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Advances in diagnosis and management of animal toxicity due to toxic plants in India

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Review Article

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ABSTRACT

Plant poisoning is emerging as a serious threat to animals as well as humans in all around the world. In India, this issue is particularly prevalent, characterized by a high incidence of poisoning cases, notably in rural regions where livestock are more frequently subjected to toxic flora. This phenomenon is frequently attributed to conventional practices that involve the utilization of phytochemical agents, in conjunction with the presence of potentially harmful plants in grazing territories. Such plants typically harbour toxic constituents including alkaloids, glycosides, oxalates, and nitrates. The impact of these toxins may vary according to the specific plant species and the amount ingested, affecting multiple organ systems and resulting in gastrointestinal, hepatic, renal, cardiovascular, or neurological impairments. In extreme instances, plant poisoning may pose a life-threatening risk if not identified and addressed in a timely manner. The probability of poisoning is further exacerbated during times of fodder deficiency, when both the quality and quantity of green forage diminish, compelling animals to ingest unpalatable or deleterious plants. Delayed recognition or late initiation of treatment often results in poor prognosis and high mortality rates. However, with the increasing incidence of plant poisoning, significant advances have been made in diagnostic and management strategies. This review covers toxic plants' risks to animal health, detailing their toxic compounds, mechanisms, clinical signs, and pathological findings, as well as recent advances in diagnosis and management strategies.

Keywords: Toxic plants, Animal poisoning, Advancement, Diagnosis, Management.

INTRODUCTION

Grazing, although beneficial for providing fresh forage and promoting animal health can also expose livestock to various illnesses and accidents. This risk increases when the availability and quality of green fodder decline, prompting animals to consume unfamiliar or unpalatable vegetation, including toxic plants. Plant poisoning in animals is a significant concern worldwide, influencing both domesticated and wild species. Numerous toxic floras encompass harmful compounds that may induce both acute and chronic health complications, ranging from gastrointestinal disturbances and organ dysfunction to neurological impairments and potential death. Such occurrences are documented across a variety of climatic zones and geographical landscapes, establishing toxic plant consumption as a worldwide concern for animal health [1].

Regions like South America and South Africa experience substantial livestock losses due to toxic plant consumption. Alkaloids, glycosides, cyanogenic glycosides, oxalates, and nitrates are the common compounds present within these toxic plants, with effects ranging from liver injury (e.g. *Lantana camara*, *Crotalaria spp.*) to cardiac arrest (*e.g.* cardiac glycoside plants like *Thevetia*, *Nerium*). Emerging threats include ornamental or imported plants causing new patterns of poisoning in pets and livestock, especially across Europe under changing climate and plant import dynamics [2].

India is home to numerous toxic plants, leading to frequent incidents of poisoning in humans as well as animals. A study found that 6-15% of poisoning cases in humans involved plants, which includes oleander, datura and castor. India's Himalayan region harbors some of the world's most toxic flora like *Aconitum ferox*, China berry and Castor bean [3]. Across regions, risk factors include drought, new pasture introductions, accidental ingestion, and plant stress. Economically, the direct losses (including mortality, abortions, and diminished quality of milk or hides) and the indirect expenses (pertaining to treatment and management) are significant on a global scale. In India, the paucity of institutional data poses a considerable obstacle to both public health initiatives and veterinary interventions. Concurrently, with the rise in incidents involving toxic flora, there has been a corresponding advancement in diagnostic and management techniques that facilitate the early detection of toxic plants, thereby potentially preserving animal life through various enhanced management strategies [4].

COMMON TOXIC PLANTS OF INDIA AND ASSOCIATED TOXICITY

1. Oleander Poisoning

Common Name- Oleander, Nerium, Kaner

The oleander is a shrub that is abundantly distributed in tropical climates, including India, represents a frequent source of toxicity in animals. Both *Nerium oleander* (Pink oleander or Kaner or Karavira) and *Thevetia peruviana* (Yellow Oleander) (Figure 1) belong to the same family, Apocynaceae, and are known to produce cardenolide glycosides [5].

Figure 1: *Thevetia peruviana* (Yellow Oleander)

Species Affected: Cattle, sheep, goat, horses and dogs are most frequently affected. Cats and poultry are affected occasionally.

Toxic Principle: The toxic principles in *Nerium oleander* are Oleandrin, oleandrine and oleandrigenin and those in *Thevetia peruviana* are Thevetin A and Thevetin B. The entire plant of oleander is considered toxic. It has been documented that the ingestion of a singular leaf can result in fatal consequences [6].

Mechanism of Toxicity: Both synthesize cardiac glycosides that work similar to digoxin, by inhibiting the Na-K-ATPase pump within cardiac myocytes. This inhibition results in an accumulation of intracellular sodium (Na) that influences the sodium-calcium exchanger, resulting in an elevation of intracellular calcium (Ca) levels which in turn enhances cardiac contractility [7].

Clinical Signs and Pathology*:* Initial clinical manifestations comprise of nausea, vomiting, abdominal pain, and diarrhoea [8]. Progressive symptoms include cardiovascular manifestations characterized by varying degrees of atrio-ventricular block, acute myocardial infarction (in case of yellow oleander poisoning) as well as ventricular tachycardia and ventricular fibrillation, which are deemed severe [9]. Neurological manifestations include tremors, drowsiness, ataxia, and seizures. Most of the deaths occur within first 24 hours of ingestion. 5-15 leaves or 15 gm of root in case of pink oleander and 8-10 seeds or 15-20 gm of root in case of yellow oleander is considered as lethal. In general, the toxicity caused by yellow oleander is more compared to pink oleander [10].

Case Highlights: Sykes et al [11] examined 21 documented cases of naturally occurring oleander poisoning in horses, confirmed by the presence of oleandrin in their samples out of which microscopic renal lesions were noted in all the cases and out of which gross renal lesions were observed in 7 of them showing congestion, haemorrhages and swollen kidneys. In Italy, 50 dairy cows were poisoned after accidentally ingesting oleander mixed with their fodder, resulting in 13 deaths. Oleandrin was detected in milk and cheese, highlighting a potential health risk for consumers. This incident marks the first documented case of oleandrin transfer into dairy products from poisoned animals [12].

Diagnosis: Diagnosis is based on history, clinical signs, vomitus analysis, ECG, and serum potassium levels, with severity assessed by factors like amount ingested, time elapsed, ECG abnormalities, and hyperkalemia [13-14].

Rubini et al [15] developed a method using liquid chromatography-high resolution mass spectrometry (LC-HRMS) to detect and quantify oleandrin in ruminal contents and forage materials.

Management: Management involves gastrointestinal decontamination, treating hyperkalemia, administering digoxin antibodies, supportive care, and monitoring, with potential consideration for temporary pacing [16].

2. *Pteridium aquilinum* (Bracken fern) poisoning

Common Name- Bracken fern

Bracken ferns (*Pteridium* genus) (Figure 2) are one of the most common plants on Earth, found in temperate and subtropical regions worldwide. They thrive in diverse environments, except extremely arid, waterlogged, or frosty areas, with growth cycles varying between temperate (seasonal) and tropical (persistent) regions [17].

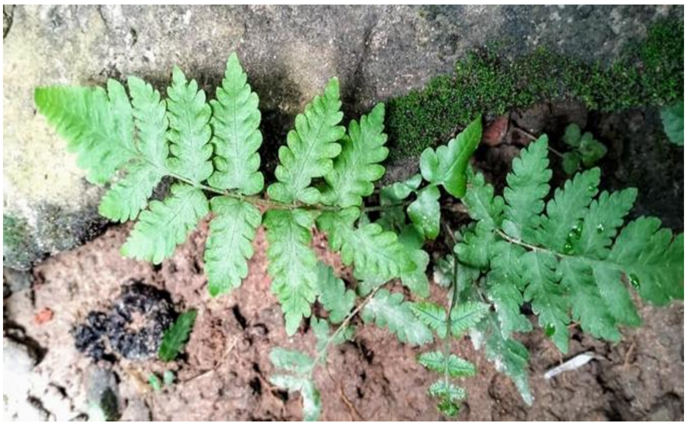

Figure 2: *Pteridium aquilinum* (Bracken fern)

Species Affected: Cattle and horses are affected most frequently compared to sheep and farmed rabbits.

Toxic Principle: Bracken fern toxicity is attributed to various compounds, including illudane-type glycosides (ITGs), with ptaquiloside (PTQ) being the most studied. Other ITGs include caudatoside, ptesculentoside, and isoptaquiloside [18]. Bracken-derived compounds, particularly PTQ, can leach into water sources, contaminating surface water, groundwater, and even drinking water supplies [19-20].

Mechanism of Toxicity*:* Bracken fern consumption causes thiamine deficiency due to the presence on thiaminase enzyme within the plant.

Clinical Signs and Pathology: In non-ruminants, bracken fern consumption causes thiamine deficiency. Besides this, consumption of this plant in ruminants causes acute poisoning, anaemia, leukopenia, urinary bladder tumours and chronic intermittent haematuria in cattle and retinal degeneration in sheep and goat [21]. Ptaquiloside (PTQ) and related compounds are identified as the mutagenic and carcinogenic agents [22]. Bracken fern habitats also harbour ticks (*Ixodes* spp.) that transmit diseases like Lyme borreliosis and others [23].

Research suggests that PTQ from bracken fern may contribute to human cancer development by causing specific mutations, suppressing the immune system, and potentially interacting with viruses like human papillomavirus and Helicobacter pylori to promote cancer growth [24]. Post mortem lesions revealed pale bone marrow, petechial and ecchymotic haemorrhages on mucous membranes [25]. Histopathological studies revealed abnormal cell growths in the urinary bladder, some attached by stalks or broad bases, with associated inflammatory and precancerous changes. In the esophagus, cancerous cells and ulcers were observed in the mucosal lining [21].

Case Highlights: A study in southern Brazil found that cattle with squamous cell carcinomas (SCCs) in the upper digestive tract showed symptoms like weight loss, difficulty swallowing, and coughing. The cancers were most common in the oropharynx and rumen, with many cases having metastases to nearby lymph nodes. Papillomas were also present in all affected cattle [26]. Another study in Himachal Pradesh, India, found a link between cattle eating ferns and Enzootic Bovine Haematuria (EBH), a condition causing urinary tract issues. Out of 103 plant samples, 95 were ferns, suggesting fern ingestion as the likely cause of EBH in the affected cattle [27].

Diagnosis: Diagnosis is based on history, clinical signs, and lesions, with haematological findings showing thrombocytopenia, decreased RBC and WBC counts due to bone marrow suppression. Bracken fern poisoning in farm-raised rabbits and ruminants can cause severe anemia, low white blood cell count, and changes in blood chemistry, including elevated liver enzymes (AST, ALT, ALP), increased urea and creatinine, and decreased thiamine, protein, and albumin levels [21].

Management: No specific antidote is available. In cattle, blood transfusion is required. Batyl alcohol (2 gm), Tween 80 (5 gm) and 1 % NaCl (100 ml) @20-50 ml/day. Batyl alcohol is known to stimulate the bone marrow. Supplementations of antihistamines, antibiotics, vitamin B complex and oral ruminotorics were given to stimulate appetite [28].

Control methods include cutting, physical removal, herbicides, and alternative land use like tree planting or harvesting for biofuel, which can improve grazing land, reduce tick-borne diseases, and promote biodiversity [29].

3. *Canabis sativa* Poisoning

Common Name- Hemp, Marijuana, Bhang, Ganja, Jia

Cannabis (Figure 3), an Asian herb, can refer to hemp (≤0.3% THC) or marijuana (>0.3% THC), with THC (tetrahydrocannabinol) being the psychoactive compound found in varying concentrations, especially in trichomes and female flowers [30].

Figure 3: *Canabis sativa* (Marijuana)

Species Affected: Dog (most common), cat, horse, birds (e.g., cockatoos), reptiles (e.g., iguanas), ferrets

Toxic Principle: Tetrahydrocannabinol (THC) is the main psychoactive cannabinoid. Other cannabinoids include cannabidiol and cannabinol.

Mechanism of Toxicity: CBD interacts with brain receptors, affecting neurotransmitters like norepinephrine, dopamine, and serotonin [31].

Clinical Signs and Pathology: As cannabis use rises, pets are at increased risk of exposure. In dogs and cats, cannabis toxicosis can cause symptoms such as lethargy, vomiting, seizures, and abnormal heart rates. Dogs may experience anxiety and hallucinations, while cats may develop bradycardia and respiratory depression. Most dogs recover within hours, but prompt veterinary care is essential [30].

Case Highlights: A survey conducted in North America on the prevalence of cannabis toxicosis in pets found that cases were reported most often in dogs, followed by other species such as cats, ferrets, horses, iguanas, and cockatoos. Both Canada and the United States showed a marked rise in reported cases. The most frequently observed clinical signs included urinary incontinence followed by disorientation, ataxia, lethargy, hyperesthesia and bradycardia [32].

Another study in Colombia found that 32.37% of 349 cases reviewed were cannabis poisoning in dogs. Puppies and small breeds were most affected. Edible cannabis products were the main source of exposure (67.27%), followed by ingesting the plant and inhaling smoke [33].

Another study was conducted in Michigan State University and Texas A & M University with a total of 223 dogs that ingested marijuana found common symptoms included ataxia (88.3%), hyperesthesia (75.3%), lethargy (62.5%), urinary incontinence (45.7%), and vomiting (26%). Symptoms typically appeared within 1.25 hours and most dogs showed signs within 4 hours [34].

Diagnosis: Diagnosing cannabis toxicity relies on exposure history and symptoms. Detecting cannabinoids in body fluids is challenging due to their low concentrations and high lipid solubility. Advanced tests like GLC (Gas-liquid chromatography), HPLC (High-Performance Liquid Chromatography), TLC (Thin Layer Chromatography) and GC-MS (Gas Chromatography-Mass Spectrometry) are needed for confirmation, as common urine tests can yield false negatives or be affected by adulterants [35]. Serum biochemistry in cases of marijuana toxicosis often reveals mild hyperkalemia and mild hypercalcemia [36]. More recently, a novel technique Ultra-Performance Liquid Chromatography-Tandem Mass Spectrometry (UPLC-MS/MS) has been developed to detect and quantify marijuana metabolites in the urine and serum of animals suspected of cannabis intoxication [37].

Management: Treatment involves decontamination within 30 minutes, supportive care like fluid therapy and temperature control, and medications for sedation, seizures, and respiratory support. In severe cases, IV lipid emulsion therapy may be used. Most animals recover within 1-3 days, but fatalities can occur in rare cases [38-39].

4. Castor Bean (*Ricinus communis*) poisoning

Common Name- Castor oil plant

The Castor bean plant (*Ricinus communis*) (Figure 4) is a versatile plant originating from Africa, now found globally in subtropical and tropical regions. It is used in industries for lubricants, cosmetics, and potentially biofuel production, with India, China, and Brazil being major producers [40].

Species Affected: Horses being most sensitive and chickens most resistant. Other animals like cattle, sheep, goat, pig, dog and cat also affected.

Figure 4: *Ricinus communis* (Castor Bean)

Toxic Principle: The main toxic principal present is ricin, which is mainly present in seeds and can be used as animal feed or fertilizer after detoxification [40].

Mechanism of Toxicity: Ricin, found in seeds (up to 5%), disrupts cellular function by inhibiting protein synthesis and inducing cell death. Its effects are time and dose-dependent, with a delayed onset of symptoms (often 6-24 hours) [41].

Clinical Signs and Pathology: Symptoms in animals include weakness, diarrhoea, dehydration, and respiratory distress, similar to those in humans [42]. On post mortem analysis, there may be severe gastroenteritis with haemorrhages in the mucosa of GIT and respiratory tract, hepatic and renal degeneration. In fowl, there is inflammation of crop, oesophagus and proventriculus [43].

Case Highlights: Ricin poisoning cases have been reported globally in domestic animals, particularly dogs, due to ingestion of castor bean seeds or contaminated products. Fatality rates vary, with a study reporting 7%, while analysis showed higher rates of 35.3% in Germany and 23.5% globally, suggesting dogs are highly susceptible to ricin toxicity. Stricter regulations on ricin content in fertilizers are needed [44].

Diagnosis: Diagnosing ricin poisoning involves clinical signs, exposure history, and lab tests. Symptoms in dogs include vomiting, diarrhea, and abdominal pain. Lab tests like ELISA can detect ricin with high sensitivity, while faster tests like lateral-flow assays are available for on-site detection but are less sensitive [45].

Apart from antibodies, DNA/RNA aptamers can detect ricin. However, both methods may yield false positives. Mass spectrometry (MS) technologies, like MALDI-TOF or LC-MS/MS, can accurately identify ricin with high sensitivity, making them a powerful tool for detection in complex samples [46].

Management: There is no approved antidote for ricin poisoning. Treatment involves supportive care, such as IV fluids and managing symptoms. Anti-ricin serum and antibodies have shown promise, particularly if administered soon after exposure. Research is ongoing to develop humanized monoclonal antibodies to reduce side effects and improve treatment efficacy [47].

Researchers are exploring small molecules, glycostructures, and aptamers to counter ricin toxicity, with some promising results in animal studies [48]. A vaccine, RiVax, is in advanced stages of approval, showing potential in inducing protective antibodies against ricin [49].

5. *Lantana camara* Poisoning

Common Name- Lantana, Red sage

Lantana camara (Figure 5) is a flowering plant that thrives in tropical and subtropical climates. It is widely known for its colourful, clustered flowers that can be red, orange, yellow, pink, or white, often changing colours as they age. While it is valued for its ornamental beauty, it is also considered a highly invasive weed in many regions, including India, Australia, and Africa [50].

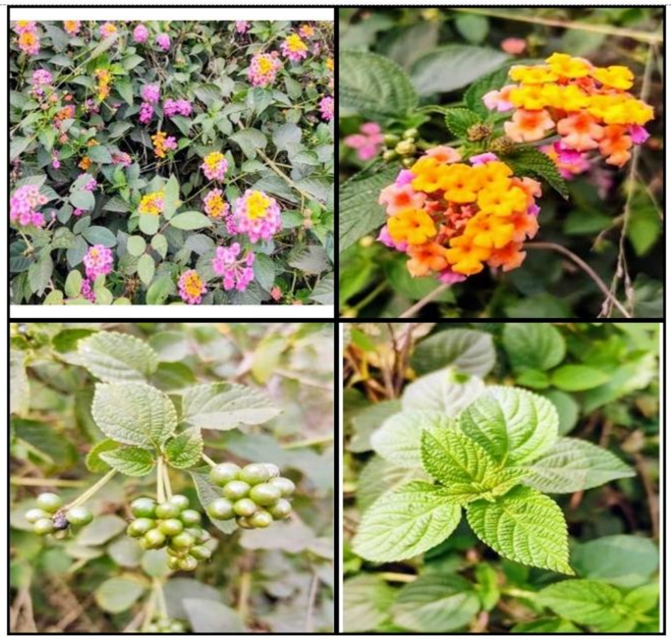

Figure 5: *Lantana camara*

Species Affected: Ruminants (Cattle, sheep and goat) are most common, horses (rare)

Toxic Principle: The toxicity of *Lantana camara* in animals, especially in ruminants is mainly due to pentacyclic triterpenoids, primarily lantadenes (A, B, C, D) found in the leaves and unripe berries of the plant.

Mechanism of Toxicity: After ingestion, lantadenes are absorbed into the small intestine which then transported to the liver where they undergo biotransformation and causes hepatocellular injury, particularly affecting bile canaliculi which finally results in Cholestasis. Due to this, phylloerythrin (a chlorophyll breakdown product) accumulates in the blood. This leads to secondary photosensitization when phylloerythrin reacts with sunlight, causing skin damage in non-pigmented areas [51].

Clinical Signs and Pathology: The toxic dose is 4-8 gm/ kg body weight. Within 24–48 hours of ingestion the animal develops anorexia, depression, constipation and ruminal stasis. Then, it progresses to Icterus, photodermatitis (especially on light-coloured skin), ulceration, dry faeces and haematuria, which leads to hepatic failure and death in severe cases [50]. On necropsy findings, there is enlarged, yellowish and ochre coloured liver with bile retention, Bile duct proliferation and fibrosis (chronic cases) and photosensitized skin lesions [52].

Case Highlights: A cattle herd exposed to Lantana camara during a storm resulted in 74 deaths out of 170. Symptoms included severe photosensitivity, jaundice, liver damage, and kidney issues, developing over 2-15 days [53]. *Lantana camara* poisoning is common in India, particularly in Himachal Pradesh. A study found highest toxicity levels in Mandi, Shimla, and Kullu districts, with more cases during the rainy season. Mature leaves were more toxic than immature ones due to higher lantadene levels [54].

Diagnosis: Diagnosis is based on clinical signs like liver dysfunction, photosensitization, and recent exposure. Lab tests show elevated liver enzymes, hyperbilirubinemia, hypoalbuminemia, and increased BUN/creatinine if kidneys are affected. Recent research has moved towards specific detection of lantana toxins using HPLC, LC–MS MS and Fourier-Transform Infrared Spectroscopy (FTIR) [55].

Management: Treatment is symptomatic, involving measures like keeping animals out of sunlight, administering antihistamines, antibiotics, and hepato-protective agents. Activated charcoal can be given to bind toxins if ingestion is recent [56].

6. Sorghum Poisoning

Common Name- Jowar, Great millet, guinea corn, cholam

Sorghum bicolor (Figure 6) (formally *Sorghum vulgaris*) is a cereal crop, erect grass-like plant mainly grown in arid and semi-arid regions of the world, particularly in Africa, Asia, and parts of India. It is rich in carbohydrates, dietary fibre, and micronutrients like iron and phosphorus and also contains antioxidants and phytochemicals [57].

Figure 6: *Sorghum bicolor*

Species Affected: While Sorghum is commonly used as fodder and forage, under certain conditions, it can become toxic to animals, particularly ruminants (cattle, sheep, goats) and horses (occasionally).

Toxic Principle: The main toxic compound is Dhurrin (a cyanogenic glycoside) which is present in young or regrowing sorghum plants. In India, this plant is the most common source for cyanogenic plant poisoning in livestock [58]. Besides, the plant can also accumulate nitrates in its stems and leaves under heavy nitrogen fertilization, drought stress, cloudy or cold weather [59].

Mechanism of Toxicity: When the animals ingest the plant, dhurrin is hydrolyzed by enzymes (beta-glucosidases) into hydrogen cyanide (HCN) which is absorbed into the bloodstream and binds to cytochrome oxidase enzymes in mitochondria. This prevents cells from using oxygen leading to cellular hypoxia even when oxygen is present in the blood [58]. Similarly, after ingestion, the nitrates get converted into nitrites in the rumen which oxidizes haemoglobin to methaemoglobin, which cannot carry oxygen resulting to tissue hypoxia.

Clinical Signs and Pathology: Clinical signs due to HCN poisoning include rapid breathing, muscle tremors, salivation, bright red mucous membranes, collapse and sudden death due to rapid respiratory failure. Post mortem finding include congestion in the trachea, abomasum and small intestine, bitter almond smell of the rumen, bright red unclotted blood. On nitrate toxicity, the animal experienced laboured breathing, cyanosis, chocolate coloured blood, weakness and death in severe cases [60].

Diagnosis: Diagnosis is based on sudden death after grazing, bright red blood, and detection of hydrogen cyanide in tissues using the picrate paper test, which turns reddish-brown in positive cases [61]. It should be followed by GC-MS for confirmation [62]. Nitrate/nitrite poisoning in animals can be diagnosed initially with diphenylamine (DPA) Spot Test, followed by spectrophotometric methods, Ion Chromatography, Flow Injection Analysis (FIA) and HPLC for confirmatory diagnosis [63]. Recent research is making nitrate detection faster, portable, and ultra-sensitive through enzyme-based biosensors, nanoparticle-enhanced sensors and paper microfluidic devices [64].

Management:

Cyanide Poisoning

Sodium nitrite (20% solution at the rate of 15–25 mg/kg body weight IV slowly) followed by Sodium thiosulfate (25% solution at the rate of 500–660 mg/kg body weight IV). Sodium nitrite converts haemoglobin to methaemoglobin, which binds cyanide and forms cyanomethaemoglobin followed by administration of sodium thiosulfate that converts cyanide into thiocyanate, which is excreted in urine. Along with that, oxygen therapy, keeping animal calm and reduce stress and activated charcoal (if ingestion was recent) can be used as a supportive care [65].

Nitrate/Nitrite Poisoning

Methylene blue (1% solution at the rate of 1–4 mg/kg body weight IV slowly) as it reduces methaemoglobin back to functional haemoglobin. Along with that provide fresh water and remove nitrate-rich feed and offer high-energy diet to support rumen microbes [60].

7. *Calotropis gigantea* (milk weed) Poisoning

Common Name- Giant Milkweed, Crown flower

Calotropis gigantea (Figure 7) is a perennial shrub commonly found in tropical and subtropical regions of Asia and Africa. Livestock can be accidentally poisoned by this plant when grazing during fodder scarcity or when its leaves are mixed with other fodder [66].

Figure 7: *Calotropis gigantea*

Species Affected: Goats, sheep and cattle are most frequently affected with this plant. Equines and camels are occasionally affected.

Toxic Principle: The toxic compounds present are calotroxin, calactin, gigantin and uscharin. All parts of the plant are toxic [67].

Mechanism of Toxicity: They inhibit Na^+/K^+-ATPase in cell membranes which lead to increased contractility initially but later cause arrhythmias and cardiac arrest [67].

Clinical Signs and Pathology: Irritation to skin and mucous membranes, acute gastroenteritis and cardiotoxicity. On post mortem analysis, there is congestion of gastrointestinal mucosa and hepatic sinusoids, myocardial and renal tubular degeneration [68].

Diagnosis: Based on history, clinical signs and post mortem analysis. On laboratory tests, ECG should be done on suspected cases where arrhythmias (AV block, ventricular tachycardia, and fibrillation) can be observed along with hyperkalaemia on serum biochemistry. Besides, cardiac glycosides can also be detectable in gastrointestinal contents, serum and liver using specialised techniques like LC-MS or TLC [67].

Management: Only symptomatic treatments can be done like gastric lavage with activated charcoal, anti-arrhythmic drugs like atropine and lidocaine and fluid therapy. In severe cases if ingested in high amount, emergency rumenotomy should be performed [68].

8. *Parthenium hysterophorus* poisoning

Common Name- Santa-Maria, Congress grass, Carrot weed

Parthenium hysterophorus (Figure 8) was native to America, was accidentally introduced to India in the 1960s through contaminated wheat imports. It first appeared in Pune, Maharashtra, and spread rapidly across the country, thriving in crops, fields, and wastelands [69].

Figure 8: *Parthenium hysterophorus*

Species Affected: Cattle, buffaloes and goats are most often affected, while sheep, equines and camels are only occasionally involved. Cases in dogs, cats and pigs are rare.

Toxic Principle: The major toxic compound present in this weed is parthenin which is a photodynamic substance. The milk of affected cattle appears bitter in taste due to the presence of parthenin in milk [70].

Mechanism of Toxicity: Ingestion of plant causes primary photosensitization, causing liver damage and skin lesions. In human, allergic contact dermatitis has noted in farm labourers those who engaged in removal of this plant manually. In India, the first incidence of parthenium-induced contact dermatitis in farm labourers reported at Pune (Maharashtra) in the year 1971 [69].

Clinical Signs and Pathology: As it is a photodynamic substance, there will be cutaneous lesions like erythematous eruptions and patches on the areas with direct contact with sunlight. On necropsy finding, there will be degenerative changes in live, lungs and kidneys [69].

Cases Highlights: *Parthenium hysterophorus* is considered as a global threat as an invasive species to many countries. A comprehensive investigation conducted by Khan and Fahad [71] showed that *Parthenium* significantly reduces agricultural yields (up to 40%) and causes health issues like skin irritation, asthma, and allergies in humans and animals. It is highly invasive, producing many seeds that can remain viable for up to 10 years and spread through various means.

Diagnosis: Based on history of grazing in fields heavily infested with these weeds and presence of cutaneous lesions. Serum biochemistry reveals marked increase in liver enzymes like ALT, AST, ALP and GGT [72].

Management: There is no specific treatment. Only symptomatic treatments can be done like treating cutaneous lesions with antihistamines, antibiotics for secondary bacterial infection and topical ointments and also improving liver functions with some hepatoprotective agents. The animals should be kept in out from direct sunlight [72].

Control: *Parthenium hysterophorus* can be controlled through manual uprooting, chemical herbicides like glyphosate, or biological methods using insects like Zygogramma bicolorata and Epiblema strenuana, as well as rust fungi. Protective gear like gloves is recommended during manual removal [71].

9. *Ipomoea carnea* poisoning

Common Name- Pink or Bush morning glory

Ipomoea carnea (Figure 9) is a perennial shrub found in tropical and subtropical regions, including India. It is a hardy, drought-tolerant plant that grows in various areas and contains toxic compounds that can harm livestock with prolonged ingestion [73].

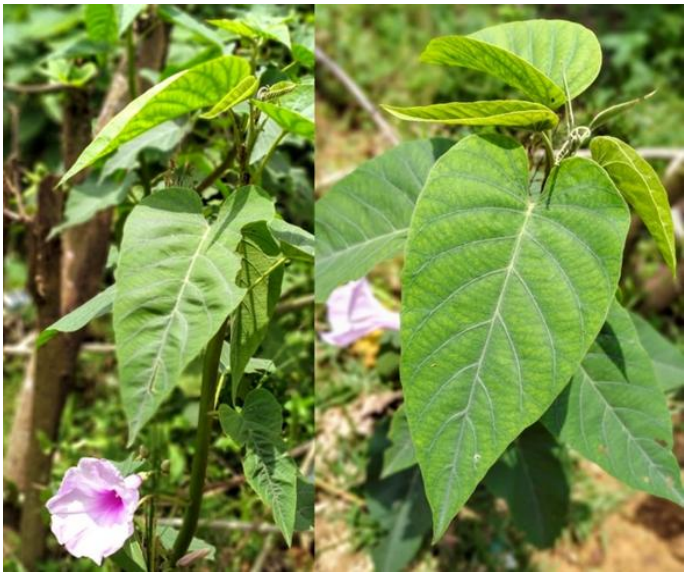

Figure 9: *Ipomoea carnea*

Species Affected: Goats and sheep (highly susceptible), cattle and buffaloes (occasionally), and equines (rarely).

Toxic Principle: Swainsonine (indolizidine alkaloid) and calystegines (polyhydroxy nortropane alkaloids) are the main toxic compounds of these types of plants.

Mechanism of Toxicity: Swainsonine causes a lysosomal storage disorder by inhibiting α-mannosidase, leading to oligosaccharide buildup, neuronal damage, and resulting in neurological and behavioral issues [74].

Clinical Signs and Pathology: Affected animals exhibit neurological signs such as incoordination, ataxia, and muscle tremors, with lumber paralysis being common in goat and sheep. Additional signs include loss of body condition, reduced milk production, infertility, and abortion in severe cases. Post-mortem findings reveal vacuolation in neurons (notably in cerebellar Purkinje cells and brainstem nuclei), endocrine glands, and hepatic degeneration [74].

Diagnosis: Based on history, clinical signs and histopathology (Neuronal and visceral cell vacuolation).

Management: Treatment involves symptomatic and supportive therapies, including Vitamin B complex, antioxidants, and liver tonics, alongside providing a nutritious and balanced diet with high-quality fodder to support overall health [75].

CONCLUSION

Plant poisoning in animals is a significant global veterinary and economic challenge, with its impact being particularly pronounced in biodiverse nations such as India. The vast diversity of flora across different agro-climatic zones increases the likelihood of livestock coming into contact with toxic plant species. The consequences of plant poisoning extend beyond animal mortality; they lead to reduced productivity, reproductive losses, and long-term economic burdens for farmers and the livestock industry. This review spotlights the toxic compounds, underlying mechanism of toxicity and incidence of poisoning from local poisonous plants. It also covers recent diagnostic advances, especially various spectrometric techniques and treatment options, aiming for earlier toxin detection and better recovery. Besides this, farmer and veterinarian education is a crucial component, ensuring that those directly handling animals can recognize early signs of poisoning and take swift action. In parallel, advancements in diagnostic techniques ranging from field-level rapid tests to sophisticated laboratory analyses must be integrated into routine veterinary practice. Development and dissemination of evidence-based management and treatment protocols will further improve survival rates and minimize economic losses. By combining proactive prevention, early detection, and effective intervention, plant poisoning in animals can be significantly reduced, safeguarding animal welfare and rural livelihoods both in India and globally.

Conflict of interest

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