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Lantana Toxicosis in Animals

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Abstract:

Lantana is an ornamental plant that contains triterpenoid chemicals and their poisonous metabolites. Lantadenes cause intrahepatic cholestasis by injuring the canaliculi (small bile ducts. The affected animals will show anorexia, dehydration, and jaundice, with ruminal stasis and photosensitization. Rumen stasis increases exposure to and absorption of the toxin; rumenotomy may be beneficial. At necropsy, the liver maybe enlarged, golden-yellow, hard or friable, and the gallbladder will be usually distended. Cholestasis and bile duct growth can be seen under a microscope, along with bilirubinuric casts in the renal tubules. Treatment protocol consists of supportive therapy along with administration of antidote.

Key Words: Lantana camara; Lantadene; animal; hepatotoxicity; photosensitization

Introduction:

Lantana camara (common name, lantana, wild sage, bunch berry: family *Verbenaceae*) is one of the most toxic plants in the world. It was introduced to India in the early 19th century as an ornamental plant. Now, this plant has naturalized in numerous areas of the country. Lantana takes up a substantial amount of space in meadows, woodlands, so-called waste land, and orchards. *Lantana camara* plants, contain red, yellow, pink, white, or intermediate-coloured flowers, are widespread in different parts of India (Gupta and Gupta, 2019).

In tropical and subtropical regions of the world, lantana plants grow lushly and are incredibly resilient shrubs that can reach elevations of around 2000 meters above sea level. It may thrive in unfavourable soil and climatic conditions. Lantana plants can reproduce quickly through stumps or cuttings, but natural multiplication appears to be from seeds disseminated by birds through their droppings or the faces of wandering herds of sheep and goats. The Lantana plant has a number of

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negative interactions with the environment. It causes hepatotoxicity and photosensitization in grazing livestock, as well as growth suppression in neighbouring plants, causing the ecological balance to shift rapidly. People who work in lantana-infested pastures and orchards experience discomfort, itching, and giddiness due to the scratchy, odorous plant leaf (Lonare *et al.*, 2012).

Lantana Toxicosis in Animals:

There have been some field reports of lantana toxicity from different parts of the World including India. Cattle will only eat lantana if there is a shortage of



Fig. Lantana camara

alternative feed because it is a very pungent plant. Ruminants and camelids are the most susceptible species. Humans cannot be poisoned by lantana unless they consume the berries. Compared to *Bos indicus* cattle, *Bos taurus* cattle are more vulnerable to lantana poisoning. When there is a shortage of fodder because of drought or flooding, the incidences range from isolated cases to widespread outbreaks occur. Additionally, cases of lantana poisoning have been documented when animals are transported from areas free of lantanas to areas afflicted with them. Regarding the consequences of plant toxicosis, it is generally noted that mortality rates by themselves do not provide accurate estimations of economic losses. Priority must also be given to indirect losses brought on by morbidity, the impact on weight growth, abortions, and birth abnormalities (Abdisa and Dinbiso, 2024). Poisonous weeds, such as lantana, are like a double-edged sword: (a) they have an advantage over other plants that provide feed, causing pastures to disappear, leaving no grasses or other vegetation, and (b) grazing in pastures infested with lantana causes toxicosis (Machado *et al.*, 2023).

Chemical Nature of the Toxin:

Pentacyclic triterpenoids known as lantadenes are the hepatotoxins found in lantana. Lantadene A, B, C, and D are some of the primary lantadenes; they all have the 22-hydroxy-olcanonic acid core structure. Minor lantadenes, such as reduced lantadene A and mid-reduced lantadene B, are also present in lantana leaves. The main hepatotoxin in the leaves of the toxic taxa of lantana plants is lantadene A, which has been shown to be toxic to guinea pigs and sheep. Although lantadene C administered orally causes extreme toxicity in guinea pigs, its effects on ruminants have not been studied (Goyal and Sharma, 2015).

Mechanism of Toxicity:

Consumption of Lantana leaves causes hepatotoxicity and secondary photosensitization. The three stages of lantana poisoning in animals are the gastrointestinal (GIT), hepatic, and post-hepatic phases. There is a lack of information regarding the release of toxins in various GIT regions. The rumen contents of animals poisoned with lantana become more poisonous with time, maybe because they become more liquid and improve absorption. Experiments in sheep revealed that lantana toxins are absorbed from every section of the digestive system, with the small intestine absorbing the most of them. Bile's presence in the

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digestive system has little bearing on how well lantana poisons are absorbed. The portal blood is primarily responsible for carrying the poisons to the liver. Whether the poisons are absorbed in their natural state or during biotransformation is unknown. In the guinea pig's caecum, lantadene A and B have been shown to undergo biotransformation (Kumar *et al.*, 2016).

Cholestasis, which is the hepatic phase of lantana poisoning, is caused by the interaction of the absorbed toxins with the biomolecules on or in hepatocytes, followed by a series of biochemical events. It is unknown what metabolic event on the hepatocytes causes the harmful effect. When lantana toxins are consumed, the gall bladder becomes immobile and the bile canaliculi close, which most likely results in a reduction in bile flow. Bile regurgitation brought on by cholestasis increases blood levels of bilirubin and phylloerythrin, a consequence of the biodegradation of chlorophyll. Phylloerythrin and bilirubin both bind proteins. When exposed to light, they go through photochemical reactions that result in photosensitization and related skin lesions (Sharma *et al.*, 2019).

Clinical Signs:

Liver and kidneys are the most affected organs. Hepatotoxicity, cholestasis, and photosensitization are caused when grazing animals consume lantana plants or when powdered lantana leaves are administered orally. Leaves of the lantana plants contain hepato-toxins. Nearly 5g of dry leaf powder per kilogram of body weight is the hazardous dose. Rarely do grazing animals on lantana-infested meadows decide to consume it. Acute cases develop haemorrhagic diarrhoea. Chronic poisoning with Lantana results in constipation. When animals kept on stall feeding for extended periods of time are allowed to graze in pastures infested with lantana, they typically browse on it first (Shridhar *et al.*, 2023).

Similarly, animals that lack the inclination to avoid lantana eat its leaf from hedges or roadside during land movement. Farmers in lantana-infested areas are well aware of the signs of lantana poisoning, and there are few instances of incorrect reporting. There is ruminal stasis, and animals go off-feed within a few hours of consuming lantana leaf, becoming extremely constipated. Within 24 to 48 hours, they develop acute jaundice with other symptoms such as difficulty in breathing, icteric conjunctiva and mucous membranes of the rectum and vagina. The eyelids swell, and fissures form on the muzzle, ear tips, and non-hairy regions of the body. Affected animals also develop terminal renal failure. Exposing light-skinned animals to bright sunlight causes photosensitization, muzzle may become inflamed, moist, ulcerated and very painful (pink nose) (Negi *et al.*, 2019).

Clinical Pathology:

Animals poisoned with lantana have shown biochemical alterations in their cytosol, canalicular plasma membrane, microsomes, lysosomes, and liver mitochondria. The plasma membrane, mitochondria, and microsomes are all enriched in cholesterol. Oxidative enzyme activities in the cytosol or mitochondria are increased. Conversely, there is a decrease in the activity of microsomal enzymes linked to drug

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metabolism. Glutathione-S-transferase activity in the cytosol is also markedly reduced. The liver's lysosomal enzymes may leak. Animals inebriated with lantana have shown notable changes in blood components. Changes in haemoglobin, urea, erythrocyte and leukocyte counts, and increased plasma bilirubin (conjugated type) level. Decrease arterial blood pressure, increase SGOT, SGPT, creatinine, sodium level in the blood and decrease normal potassium level (Sharma *et al.*, 2019).

Post mortem Lesions:

The liver appears orange and greatly swollen. There is significant enlargement of the gall bladder. Often, the contents of the rumen are dry and undigested. Faecal impaction is present in the colon close to the spiral. The thicker cortex turns yellow, and the adrenal glands swell (Sharma *et al.*, 2019).

Diagnosis:

- (1) History
- (2) Clinical signs
- (3) Clinical pathology
- (4) Post mortem lesions

Differential Diagnosis:

The ability to differentiate lantana toxicity from other hepatotoxicity and secondary photosensitization conditions is one of the main concerns of the clinician. This is accomplished by comparing the clinical and biochemical picture with the flora present on the pasture in which the animal grazed before becoming intoxicated; if the pasture had lantana bushes, the clinical history and plasma bilirubin measurements would support the diagnosis of lantana poisoning. The most toxic variety of lantana is *Lantana camara* var. *aculeata*, which has been found to produce red flowers (in a range of yellow to red). Lantana plants with pink flowers have been shown to be mostly nontoxic (Hussain *et al.*, 2013).

Treatment: (Baruti *et al.*, 2018)

- ➤ **Antidote:** No specific antidote is available. Physostigmine may trigger significant reversal of some of the signs within minutes.
- **Symptomatic treatment** needs to be given (Kachhawaha, 2014)
 - **1.** Prevent the animals from being exposed to harmful weeds any longer.
 - 2. If photosensitization occurs, keep the animal out of direct sunshine and in well-shaded places.
 - **3.** Give large doses of glucose saline solution intravenously.
 - **4.** Use hepatoprotective medications to improve liver function
 - **5.** Remove toxic ruminal contents through rumenotomy
 - **6.** Use saline purgatives to aid in the evacuation of gastrointestinal contents
 - **7.** Replace ruminal contents with a suspension of electrolytes, chaffed fodder, and rumen liquor from a healthy animal.

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- **8.** To inhibit additional absorption of the toxin in the rumen, administer a single dose of activated charcoal (5 g/kg).
- **9.** Treat lantana poisoning in cattle, administer 2.5 kilogram of powdered activated charcoal in 20 liters of multiple electrolyte solution via stomach tube. For sheep, 0.5 kg charcoal in four liters of fluid is sufficient.
- **10.**To treat photosensitization lesions and subsequent bacterial infections, provide H1 antihistaminic and antibiotics.

Prevention: (Desta, 2019)

- **1.** Identify the poisonous plants grown in the nearby area and removal of the same can be practiced or fencing off
- **2.** Try to have information regarding the conditions under which the toxic plants can be dangerous to animals such as wetness, temperature *etc*
- **3.** Formulate a grazing strategy that limits the time of grazing in an area where poisonous plants are growing
- **4.** Excessively hungry or stressed animals should not be moved to an area where poisonous plants are present
- **5.** Animal should be provided with ample supply of water, salt and other supplements
- **6.** If animals started showing symptoms, immediately consult a veterinarian

Conclusion:

There is currently no logical treatment based on the molecular mechanism of action and the structure of lantana toxins. Therefore, the best approach is to keep animals away from lantana leaves. If an animal is poisoned, it should also be shielded from the sun to prevent secondary problems like photosensitization. The animals should not be provided green feed during the course of illness.

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