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Comparative pathology of different plant toxins in grazing animals

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Abstract
Grazing animals in open places is quite common in animal large animal rearing owing to meet the daily need of roughages. Scarcity of sufficient fodder further forces farmers to graze in open land. Prevailing climatic conditions harbours different kinds of plants as part of biodiversity. Many of these plants and small shrubs are edible and non-toxic, however, many are found to be potentially toxic when consumed in larger amount. There different kinds of derangements exhibited in form of clinical manifestations upon consumption. These major and to some extent minor poisoning caused are discussed in this topic.

Keywords: Pathology, plant toxins, grazing animals

Introduction
Plants that, in whole or in part, will, under all or specific circumstances, and in an amount likely to be taken or come into contact with an organism, exert harmful effects or cause death, either immediately or through cumulative action of a toxic property due to presence of known or unknown chemical action, are considered poisonous plants.The toxicity of the entire plant or any portion of it may result from the formation of toxic compounds, many of which are detrimental to human and animal health, such as alkaloids, glycosides, amines, toxalbumins, micro toxins, resins, saponins, and tannins. (Katewa *et al*., 2006) [27].

All continents, even arid regions, are home to poisonous plants (Johnson, 2009) [28]. Its is commonly utilized in food, drugs, ornamentals, and poison preparation. Animals have evolved to withstand little amounts of toxins, or they avoid consuming the toxic components. There are toxic plants that are too dangerous to eat, but they can also be useful. Along with periwinkles and foxglove, oleander is avoided by deer and goats. Although poisonous, plants like poinsettias, frangipani, and yellow bells are used as ornaments. Certain toxic plants are utilised as a source of poison to get rid of rats and other pests. Additionally, poisonous plants have therapeutic uses. Plants have been used to make medicines in a variety of dosages. Plants that are poisonous can both kill and heal (Johnson, 2009) [28].

India boasts a diverse array of flora and wildlife, reflecting the great geographical variances found across the nation. Herbivores rely only on plants for their nourishment, although certain plants or herbs pose a threat to their well-being and existence. The poisonous substances found in plants are typically defence mechanisms against predators. They have a disagreeable taste or odour and are not grazed more readily. However, a lack of food drives the animals to eat these toxic weeds. When these toxic plants are consumed, harmful symptoms such as dermatitis, alopecia, nausea, vomiting, inappetence, tympani, diarrhoea, stomach discomfort, and in rare circumstances, even death, emerge (Samal *et al*., 2016) [16].

Cyanogenic plants
One of the strongest toxins found in plants naturally is cyanide, which can harm all animals, but particularly cattle. This poisonous element can readily influence cognition when it is consumed through a variety of natural plants, such as Johnson grass and sorghum. Cyanogenic glycosides are found in most plant species, and in the bodies of cattle, they are hydrolysed to produce hydrogen cyanide. Following hydrolyzation, methaemoglobin and hydrogen cyanide

combine to generate metalloprotein haemoglobin, which inhibits oxidative phosphorylation. This process may have fatal consequences for the impacted contemplative creatures. Animals are quickly affected by this poisonous cyanide. The classic method of detecting cyanide poisoning in cattle is to observe a shift in the blood's colour from cherry red to cherry red. Early death signs are also indicative of cyanide poisoning and can be seen during a necropsy. The poison can be neutralised with quick treatment, but because it acts quickly, most animal deaths are caused by the toxin. Preserving livestock against exposure to harmful substances is of greater importance (Sankaran *et al*., 2020) [17].

Plants Producing Lathyrism

It is also called as crippling diseases in animals caused by eating legumes of genus Lathyrum (Wild pea,) Osteolathyrims and Neurolathyrism. The Osteolathyrism is most commom form *L. odoratus* (Sweet pea), *L. pusillas* and *L. latifolius* this have B-N-aminopropionitrile (BAPN) as a toxic principle which causes skeletal deformities and aortic rupture due to defective synthesis of cartilage and connective tissue.

BAPN inhibits formation of elastic fibres in the wall of arteries by increased proliferation of fibroblast and also irregularities in the synthesis of collagen fibres. BAPN irreversibly inhibits lysyl oxidase (required for cross linking in collagen synthesis) (required for cross linking in collagen synthesis). The Clinical signs include lameness of hind limbs, exostoses, pain in feet and disinclination to rise. In experimental infection it produces severe connective tissue and skeletal deformities.

Neurolathyrism occurres due to long-term feeding of Lathyrus sativus (Grass pea or Kesari Dal). It is less reported now days. The toxic principle is B- oxalylaminoalanine or BOAA. This is a structural analogue of the neurotransmitter glutamate. It causes excitatory action on lumbar and thoracic spinal nerves. There is Symmetrical axonal degeneration of pyramidal tracts in the spinal cord complete demyelination of white columns of spinal cord. The Clinical signs included Stiffness, paralysis of legs, bladder or laryngeal muscles leading to dyspnoea and death. Horses are mostly affected (Singh and Rao 2013) [20].

Over the past 30 years, new concepts have emerged that will impact the future of L. sativus. At a recent conference, it was dubbed the "golden pulse of the future." Almost all current research publications emphasise that there is no neurolathyrism when pulses are consumed as part of a typical diet, which is particularly true in India. Historically, only L. sativus has been used as a staple food. Recent research on L. sativus offers potential strategic options, which will be briefly discussed. Although conventional plant breeding approaches have made headway in developing low ODAP seeds of L. sativus, this approach has yet to realise its promise due to many factors. (Girma and Korbu *et al*., 2012) [8].

Bracken Fern Poisoning

One of the top five vascular plants in the world, bracken fern (*Pteridium aquilinum*) is distributed all over the world. The species has a wide range of subspecies and variants, which accounts for the variation in plant size. Frond lengths range from 0.5 to 4.5 m. With upright deciduous fronds that stay green until they are damaged by frost or dryness, bracken fern is a perennial plant. Primarily dispersing via extensive rhizome networks, it can take over plant ecosystems, particularly those that have been burned or disturbed. Though it can be found in many different places, bracken fern is most frequently found in open, semi-shaded woodlands that have good drainage.

These plant groups include *Pteridium aquilinum* which have thiaminase catalyses as a toxic principle and produce thiamine deficiency in animals. Along with this *Euisetum arvense*, *Marsilea drumondii*, *Cheilanthes siederi aposonoingre* are the plants which causes bracken fern toxicity. Bracken fern poisoning has been linked to several disorders. The type of poisoned animal, its dosage, period of exposure, and other factors all play a major role in determining these symptoms. (Sonne *et al*., 2011) [22].

Enzootic Hematuria

The most prevalent type of bracken fern poisoning, enzootic hematuria, mostly affects cattle and affects sheep less commonly. It is distinguished by sporadic hematuria and anaemia. The majority of poisoning cases happen in the late summer when there isn't much other feed available or when animals are fed hay that contains bracken fern. Because afflicted livestock must consume bracken fern for several weeks to years before disease develops, poisoning requires prolonged exposures (Tiwari *et al*., 2016) [25].

Cattle that are affected exhibit weakness, fast weight loss, and fever (106–110 °F; 41–43 °C). Calves' mucosal membranes are pale, and they frequently have trouble breathing. There are different types of haemorrhages: exuberant bleeding, mild mucosal petechia, and occasionally, big blood clots that pass through the stools. Prolonged coagulation and noticeable, profuse bleeding can occur even from tiny wounds like scratches or bug bites (Hopkins 1986) [9].

Poisoning almost invariably results in death for animals if they exhibit clinical symptoms. Upon postmortem examination, many bruising or haemorrhages are typically found throughout the body. Additionally, the GI tract may have hemorrhagic and necrotic ulcers. Small haemorrhages, dilated arteries, or vascular, fibrous, or epithelial neoplasms are frequently seen in the bladder mucosa. There have also been reports of other neoplasms in the upper GI tract of cattle and other species. Most often, a combination of neoplastic and haemorrhagic lesions are discovered. The main source of enzootic haematuria has been identified as ptaquiloside, a norsesquiterpene glucoside, even if not all bracken fern toxins have been fully identified. Quercetin, isoquercetin, ptesculentoside, caudatoside, astragalin, and other tannins are some more hazardous and possibly cancer-causing substances.

Ptaquiloside is a strong radiomimetic that causes cancer after first harming the bone marrow (primarily producing urinary tract neoplasia in ruminants). Ptaquiloside and bracken fern have been used in experiments to mimic the hemorrhagic syndrome and uroepithelial neoplasms. A few months of high ptaquiloside doses cause the typical hemorrhagic illness. This has been linked to the radiomimetic harm that ptaquiloside causes to growing bone marrow stem cells. This is typified by erythrocytic and leukocytic hypoplasia as well as the depletion of bone marrow megakaryocytes. A mixed response is often seen in the ensuing leukogram. During the first stages of poisoning, monocytosis is frequently evident and is followed by granulocytopenia and thrombocytopenia. Leukopenia, hypergammaglobulinemia, and severe thrombocytopenia are characteristics of the last stages. Proteinuria and haematuria are typically seen in urinalysis. Affected animals are more prone to infections and have a propensity for bleeding on their own (Xu, 1992) [26].

It seems more plausible that bracken fern at lower doses for

longer periods of time causes cancer. Since animals are exposed over an extended period of time, the effects appear to be cumulative. Clinical illness frequently does not show symptoms for weeks or even months after animals have been taken off pastures and ranges infested with bracken fern. Bracken fern and ptaquiloside have been shown to have carcinogenic potential in rats, mice, guinea pigs, quail, and Egyptian toads in addition to livestock (Solcan *et al*., 2016) [21].

Animal poisoning excretes ptaquiloside in both their urine and milk, and contaminated milk has been reported to cause gastrointestinal neoplasms in rats. Numerous researchers have hypothesised that bovine papillomavirus infection could increase or exacerbate ptaquiloside neoplastic transformation. This could be a secondary alteration brought on by the myelodysplasia linked to bracken fern and the ensuing immunosuppression, which will probably encourage papillomavirus infection.

Bright Blindness

It is a less frequent symptom of ptaquiloside poisoning. Clinically, it is known as tapetal hyperreflectivity, and in some regions of England and Wales, sheep are the most frequently reported cases. Sheep with the condition become permanently blind and develop a recognisable alert demeanour. The funduscopic examination in advanced disease is characterised by narrowing of arteries and veins and a pale tapetum nigrum with small cracks and grey patches. The pupils react poorly to light. Histologically, the lesion is characterised by significant atrophy of the outer nuclear layer, rods, and cones of the retina, with the tapetal region of the retina showing the greatest degree of atrophy. Numerous additional pathologies linked to bracken fern, including bleeding, immunosuppression, urinary tract neoplasia, and bone marrow suppression, are frequently seen in affected animals.

Bracken Staggers

When horses ingested tainted hay, bracken fern poisoning in monogastric animals was initially identified as a neurological illness. A typical poisoning would involve feeding hay contaminated with 20–25 percent bracken fern for three months or more, which would involve relatively large dosages over an extended period of time. Anorexia, weight loss, lack of coordination, and a crouching position with the back and neck arched and feet spread widely are the hallmarks of equine bracken staggers. Muscles are observed to tremble when pressed to move. Seizures, clonic spasms, and opisthotonos frequently precede death, which typically happens 2–10 days after commencement. In severe cases, tachycardia and arrhythmias may also occur. Because the symptoms of the poisoning resemble vitamin B1 insufficiency, bracken fern thiaminases have been implicated in the poisoning. Thiamine treatment works for most animals (Stegelmeier *et al*., 2020) [24].

Oxalate poisoning

There are two types of oxalic acid that are commonly found in plants: soluble and insoluble oxalates. In soluble oxalate, monovalent counterions like sodium (Na+), potassium (K+), and ammonium (NH4+) are typically used in its formation, while divalent ions like calcium (Ca2+), magnesium (Mg2+), and iron (Fe2+) are used in insoluble oxalate (Savage *et al*., 2000). The cleavage of ascorbate and isocitrate, hydrolysis of oxaloacetate, and photorespiratory glyoxylate oxidation are some of the routes for oxalate formation that have been suggested (Nakata, 2003) [29]. It has been suggested that in oxalate-accumulating plants, glyoxylate oxidation is a more direct and effective precursor for oxalate production (Fujii *et al*., 1993) [7].

Forage plants contain a variety of anti-nutrients, one of which is soluble oxalate. By binding calcium (Ca), magnesium (Mg), and other trace minerals like iron (Fe), it prevents the body from assimilating these minerals. This results in abnormalities in the metabolism of calcium (Ca) and phosphorus (P), as well as an overabundance of bone mineral mobilisation. In horses, the demineralized bones become fibrotic and malformed, resulting in lameness and a "bighead" (McKenzie *et al*., 1981) [12]. Although ruminant animals are less impacted, continuous grazing on specific tropical grasses by sheep and cattle can cause severe hypocalcaemia (Seawright *et al*., 1970) [30]. It was thought that one of the main causes of urolith formation in grazing animals was the high oxalate content of pasture plants (McIntosh *et al*., 1974) [31]. Another study found that elevated oxalate levels in

Plants producing photosensitization

Syndrome of abnormal sensitivity of the lightly pigmented areas of skin to sunlight due to presence of photodynamic agent in the peripheral circulation most common in cattle and sheep

Primary Photosensitization: Photodynamic agent directly ingested or bio transformed to a photodynamic agent and react with UV light. Hypericum Spp and Fagopyrum spp. Phenothiazine, sulphonamides, tetracycline and acridine dyes.

Secondary Photosensitization/ Hepatogenous:- Ingestion of substances leading to liver dysfunction or bile duct obstruction No photodynamic activity itself but damage liver and causes deposition of plant pigments phylloerythrin causing photosensitization Plants causing liver damage:- Senecio spp., Crotolaria spp., and Helitropium Plants causing biliari stasis:- Tribulus trrestris and panicum colaratum Lantana camara:- Lantadene A-D.

Photosensitization due to abnormal porphyrin metabolism: Porphyrins are pigments normally present in haemoglobin. Inherited congenital porphyria, where there is excessive production of porphyrins in body which are themselves photodynamic. Usually inherited due to an enzyme deficiency, which results in abnormal synthesis of photodynamic agent's oroporphyrin and coproporphyrin.

Clinical Signs

Lesions occur in areas which have little protection with pigment hair or parts of the body exposed to sun.
The areas mostly affected are
Horses - muzzle, coronary band, cannon and pastern.
Cattle - teats, udder, perineum and muzzle.
Sheep - pinnae, eyelids, face, muzzle and coronary band.
Sheep may develop extensive edema of head called, "swelled head" and "facial eczema". Affected non-pigmented skin: Hair loss, skin reddening, peeling, skin ulceration, bleeding, and crusting.

Datura and related plant toxicity

These categories of plants include *Datura strmonium, Atropa belladonna, Hyoscyamus niger* and *Physalis virginiana.* It contain alkaloids or glyco-alkaoids having similar

pharmacological and toxicological actions with scopolamine, atropine, hyoscyamine, daturine and solanine.

Plants that induce heart failure

The natural Brazilian tree Ateleia glazioviana (Leg. Papilionoideae) grows to a height of 5 to 15 metres. It is typically found in southern Brazil's roadsides, forests, and invading pastures. When green herbage is sparse due to extended droughts or overcrowding, cattle can easily and happily devour the plant; shipping appears to be a risk factor for plant ingestion (Gava & Barros, 2001) [32]. Cattle that consume *A. glazioviana* suffer significant losses as a result of the toxicosis, which is linked to three clinical manifestations: I miscarriage; (ii) neurological disease marked by lassitude and sporadic blindness; and (iii) acute or chronic heart failure. Myocardial fibrosis and degeneration are two types of myocardial lesions (Gava & Barros, 2001) [32].

Plants containing toxic lectins/Castor Bean and Rosary Pea Poisoning

The castor oil plant (*Ricinus communis* L), is a plant species of the family *Euphorbiaceae* and the sole member of the genus *Ricinus* and of the subtribe *Ricininae*. *Ricinus communis* is a perennial, erect, branched, herb, typically less than 2 meters in height. The beans are oblong and light brown, mottled with dark brown spots. The seed is only toxic if the outer shell is broken or chewed. Ricin is contained in the bean is a toxic principle in this beans. It is a protein toxin (toxalbumin). It is a glycoprotein lectin composed of 2 chains, A and B, linked by a disulfide bond. The B chain is a lectin and binds to galactose-containing glycoproteins and glycolipids expressed on the surface of cells, facilitating the entry of ricin into the cytosol. The A chain inhibits protein synthesis by irreversibly inactivating eukaryotic ribosomes through removal of a single adenine residue from the 28S ribosomal RNA loop contained within the 60S subunit. This process prevents chain elongation of polypeptides and leads to cell death. Toxicity results from the inhibition of protein synthesis, but other mechanisms are noted including apoptosis pathways, direct cell membrane damage, alteration of membrane structure and function, and release of cytokine inflammatory mediators. The castor bean plant also contains another glycoprotein lectin, the *Ricin communis agglutinin*, which, unlike ricin, is not directly cytotoxic, but does have affinity for the red blood cell, leading to agglutination and subsequent haemolysis. *Ricin communis agglutinin* is not significantly absorbed from the gut and causes clinically significant haemolysis only after intravenous administration (Al-Tamimi *et al*., 2008) [1].

Gossypol toxicity/Cottonseed toxicity

It is a Phenolic compound, lipid soluble. Directly absorbed from GIT Binds to Protein and amino acids (lysine and methionine) Resulting in disturnance in heme synthesis and anemia Also acts as an inhibitor for several dehydrogenase enzymes and reversible blocks calcineurin and binds to calmodulin. Inhibition of testicular LDH affects male reproductive system along with this inhibits the glutathione S-transferase which impairs the livers ability to metabolose xenobiotics.

It causes Hepatotoxicity, reproductive toxicity and immunotoxicity in all types of animals Mon gastric animals and young animals more susceptible Reduces rate of pregnancy, increases abortion rate, affects ovarian functions. Respiratory distress impaired body condition, anorexia and weakness and death. Pathological Findings in ruminants include pulmonary oedema, gastroenteritis and centrilobular liver necrosis and in calves ascitis, Visceral oedema, Kidney damage and Cardiovascular lesions.

Conclusion

In summary, the intricate relationship between plants and organisms highlights the dual nature of poisonous plants – both harmful and, in some cases, beneficial. Understanding and managing the risks associated with poisonous plants are essential for safeguarding the well-being of humans and animals alike.

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