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Review Article

Documentation of Major Poisonous Plants and Their Toxic Effects on Livestock: A Review

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Abstract: Plants and plant products are the main source of feed for herbivore animals throughout the world. But all plants are not safe for animals since some plants have toxic properties. Poisonous plants have particular importance in areas where extensive pastoral management is practiced. The large array of toxic chemicals produced by these toxic plants (phytotoxins) is usually referred to as secondary plant compounds that cause physiological changes in the host. They include alkaloids, glucosides, oxalate, cyanide, hypericin, amines, toxalbumins, picrotoxins, resins, and saponins, many of which are dangerous to human and animal life under particular conditions. The poison plants cause direct and indirect economic losses to the livestock industry around the world. The direct economic losses of poison plants include livestock deaths, abortions, birth defects, weight losses, a lengthened calving interval, decreased fertility, photosensitization, immune responses, and dysfunction of the organs. This leads to a significant economic loss for the livestock industry. Most poisonings occur in the early spring or during a drought period when feed is short, which causes animals to overgraze or change their grazing habits. Livestock poisoning by plants can often be traced to problems of management or range conditions rather than simply to the presence of poisonous plants. Thus, timely diagnosis of toxic plant or biotoxin poisoning is very important and heavily relies on identification of the toxins in the feed, pasture, or ingesta along with appropriate clinical and pathological findings. This review presents the current knowledge of the identified poisoned plants and their toxic effects on livestock. Research is needed to identify and document all poisoning plants that have potential risks for animal health and production and to determine the photochemistry and toxicology of plants.

Keywords: Poisonous Plant, Toxin, Livestock, Economic Impact, Secondary Metabolic Products

1. Introduction

A poisonous plant contains a specific substance that produces deleterious effects on the health of animals when taken in small or moderate amounts [31]. The poisoning plants are a major concern for every veterinarian and livestock producer in the world since they cause significant economic loss on livestock industry by their secondary toxic metabolic products. Ethiopia has a high level of plant biodiversity both native and naturalized [8]. The number of problematic toxic plants is higher in countries with higher plant biodiversity [16]. Animals are poised by toxic plants either accidental ingestion or planned intake of poisonous plants when pasture is dry. The poisonous plants remain green throughout the year. Poisoning of animals moreover happens when animals are introduced to new areas since they become unfamiliar with the surroundings, which exposes them to ingesting toxic plants. The majority of poisons plants stay green and eye-catching to hungry and thirsty animals. Animal poisoning happens when animals ingest large amounts of plants in a short period of time. Many common plants found in the home, outdoors in gardens, woods, and fields can be harmful if consumed or come into contact with the skin or eyes. Poisonous plants can cause toxicity and death of affected animals, which disturbs the financial status of farmers and the nation's economy. Severity of plant poisoning mainly influenced by a variety of

determinants including chemical nature of the toxin, amount and time of toxin eaten, parts of plant eaten, maturity stage of plant, species of the animal, age, size, sex and condition of the anima [31].

There are a variety of plants that cause toxicities in livestock by producing different toxic compounds. The most common toxic compounds produced by plants include oxalate, cyanide, photodynamic, hypericin, ptaquiloside and nitrate (nitrite) in the animal's body after being consumed by them, causing poisoning or photosensitization in different parts of the host. Animals are exposed to nitrate poisoning when they consume excess forage that is high in nitrates and urea or nitrogen fertilizer spilled in the filled or left-over area where the animals can find it. There are different plants that cause photosensitization in plants. Animals that are exposed to sunlight after consuming or coming into contact with plant pigments or secondary products that are ultraviolet (UV) reactive suffer from a severe dermatitis condition known as photosensitivity [14]. St John's wort, containing light-sensitive compound hypericin, is one of the most common plants causing primary photosensitization when activated by light [29].

Oxalic acid and its salts are metabolic byproducts in a variety of plant tissues since it binds to calcium, potassium and other minerals. Oxalic acid promotes stone formation in the urinary system [6]. Hydrogen cyanide is the most swiftly acting toxin that affects cattle following ingestion of cyanogenic plants [21]. Awareness of poisonous plants and their associated secondary metabolites, clinical signs, and diagnosis is instrumental in making treatments and managemental practices. Therefore, the aim of this paper is to review some major toxic plants that affect livestock health and productivity, to illustrate different poisonous compounds found within different toxic plants, and to suggest certain management practices to prevent and control plant-based poisonings.

2. Major Livestock Toxic Plants and Their Toxic Effects

Toxic plants are a significant source of financial loss in the cattle sector. The poisonous nature of a whole plant or any plant part can be attributed to the synthesis of toxic compounds such as alkaloids, glucosides, oxalate, cyanide, hypericin, amines, toxalbumins, picrotoxins, resins, and saponins, many of which are dangerous to man and animal life under particular conditions [35]. Different animal species are affected by poisonous plants in different ways. Some plant species cause toxic reactions in specific types of animals, and some plants only become hazardous at specific times during their life cycles. Only specific plant parts might be poisonous. Poisoning happens when situations emerge that allows or causes the animal to consume an excessive amount of food in a short period of time. Livestock owners can better safeguard their cattle from plant poisoning by being aware of the poisonous plants and the circumstances that encourage poisoning. The following are some notable and significant plant metabolic end products that have toxic effects on livestock.

3. Plants Associated with Nitrate Poisoning

Nitrate poisoning is a common ruminant toxicity that results in rapid mortality. Plant roots absorb nitrate (NO3) from the soil and convert it to nitrite (NO2) and ammonia. Ammonia is then incorporated into plant amino acids to form plant proteins. If plants absorb excess nitrates and are consumed by livestock before they are converted to proteins, nitrate poisoning can occur [11]. Livestock is fatally affected by plants with more than 1.5% dry weight of nitrates (NO3). The amount and rate of nitrate consumption affect the degree of toxicity. Generally speaking, toxicity happens when livestock eat a lot of fodder that contains 1.76% or more nitrate ions on a dry matter basis [38]. Many plants, including crops and weeds, store nitrate molecules that can induce nitrate poisoning in animals. Crops that accumulate nitrate molecules include oat hay, sorghum, corn, Sudan grass, Johnson grass, and beets, whereas weeds such as careless weed, kochia, pig weed, Russian thistle, and nightshade [37] are sources of nitrate molecules.

The nitrate concentration of young plants is normally the highest and declines as the plant matures. Despite the fact that plant nitrate content may be high at maturity due to high soil nitrate levels or growth stress situations, nitrate levels are highest soon before flowering and rapidly fall following pollination and seed development. Nitrate concentrations are higher in lowest part of the plant stem. Leaves contain less than stalks or stems. It gets gradually diluted as it approaches the top of the stem. The seeds (grain) and flowers contain very little or no nitrate [17]. The majority of the plant nitrate is concentrated in the lower part of the stalk. Many animals are vulnerable to nitrate and nitrate poisoning, although ruminants are the most commonly affected. Ruminants are spatially fragile because the ruminant flora uses nitrite to convert nitrate to ammonia. Because nitrate is reduced to nitrite in the cecum of equines, the toxicities of nitrate in equines are not as severe as in ruminants [10].

3.1. Pathogenesis /Mechanism of Action

Nitrates are generally found in forages and are transformed to nitrite by the digestion process, which is then turned to ammonia. Bacteria in the rumen then convert the ammonia to protein. If cattle consume large amounts of plants containing high levels of nitrate, nitrite will accumulate in the rumen. Nitrite is ten times more poisonous to cattle than nitrate [13]. Nitrite enters red blood cells and binds to hemoglobin (an oxygen-carrying molecule) to generate methemoglobin. The heart rate and respiration of the animals increase since methemoglobin does not transport oxygen as efficiently as hemoglobin [18].

3.2. Clinical Findings

During the acute poisoning stage, blue color of the mouth, eyes, shortness of breath, stumbling walk, demise, chocolate

brown blood, cloudy, cyanotic mucus membranes, and rumen and abomasum congestion are detected. Watering eyes, unkempt appearance, decreased milk yield, decreased weight gain, miscarriage, and infertility are clinical indicators of sub-acute acute poisoning [18]. The clinical signs of nitrate toxicity are similar to those of cyanide, urea, pesticides, toxic gases (carbon monoxide, hydrogen sulfide), chlorates, aniline dyes and drugs (sulfonamides and acetaminophen). The signs are also similar with infectious and non-infectious diseases such as grain overload, hypocalcemia, hypomagnesemia, pulmonary adenomatosis, or emphysema [35].

3.3. Diagnosis

Clinical symptoms and laboratory tests of nitrate and nitrite levels in serum, blood, ocular fluid, rumen contents, and fodder are used to make the diagnosis. Nitrate levels of 0.3% or more in forage are potentially dangerous [26]. Nitrate levels in premortem and postmortem materials can be tested to identify excessive nitrate exposure. Plasma is the preferable premortem collection because serum may result in the loss of some plasma protein-bound nitrite in the clot. Because nitrite in whole blood continues to react with hemoglobin in vitro, these specimens must be centrifuged immediately and plasma separated to avoid erroneous hemoglobin and nitrite results. Other postmortem specimens from abortions include fetal pleural or thoracic fluids, fetal stomach contents, and maternal uterine fluid.

3.4. Treatment and Prevention of Nitrate Poisoning

A slow intravenous (IV) injection of 1% methylene blue in distilled water or isotonic saline should be given at 4–22 mg/kg, depending on the severity of exposure. Lower dosages may be repeated in 20–30 minutes if the initial response is not satisfactory. Lower dosages of methylene blue can be used in all species, but only ruminants can safely tolerate higher dosages. If additional exposure or absorption occurs during therapy, re-treating with methylene blue every 6–8 hours should be considered. Rumen lavage with cold water and antibiotics may stop the continuing microbial production of nitrite [34]. The best alternatives for preventing nitrate poisoning in cattle are to control the type and quantity of forage supplied to animals, avoid forages with potentially lethal levels of nitrate, or dilute with feeds low in nitrate.

4. Plants Causing Photosensitization

Photosensitivity in animals is defined by severe dermatitis produced by an increase in the reactivity of skin cells and associated dermal tissues to sunlight following intake or interaction with UV or light-reactive plant pigments or secondary metabolites (photodynamic agents). Photosensitivity in animals is mediated by a light absorbing secondary plant products. Non-pigmented skin has a more severe response since it is less protected against light exposure. Photosensitization differs from sunburn and photodermatitis though its appearance superficially very similar. Without the use of photodynamic agents, skin pathologies such as sunburn and photodermatitis develop [27]. Sunburn is brought on by prolonged exposure of healthy skin to harmful UV radiation. Affected animals are photophobic and develop skin bullae, ulcers, and necrosis. There are four types of photosensitization based on the sources of the photodynamic agent. These are primary (type I) photosensitivity, secondary (type III) photosensitivity, aberrant endogenous pigment synthesis (type II) photosensitivity and idiopathic (IV) sensitivity [14]. Most compounds that are significant causes of photosensitivity in animals are plant derived metabolites. Primary photosensitization occurs following the ingestion of plants containing photodynamic substances. The photodynamic substances reach the skin following absorption from the alimentary tract. Plants in the families of Umbelliferae and Rutaceae contain photoactive furocoumarins (psoralens), which cause photosensitization in livestock.

Primary photosensitivity has been linked with ingestion of *Hypericum perforatum* (St. Johnswort), *Trifolium* spp. (clovers), *Vicia spp*. (vetches) and *Fagopyrum esculentum* (buckwheat), *Ammi majus* (bishop's weed), *Lantana camara, Cymopterus watsonii* (spring parsley), *A. visnaga* seeds, *Trifolium Spp.*, *Medicago* (clovers and alfalfa), *Erodium*, *Polygonum*, and *Brassica* plant species in livestock (29). These plant species produce different toxic metabolic products.

There are various elements that influence how sensitive cattle are to photosensitizing substances. These factors include the animal's breed, species, skin pigmentation, age, level of health, and environmental factors including temperature, humidity, and rainfall (27). Animals are shielded from the damaging effects of UV radiation by their hair and skin colour. The impact will also be greater on animals with thin, light-colored coats or fleece than on those with thick, pigmented coats [12]. Dermal photosensitization is more likely to affect young, ill, non-pigmented animals than adult, healthy, pigmented, or hairy animals [27].

St. John's wort (*Hypericum perforatum):* It is a persistent, invasive noxious weed that thrives in temperate regions' sandy, poor soils [27]. The plant has glands on leaves, stems and petals containing the light-sensitive compound hypericin. Hypericin appears as small clear dots in the leaves and present during all stages of the plant's growth. The hypericin acts as a photodynamic agent. The plant has orange yellow flowers with five petals. Animals consume significant amount of this weed when the plant is prolific, young tender shoots, and feed is scare. Horse is more sensitive to this toxic plant. The bioactive compound that causes photosensitivity in the plant is photo cytotoxicity and highly fluorescent pigment hypericin.

Buck wheat (Fagopyrum esculentum): It contains a photodynamic compound fagopyrin [29]. The fagopyrin and dianthroquinone toxins are present in both the green and dried plant parts but not in the ripe seeds. When animals ingest this poisoning plant in sufficient quantity, they become phototoxic. The fagopyrin toxin that is absorbed from the gastrointestinal tract into the blood vessels of non-pigmented skin reacts with ultraviolet waves from sunlight. Radiant energy in the form of

fluorescence produced by fagopyrin toxin exposed to sunlight causes damage to the blood vessels. This primary photosensitization can be severe enough to cause cell death and sloughing of the skin. Cattle, horses, sheep and goats are susceptible to the poisoning plants.

Lantana (Lantana camara): Lantana camara is toxic ornamental shrubs. It is one of the world's ten worst weeds in tropical, subtropical and worm temperate climates. Its weedy nature and subsequent potential to poisoning livestock are exacerbated by its allelopathic nature that reduces alternative forages. The plant dominates many plant communities [32]. The plant propagates from stumps or cuttings and from seeds disseminated by birds through their droppings [31]. There are numerous natural species of America and Africa in the genus Lantana. The species *L. camara aculeate* is responsible for livestock poisoning among different spread species. The fruits of the plant contain high concentration of toxic substances. *Lantana camara* induce hepatotoxicity that causes high morbidity and mortality. Lantadens A and Lantadene B are pentacyclic triterpenoids, which are two of the principal hepatotoxic and allelopathic secondary metabolites. The toxins are present in the roots, leaves, stems and unripe fruit [32]. The toxic signs of *L. camara* are mainly due to the presence of toxic substances called lantadenes (lantadene A, lantadene B, lantadene C, lantadene D and Icterogenin). Lantadene A is considered as most hepatotoxic among all these toxic substances [31]. Due to the persistence of phototoxins in the rumen, ruminants experience more severe effects over a longer length of time than non-ruminants. Livestock consume the plant when there is drought, and animals are naïve.

4.1. Pathogenesis

When phototoxic plant substances or their metabolites are ingested by an animal or get localized in the skin's cellular layers, primary photosensitization takes place. This might be caused by direct contact with the plant or by ingestion and spread in the herbivore to cutaneous tissues via absorption from the gut and dissipation in the circulatory system. These photodynamic agents have the ability to absorb light energy and form unstable, high-energy molecules when they react with photons [4]. The high-energy molecules initiate reactions with substrate molecules of the skin, causing the release of free radicals that in turn enhance the chain reaction of phospholipids, which results in increased permeability of the outer cell and lysosome membranes. Damage to outer cell membranes allows for leakage of cellular potassium and cytoplasmic extrusion. Lysosome membrane damage releases lytic enzymes into the cell. This can lead to skin ulceration, necrosis, and edema. This can happen as a result of a diseases brought on by gene abnormalities in specific mammalian systems that result in a lack of the enzymes necessary for the formation of hemoglobin. These substances have the potential to directly harm skin cells through cytotoxic mechanisms or, much less frequently, by activating an immunological (photoallergic) response. A highly varied onset can be seen in photocytotoxic photosensitization [27].

4.2. Clinical Signs and Lesions

Clinical signs are observed several days after ingestion, what Erythema and edema of unpigmented areas of skin, followed by blistering and sloughing of the skin. Sere irritation, restlessness, rubbing, shaking of head and ears and seeking shade are other observable clinical signs. In severe cases the surface of the skin crack and turn black [29]. Lameness, sloughing skin in affected areas, with underlying tissues inflamed and scabs forming over the inflammation and jaundice are all possible symptom [27]. In domestic animals, hypericin photosensitization appears as standard clinical indications of UV photosensitivity, such as reddening and oedema of snout, eye, and ear tissues, as well as increased rectal temperature [12].

4.3. Diagnosis and Prevention

Postmortem and laboratory findings results of the complete blood count and serum biochemistry, values for electrolytes, and the whole blood count differential were all within normal reference ranges, and there were no apparent difference in values recorded from affected and unaffected animal. First aid methods are aimed at removing the dietary cause and protecting from sunlight, remove stock from the paddock where the trouble is occurring, ideally put affected stock in a darkened shed, provide stock with water and cereal hay or lower quality pasture hay with no green color [29]. The use of fly sheets and boots to minimize UV light exposure is other alternatives in mild cases [14].

5. Plants Associated with Ptaquiloside Poisoning

Ptaquiloside poisoning caused by the ingestion of *Pteridium aquilinum (*Bracken fern), *Pteridium species*, Cheilanthes, and *Onychium contiguum* spcies of plants. Bracken fern poisoning is the fifth most widely distributed and common weed found throughout the world. A high incidence of vesicular carcinomas observed in cattle and sheep that grazed bracken for extended periods of time. The plants produce cyanogen glycosides, thiaminases, thiamine inhibiting compounds, ptaquiloside, isoptaquiloside, caudatoside, and radiomimetic toxins. Ptaquiloside and its metabolites are mutagenic, clastogenic, and carcinogenic. It primarily affects the dividing cells of bone marrow and gastrointestinal tracts. High ptaquiloside concentrations are found in the vegetative plant parts, whereas rhizomes, roots, and spores contain very low ptaquiloside concentrations [32]. The toxin primarily affects ruminants. Cattle and sheep are most often poisoned. Poisoning necessitates lengthy exposures, as infected livestock must consume bracken fern for several weeks to years before sickness develops.

5.1. Clinical Signs

Acute hemorrhagic disease, bovine enzootic hematuria, bright blindness, upper alimentary carcinomas, and thiamine

deficiency are among the clinical signs that the bracken fern poisoning plant can identify. Affected cattle are weak and rapidly lose weight. They become febrile and dyspneic with pale mucosal membranes.

5.2. Prevention and Treatment

The initial step in preventing animals from poisoning is removing poised animals from bracken-fern-containing pastures. Avoiding exposure by improving pasture management and increasing the production of alternative forage is essential. Thiamine deficiency can be treated if it is detected early. Antibiotics may be useful to prevent secondary infections. Most animals that develop hemorrhagic and neoplastic diseases do not recover.

6. Plant Poisoning Associated with Cyanide Toxicity

Cyanide is a potent, rapidly acting poison. Toxicity of cyanide is due to production of hydrogen cyanide after digestion of plant that contains cyanide. Cyanide glycosides are substance present in many plants that can be produce hydrogen cyanide poisoning. The young growing plant and seed contains more concentration. About 2000 species of plants known of containing cyanide glycosides with a potential to produce HCN poisoning [24]. Some of the plants that are source of cyanide poisoning in cattle are Hairy vetch (*vicia villosa*), Johnson grass *(Sorghum halepense*), and Laurel (*Prunus laurocerasus*) [21]. Frost and drought condition may increase cyanogensis, also cool moist growing condition enhance the conversation of nitrate to amino acid and cyanogenic glycosides instead of plant protein. Ruminants are more susceptible to cyanide poisoning because the ruminant micro flora contain enzyme, that is in the presence of water are also capable of converting cyanogenic glycosides in plant to free cyanide gas.

6.1. Mechanism of Action (MOA)

Cyanide is a mitochondrial toxin that impure cellular respiration causing morbidity or mortality within a short period of time. The toxicity is mediated through inhibition of cytochrome oxidase of and end chain enzyme of mitochondrial respiration. But recent study shows that cyanide inhabit multiple enzymes and alter several vital intracellular processes depending on animal morphology [24]. As ruminants consume this plant material, hydrogen cyanide gas is liberated in the rumen and rapidly absorbed in to the blood stream. Cyanide ultimately prevents hemoglobin in red blood cells from realsed its oxygen to the tissue and animal die through lack of oxygen [5].

6.2. Clinical Signs and Lesions

Clinical signs of cyanide poisoning observed within 20 minutes to a few hours after animals consume the toxic forage. Animals often are found dead. Clinical signs, when noticed, occur in rapid succession, excitement, rapid pulse and generalized muscle tremors occur initially, followed by rapid and labored breathing, staggering and collapse. Signs also may include salvation (drooling), lacrimation (runny eye) and voiding of urine and faces the mucus membrane are usually bright pink eye and blood will be characteristics bright cherry red [33].

6.3. Diagnosis and Treatment

Appropriate history, clinical signs, postmortem findings, and demonstration of HCN in rumen (stomach) contents or other diagnostic specimens support a diagnosis of cyanide poisoning. The treatment for cyanide poisoning consists of re-establishing oxygen transport at the cellular level. Sodium nitrite is injected intravenously to convert hemoglobin to met-hemoglobin, which reacts with cyanide from the cyanide-cytochrome complex to form cyanomethemoglobin. A simultaneous injection of sodium thiosulfate provides sulfur to convert cyanomethemoglobin to the less toxic thiocyanate, which is excreted in the urine. The remaining met-hemoglobin is converted by other enzymes to hemoglobin, which then is available to transport oxygen normally [33].

7. Plants Causing Oxalate Poisoning in Livestock

Oxalic acid and its salts occur as end products of metabolism in a number of plant tissues. Some of the plants including Chenopodium, Rumex, Elephant grass (Panicum spp), pigweed (*Amaranthus spps*) have extremely high amount of oxalate that leads to oxalate poisoning of animals. Oxalate content is highest in the leaves and seeds with the lowest level in the stems. Humans and most animals lack enzyme capacity to metabolize oxalate, so execration is necessary to avoid pathological consequence from a high circulating oxalate [25]. Ruminants tend to be more tolerant of oxalate than non-ruminants, because rumen bacteria degrade oxalate into harmless formic acid and carbon dioxide but hungry and unexposure ruminants are the ones most susceptible to oxalate intoxication [28].

7.1. Mechanism of Action (MOA)

If large amount of oxalates are consumed by ruminants, it binds with free calcium in the rumen and excreted in the feces or be absorbed in the blood stream to affect tissue and serum calcium level. After entry in to the circulation, the oxalate binds with calcium ion forming insoluble calcium-oxalate complex. This results in hypocalcemia and tetany (acute cases) or derangement in bone growth or milk production from lowered intake of calcium. It may also crystallize in vasculature and renal tubules and infiltrate vessel walls causing vascular necrosis, hemorrhage, anuria and uremia [30].

7.2. Clinical Signs and Lesions

Oxalic acid ingestion results in corrosion of the mouth and

gastrointestinal tract, gastric hemorrhage, renal failure and hematuria. Other associated problems include low plasma calcium, which may cause convulsions, and high plasma oxalates. Most fatalities from oxalate poisoning are apparently due to the removal of calcium ions from the serum by precipitation. High levels of oxalate may interfere with carbohydrate metabolism, particularly by succinic dehydrogenase inhibition [30]. Acute oxalate poisoning causes a sudden decrease in serum calcium which impairs normal cell function and causes animals to develop muscle tremors and weakness, leading to collapse and eventual death. In chronic oxalate poisoning, filtration of insoluble calcium oxalate by the kidneys causes severe damage to the kidney tubules [28].

7.3. Diagnosis and Treatment

Diagnosis is based on history, clinical signs and necropsy lesions. The necropsy lesion includes pale, swollen and edematous kidneys with striking striation in the renal cortex. Recognition of oxalate crystals in the kidney or vasculatures, Calcium borogluconate (25%) solution should be given via IV or S/C in dose of 300-500ml in cattle, and 50-100ml in sheep and usually effective recovery. The use of saline glucose solution to produce diuresis and to combat alkalosis is rational.

Table 1. *Major poisonous plants and their toxic effect found in Ethiopia.*

Scientific name	Toxic part	Toxins	Effect/clinical signs	Species affected	Source	Growth forms	Reference
Brackenfern (Pteridium aquilinum	All parts	ptaquiloside	Bloody urine, nervous signs	Bovine, equine	Wild	Herb	[39]
Lantana camara	All parts	Lantadenes (A, B), C	Photosensitization, bloody urine	Camel, bovine and caprine	Wild	Shrubs	[32]
Castoroil (Ricinus communis)	Seed and fruit	ricin	Weakness, salivation diarrhea,	Bovine, Ovine	Both	Tree	[39]
Amaranthus spps	Leaf and seed	Oxalate		Bovine	wild	Herb	
Sudan grass	All parts	Nitrate	blue color of the mouth, eyes, shortness of breath, stumbling gait	Bovine, ovine	Wild and cultivate	Herb	[37]
Hypericumb perforatum L. (St. John's wort)	Leaf	Hypericin	Photosensitization		Wild	Herb	[27]
Fagopyrum esculentum Moench (buckwheat)	Leaf	fagopyrin	hematuria, bright blindness, carcinomas and thiamine deficiency	Bovine, horse	Wild	Herb	
Ammi majus L. (Bishop's weed)	All parts	Xanthotoxin, bergapten, ammirin	Photophobia, edema of muzzle, ear, inflammation of skin	Bovine, ovine	Wild	Herb	[15]
Arisaema Eunaephyllum	Other parts	calcium oxalate crystals	Pupils dilate, thirst, dry and	Bovines, caprines	Wild	Herb	[7]
Amaranthus spp	Leaf	nitrate and oxalates	Bloating, diarrhea	Bovine, ovine and caprine	Wild	Herb	[36]
Trifolium hybridum	Leaf	phytotoxin	Bloating, diarrhea	Bovine ovine	Wild	Herb	
Sorghum bicola	Leaf	hydrocyanic acid, the alkaloid	Bloating, diarrhea	Bovine, Ovine	Cultivated	Herb	[22]
Urtica doca	Leaf, stem	flavonoids	Skin burn	All species of animals	wild	Herb	[22]
Grass species	All parts	-	Bloating	Bovine, ovine	wild and cultivated	Herb	[15]
Medicago burweed	All parts	Isoflavonoids	Bloat	Bovine, ovine	Wild	Herb	[22]
Capparis tomentosa	Leaf, Seed pod	-	Bloat	Camel	Both cultivated and wild	Shrub	[15]
Prosopis juliflora	Leaf and seed pod	-	Bloating, Lower jaw dislocation	Camel, ovine, caprine, bovine	Cultivated and wild	Shrub	[22]
Parthenium hysterophorus	All part	*sesquiterpene lactones*	Anuria, hematuria Lantana	Camel, ovine, caprine, bovine	Cultivated andwild	Herb	[22]
Snowdonia polystachia	All parts		Bloating, diarrhea Bloating,	Camel, bovine, caprine	Cultivated	Herb	
Acacia absynica	Leaf		Bloat	Bovine, caprine	Wild	Tree	[3]
Prunus africana Medicago	Leaf		Bloating, salivation, Colic	Bovine	Wild	Herb	[3]
Plantago lanceolata	All parts		Bloating	Bovine, ovine	Wild	Herb	[3]
Rhizophoraceae	Leaf		Bloating, Colic	Bovine	Wild	Tree	[9]
Medicago polymorha	Leaf		Bloating Colic	Bovine	Wild	Herb	[9]
Trifolium burchellianum	Leaf		Depression, Erection of Hair, Bloating	Bovine	Wild	Herb	[9]
Medicago sativa	Leaf		Bloat/diarrhea	Bovine/caprine	Cultivated	Herb	[1]

Scientific name	Toxic part	Toxins	Effect/clinical signs	Species affected	Source	Growth forms	Reference
Brucea antidysenterica	Leaf		Alopecia/skin lesion	Bovine, equine	Wild	Tree	[1]
Euphorbia schimperiana	leaf		Inflammation of vulva/ penis/ eyes	Bovine/ovine/caprine	Wild	Herb	[1]
Pavetta gardeniifolia	Fruits and leaves		Bloat diarrhea, blood in urine	Camel, goats, sheep and cattle	Wild	Shrubs	[2]
Euphorbia nubica	Leaf and stems	*diterpene ester*	Diarrhea Irritates eye, blindness	Camel, goats, sheep and cattle	Wild	Shrub	[2]

8. Prevention and Control of Plant Poisoning in Livestock

Prevention and control of plant poisons is important before the animal exposed to toxin. There are different types of prevention and control method of poisoning plants, including chemical, mechanical and biological methods. One class of livestock is often more resistant to a toxic plant than others so be cautious when introducing livestock from other geographic locations. The main prevention and control method of plant poisonings are hungry livestock shouldn't realsed into pastures infected with toxic plants; make certain livestock have free access to salts and mineral supplements; provide adequate and clean water, be aware of special environmental conditions to restrict animal movement or change diet selection [23]. The other prevention and control methods are burning of herbaceous perennial plants, chaining, railing, root plowing. Biological control methods; use living organisms to feed upon toxic plants. This can be particularly effective when the plant is toxic to one species of livestock and not to another, because the non-affected species can be used to control the plant. Chemical is often the most economical and effective method of controlling poisoning plants. Toxic plants, depending upon their growth habits and susceptibility to herbicides, may be controlled with individual plant treatments [19].

9. Economic and Health Impact of Plant Poisons on Livestock

The livestock business has suffered significant losses due to a range of poisonous plants, primarily in East Africa, especially Ethiopia. Poisonous plants produce their toxic effects after being ingested and/or absorbed by animals which include physical upset, loss of productivity and death [3]. The poisons plants have direct and indirect economic losses to the livestock sector in the world. The direct economic losses of poisons plants includes death of livestock, abortion, birth defects, weight losses, lengthened calving interval, decrease fertility and immune response and dysfunction of the organs [10]. Costs that are incurred for livestock to prevent losses or costs incident to livestock poising plants, herding livestock to prevent poisoning, supplement feeding to prevent poisoning, and medical costs are some of the indirect losses linked to poisoning plants. Poisonous plants affect most of the major organ systems of the body. Each plants poison may affect specific organ systems like gastro intestinal system, circulatory system, urinary system, nervous system, musculoskeletal system, connective tissue and skin which lead to loss of function and general health disturbance of the animal [10, 20].

10. Conclusion and Recommendations

This review highlights some of the potential poisonous plants and their metabolic byproducts. These plants have a significant negative impact on the health and productivity of animals, which results in economic losses for the livestock business. Thus, timely diagnosis of toxic plant or biotoxin poisoning is very important and heavily relies on identification of the toxins in the feed, pasture, or ingesta along with appropriate clinical and pathological findings; i e., only the presence of poisonous plants or suspected feed in the environment is insufficient diagnostic evidence for toxicity without evidence of consumption (sign of grazing or presence of such feed in the ingesta) by the animal.

Therefore based on the above conclusion the following recommendations are forwarded:

1. Livestock owners should manage the feeding system of their livestock to reduce exposure to poisons plants.
2. Adequate feeding should ensure cattle need not graze toxic plants especially the green fronds during early summer.
3. Identification of poisonous plants and the proper management of animals and pastures will help to minimize the potential for poisoning animals from poisonous plants.
4. Avoid overgrazing and turning of animals into new pastures.
5. Creating awareness of the season when most poisonous plants are growing and causing problems for animal health.

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