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Monograph

Nerium Oleander poisoning in farm animals

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Introduction

Nerium oleander (oleander, kaner, rose laurel) is a drought-tolerant, evergreen flowering shrub that belongs to the Dogbane family, *Apocynaceae*. It is frequently grown as an ornamental plant in gardens and parks as well as highway median divider or hedge around yards or orchards. Oleander is originally a Mediterranean and Asian plant and is widely distributed in the world, especially in tropical and subtropical regions. (Aslani, 2004). Two common oleanders are Nerium oleander (common oleander) and Thevetia peruviana (yellow oleander). All parts of the plant either fresh or dried are toxic to humans, animals and certain insects (Langford and Boor, 1996).

Red flowered varieties of oleander appear to be more toxic. A single leaf can be lethal to a child eating it, although mortality is generally very low in humans. The lethal dose of the green oleander leaves for cattle and horses has been found to be 0.005% of the animal's body weight. The minimum lethal dose of oleander for cattle was found to be 50 mg/kg body weight. Horses given 40 mg/kg body weight of green oleander leaves via nasogastric tube consistently developed severe gastrointestinal and cardiac toxicosis.

Mechanism of toxicity: -

Oleandrin and **nerine** are two very potent cardiac glycosides (cardenolides) found in all parts of the plant. Cardiac glycosides that act by inhibiting the cellular membrane sodium-potassium (Na+-K+ ATPase enzyme system) pump with resulting depletion of intracellular potassium and an increase in serum potassium. This results in progressive decrease in electrical conductivity through the heart causing irregular heart activity, and eventual complete block of cardiac activity, and death.

Clinical Signs: -

The clinical picture of oleander poisoning is characterized by polymorphous symptoms, whose onset and severity vary according to the number of active principles ingested (Galey *et. Al*.,1996). Indeed, we observed a rapid onset of symptoms within the first 24 h after the ingestion of the plant, followed by the death of the animal within 24 h, and death is due to ventricular fibrillation. The main clinical signs are related to disorders of the **cardiac, gastrointestinal, and nervous systems** (Ozdemir *et. Al*., 2011, Soto-Blanco *et. Al*., 2006).

- **The cardiac vascular sign:** alterations of the cardiac rhythm i.e., premature ventricular complexes and paroxysmal ventricular tachycardia with S-T segment slanting and followed by complete heart block.
- **The gastrointestinal tract sign:** involvement in ruminants results frequently in abdominal pain, atony and tympanism; however, diarrhea has been observed in acute accidental oleander poisoning in cattle as well as in other animal species (Galey *et. Al*., 1996, Aslani, 2004).
- **The nervous systems sign:** Confusion, dizziness, drowsiness, weakness, visual disturbances, mydriasis and convulsions are central nervous system manifestations of toxicity (Langford *et al.*, 1996).

Postmortem findings: -

- Microscopically, indications of hepatitis and nephrosis
- Hemorrhages on the gastric and intestinal mucosae, heart, gall bladder, meninges etc.
- Severe gastroenteritis.
- Generalized congestion.

Diagnosis: -

- Based on the history.
- Based on clinical Signs.
- PM lesions: reveals presence of obnoxious material/plant in the stomach/rumen.
- Lab diagnosis: detection of cardiac glycosides in urine or tissues.

Treatment: -

- There is no specific treatment for the poisoning.
- Administered a single dose of activated charcoal (5g/kg) to bind the toxin in the rumen and prevent further absorption, can be efficient in the early stages.
- Tachycardia can be treated by application of the adrenergic blocking drugs such as propranolol, which can be accompanied by atropine to reverse atrium-ventricular block. Other antiarrhythmic drugs include potassium chloride, procainamide, lidocaine, dipotassium EDTA and atropine sulphate.
- Complementary therapies include rehydration of animals suffering from diarrhoea and acidification of the rumen content.
- Elimination of ingested oleander from rumen can be obtained by rumenotomy.
- The digoxin specific antibodies must be given early in the course of poisoning to be effective for acute poisoning.
- Fluid for intravenous therapy should not contain calcium because this improves the action of cardiac glycosides (Cheeke, 1998; Knight and Walter, 2001).

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