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Review Article

Common poisonous plants affecting livestock of Himachal Pradesh: A Review

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Abstract

Most of the cited literature on plant poisoning among animals in Himachal Pradesh is not available to the majority of interested researchers, grassland managers and animal rearers dealing with livestock management. Therefore, a review of common poisonous plant affecting livestock was conducted in Himachal Pradesh. Based on this, the study was mainly focussed on local weeds (*Lantana* and *Ageratum* spp.), Napier and Sudan grasses, oak, Subabul trees and bracken fern poisoning for information on their toxins, clinical signs of phytotoxicity, its treatment and control methods in affected livestock. *Lantana camara* and *Ageratum* plants when taken along with other grasses or are grazed accidentally cause death of animals due to presence of poisonous alkaloids, further causing massive losses to livestock owners.

Key words: Hills, livestock, phytotoxicity, treatment.

Himachal Pradesh is having large amount of land under pastures. In recent years, majority areas of these pastures and grasslands have been covered by the obnoxious weeds. During scarcity of fodder and green grasses, livestock while grazing often eats these toxic plants, which leads to toxicity and death of affected animals and disturbs financial status of farmers. Livestock in the Himachal Pradesh rarely have problems from poisonous plants, this is generally true; however, severity of plant poisonings is mainly influenced by following factors such as chemical nature of the toxin, amount and time period of the toxin eaten, parts of the plant eaten, general condition and stage of maturity of the plant, environmental conditions in which the plant is growing, species of the animal and age, size, sex and general condition of the animal.

A poisonous plant contains specific substance which produces deleterious effects in the body of animals when taken in small or moderate amount. In this review article, we have discussed the most commonly prevalent toxic plants species which affects livestock of the state, clinical signs of phytotoxicity, its treatment and control methods.

1. Lantana Poisoning (Lal Phoolnu) (*Lantana camara)*

Lantana is considered as one of the world's ten worst weeds, was brought to India in the early part of nineteenth century as an ornamental plant (Matthew, 1971). It can propagate readily from stumps or cutting and from seeds disseminated by birds through their dropping (Gujral and Vasudevan 1983). The first report of lantana poisoning was from Townsville (Australia) in 1910. From India field reports of *Lantana* toxicity have been frequently observed which have accounted for high morbidity and mortality causing huge economic losses (Mandial 1996; Singh *et al.* 2003).The problem is more severe in hilly temperate parts of the country where its growth is more luxuriant, and the plant has covered all pastures. In Himachal Pradesh, the roadside and grasslands are covered with lantana plant in

abundance and this weed is also known by the name of Lantana, wildsage, bunch berry, lal phulanoo and punch phulbuti. In this state incidence varies from sporadic to heavy outbreaks reported from Hamirpur, Una, Bilaspur, Rampur-Bushair and Kangra district areas (Sharma *et al*. 1981; Sharma and Makkar 1981; Pal *et al*. 1997; Mandial *et al*. 2000; Singh *et al*. 2003). There are about 40 species of the genus *Lantana*. Amongst various spreads species *L. camara aculeata* has been responsible for livestock poisoning. *Lantana* plant of yellow, red variety is the most widespread and most poisonous in this part of the country (Sharma *et al*. 1981). The plant starts flowering in April-May and the fruiting continues till November-December (Gujral and Vasudevan1983).

The toxic signs of *Lantana camara* are mainly due to the presence of toxic triterpenoids namely lantadenes (lantadene A, lantadene B, lantadene C, lantadene D and Icterogenin). Lantadene A is considered as most hepatotoxic among all these (Sharma *et al*. 1992).

Clinical findings: Livestock reared in the area infested with *Lantana* shrub normally do not consume it as a matter of choice, but they do so during the scarcity of other green vegetation in the pasture (Sharma *et al*. 1981).The clinical symptoms of lantana poisoning are very characteristic. The signs develop in a proper and progressive way. Usually, the cases are reported after the lapse of a period of about 2 days when the poisoned animals become anorectic and are unable to defecate even after strenuous efforts. These are the earliest signs which the farmers report at their own. Some of the affected animals are presented with some initial signs of photosensitization in the form of swollen and oedematous ears and eyelids (Tokarnia *et al*. 1984 and Ahmed and Roy 1991). After ingestion of lantana foliage, the animal becomes partially off-feed within 2-6 hours, completely anorectic 24 hours onwards with complete ruminal atony from 48 hours onwards. Thus, the animal suffers from acute constipation/impaction (Tokarnia *et al*. 1984; Sharma and Wadhwa 2011).

The rumination is suspended or completely ceased, and the poisoned animal soon becomes dull and depressed with dry muzzle. The signs of photosensitization are observed in animals which continue to take green fodder. These are in the form of swollen and oedematous ears and eye lids with cracks/fissures on the muzzle and other non-hairy parts. Later, the photosensitization is so severe that the skin starts peeling off at various parts, particularly at the head and face regions, leaving behind raw surfaces with wounds (Mandial *et al*. 2000). The signs of jaundice include pale conjunctival, vaginal or vulvar mucous membrane and sclera of eye and dehydration is pronounced as the disease advances. There is recumbency and subnormal temperature in the terminal stages of the disease (Ali *et al*. 1995).

Treatment

There is no specific treatment available against the *Lantana* toxicity, resulting in heavy toll of livestock every year causing considerable economic loss to the farmers. However, treatment could be followed during the course of diseases, in gastro-intestinal phase during ingestion of lantana foliage which results in constipation could be cured with the purgatives. Sporadic efforts have been made to use oily purgatives to evacuate the undigested or semi-digested leaves with variable success (Sharma *et al*. 1988). Saline purgative to clear gastrointestinal tract comprising of magnesium sulphate ($MgSO_4$) have also been used but with great caution keeping in consideration the presence of dehydration. In advance cases of dehydration, it is always better to administer fluids before giving saline purgative (Pass and Stewart 1984). Mandial and Randhawa (1996) administered activated charcoal (to adsorbs toxic triterpenoids and further preventing its systemic absorption in the gastrointestinal tract) @ 5 g/kg body weight in 5 litres of multielectrolyte solution (Sodium chloride, Potassium chloride, Sodium citrate & Dextrose) orally, along with symptomatic and supportive therapy was found effective. It is worthwhile to use rumen cud transplantation and rumenotorics to promote forestomach function also. In Hepatic Phase, the use of liver tonics is justified provided damage to the liver is reversible. To counteract the effect of circulating toxins/metabolites, the specific antidote(s) may play a decisive role, however, so far, no antidote (s) could be developed, perhaps, because of inadequate information on the chemistry and geometry of the various lantana toxins. In extra hepatic phase, photosensitization develops because of photochemical reactions in the cutaneous system (Lau

1990 and Mandial *et al*. 2000), at this stage housing of the animals in shade along with the use of soothing agents, anti-inflammatory and antiseptics is useful. In case of secondary bacterial infections systemic antibiotics are helpful. Pal *et al*. (1997); Mandial *et al*. (2000) and Sharma (2003) successfully treated lantana poisoning cases in cattle with combined therapy comprising of five percent dextrose given parenterally, liver extract with B-complex, antihistaminic drug, rumenotorics, along with oral administration of magnesium sulphate purgative (@ 2.5 g per kg b.wt.).

Vaccination was also thought as a possible preventive mean in lanatana toxicosis. The toxic triterpene acid lantadene A and lentadene B were isolated and conjugated to bovine serum albumin or haemocyanin. These were emulsified and injected into sheep and cattle. After inducing lantana poisoning, cholestasis was found less severe in vaccinated animals (Stewart *et al*. 1988).

2. Bracken fern poisoning (*Pteridium acquilinum)*

Bracken is most widely distributed of all fern species. It is an aggressive colonizer of open ground and readily invades pastures and fields. Once established, the deep-set rhizomes are nearly impossible to eradicate. Because of its ability to render land unfit for livestock and its tendency to shade out other plant species (including some of conservation concern), bracken is among the world's worst weeds and is listed as an invasive species in some places.

The toxic signs are mainly due to the presence of Ptaquiloside, the major well-known carcinogenic principal present in bracken (Alonso-Amelot and Avendano 2002). There are also thiaminase factor, enzyme that effects the use of thiamine by the animal and aplastic anaemia factor causing bone marrow suppression. Joshi and Prasad (1975) revealed low blood serum levels of Thiamine in cattle affected with chronic bovine haematuria. Acute disease and death in cattle can result following ingestion of young bracken fronds.

Clinical signs

It is an economically important chronic disease of hill cattle in India and elsewhere. Diagnosis of enzootic haematuria in cattle is based on epidemiological, clinical, and pathological findings similar to the ones previous reports of this disease (Garpha 2017), combined with the botanical identification of the plant. In cattle, bracken fern chronic toxicity causes multiple tumours in the bladder wall and haemorrhages in the bladder mucosa, giving rise to so-called bovine enzootic haematuria (BEH) (Meuten *et al*. 2004). It is characterised by intermittent haematuria, chronic cystitis in cattle with possible straining and pain during urination and a variety of benign and malignant neoplastic conditions of the urinary bladder (Smith *et al*. 1988). Ingestion of bracken over several weeks when pasture is sparse can lead to toxicity. Consumption of bracken results in the depression of bone marrow (red and white blood cell and platelet production) causing aplastic anaemia (Prasad and Iyer 1986) and the plant has a direct or indirect anti-coagulant property. Cattle show signs after grazing bracken for 1 to 2 months, although death may occur within this time frame as well. Chronic bracken poisoning can occur in different times of the year and affects cattle over four years of age. The disease occurs when there is ingestion of less than 10 g/kg/day of the plant for one year or more. Enzootic haematuria is rarely seen in young animals (Tokarnia *et al*. 2012). Affected cattle show increased temperature, weight loss, and exhibit increased bruising and bleeding. Wadhwa *et al*. (2002) reported regular haematuria, gradual weakness in chronic enzootic bovine haematuria. Due to the excessive bleeding, cattle are anaemic, and can die within a week of showing signs. Disease caused by lack of blood cells and clotting factors may present as sudden death, but anorexia, marked pyrexia due to secondary bacterial infection, haemorrhages and blood from the nasal passages and vagina are more common signs.

Treatment

Treatment consisting of Inj. Vincristine in standard doses, bone marrow stimulator: N- Butanol (N-Butyl alcohol)-5ml (1g) in 10 ml olive oil preferably intramuscularly could be given in cattle. Symptomatic therapy comprising of antibiotic, haemostat, haematinic mixture (copper, cobalt & iron) and mineral mixture has led to mild and transient response suggesting long term therapy to increase longevity of affected animal (Garpha 2017).

3. Sorghum Sudan grass poisoning (*Sorghum sudanensis*)

Prussic acid poisoning occurs when livestock are pastured on sorghum-type plants, including grain sorghum, forage sorghum, sudangrass, sorghum sudangrass crosses, Johnson grass, sweet sorghums, and other sorghum-type plants. Prussic acid poisoning (also known as cyanogenesis) is caused by hydrocyanic acid, one of the most toxic and rapidly acting poisons (Vogel *et al*. 1987). Although many plants contain hydrocyanic acid, sorghum-type plants contain amounts that can cause severe poisoning. Cattle, sheep, and goats are most susceptible, while horses and swine are generally not susceptible. Dhurrin (Hydrocyanide poison) is the main toxic constituent and young shoot, and leaves are the toxic parts.

Clinical signs

Prussic acid poisoning can occur within a few minutes after an animal consumes forage high in prussic acid. In acute cases death occurs within five minutes. The first symptom of prussic acid poisoning is accelerated and deep respiration. The nose and mouth may become filled with foam, and in some cases, involuntary urination may occur. Cerebral anoxia with muscle tremors and clonic convulsions are few other clinical signs. The mucous membrane & blood colour is bright red to dark red. Dyspnea & cyanosis of mucosa in terminal stages. These symptoms are followed by depression, inability to stand, severe difficulty in breathing and finally death (Boyles 2020).

Treatment

Since prussic acid is one of the fastest acting poisons known, prompt diagnosis and treatment are required. Extremely low levels of prussic acid can cause toxicity. Only 2 mg per pound of body weight per hour can kill an animal. Two antidotes are used to treat prussic acid-poisoned animals: sodium nitrate and sodium thiosulfate. Recommended treatment is an intravenous injection of 1.2% sodium nitrate and 7.4% sodium thiosulfate in a 125-250 cc dose or sodium nitrite 5 g, sodium thiosulphate 15 g mixed in 200 ml distilled water intravenously in cattle could be used (Sinha *et al*. 2019 and Rajasokkappan *et al*. 2020). If the injection is given before the heart stops, the animal may be saved. Nonspecific treatment includes use of respiratory stimulants & artificial respiration.

The symptoms of prussic acid and nitrate poisoning are similar. However, nitrate poisoning usually occurs several hours after the animal has eaten high nitrate forage, and the animal's blood is dark chocolate brown in contrast to the red venous blood of the prussic acid-poisoned animal. If a nitrate poisoned animal is treated with sodium nitrate and sodium thiosulfate, it will die.

4. *Ageratum* poisoning (Neela Phoolnu)

Ageratum conyzoides and *Ageratum houstonicum* species are the most prevalent *Ageratum* in Kangra valley of Himachal Pradesh (Negi 1988). *A. houstonicum* commonly known as blue celestine or bill goat weed is an attractive member of the family Compositae and is one of the most obnoxious weeds in the world (Holm and Herberger 1969). The plant is easily identifiable by small blue terminal flowers in axillary heads and capitulate flowers. The flowers of *A. houstonicum* are slightly large and remain blue on maturity also. Being perennial, is available abundantly during August to December months in district Kangra, Sirmour, Bilaspur, Hamirpur, Mandi, Solan and Una of Himachal Pradesh near water channels, orchards, forests, waste lands etc. (Holm *et al.* 1979).

Ageratum is an annual plant that tends to become a pest in gardens and pastures. Fresh green leaves and stem are mainly toxic. The toxic signs are mainly due to the presence of constituents like Precocene-I, Precocene-II and Pyrrolizidine alkaloids (Ravindernath *et al.* 1987).

Clinical signs

Generally, animals do not consume this weed but during stress period or along with other green fodder, if consumed in sufficient quantity it results in health hazards. Clinical signs for *Ageratum* toxicosis in cattle are characterized by either an acute haemorrhagic course or sub-acute photodynamic dermatitis. Reduced feed and water intake, general depression, atony of GIT, tenesmus and blood-tinged black faeces are the other common clinical signs. The toxicosis has often been associated with Holstein-Friesian or crossbreed Holstein cattle less resistant to tropical climate conditions. The symptoms of *Ageratum houstonicum* toxicity in cattle was reported as haemorrhagic and photosensitization syndrome by Alfonso *et al.* (1989). Haemorrhagic syndrome shows

voluminous expulsion of frank blood from the gastrointestinal tract, diarrohea, tenesmus and atony of GIT, increased clotting time, increased cardiac and respiratory rates, and haemorhages in sclerotic membranes. *A. houstonicum* was first suspected for hepatotoxicity in North Sumatran cattle (Murdiati and Stoltz 1987). The plant has also been found to cause skin and eye irritation to human beings while doing hand weeding (Kanwar and Kharwara1988). Photosensitization was seen in survivors and exhibited especially in hairless or white-haired parts. Pathogenesis is not clearly understood but vascular endothelial damage, impairment of Vitamin K and hepatic damage are suspected to play pivotal role in generation of clinical symptoms (Alfonso *et al.* 1989).

Treatment

There is no specific antidote, however, symptomatic treatment using combination of 5% Dextrose saline and Liver extract together with haemostatic and haematinic drugs can reduces mortality upto 18 % (Singh 1995). Alfonso *et al.* (1989) successfully treated cases of experimentally induced *Ageratum* toxicity by giving vitamin K (haemostatic) preparations and liver protectants. Repeated removals before seed setting can be an effective method of its eradication. Therefore, for efficient control of *Ageratum* regular use of recommended herbicides for weed control in different crops should be ensured which will result in minimizing the population of this weed in concerned crop.

5. Ohi poisoning (*Albizia stipulata)*

Albizia, a genus of medium sized trees, found in abundance in Himachal Pradesh where they are grown to provide shade to the tea bushes and for the manurial value of its fallen leaves. *Albizia stipulata* is known by different regional names viz., Ohi (Punjabi, Pahari), Siran (Hindi), Chakua (Bengali), Laeli (Marathi), Hottabage (Kannada) and Pottavaga (Malyalam). The leaves of *A. stipulata* have been used as cattle fodder (Laurie 1945). In Himachal Pradesh, its leaves are fed to the animals in the lean months of November and December, when there is scarcity of green fodder. However, the local farmers do not feed the leaves of *A. stipulata* to their animals until they cross their succulent stage, as the immature leaves are considered to be toxic. Saponin and tannin are the main toxic constituents and young shoot leaves are highly toxic (Lohan *et al*. 1983; Humphrey 1988). Norton (1994) reported that *Albizia* species possessed antinutritive and toxic factors.

Clinical signs

Immature succulent leaves of *A. stipulata* are mainly responsible for toxicosis in ruminants. Depression, dullness, anorexia, grinding of teeth, dyspnea, oliguria, loose mucoid faeces and muscular stiffness are commonly seen clinical signs in ruminants with ohi poisoning (Wadhwa *et al*.1993).

Treatment

Although there is a no specific antidote, however, a combined treatment of saline purgatives, liver extract, dextrose saline 5% and rumen cud transplantation could prevent the appearance of clinical symptoms and could also lead to recovery of the animal (Manuja 1996; Wadhwa *et al*. 1993). If timely provision of combined therapy is practiced, there is possibility of the survival of the case, thereby preventing the economic loss caused by this toxicosis to the farming community.

6. Oak poisoning (Acorn, Bun) (*Quercus incana)*

Oaks are trees with leaves that turn brown but hang on through the winter. The fruit is the familiar nut borne in a scaly cup and called an acorn. Acorns from *Quercus* spp. can present a serious problem on pastures with oaks after autumn storms. The toxic signs are mainly due to the presence of toxins like Hydrolysable gallo tannin (Gallic acid), Pyrogallol (Tannic acid) and Phenol (Blakley 2013; MSD manual; Shi 1988).Tannins in acorns cause serious, often fatal, kidney damage. The leaves of young buds are mainly toxic.

Clinical signs

Cattle (especially less than 2 years of age), sheep and deer are susceptible (MSD Manual). Goats and swine are more resistant to poisoning while horses are rarely affected. The most encountered oak poisoning is of chronic nature. Symptoms include depression, lack of appetite, a gaunt and emaciated appearance, poor or rough hair coat, dependent edema (fluid build-up under the skin under the neck, abdomen or on the legs), digestive disturbances (both diarrhoea and constipation have been reported, with mucus covered or tarry stools), increased drinking, passage of copious amounts of urine which may contain blood, and death (Sandusky *et al.* 1977). Sudden deaths may occur but anorexia, depression and bloat due to ruminal stasis

are more common signs (Yeruham *et al.* 1998). Initially there is constipation and associated straining progressing rapidly to foetid tarry diarrhoea. There could be an extensive nephro- and hepatotoxicity in the affected cattle due to hydrolysable tannins and simple phenols in the oak leaves (Garg *et al.* 1992). Oak poisoning due to eating of immature tender oak leaves has caused 70% mortality in crossbred cattle and the animals exhibited anorexia, severe constipation and brisket edema.

Treatment

There is no specific treatment. Calcium hydroxide (15% of the ration) is found to be one of the effective measures under experimental condition (Zhu and Filippich1995). Supportive and symptomatic treatment including large volumes of intravenous fluids, administration of liquid paraffin or purgatives, rumenotoric drugs and injection of Liver extract could be effective (MSD manual).

7. Subabul poisoning (*Leucaena leucocephala)*

Leucaena leucocephala is a vigorous, rapidly growing, drought tolerant, palatable and high yielding tropical or subtropical legume. It's a branching, thornless, shrub or tree that can reach heights of 7-18 m. Mimosine (Aminopropionic acid) is the main toxic constituent and young leaves of *L. leucocephala* are more toxic than older leaves and seeds. Toxicity of mimosine may occur due to inhibition of tyrosine utilizing enzyme or incorporation of mimosine into biologically vital proteins in place of tyrosine (Crounse *et al*. 1962).

Clinical signs

Alopecia, loss of appetite, excessive salivation, in coordination of gait, enlarged thyroid gland and poor breeding performance are the main clinical symptoms seen with subabul poisoning in cattle (Ghosh and Bandyopadhyay 2007). Poor wool growth and haemorrhagic cystitis were observed in sheep fed on *Leucaena* diet (Koli *et al.* 2018).

Treatment

Mimosine containing feeds should not be fed exclusively to the animals. The supplementation of mineral mixture containing iron and drying of feeds is recommended, as sun drying usually destroys some mimosine content of the feed (Vijayakumar and Srinivasan 2018; Hatzade *et al.* 2020).

8. Napier grass poisoning (*Pennisetum purpureum)*

Pennisetum purpureum, also known as Napier grass, elephant grass or Uganda grass, is a species of perennial tropical grass and is one of the highest yielding tropical grasses. Nitrate (potassium nitrate) and nitrite are the main toxic constituents of *Pennisetum purpureum* roots and stems parts of the grass are mainly toxic. Nitrite is toxic to erythrocytes because it converts haemoglobin into the methaemoglobin. This is an oxidized form of haemoglobin, whose Fe^{2+} from the heme group is oxidized to the ferric state (Fe^{3+}), which cannot bind to oxygen (O_2) making it difficult to release oxygen to tissues and causing tissue anoxia (Haymond *et al.* 2005).

Clinical signs

Clinical signs with Napier grass toxicity include weakness, staggering gait, cyanosis, salivation, abdominal pain, diarrhea, vomiting, and dyspnoea (Sidhu *et al.* 1996). Post-mortem findings include typical chocolate brown blood resulting from methaemoglobinaemia (Seiler *et al.*1979).

Treatment

No Specific antidote is available for Napier poisoning. However, intravenous injection of methylene blue @ 4-8 mg /kg b. wt. (1% solution) followed by repetitive treatment at interval of 6-8 hours has found to be effective (Seiler *et al.* 1979).

9. *Setaria* grass poisoning (Golden Timothy) (*Setaria sphacelate)*

Setaria grass has variable levels of oxalates. The highest levels of oxalate occur at night, and the lowest in mid-afternoon. Oxalate levels are higher in fresh growth and exacerbated by nitrogen and potassium fertilization. The oxalate levels above 3.5% have been reported with toxicity in grazing animals (Cook *et al*. 2005; Stewart *et al.* 2010). The soluble oxalate in the form of potassium salt are the main toxins and young and growing leaves are the main toxic parts of the *Setaria* (Rahman *et al.* 2014).

Clinical signs

Main clinical signs with *Setaria* grass poisoning in ruminants include anorexia, paresis, muscle tremor, stumbling gait and dyspnea (Allison *et al*. 1985). Recumbency with head turned into flank,

hypocalcaemic Syndrome, pupillary dilation and death have been reported in sheep (Moore 2019).

Treatment

Calcium borogluconate parenterally have been found effective in the treatment of *Setaria* grass toxicity in animals.

Conclusion

This review highlights some of the potential toxic plants in the state of Himachal Pradesh, India. These toxic plants are causing huge economic losses to poor farmers of state in terms of loss of productivity and death of animal. Diagnosis may be fastidious in many cases since symptoms are non-specific, so focus should be on revealing history of exposure for proper diagnosis. Once diagnosis is established, removing animals from toxic source should be the first step. Immediate, lifesaving measures may be needed initially. Beyond this treatment consists of preventing further absorption of poison, providing supportive treatment and administering specific antidotes, if available. The information on toxic plant chemistry is mostly limited to their nitrate or cyanide content. Research is needed not only to determine which plants represent a potential risk for animal health and production but also to investigate their phytochemistry and toxicology. It would be very useful if veterinarians were able to document plant poisoning cases through government reporting services. Furthermore, university and government scientists, veterinarians, and extension personnel could fully investigate the various toxicoses and publish their findings in specialized journals. This would help to identify toxic species for further phytochemical and toxicological studies and possibly pharmacological activity.

Conflicts of interest: The authors declare that there is no conflict of interest in this review article.

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