinity of the farm for aesthetic value can be menacing. There have been many cases of poisoning, both in animals and humans, around the globe, and it is a clinical emergency. The main clinical features caused by the ingestion of oleander are nausea, vomiting, abdominal pain, diarrhea, dysrhythmias, and hyperkalemia. Management is done with symptomatic and supportive therapy. If therapy is given time, there is a good chance of recovery as seen in our case study.

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CONFLICTS OF INTEREST

The authors declare no conflict of interest.

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