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Pragnyasmita Mishra
Department of Veterinary pathology, College of Veterinary Science and animal Husbandry, Odisha University of Agriculture and Technology, Bhubaneswar, Odisha, India

Prasana Kumar Rath
Department of Veterinary pathology, College of Veterinary Science and animal Husbandry, Odisha University of Agriculture and Technology, Bhubaneswar, Odisha, India

Pratyastha Singh
Department of Veterinary pathology, College of Veterinary Science and animal Husbandry, Odisha University of Agriculture and Technology, Bhubaneswar, Odisha, India

Jeevan Ranjan Dash
Department of Veterinary Pharmacology and Toxicology, College of Veterinary Science and animal Husbandry, Odisha University of Agriculture and Technology, Bhubaneswar, Odisha, India

Debasish Hota
Department of Veterinary pathology, College of Veterinary Science and animal Husbandry, Odisha University of Agriculture and Technology, Bhubaneswar, Odisha, India

More Sumit Vitthal
Department of Veterinary pathology, College of Veterinary Science and animal Husbandry, Odisha University of Agriculture and Technology, Bhubaneswar, Odisha, India

Sudhir Kumar Sahu
Department of Veterinary Microbiology, College of Veterinary Science and animal Husbandry, Odisha University of Agriculture and Technology, Bhubaneswar, Odisha, India

Arundhati Nayak
Department of Veterinary Medicine, College of Veterinary Science and animal Husbandry, Odisha University of Agriculture and Technology, Bhubaneswar, Odisha, India

Keertti Mohanty
Internee Scholar, College of Veterinary Science and Animal Husbandry, Odisha University of Agriculture and Technology, Bhubaneswar, Odisha, India

Kalpita Subhalagna Dey
Internee Scholar, College of Veterinary Science and Animal Husbandry, Odisha University of Agriculture and Technology, Bhubaneswar, Odisha, India

Corresponding Author:
Pragnyasmita Mishra
Department of Veterinary pathology, College of Veterinary Science and animal Husbandry, Odisha University of Agriculture and Technology, Bhubaneswar, Odisha, India

An insight to different plant poisoning in domestic animals: An overview

Pragnyasmita Mishra, Prasana Kumar Rath, Pratyastha Singh, Jeevan Ranjan Dash, Debasish Hota, More Sumit Vitthal, Sudhir Kumar Sahu, Arundhati Nayak, Keertti Mohanty and Kalpita Subhalagna Dey

Abstract
Domestic animal may ingest toxic plants accidentally while grazing. Sometimes they get bored with the same regular feed, so, used to consume unpalatable weeds or ornamental plants growing along fences. The severity of poisoning depends on quantity consumed, species of animal, portion and condition of the plant, age and health of the animal etc. We often underestimate the danger from poisonous plants to our domestic animals. They might be so toxic that a small quantity can cause serious illness even death of the animals. Thus it is important to know which plants are toxic to domestic animals.

Keywords: Domestic animals, phytotoxin, phytotoxicity, remedies

1. Introduction
Plant toxicity, including alkaloids, glycosides, proteins, and amino acids, cause significant economic losses to the livestock industry due to poisonous plants and the toxins they produce (James *et al*., 1992) [1]. Different plant extracts like citrus fruits such as amla, mousambi peel powder (Giriprasad *et al*., 2015) [2], plant products such as barnyard millet flour, rice flour (Mishra *et al*., 2014; Mishra *et al*., 2015) [3, 4] etc. are used as extender in the preparation of various value added livestock products (Mishra *et al*., 2015a) [5] with some medicinal and anti-oxidant value as well as enhanced storage stability. Thorough exclusion of alternative causes are required, because many bacterial, viral, immunological disorders generate clinical indications, biochemical changes and lesions that are identical to those induced by poisonous plants (Stegelmeier *et al*., 2020) [6]. The dog accounted for 61.8% of all calls about poisoned pets, with the cat coming in second (26 per cent) according to Italian survey (Caloni *et al*., 2013) [7]. According to the World Health Organization (WHO), over 80% of the inhabitants in underdeveloped nations rely on traditional medicine as their major source of healthcare which also cause toxicity (Jamloki *et al*., 2022) [8]. Chemical, physical, biological, and environmental elements all play a role in determining whether or not a given plant species is poisonous (Mendieta *et al*., 2014) [9]. Poisoning can result from skin contact, ingestion, absorption, or inhalation (Tamilselvan *et al*., 2014) [10]. Plant poisons include anti-cholinergic, nicotine-like alkaloids, calcium oxalate crystals, gastrointestinal, cardiovascular, convulsive and cellular respiration toxins (Banasik and Stedeford, 2014) [11]. Foreign plant incursions in non-native places have an impact on local fauna. Because most poisons have no particular antidote, treatment of plant poisoning is primarily symptomatic and supportive. Poisoning treatment comprises the delivery of activated charcoal and the resuscitation of animals with fluid therapy. Plants cannot escape from animals or fungi, bacteria, and other microbes, so they must find other strategies to defend themselves (Dauncey and Larsson, 2018) [12]. Chemicals that inhibit feeding and infection are one method.
The risk of domestic animal poisoning can be reduced by management measures such as the treatment of pasture and forage to eradicate poisonous plants and hazardous compounds. The most frequent plant toxins faced by domesticated animals and their specific therapeutic treatments are discussed in this article which will refresh the knowledge of the field veterinarians in managing such conditions in a better way.

2. Common plant poisoning in domestic animals
2.1 Cyanogenic plant poisoning
Hydrocyanic acid is another name for cyanide. First extracted from Prussian blue, hydrogen

cyanid is also called "prussic acid". Hydrocyanic acid is a colourless, odourless gas (Clarke *et al*., 1981) [13]. Sorghum (*Sorghum bicolor*), Sudan grass (*Sorghum x drummondii*), corn (*Zea mays*), lima beans (*Phaseolus lunatus*), cherry, apple, peach, and apricot are the most prevalent plants. These plants produce a broad range of lethal cyanogenic glycoside principles such as Amygdalin, Taxiphylyn, Prunacin, Lotaustralyn, Vicianyn, Linamaryn, Dhurryna and Proteacyn (Gracia and Shepherd, 2004) [14].

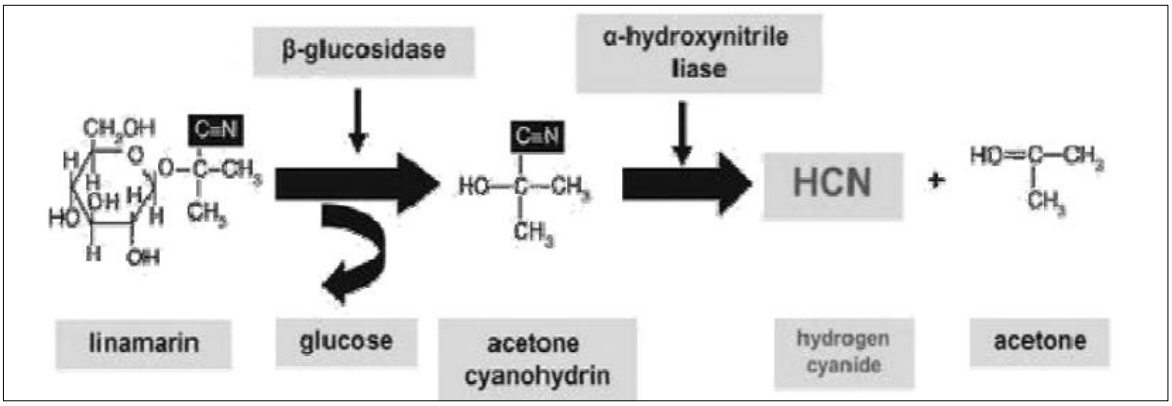


Fig 1: Release of HCN from cyanogenic plants (Source: Gomez and Stuefer, 2006) [15a]

2.1.1 Mechanism of action

In the rumen and abomasum, an alkaline pH promotes toxicity. There in rumen, hydrogen cyanide from cyanogenic plants interacts with methaemoglobin to generate cyanmethemoglobin. This combination inhibits the final phase of oxidative phosphorylation by deactivating the cytochrome oxidase enzyme. The absence of oxygen utilisation results in the termination of cell respiration. The absence of oxygen utilisation results in the termination of cell respiration. The cause of death in cyanide poisoning is histotoxic anoxia. These poisons are actually inert outside of living organisms, but once they've been hydrolyzed inside of organisms, their toxicity becomes apparent. Due to the enzymes that are liberated while processing, these plants become more deadly. According to scientific research, the level of toxicity increases as a result of factors such as ruminal pH and microbiota, quick consumption, swallowing a huge volume of immature cyanogenic plant, and the amount of cyanogenic glycoside or free HCN in the eaten plants.Over utilization of nitrogen fertilisers and herbicides such as 2,4-D increases cyanide poisoning sensitivity (Arnold and Gaskill, 2014 ; Borron, 2006) [16,17]. The maximum fatal dose of HCN for ruminants is approximately 2 mg/kg B.W. Plants with more than 200ppm of these glycosides are considered lethal.

2.1.2 Clinical signs

Inability to conceive, abortion, head tremors, incordination, decrease in productivity and pawning of the posterior limbs can be seen in patients (Gurnsey *et al*., 1977) [18]. Clinical indications of intoxication in animals include laboured breathing, dyspnea, restlessness, tremors, terminal clonic convulsions, and opisthotonos. Because of the amount of oxygenated blood, the mucous membranes are initially brilliant and cherry-red in colour (Borron, 2006; Gurnsey *et al*., 1977) [17, 18]. Due to hypoxia, it eventually gets cyanotic. If not treated immediately, the animal will enter a coma stage and die (Gurnsey *et al*., 1977) [18]. In few cases, myelomalacia and urinary incontinence have also been observed. Chronic toxicosis has been associated with arthrogryposis.

2.1.3 Diagnosis: Mostly done through taking proper history of feeding, Clinical signs and necropsy examination. Specific test-The picric paper method is used to make a qualitative assessment of cyanogenic substances in rumen content or plant specimens (Vetter, 2000) [19].

2.1.4 Pathological lesions

In the post mortem, cyanide intoxication is characterised by a bitter almond odour (Gurnsey *et al*., 1977; Arnold and Gaskill, 2014) [16, 18]. Blood remain unclotted., bright red color,congestion and haemorrhages in abomasum.

2.1.5 Treatment

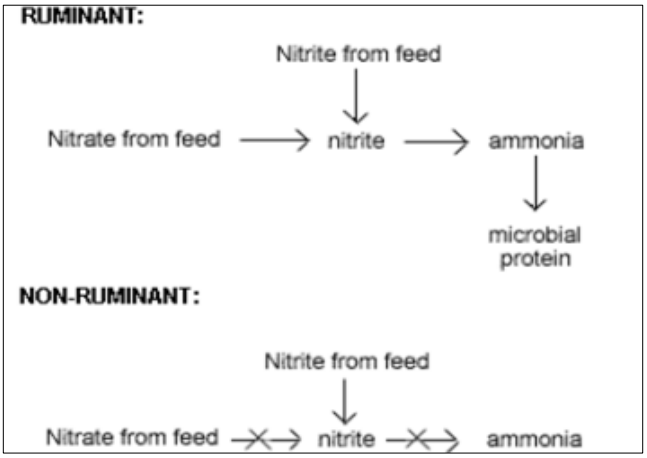
For intravenous infusion, a combination of 1 ml of 20% Sodium nitrate and 3 ml of sodium thiosulfate can be administered to cure cyanide poisoning at a dosage rate of 4 ml of combination/45 kgbwt. Patient's rumen contents should be replaced with that of healthy animal (Borron, 2006; Arnold and Gaskill, 2014) [17, 16].

2.2 Nitrate containing plants toxicity

Certain forages, such as Oats, Cape weed, Sorghum, Maize, Lucerne, Turnip tops, Sudan grass, Wheat, and Barley, when taken in abundance, can result in nitrate poisoning. Normal levels of nitrate in feed are changed into ammonia and then bacterial protein in the rumen of ruminant animals. High nitrogen fertiliser and drought situations cause plants to accumulate nitrate.

2.2.1 Mechanism of action

Nitrate is converted to nitrite at a significantly higher pace than nitrite is converted to ammonia. However, excessive nitrite accumulates in the rumen when nitrate-containing plants are overfed. An animal's mortality from a lack of oxygen is caused by nitrite getting into the bloodstream and converting haemoglobin to met-hemoglobin. Nitrate levels in plants vary with parameters such as species, growth stage, and organ. The nitrate content of immature plants is higher than that of fully developed plants. Since most of the nitrate is concentrated in the plant's lower third, the leaves and flowers don't have as much of it.



2.2.2 Clinical signs
Clinical signs of nitrate toxicity comprise saliva production, vomiting, diarrhoea, tachycardia, blue-brown discoloration of the mucosal membranes, Severe abdominal pain, limited response to analgesics and antispasmodics, tremors, stumbling, dyspnoea, quick and loud breathing (Oruc *et al*., 2017) [20].

2.2.3 Pathological lesion
The most prevalent lesions among stillborn calves were hydroperitoneum and ascites. Lung edoema and bleeding were noted (Ozmen *et al*., 2005) [21]. Peritoneum, respiratory mucous membranes, Epicardium, and digestive tract serous coats develop petechial haemorrhages and Abdominal mucus congestion. Poorly clotting blood with coffee brown colour is have been seen (Davidson *et al*., 1941) [22].

2.2.4 Diagnosis: Mostly done through taking proper history of feeding, Clinical signs and necropsy as well as haemato-biochemical examination.

2.2.5 Treatment
Antidote for nitrate toxicity is intravenous infusion of methylene blue, A 2% methylene blue solution helps to convert methemoglobin to haemoglobin and correcting poisoning.

2.3 Ricin toxicity
The intake of castor bean seeds that is *Ricinus communis* causes ricin poisoning (Bradberry *et al*., 2003) [23]. This plant's poisonous principle is ricin, a water-soluble ribosome-inactivating protein that is particularly high in its seeds. Ricin can also be eliminated through heating. It is come under Category B agent by the U.S. Centers for Disease Control and Prevention (Gal *et al*., 2017) [24].

2.3.1 Mechanism of action
Toxins specifically target eukaryotic ribosomes. Ricin interacts cell surface sugars and impede protein synthesis by inactivating the 60S ribosomal subunits (Olsnes *et al*., 1974; Bradberry *et al*., 2003) [25, 23] that is why it is also called as protein toxin (Griffiths, 2011) [26]. Ricin contains a glycoprotein known as lectin (agglutinins) that has a stronger affinity for red blood cells, resulting in agglutination and hemolysis. Inhalation and injection are the most dangerous modes of exposure.

2.3.2 Clinical signs
Within 48-72 hours following the onset of clinical symptoms, the patient died from diarrhoea, dehydration, depression, instability, cramps, tachycardia, dyspnea, and permanent lateral recumbency (Albuquerque *et al*., 2014) [27].

2.3.3 Pathological lesions
Extensive ulceration and bleeding of the stomach, Necrosis of liver tissue, notably Kupffer cells, and nephritis are detected, necrosis of mesenteric lymph nodes and spleen (Griffiths, 2011) [26].

2.3.4 Diagnosis: Mostly done through taking proper history of feeding, Clinical signs and necropsy examination. Blood and Biochemical tests revealed elevated PCV, serum creatine kinase and AST levels, and hyperbilirubinemia, PCR, ELISA, mass spectroscopy, surface Plasmon resonance, immune affinity columns (Griffiths, 2011) [26].

2.3.5 Treatment
Since there is no specific antidote, activated charcoal can be administered orally. The administration of fluids to combat dehydration and electrolyte loss in affected animals. Anti ricin antibody base therapy (Gal *et al*., 2017) [24].

2.4 Oak poisoning
Seeds, branches, bud as well leaves are poisonous. Toxic ingredient is tannins (Panciera, 1978) [28]. Despite part of many animals' regular diet, it can induce poisoning especially during hot seasons (Bates, 2019) [29].

2.4.1 Mechanism of action
Leaves and acorns have a high concentration of tannins, gallo tannins, phenols, and other bioactive compounds. Tannins and their metabolites are believed to be the toxic element (Panciera, 1978) [28].

2.4.2 Clinical signs
Gastrointestinal and renal dysfunction are prominent disease symptoms. Due to toxic renal tubular necrosis, renal impairment has been found (Panciera, 1978) [28], bradycardia, sluggishness, Anorexia, diarrhoea, emaciation, decreased cutaneous turgidity, crusty ocular discharge, and nasal discharge. The patient's body temperature was normal (Stober *et al*., 1976) [30].

2.4.3 Pathological lesions
Perirenal edoema and petechial haemorrhage, ascites, hydrothorax, and hemorrhagic enteritis. A histologic examination of the kidney revealed multifocal necrosis of the proximal convoluted tubules. Kidney that is pale and swelled up with petechial haemorrhages (Panciera, 1978) [28].

2.4.4 Diagnosis: History of feeding, Clinical signs, Detection of glycosides in urine or tissue and Post-mortem report. The levels of blood urea nitrogen have been observed to increase. Hyperkalemic, hypochloremic, hypocalcemic, hyponatremic, hypoalbuminemic, and hypoproteinemic conditions have been established (Panciera, 1978) [28].

2.4.5 Treatment
Calcium hydroxide, activated charcoal, ruminatoric and purgatives and magnesium sulphate are the affected antidots. For symptomatic treatment like fluid therapy, transplantation of microflora, polyethylene glycol (1 g/kg) administrated in feed and water will bind tannins and reduce tissue damage.

Based on the findings, it is likely to be drawn that cutting followed by water soaking is the most nutritionally treatment strategy for detannifying of leaves Ajith *et al*., 2014 [31].

2.5 Abrin poisoning

An ornamental plant name *Abrus precatorius* (Rosary pea, precatory bean, crab's eye) with pale reddish purple flowers, pea shaped pods with 3-5 red seeds with black spots. Abrin toxin is identical to ricin poisoning as both are responsible for apoptosis and acute necrosis at cellular level as well as do vascular leakage syndrome (Bhasker *et al*., 2014) [32]. The plant's poisonous component is a lectin which is known as abrin, which is present in seeds (Haritha *et al*., 2019) [33].

2.5.1 Mechanism of action

Abrus precatorius generates abrin, a type of lectin that inhibits protein production through the inactivation of the ribosomal subunit (Haritha *et al*., 2019) [33]. Cattle were determined to be the domestic species most susceptible to poisoning (Haritha *et al*., 2019) [33].

2.5.2 Clinical signs

Toxic indicators include gastrointestinal disorders, vomiting, and diarrhoea that result in circulatory collapse. Conjunctivitis and dermatitis is also have been founded (Haritha *et al*., 2019) [33].

2.5.3 Pathological lesions: Brain histopathology demonstrated cortical demyelination (Bhasker *et al*., 2014) [32].

2.5.4 Diagnosis: History of feeding, Clinical signs, Post-mortem report Neurodegeneration and neurotoxicity mediated by oxidative stress, AChE inhibition, lipid peroxidation, and reduction of MBP, Spectrofluorometry found a rise in 5-HT and Norepinephrine (Bhasker *et al*., 2014) [32].

2.5.5 Treatment

Abrine poisoning is treated by administering anti-abrine serum. Alkalinizing urine using saline purgatives aids in the elimination of toxins. Fluid replacement or electrolyte therapy to cure dehydration. For large ruminants 250-500g of activated charcoal can treat abrin toxicity (Haritha *et al*., 2019) [33].

2.6 Parthenium Weed Poisoning

The parthenium hysterophorous is commonly known as congress grass or carrot grass. Weed causes substantial losses in crop productivity across the entirety of India. This plant depletes the soil of its nutrients, leaving the field unusable also causes respiratory allergies, skin rash, and mutagenicity in livestock (Patel, 2011) [34]. Detoriation of milk meat quality of affected animals.

2.6.1 Mechanism of action

In parthenium, parthenin, a photodynamic agent that causes primary photosensitization, liver damage, and skin responses.

2.6.2 Clinical signs

Inflammation, rheumatism, diarrhoea, UTIs, dysentery, malaria, neuralgia, alopecia and as well as pigment loss, mouth ulcer, pruritis, eye irritation (Patel, 2011) [34].

2.6.3 Pathological lesions

Ulcer in muzzle, dental pads, tongue and palate, distended gallbladder, punch out ulcer on oesophagus and abomasol folds, oedema of lungs. Inhibition of dehydrogenase enzyme cause degeneration of liver and kidney (Patel, 2011) [34].

2.6.4 Diagnosis: History of feeding, Clinical signs, Post-mortem report Skin prick test give positive skin reaction to mAb-2 (Patel, 2011) [34].

2.6.5 Treatment

Liver supplements and antipruritics are typically used in treatment. It has been eradicated by burning, chemical herbicides, eucalyptus oil, and biological control by, stem-galling moth, stem-boring weevil, leaf-feeding beetle and fungi (Patel, 2011) [34].

2.7 Ipomoea poisoning

Most common name is morning glories. Goat which gaze on the leaves of ipomoea carnea, a toxic plant specially during summer and draught period, found in Brazil, prevalent in India. Active principle is scammonin (jalapin).

2.7.1 Mechanism of action

They release a number of hallucinogenic lysergic acid alkaloid compounds. Significant reduction in haemoglobin concentration, increase in alanine aminotransferase (Damir *et al*., 1987) [35].

2.7.2 Clinical signs

Copious salivation, diarrhoea, mydriasis, shivering, incoordination, stumbling gait, ataxia, limb paralysis, lateral recumbency, hypotension, and death (Damir *et al*., 1987) [35].

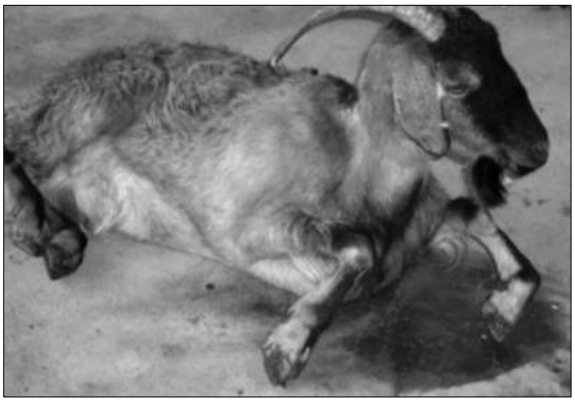


Fig 2: Goat poisoned with Ipomoea carnea showing difficulty to rise and stand (Source: Rios *et al*., 2012 [36])

2.7.3 Pathological lesions

Investigation found degenerative vacuolar abnormalities in the liver, pancreas, thyroid and kidney cells, oedema in the lungs, congestion in the kidneys, and subendocardial haemorrhages (Damir *et al*., 1987) [35].

2.7.4 Diagnosis: History of feeding, Clinical signs, Post-mortem report.

The serum contains a decrement in calcium, magnesium, and total protein. Serum concentrations of aspartate aminotransferase and ammonia rise. Normocytic normochromic anaemia is also detected (Damir *et al*., 1987) [35].

2.7.5 Treatment

Intravenous infusion of 5 percent dextrose at the rate of 10 ml per kg bwt., Vit C 50 mg per animal orally, neurobion forte 2

ml per animal i/m. Symptomatic treatment and liver tonic is also recomended (Damir *et al*., 1987) [35].

2.8 Bracken fern poisoning

This is found in temperate region. Bracken fern poisoning in monogastric animals was first recognized as neurologic disease when horse consumed contaminated hay due to its high silicon content (Ugochukwu, 2019) [37], which diminishes palatability, this noxious weed is not preferred by ruminants. Poisoning in pigs is rare. Carcinogenic property might be arise due to interaction of toxin to DNA (Tiwari *et al*. 2016) [38].

2.8.1 Mechanism of action

Consumption of bracken fern induces thiamine poisoning in animals by the release of the toxin thiaminase and causes neurotoxic syndrome (Tiwari *et al*. 2016) [38]. Excessive ingestion of these weeds produces bovine enzootic haematuria, which is characterised by red-colored urine.

2.8.2 Clinical signs

High fever, appetite loss, depression, dyspnea, excessive salivation, bleeding from oral and nasal orifices or epistaxis, haematuria, and bladder cancer (Ugochukwu, 2019) [37], in ovine there is retinal neuroepithelium degeneration is found, incordination in horse due to thiaminase content (Ugochukwu, 2019) [37]. Bright blindness in cattle and sheep (Tiwari *et al*. 2016) [38].

2.8.3 Pathological lesion

Multiple haemorrhages or bruises throughout the carcass. Ulcer in GIT. Dilated vessels, vascular fibrous or epithelial neoplasms (Ugochukwu, 2019) [37]

2.8.4 Diagnosis: History of feeding, Clinical signs, Post-mortem report

Blood test -Anaemia, leucopenia, hypergammaglobulinaemia, microhaematuria, monocytosis, thrombocytopenia. Urine-proteinuria, bone marrow depression. Biochemical test-ALT, AST, ALP, urea and creatinine is increased

2.8.5 Treatment

Untreatable it is most easily controlled by preventing exposure and there is no specific antidot. In cattle blood transfusion is most effective therapy.in calf 0.4 litres and in adults 1 to 4 litres along with 3% of sodium citrate to counteract the effect of heparin,1% protamin sulphate is used (Tiwari *et al*. 2016) [38].

2.9 *Lantana camara* Poisoning

Lantana (*Lantana camara* Linn) is a hazardous weed found worldwide, particularly in tropical and subtropical regions. Cholestasis and hepatotoxicity in grazing animals have been linked to intake of Lantana leaf. Ruminant as well as non ruminant both are susceptible to this toxic plant Triterpenes ester metabolites Lantadene A and Lantadene B, which are present in leaves and young berries, create a poisonous impact (Sharma *et al*., 2007) [39]. It is harmful and (Sharma *et al*., 2007) [39] lethal to livestock. It also reduce productivity of animals. It also contains anticancer, antibacterial, antidiabetic, antiinflammatory, antimotility, antifeedant, anti-oxidative, antiulcer, analgesic, antifungal, antibacterial, anticonvulsant activities despite its toxicity.

2.9.1 Mechanism of action

Lantana toxicity manifested in three distinct phases:

1. Digestive system the release and uptake of toxins in the digestive tract (Sharma *et al*., 1988) [40]
2. Hepatic system-cholestasis, hyperphylloerythrinaemia, hyperbilirubinaemia (Sharma *et al*., 1988) [40]
3. Tissue -cell injury cause bilirubin and phylloerythrin accumulation (Sharma *et al*., 1988) [40]

2.9.2 Clinical sign

Loss of appetite, unpigmentation, photosensitization, icterus of mucous membrane.

2.9.3 Pathological lesions

Liver is swellon and round edges. Gall bladder is distented, Kidney is swellon and pale. In stomach gas is accumulated. Pale mucous membrane (Kumar *et al*., 2016) [41]. In sheep centrilobular cell vacuolation with bile. In cattle hyperplasia of bile duct. In goat haemorrhage in inter sinusoidal space (Kumar *et al*., 2016) [41].

Fig 3: Pale enlarged liver with swollen gall bladder of an affected sheep (Sourse: Gupta *et al*., 2019 [42])

2.9.4 Diagnosis: History of feeding, Clinical signs, Post-mortem report

Blood test -Increase blood clotting time as well as hematocrit value rise but ESR decrease in large ruminant, thrombocyte decrease in ovine. Biochemical test-bilirubin increase, phylloerythrin level rises. AST, ALT, GGT and BUN rise (Sharma *et al*., 2007), (Kumar *et al*., 2016) [39, 41].

2.9.5 Treatment

Administering laxatives as part of a treatment plan is one way to help the body get rid of harmful substances. Activated charcoal is given to patients to help them digest and excrete harmful substances. supportive therapy is also added to treatment (Sharma *et al*., 2007), (Kumar *et al*., 2016) [39, 41]. Chinese herbal tea Yinzhi Huang clears bilirubin, as per recent findings (Sharma *et al*., 2007) [39]. Liver tonics, keep the animal away from light, ruminotomy, mineral mixture, use of bilirubin oxidase to eliminate bilirubin from animal (Kumar *et al*., 2016) [41].

2.10 Nerium oleander toxicity

The oleander is a beautiful plant that flourishes in tropical and subtropicalzones. Come under members of the Apocynaceae family. These shrubs generate the cardiotoxic cardiac glycosides such as oleandrin, folinerin, and digitoxigenin. This toxin inhibits the Na^+/K^+ ATPase pump, hence impeding electrolyte transport and altering the electrical conductivity of the heart by elevating intracellular calcium ion concentration (Soto-Blanco *et al*., 2006) [43]. It has some medicinal utilisation against diabetes, skin disease and rheumatic and also expresses analgesic, anti-inflammatory, hepatoprotective, cardioprotective and neuroprotective properties [43].

2.10.1 Clinical signs

Hyperpnea, vocalisation, depression, incordination, diarrhoea, colic, polyuria, second degree atrioventricular block (Barbosa *et al*., 2008) (Soto-Blanco *et al*., 2006) [44] [43], ventricular arrhythmia resulting in fibrillation of the ventricles, followed by the demise of animals. It produces rumen abnormalities in ruminants, such as ruminal atony and tympany, minor stomach pain, frequent urination, tachycardia, dullness (Langford and Boor, 1996) [45].

2.10.2 Pathological lesions

renal necrosis in the convoluted as well as in collecting tubules, myocardial degeneration have been seen after staining(Langford and Boor, 1996) [45].

2.10.3 Diagnosis: History of feeding, Clinical signs, Post-mortem report, Biochemical test –ALT AST elevated (Barbosa *et al*., 2008) [44]

2.10.4 Pathological lesion

Coagulated necrosis in cortical tubules, generalised congestion, petechial and ecchymotic haemorrhages on meninges and on other organs. Hepatitis as well as nephrosis (Barbosa *et al*., 2008) [44]

2.10.5 Treatment

Activated charcoal, emetics, purgatives, phenytoin to improve the cardiac conditions (Barbosa *et al*., 2008) [44]

3. Conclusion

There are many toxic weeds and plants for our domestic animals. So toxic weeds and plants should be avoided in your home if your domestic animals are there. When they go out for grazing pay attention what they take. And if it happens go to the veterinarian as early as possible. Present article will refresh the knowledge of the field veterinarians regarding early diagnosis and advocating suitable theraphy for various suspected plant poisoing cases in animals.

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