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Sonu Kumar Yadav
Department of LPM, College of Veterinary Science & AH, Rewa, Madhya Pradesh, India

Nirbhay Bhavsar
Division of Extension Education, IVRI Izzatnagar, Bareilly, Uttar Pradesh, India

Anjani Kumar Mishra
Division of Extension Education, IVRI Izzatnagar, Bareilly, Uttar Pradesh, India

Pramod Sharma
Department of ILFC, College of Veterinary Science & AH, Jabalpur, Madhya Pradesh, India

Bhavna Aharwal
Department of LPM, College of Veterinary Science & AH, Jabalpur, Madhya Pradesh, India

Corresponding Author:
Sonu Kumar Yadav
Department of LPM, College of Veterinary Science & AH, Rewa, Madhya Pradesh, India

Farm animals afflicted by plant poisoning

Sonu Kumar Yadav, Nirbhay Bhavsar, Anjani Kumar Mishra, Pramod Sharma and Bhavna Aharwal

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Abstract
In India, animal poisoning is a significant concern for marginal and small-scale farmers due to the grazing practices that expose cattle to various toxic plants. Such poisoning can lead to physical discomfort, diminished productivity, or death in animals, particularly those that are malnourished. Factors influencing toxicity include animal species, body size, sex, dosage, and overall health, with liver or renal dysfunction potentially exacerbating the effects of toxicant exposure. Different types of toxicity include acute toxicity, Mild toxicity, and chronic toxicity. Grazing on meadows with toxic herbs is a common cause of livestock poisoning. To reduce the chance of poisoning, it's critical to recognize these dangerous plants. They may be so hazardous that even a tiny amount can kill animals or cause severe disease. Knowing which plants are poisonous to domestic animals is crucial.

Keywords: Brakenfern poisoning, cyanogenic plants poisoning, animals, livestock poisoning, specific treatment

1. Introduction
In the agrarian country of India, where a significant number of cattle are held by marginal and small-scale farmers, animal poisoning is more prevalent. There are several poisonous plants that are known to affect the cattle industry (Rangnekar, 2006) [26]. While grazing is considered a normal practice for livestock management, it exposes the animals to a variety of hazardous plants, particularly when there are less physiological issues (Abdisa & Dilbato, 2024) [1].
Toxic plant consumption can cause physical discomfort, reduced output, or even death in animals. Animals who have become malnourished are more susceptible to plant poisoning. Animal species, size of body, sex, dosage, physical and chemical composition, exposure level, and general health are some of the variables that might affect how toxic substances behave. Liver or renal dysfunction may increase poisoning because of slow excretion of toxicants or inadequate metabolism (Matches 1992) [20].

2. Different types of toxicity
- **Acute:** It happens whenever an individual receives just one high dosage; signs and poisoning markers show up immediately and it can become lethal. (Smolinske *et al*., 1990) [31]. Affected animals may pass away unexpectedly without displaying any obvious clinical or hemato-biochemical changes (Sadariya *et al*., 2023) [27].
- **Mild:** The effects of frequently being exposed to low doses (sub toxic) of a substance over a ninety-day period. When poisoning develops gradually, animals may exhibit reduced food intake, slower development, specific hematobiochemical changes, and pathological changes.
- **Chronic:** Chronic describes the result of prolonged exposure to a chemical at very low levels (more than six months). Toxicology happens gradually when an animal shows signs of reduced food intake, slower development, specific hematochemical and pathological changes, and other symptoms (Biswas *et al*., 2000) [5].

3. Common Livestock Poisoning

Animals graze in wide pastures and on slopes. During this period, they inadvertently include poisonous herbs in their diet. As a result, poisoning might happen sometimes. It is essential to identify hazardous plants and comprehend the potential for poisoning.

3.1 Cyanogenic Plants poisoning

Common cyanogenic plants include sorghum, Sudan grass, corn, lima beans, cherries, apples, peaches, and apricots. The toxic element in cyanogenic plants is cyanogenic glycosides, which include amygdalin, prunacin, linamarin, lotaustralin, dhurrin, and taxiphyllin. Most glycosides are harmless by themselves, but they become poisonous when the body hydrolyzes them. When plants are processed frozen, chopped, or chewed enzymes are released, making the plants more dangerous (Urugo & Tringo 2023) [34]. Numerous variables have been shown to increase the level of toxicity, such as ruminal pH and microbiota, fast ingestion, ingesting large volumes of immature cyanogenic plants, and the quantity of cyanogenic glycoside or free HCN in the ingested plants (Gensa, 2019) [9]. As the rumen and abomasum's pH levels rise, the toxicity increases. It has been shown that when the pH falls below 5.0, the enzymes that break down the glycosides stay dormant and the risk of toxicity decreases (Attia 2015) [4].

3.1.1 Mechanism of Action (MOA)

A pH of alkaline encourages toxicity in the rumen and abomasum. Methemoglobin and hydrogen cyanide from cyanogenic plants combine in the rumen to form cyanmethemoglobin. By deactivating the cytochrome oxidase enzyme, this combination prevents the last stage of oxidative phosphorylation. When oxygen consumption is absent, cell respiration stops. The overuse of nitrogen fertilizers and herbicides, such 2, 4-D, makes people more susceptible to cyanide poisoning (Arnold and Gaskill, 2014; Borron, 2006) [3, 6]. For ruminants, the most lethal dosage of HCN is roughly 2 mg/kg B.W. Plants that contain more than 200 parts per million of these glycosides are thought to be deadly.

3.1.2 Clinical sign

Animals may experience infertility, abortion, head tremors, incoordination, decreased productivity, and posterior limb pawning (Gurnsey *et al*., 1977) [10]. Others clinical indications of intoxication in farm animals include breathing difficulties, opisthotonos, tremors, restlessness, dyspnea, and terminal clonic convulsions. The mucous membranes are initially bright and cherry-red in color due to the volume of oxygenated blood. (Borron, 2006; Gurnsey *et al*., 1977) [6, 10]. It gradually becomes cyanotic as a result of hypoxia. The animal will go into a coma and eventually die if treatment is delayed (Gurnsey *et al*., 1977) [10]. Urinary incontinence and myelomalacia have also been noted in a small number of patients. Arthrogryposis has been linked to chronic toxicosis.

3.1.3 Specific treatment

To treat cyanide poisoning, an intravenous infusion of 1 milliliter of 20% sodium nitrate and 3 milliliters of sodium thiosulfate can be given at a dosage rate of 4 milliliters of mixture per 45 kilograms of body weight. The contents of the animals rumen should be swapped out for those of a healthy animal (Borron, 2006; Arnold and Gaskill, 2014) [6, 3].

3.2 Nitrate containing plant poisoning

Ruminants that consume forages contain excessive amounts of nitrate, such as oats, capeweed, sorghum, maize, lucerne, turnip tops, Sudan grass, wheat, and barley, may get nitrate poisoning. The kind of plant species, maturity stage, and plant part all affect the amount of nitrate in the plant tissues. Compared to fully grown plants, immature plants have a higher nitrate content.

3.2.1 Mechanism of Action (MOA)

Under normal circumstances, ruminants consumption of nitrate is transformed to ammonia and then to bacterial protein in the rumen, where the rate at which nitrate is converted to nitrite is far faster than that of nitrite to ammonia. When nitrate-containing plants are consumed in excess, nitrite builds up in the rumen. When this nitrite enters the bloodstream, it changes hemoglobin into methemoglobin, which stops oxygen from being transported. As a result, anoxia from nitrate poisoning causes the animal to die (Kamra *et al*., 2015) [16].

3.2.2 Clinical signs

Clinical manifestations of nitrate poisoning include tachycardia, diarrhea, vomiting, blue-brown staining of the mucosal membranes, and salivation. Intense stomach discomfort, fainting, tremors, dyspnea, rapid and noisy breathing, and a limited response to analgesics and antispasmodics (Oruc *et al*., 2010) [23].

3.2.3 Specific treatment

An intravenous methylene blue infusion is the treatment for nitrate toxicity. A 2% methylene blue solution aids in the conversion of methemoglobin to hemoglobin and cures the poisoning (Haouzi *et al*., 2019) [11].

3.3 Oxalate containing plant poisoning

Plants frequently contain oxalic acid, which comes in two forms: soluble and insoluble oxalates. The way soluble oxalate works is by binding trace minerals like iron (Fe), calcium (Ca), and magnesium (Mg), preventing their absorption (Li *et al*., 2022) [18]. The fibrotic and deformed demineralized bones in horses are the cause of lameness and "bighead." (McAllister *et al*., 2020) [21].

It is commonly recognized that eating oxalate can be harmful if taken in excess. Oxalate is taken into the bloodstream because the rumen is overloaded and has no way to digest it. Oxalate in bloodstreams creates an insoluble salt that accumulates in the kidney and results in renal failure (Hogan *et al*., 2016) [13]. Plants high in oxalate are highly appetizing to animals. Oxalates and nitrates are abundant in fresh sugar tips (Rahman *et al*., 2013) [24].

3.3.1 Mechanism of action (MOA)

The affinity of soluble oxalate for calcium is high. Insoluble calcium oxalate is created when soluble oxalate binds to calcium ions and enters the bloodstream. Numerous degenerative alterations occur, and the animal with hypocalcemia, which affects normal cell membrane function, have muscle weakness and tremors before collapsing and dying. Acute nephrosis and uremia are caused by calcium oxalate crystal production and buildup in renal tubules. Rumenitis is caused by the crystallization of calcium oxalate in the ruminal capillaries. There may be a subsequent bacterial or fungal invasion of the rumenitis lesions (Ermer *et al*., 2023) [8].

3.3.2 Clinical signs

After four hours of eating oxalates-rich plants, the first signs

of oxalate poisoning include dullness, lowering of the head, loss of appetite, and isolation from the herd. Following these findings, there is a coma with profoundly erratic breathing, profuse salivation with frothing, and gradual incoordination. Additional symptoms comprise ruminal atony, minor bloating, pollakiuria, frothy blood-tinged nasal discharge, dyspnea, sitting down with head turned in to the flank, shaking and paralysis of the muscles, especially the muscles of the face, and death without any struggle (James, 1972) [15].

3.3.3 Specific Treatment
Change to pastures devoid of oxalate, 50-100 ml of 25% calcium borogluconate (sheep), 300-500 ml (cattle), administered via intravenous or subcutaneous injection. Through oral ingestion of dicalcium phosphate (25% in salt ration) or alfalfa hay pellets comprising 10% dicalcium phosphate at a dosage of 225 g per animal per day for the removal of oxalates is used. When used as a calcium supplement, calcium carbonate works in the small intestine by chelating with oxalate to stop absorption and the development of renal calculi. Because of the possibility of nephrosis, the prognosis is always cautious. Cattle who survive the acute stages of the disease may die from renal nephrosis a few days later.

3.4 *Lantana camara* Plant Poisoning
The *Lantana camara* is the most popular and appealing garden plant. It is known that ruminants, especially sheep, are poisoned by a number of L. camara complex kinds. The detrimental effect is brought on by the presence of lantadene A and B, two metabolites of triterpene esters (Ntalo *et al*., 2022) [22]. For animals, leaves and young berries are more poisonous. Lantadene A is extensively metabolized in the liver to produce additional polar molecules, which are subsequently eliminated into bile (Haritha, 2019) [12]. Jaundice is brought on by an accumulation of bilirubin as a result of biliary secretion failure (Sharma *et al*., 2007) [28]. Phylloerythrin buildup leads to photosensitization (Dervash *et al*., 2025) [7].

3.4.1. Mechanism of action (MOA)
Hepatocytes and bile canaliculi are affected by lantadenes, which cause both cell types to necrotize. They generate cholestasis and liver necrosis, have direct cytotoxic effects, and affect intermediate metabolic pathways. Gastroenteritis may also be brought on by these direct cytotoxic consequences.

3.4.2 Clinical signs
Appetite loss, photosensitization, icteric mucous membrane, and discoloration.

3.4.3 Specific Treatment
- Laxatives are given as part of the general course of treatment to help the body eliminate itself of toxins.
- Toxins in the rumen can be adsorbed and further absorption can be inhibited by administering activated charcoal.

3.5 Brakenfern poisoniong
Bracken fern is a deadly weed that are not preferred by ruminants due to high silicon concentration that decreases its palatability (Smith, 2020) [30]. It is found all across the world, growing in open pastures, hillsides, forests, and other shady areas. After consuming a lot of plants, illnesses in cows typically manifest acutely. Ptaquiloside, a nor-sesquiterpene glycoside found in plants, produces aplastic anemia in cattle with a delayed beginning of action. Bovine enzootic haematuria, which results from prolonged intake, is what gives urine its distinctive crimson hue (Lopes *et al*., 2019) [19].

3.5.1 Mechanism of Action (MOA)
Ptaquiloside and thiaminase are more frequently linked to poisoning. Bracken fern consumption produces neurotoxic syndrome and thiamine poisoning in animals by releasing the toxin thiaminase. Red urine is a sign of bovine enzootic haematuria, which is caused by consuming too many of these plants (Vetter 2010) [35].

3.5.2 Clinical Sign
High temperature, appetite loss, depression, dyspnea, excessive salivation, nasal and rectal bleeding, hematuria, and mucous membrane hemorrhages are among the clinical symptoms in sheep and cattle. Bladder tumors, leukopenia, thrombocytopenia, and anemia in cattle (Ugochukwu, 2019) [33].

3.5.3 Specific Treatment
- Saline cathartics, activated charcoal, and thiamine hydrochloride can all be used to treat poisoning.
- The best treatment for cattle is blood transfusions. Adults need 1 to 4 liters, while calves should get 0.4 liters. Heparin's effects are offset with a 3% sodium citrate solution.

3.6 Parthenium Weed Poisoning
Parthenium weed is a photodynamic substance. When Parthenium weed is consumed, it causes primary photosensitization, which can lead to skin allergic reactions and liver pathology since the weed contains parthenin, a photodynamic agent (Kumar & Aggarwal, 2024) [17].

3.6.1 Mechanism of Action (MOA)
A photodynamic material is the parthenium weed. Therefore, consumption of pathenium weed causes primary photo-sensitization, which results in liver disease and skin sensitivity, as is observed with other photodynamic drugs. Primary photosensitization, liver damage, and skin reactions are caused by parthenin, a photodynamic substance found in parthenium.

3.6.2 Clinical Signs
Pigment Loss, Mouth Ulcers, Pruritus, Eye Irritation, Rheumatism, Diarrhea, Utis, and Dysentery, Malaria, Neuralgia, and Alopecia, outbreaks of erythema across the body. Skin depigmentation and alopecia.

3.6.3 Specific Treatment
Antipruritics and hepatic medications are commonly utilized in treatment (Hussain *et al*., 2018) [14].

3.7 *Anagallis arvensis* poisoning
In the Marathwada region of Maharashtra state, *Anagallis arvensis* (Family: Primulaceae), often known as blue pimpernel, is an intercrop weed that is mostly found in crops including turmeric, wheat, maize, and cotton. The square stems of this little perennial herb branch out from the base. The glossy, stalkless leaves are on the other side. It grows in semi-exposed areas, gardens, waste areas, and damp soil. Glycosides, volatile oil, saponin (anagallin), tannin, and oxalates are among the toxic active principles found in this weed's edible portions.

3.7.1 Mechanism of Action
Higher blood calcium oxalate concentrations brought on by *Anagallis arvensis* toxicity result in oxalate nephrosis, a condition that causes renal insufficiency and the buildup of sensitive renal markers like creatinine and urea nitrogen. Because calcium oxalates are essentially insoluble, they have a tendency to precipitate in renal tubules at larger quantities, which can result in nephrosis. As a result, the kidney is the first organ to be impacted by the poisoning of oxalic acid (Rana *et al*., 2018) [25].

3.7.2 Clinical Signs
Anorexia, depression, stumbling gait, hind limb lameness with recumbency, and swelling in the perineal area are among the clinical symptoms displayed by cattle intoxicated with *Anagallis arvensis*. Significant hypothermia, tachycardia, tachypnea, and reduced ruminal motility are also present in affected animals (Al-Sultan *et al*., 2003) [2].

3.7.3 Specific Treatment
The main treatment is slow IV calcium borogluconate at a dose of 450 ml per cow.

3.8 Gossypol Poisoning
Gossypol is a major pigment and strong toxin found in cottonseed. Pigment glands in the cotton stem, leaves, seeds, and flower buds create gossypol. However, gossypol's use in animal feed is restricted due to its toxicity. Consuming cottonseed or cottonseed products containing sufficient free gossypol can result in gossypol poisoning, which is typically chronic, cumulative, and occasionally sneaky.

3.8.1 Specific Treatment
- The rate at which gossypol is absorbed is inversely correlated with the amount of iron in the food, addition of ferrous sulfate to the diet deactivates free gossypol.
- For ten to fifteen days, take two Boli of ferrous fumerate (Feritas Bolus) every day (Sihag *et al*., 2020) [29].

4. Conclusion
A lot of poisonous plants and weeds for our household animals. Therefore, if you have domestic animals in your home, you should avoid harmful plants and weeds. Pay attention to what they take when they go grazing. Additionally, visit the veterinarian as soon as you can if it occurs. The current paper will help field veterinarians become more knowledgeable about early diagnosis and appropriate treatment for a variety of suspected animal plant poisoning cases.

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Not available

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