MANAGEMENT OF INDIAN SQUILL TOXICITY IN SHEEP

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ABSTRACT
Scarcity of feed and fodder is most common situation in drought prone areas of Kadapa district. Many cases of sudden death were reported in sheep in treatment camp vaccination camp. After anamnesis it was found that the deaths were due to consumption of certain plants and the animals showed the signs of diarrhoea, ruminal atony, frequent micturation, convulsions, tremors and posterior paresis. Post mortem examination revealed enlargement and congestion of abdominal and thoracic organs, enteritis and gastritis. Liver, lungs, myocardium and kidneys show the signs of congestion and swelling. Histopathological examination revealed degenerative changes.

KEYWORDS: Sheep, Squill, Toxicity.

INTRODUCTION
Scarcity of feed and fodder is most common situation in drought prone areas of Kadapa district. Many cases of sudden death of ruminants due to grazing of certain plants are reported particularly in summer season in drought prone areas. Many cases of sudden death were reported in sheep in treatment cum vaccination camp which was organized at Ponnampalli, Mailavaram of Kadapa district. After anamnesis it was found that the deaths were due to consumption of certain plants and the animals showed the signs of diarrhoea, ruminal atony, frequent micturation, convulsions, tremors and posterior paresis.

Further it was reported that death occurs immediately after drinking water.

IDENTIFICATION OF THE TOXIC PLANT
The plant responsible for the toxicity was identified as Indian Squill (\textit{Urginea indica}), (Eng: Sea onion, Tel: Adavi tellagadda, Tamil: Nari vengayam, Hindi: Jangli kanda, Gujarati: Jangir kanda, Sanskrit: Vanapalandam). The plant is a robust perennial having globose bulb, overlapping tunic scale and small star shaped white flowers with narrowly lanceolate, glaucous leaves.
TOXICOLOGY
The plant is cyanogenic and the major toxic principles are HCN, Scilliroside, Scillaren which is a mixture of Scillaren A & B (Frank, 2004). Scilliroside is a cardio active bufadienolide glycoside, which is the main active toxic principle (Verbiscar, 1986) and have rodenticidal property (Gupta, 2007). The toxic dose in ruminants is reported to be 250 mg/ kg b.wt. (Sandhu and Brar, 2009).

SYMPTOMS AND POST MORTEM LESIONS
The initial signs of poisoning generally occur within 12 hrs and death usually occurs within three days. The toxic symptoms include ataxia, diarrhoea, ruminal atony, frequent micturation, convulsions, tremors, cardiac arrest, tachycardia, arrhythmia, bradycardia, hyperasthesia followed by paralysis and posterior paresis. Reports of death in cattle and buffaloes were reported earlier in Tarikere district of Karnataka (Narayana, 2003) and in cattle were reported in Chittoor district of Andhra Pradesh.

PM of dead animals revealed enlargement and congestion of abdominal and thoracic organs, enteritis and gastritis. Liver, lungs, myocardium and kidneys show the signs of congestion and swelling. Histopathological examination revealed degenerative changes.
DIAGNOSIS

It is based on the following points:

1) History of grazing of animals on plants and identification of the plants
2) Clinical signs and symptoms and PM lesions
3) Hyperkalemia as it is a primary manifestation of acute cardiac glycoside toxicity
4) Confirmatory diagnosis by identification of plant or its glycosides and/ or both in ingesta along with myocardial lesions. (Radostits et al., 2005)
5) Differential diagnosis with other poisoning like Albizia sps., Fluroacetate, Gossypol, Ionophore antibiotics, Vitamin E deficiency, Selenium and Copper which produces cardiomyopathy and similar clinical signs.

TREATMENT AND PREVENTION

1) No specific antidote is available. Only supportive therapy and evacuation of GIT is possible. A large number of animals survive without any treatment as because when poisoned animals are caught for treatment the fiercely struggling may aggravate the cardiomyopathy and may overburden the heart resulting in death.
2) Treatment for cyanide toxicity may be given- 
   Sodium nitrite @ 20mg/kg IV and sodium thiosulphate @ 500 mg/kg IV
3) Activated charcoal @ 1-3 g/kg b. Wt. at 4-8 hrs interval
4) Glucose and Sodium bicarbonate IV
5) KCl @ 1g/ kg
6) Atropine sulphate @ 30 mg/100 lbs, initially 1/4th to 1/3rd of the total initial dose slowly IV and rest of the dose IM or SC
7) Lidocaine @ 0.5 mg/ kg IV or Propranalol
8) Prevention can be done by removing the stock from the pasture during the draught period and grouping of young stock with the older stock as they have the experience of local grazing areas.

CONTRAINDICATIONS

Indian squill is known to produce interactions with calcium supplements, corticosteroids, Quinidine, stimulants, laxatives and diuretics.

DISCUSSIONS

The mechanism of death in Indian squill toxicity is due to cardiac arrest as Scilliroside in high doses have digitalis like action. As the death is reported immediately after drinking water as there may be hydrolysis of glycosides releasing toxic principles. The symptoms of diarrhoea,
gasritis and enteritis are due to its direct irritant action on GIT. Hyperkalemia is a primary manifestation of acute cardiac glycoside toxicity as there is redistribution of K$^+$ ions from intracellular to extracellular compartments resulting in hypomagnesemia. Hypercalcemia and hypomagnesemia exacerbate cardiac glycoside toxicity that why calcium supplements is contraindicated and the drug of choice includes the agents that promote potassium redistribution from extracellular to intracellular compartments.

Sodium nitrite and sodium thiosulphate are used as they convert met- haemoglobin (formed due to cyanide) to haemoglobin. Activated charcoal is used to bind the remaining glycosides in rumen and intestines. Glucose causes redistribution of potassium intracellular and sodium bicarbonate counteracts potassium effects, whereas alkalosis created by bicarbonate leads to redistribution of potassium intracellular. In chronic cases as there is hypokalemia so KCl should be used in addition to activated charcoal.

There is brady dysrhythmias and tachy dysrhythmias due to cardiac glycoside toxicity. Atropine abolishes vagal tone and thus increases heart rate hence, used for bradycardia and cardiac arrest. Antiarrhythmic agents like Lidocaine and Propranalol (Class IB anti- arrhythmic agents) are used as they increases electrical stimulation threshold of the ventricle and suppresses the automaticity of conduction through the tissues.

REFERENCES


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