World Journal of Animal Science Research
Vol. 3, No. 3, May 2015, pp. 1 - 17, E-ISSN: 2333 - 8946
Available online at http://wjasr.com/

Review Article

REVIEW: PLANT, FUNGAL AND FARM CHEMICAL POISONS OF VETRRINARY IMPORTANCE

[1]Samuel Derso, [1]Shewatatek Melaku

[1]University of Gondar, Department of Veterinary Clinical medicine, PO BOX 196

Phone: +251910927004 E-mail: samuelderso@gmail.com

[1]Alkan Medical and Veterinary College, Bahrdar

Phone: +251913110630 E-mail: shewavet@gmail.com

SUMMARY

Poisoning of veterinary importance could arise from different sources of which some plants, fungi and farm chemicals are important mainly associated with feeding characteristics, access to them and moreover some plants and fungi are inherently mixed with pasture. Bracken fern, Sorghum, *Duturastramonium*, and *Astragalus* are among others in plant poisoning of farm animals. Fungus can cause harm to the animal without invading animal's body through mycotoxins. Intensive use of herbicides and insecticides is also becoming a problem to livestock especially in pasture. Animals can be exposed to poisons in many ways accidentally ingestion, drought and scarcity of pasture are common exposure ways. Irrespective of their sources toxins have effects to animals such as birth defects, reproductive dysfunction, dermatitis, neurological effects and death. The loss from cases of poisoning are due to mainly death, reduced weight gain, reduced reproductive performance, veterinary fees, control costs and reduced land value. Diagnoses of poison depend on history, clinical signs and laboratory examination. Treatment for poisoning is based on three principles, prevention of further absorption, supportive and symptomatic treatment and using of specific antidotes. Most poisons may be prevented by keeping hungry animals and animals with poor condition away from pastures populated with known poisonous plants.

Key words: *Antidotes, Farm chemicals, Mycotoxins, plant, poisons*

1. Introduction

Livestock poisons come from a variety of sources and their identification and control can be very problematical. Veterinary important poisons could arise from different types of plants, fungal and farm chemicals [1].

There are many plants in the pasture that are poisonous to livestock. Most of these poisonous plants are unpalatable (have bitter taste) but different factors like drought, scarcity of pasture will lead to consumption of these plants by animals and cause illness or death. Among these plants, bracken fern poisoning, nitrate or nitrite poisoning, cyanide poisoning and photosensitization are common [2]

Molds grow on any stored feeds, the highest incidence being on feed with high moisture content, and also grow on standing plants, especially of seed heads. Feed that become mouldy and stale may be contaminated with fungi which produce toxins called mycotoxins. Several species of fungus produce mycotoxins, includes *Aspergilus flavus*, *Aspergilus ochracus* and *Aspergilus fumigatus* [3].

In these days increasing agricultural technology leads to production of different types of chemicals for different purpose such as herbicides, insecticides, fungicides and rodentcides. Improper use of these chemicals in various agricultural and animal husbandry practices leading to toxicities in animals. Accidental ingestion of chemicals or feed contaminated by these chemicals could be a source to farm chemical poisoning [3].

Arsenic poisoning, chlorinated hydrocarbon and organophosphate compound poisoning are some of them among frequently known chemicals to cause poisoning in livestock [4]. Hence the objectives of this paper are designed:

- To summarize common plant, fungal and farm chemical poisons of veterinary importance

2. Plant poisons

Throughout the world, there are many plants in pastures that are poisonous to livestock. The poisons of these plants are mostly secondary compounds, not involved in the plants basic metabolism, and they often have a bitter taste. Due to their bitter taste, grazing livestock normally ignore poisonous plants but when there is drought, prolonged dry season, hungry and thirsty animals forced to eat them [2].

2.1 Nitrate and Nitrite poisoning

Nitrates and nitrites are closely linked as cause of poisoning. Nitrates may cause gastroenteritis when ingested in large quantities but their chief importance is as a source of nitrites which may be formed after ingestion of nitrates [5].

The common sources of nitrates for farm animals includes cereal crop, certain specific plants such as red root, rye-grass (Fig. 1), winged and water from deep wells. Immature green forage crops such as wheat, corn, or maize and sorghum may accumulate excessive amount of nitrate after heavy use of nitrate fertilizers, or after heavy ran causing water runoff from adjacent heavily fertilized area. Ingestion of large quantities of this fodder over a short period causes an excess production of nitrite in the rumen, which interferes with oxygen transportation by the blood [2].

- Fig. 1 Rye grass (from: http:// www.sheltoncarpet.net/grassturf.htm)

In growing plants nitrite is always found in the form of nitrate, but when it enters to the body of animal's changes to nitrite and then oxidizes ferrous state of hemoglobin to ferric state and his forms methemoglobin which cannot carry oxygen. Then the animal will die due to lack of oxygen. Sometimes nitrate reduced to nitrite in the plant before ingested by the animals. This is occurring mostly in oaten hey [6].

Plants that accumulate more than 1.5% dry matter of nitrate potentially toxic. In accordance to London, *et al.* (1967 cited by 1) toxic dose is hard to compute because of variation in susceptibility, in production of nitrite from nitrate. In accordance to Crawford *et al.* (1966 cited by [1] the most important factor influencing susceptibility appear to be the rate of ingestion of the nitrate baring plant. Animals which are join to nitrite poisoning dies after 12-24 hours of ingestion of the toxic plants [6].

For nitrite and nitrate poisoning, there is considerable variation between species in their susceptibility. Pigs are most susceptibility followed by cattle, sheep and horse. Even though, pigs are highly susceptible but they are affected only if ingest preformed. Young pigs also have gastrointestinal micro flora capable of reducing nitrate to nitrite. Ruminants are especially vulnerable because the Ruminal flora reduces nitrate to ammonia with nitrite as an intermediate product, but mature monogastric animals, except equids, are more resistant to nitric toxicosis because this path way is age limited [1].

Clinical sign of nitrate poisoning includes sudden onset of sever dyspnea, salivation, abdominal pain, vomiting, weakness and in sever case the mucosal and conjunctiva become brown in color [1].

Analysis of tissue, ingest or stomach content or intestinal content, urine and suspected material may be necessary to confirm the diagnosis. Rapid response to treatment with Methylen blue is a good criterion for field use [1]. Intravenous administration Methylen blue has a good response to nitrite poisoning. Ruminants likely to be exposed to nitrites or nitrates should received adequate carbohydrates in their diets and traveling or hungry animals should not be allowed access to dangerous plants [2].

2.2. Cyanide poisoning

Cyanogenic glycoside is chemical which is found in many plants including grain sorghums (Fig. 2) and forages such as Sudan grass (Fig. 3) and Columbus grass. Hydrolysis of the glycoside by rumen enzymes release hydrogen cyanide (prussic acid). Under certain circumstances, the cyanide contents of such plants may high and ingested quickly in sufficient quantity then poisoning will occur. The most dangerous time is when the plants are growing rapidly after a period of retarded growth during rains after drought or during over grazing. Immature or wilted plants are also more likely to be poisonous than mature plants. The seeds or a pit of several plants such as peach have been the source of Cyanogenic glycosides in many cases. Eucalyptus species, kept as an ornamental house plant, have been implicated in death of small animals [2].

Fig. 2 Sorghum

Fig. 3 Sudan grass

(From: http:// www.sheltoncarpet.net/grassturf.htm)

Cyanogenic glycosides in plants yield free hydrocyanic acid (HCN), also known as prussic acid. When it hydrolyzed by beta glycosidase enzyme or when other plant cell structure is disrupted or damaged, it could be either by freezing, chopping or chewing, microbial action in the rumen, further release free cyanide [2].

Although apple and other fruits contain prussic acid glycosides in leaves, and seeds but little or none in the fleshy part of the fruit. In sorghum species forage grass, leave usually 2-2.5 times more HCN than do stems; seeds contain non HCN. New shoots from young, rapidly growing plants often contain high concentration of prussic acid glycosides. The Cyanogenic glycoside potential of plants can be increased by heavily nitrate fertilization especially in phosphorous deficient soils spraying of Cyanogenic forage plants with herbicides can increase their prussic acid

concentrations for several weeks after application [1]. Ruminants are more susceptible than monogastric animals and cattle slightly more than sheep. Hereford cattle have been reported to be less susceptible than other breeds [7].

Poisons animals have difficulty in breathing, bright red visible mucus membrane and collapse and convulsion are some manifestation and die shortly after wards less acute case may stagger about for one or two hours before death [7]. In addition to appropriate clinical sign, history and post mortem examination, demonstration of HCN in rumen (stomach) contents are important for diagnosis of cyanides poisoning. Specimens recommended for cyanide analysis include the suspected source (plant), rumen or stomach contents, heparinized whole blood, liver and muscle [8].

Treatment and prevention, cyanide poisoning treated with sodium nitrite (10 g per 100ml of distilled water or isotonic saline) given intravenously at dose of 20mg per kg body weight followed by thiosulphate (20%) IV at 500mg per kg. Pasture grass should not be grazed until they are 15-18 inch tall to reduce danger from prussic acid poisoning. Animals should be out to new pasture later in the day; prussic acid release potential is reported to be highest during early morning hours. Whenever there is environmental stress, grazing should be monitored properly [2]. Sorghum hay silage usually loses 50% of prussic acid content during curing and ensiling process. In a rare occurrence hazardous concentration of prussic acid may still remain in the final products. Hay has been dried at oven temperature for up to 4 days with no significant loss of cyanide potential. These feed should be analyzed before use and should be diluted or mixed with grain or forage that is law in prussic acid content to achieve safe concentration in the final product [7].

2.3. Bracken fern poisoning

Ingestion of significant quantities of bracken fern poisoning produces acute sign of poisoning related to thiamine deficiency in monogastric animals and to bone marrow depletion in ruminant. The toxic effects appear to be cumulative and may require 1-3 months to develop depending on the species of animals and quantity of the toxin consumed. Most acute poisoning is seen after periods of drought when grazing is scarce; however, the plant is toxic even when present as contaminated hay [8].

Bracken fern (Fig. 4 and 5) *(pteridium aquilium)* is a plant which is distributed in upland and marginal areas throughout most part of the world. Both leaves and rhizomes contain the toxic principles, which vary in concentration with the season [1].

Poisoning in non ruminant is due to thiaminase; the effects are essentially those of vitamin- B1deffeciency. But thiamine deficiency is generally not a problem in ruminants. The toxic cause death of precursor cells in the bone marrow and those cells with a short life span are affected first. An initial leukocytosis is followed by granulocytopenia and thrombocytopenia with results susceptibility to infection and tendency to spontaneous hemorrhage horse seem to be particularly susceptible, while disease in pigs is rare [8].

For diagnosis of bracken fern poisoning, blood thiamine level analysis is important, blood thiamine levels decrease from an average of 8.5 to 2,5 microgram per deciliter, while blood pyruvate level increase from a normal 2 to 8.5 microgram per deciliter. Intravenous injection of 5mg per kg body weight thiamine is effective treatment at early stage [9].

Fig. 4 bracken fern (growing stage) Fig.5 bracken fern (adult)

(From: http:// www.rossvet.edu.kn/poisonousplant_display.cfm?toxic=200)

2.4 *Datura stramonium poisoning* (devils apple*)*

Datura stramonium (Fig.6) also called Jimson weed, Gypsum, Locoweed, Jameston weed. Thorn apple, Anges, Trumps and Zombies cucumber are also common poisonous plants. It contains tropene alkaloids that are sometimes used as hallucination. The active ingredient is atropine, hyoscyamine and scopolamine which are classified as deliriants, or anticholinergics. *Datura stramonium* is found in many parts of the world. This plant is growing in most habitats, but thrives in high nutrient soil. Goats occasionally eat Jimson weed and subsequently die [10].

Fig.6 *Datura stramonium* (flowering stage)

(from: http:// www.hlasek.com/datura_as_a6226.htm)

There is mnemonic for the physiological effect of *Datura* or atropine toxicosis. The actual effects are reported to be cycloplegia and mydriasis, lushed, warm and dry skin, dry mouth, urinary retention and illus, rapid heartbeat, hypertension or hypotension. In case of over dose the effects are hyperthermia, comma respiratory arrest, and seizures [10].

2.5 Astragalus poisoning

Member of the genus Astragalus (Fig. 7) are known by different names. Those most widely used are locoweed and milk vetch. There are over 2000 species in this genus that are known currently, of which many are known to carry various toxins. This plant mostly found in western and Midwestern part of the world. Due to the range and toxicity, livestock poisoning by loco weed is the most wide spread toxic plant problem in the western United States [10].

Fig.7 *Astragalus* (flowering stage)
(From: https://www.google.com.et/search?q=astragalus&es)

Despite the unpleasant effect of *Astragalus* poisoning, animals often become habituated to eating, fueling their further intoxication by the plant. *Astragalus* poisons in three main ways: with indolizidine alkaloid swainsonine as in the case *Astragalus lentiginous* and *Astragalus lusetanicus*, with the nitro propanol bearing glycoside miserotoxin as in the case of *Asrgalus mser* and with toxic level of selenium metabolites as in case of *Astragalus bisutcatys* [10].

Indolizine alkaloid: the two indolizidine alkaloids of plant origin are swainsonine and castanospermine, both of them affecting cellular enzymatic activities. Swainsonine is found in *Astragalus, Oxytropis and* sswainsona species and some ipomoea species plants, ingestion of the toxic plants over a long period cause lysosomal storage disease in all animal species. The disease is common in America, Australia and China, grazing animals must ingest the plant

for atleast two weeks, more usually six weeks, before clinical sign appears. All grazing animal species are affected; if access to the plant is cease death is rare [1].

Swainsonine is a specific inhibitor of lysosomal alfa-mannosidase causing accumulation of mannose in lysosomes and thus widespread neurovisceral cytoplasmic vacuolation. The vacuoles are accumulation of mannose- rich oligosaccharides. Vacuolation reaches its greatest intensity in the central nervous system and this is probably related to the predominance of nervous sign in the disease. Vacuolation of the chorionic epithelium may be related to the occurrence of abortion and transient infertility suspected in rams to be the result of a similar lesion in the epithelium of the male reproductive tract [11].

After several weeks grazing in affected pasture adult animals begin to loss body condition and young animals cease to grow and nervous sign is followed. Pregnant ewe ingesting *Astragalus* species plants may abort or produce abnormal offspring [11].

Diagnostic confirmation is depends on identification of the alkaloids. Vacuolation in circulating lymphocytes occur in poisoning due to swainsona species, serum level of alfa-mannosidase is reduced and swainsonine levels increased. The urikne content of mannose containing oligosaccharides is greatly increased during the period of intake of swainsonine. The characteristic microscopic lesion is fine vacuolation of the cytoplasm in nervous throughout the central nervous system. Similar vacuolation is present in cell of other organs especially the kidney and the fetus in animals poisoned by *Astragalus* species, high blood and tissue level of swainsonine detected [9].

3. Photosensitization

Phylloerythrine is a break down product of plant chlorophyll in the fore stomach of ruminants, which is in healthy animals excreted through bile by the liver, but in animals impaired liver function, however, Phylloerythrine accumulates in the blood and is transported to the skin which is sensitized to the ultra violate rays of the sun as a consequence, resulting in photosensitization in lightly pigmented area of animals skin [12].

A number of plants that poison liver may result in photosensitization if eaten in sufficient quantity, including *Lantana camara* (Fig.8), it is a known cause of photosensitization where it has become tenacious weed over taking good grazing land [13].

The two important events for the occurrence of photosensitization is animals are hypersensitivity due to ultra violate rays and photodynamic compounds. Photodynamic substances have ability to absorb light energy and forms unstable

high energy which damage cells by the formation of free radicals from cellular components which enhance chain reaction of phospholipids leads to loss of cellular integrity and finally cause death [12].

When animals feed plants which contain high photodynamic substances such as hypericine and hypericum fagoprin will develop photosensitivity and called primary photosensitivity is associated with liver damage, when animals feed some plants such as *Lantana camara* and *Sencio* chlorophyll changes to Phylloerythrine by micro flora and it taken through blood to liver to be conjugated with the amino acids and excrete through the bile and then it will circulate through the skin as a result absorb ultra violate rays and finally cause photosensitization. Photosensitive animals are photophobic immediately when exposed to sunlight and squirm in apparent discomfort, they scratch or rub lightly pigmented, exposed area of skin and for confirmatory diagnosis serum analysis and liver biopsy will be taken for indication of liver disease and examination of blood, feces and urine for porphyrine are important [1]. Animals with photosensitization may exhibit lining of eyelids or conjunctiva which becomes red and swollen, which discharge from the lesion, jaundice, nervousness and digestive disorders [12].

While photosensitization continues, animals should be shade fully, or preferably, housed and allowed to graze only during darkness, and also antihistamine drugs are recommended. If *lantan camara* poisoning is a problem, attempts should be made to remove the plant from the pasture by digging out and burning [12].

Fig. 8 *Lantana camara* (flowering stage)
(From: http:// www.davesgarden.com/guides/pf/showimage/12277)

3. Mycotoxins

Fungus can cause harm to the animal without invading animal's body. Thus fungus produce harmful metabolites called mycotoxins. If the fungus was growing on a stored food and even though the fungus is no longer alive due to

unsuitable environment, if it produce a myotoxin, itself rather it could be consumption of its toxins. Several species of fungus produce mycotoxins and include *Aspergillusflavus*, *Asperlliusfumigatus*and*clavicepapurpurea* [10].

Acute or chronic mycotoxicosis can result from exposure to feed or bedding contaminated with toxins that may be produced during growth of various species of fungus on cereals,hay,straw, pastures or any other fodder. Mycotoxinc diseases have generally its some characteristics. They are not transmissible from animal to animal, the cause may not be immediately identified [3]. Treatments with drug or antibiotics have little effect on the course of the disease, and outbreaks are usually seasonal because particularly climatic sequence may favor fungal growth and toxins production [3].

Confirmatory diagnosis of mycotoxic disease requires a combination of information. Detection of fungus spore alone, even at a high concentration, is not sufficient for diagnosis; fungal spores even molds growth may not present without formation ofmycotoxins. Especially important in diagnosis is the presence of the disease documented to be caused by known mycotoxins, combined with detection of the mycotoxin in either feed stuffs or animal tissues. Sometimes, more than one mycotoxin may be present in feed stuffs, and their different toxicologic properties may cause clinical sign and lesions that are not consistent with those seen when animals are closed experimentally with pure single mycotoxin [13].

Several mycotoxins are immune suppressive which may allow virus, bacteria or parasite to create secondary diseases that are more obvious than the primary [10]. In reaching a diagnosis of mycotoxicosis characterized by reduced feed intake, reproductive failure, or increased infectious disease due to immunosuppressive. Differential diagnosis must be carefully established and eliminated by a combination through clinical and historical evaluation [13].

There are no specific antidotes for mycotoxins and removal of the source of the toxin eliminates further exposure. The absorption of some mycotoxins has been effectively prevented by aluminosilicate [10].

4.1 Aflatoxicosis

These are produced by toxigenic strains of *Aspergillusflavus*and*Aspergillusparasiticus*on peanuts, soybeans, corn or maize, and other cereals either in the field or during storage where moisture contents and temperature are sufficiently high for mold growth. Usually, this means consistent day and night temperature should be greater than

70^0F. The toxic response and disease in mammals and poultry varies in relation to species, sex, age, nutritional status and duration of intake and level of aflatoxind in the ration [3].

Aspergillusflavus rarely invades stored grains alone; various species of fungus will normally grow on a substrate prior to the invasion by *Aspergillus*such as *AspergillusGlavcus* and *candida pseudo tropicalis*, and even if they are known as aflatoxins producer, the amount of mycelial growth that occur in animal feed, with several species of fungi is involved [10].

Aflatoxin occurs in many part of the world and affect growing poultry (especially ducks and turkey). Young pigs, pregnant sows, calves and dogs. Adult cattle, sheep and goats are relatively resistant to the acute form of the disease but they will become susceptible of toxinc diets are fed over long periods. Aflatoxicosis is manifested my non-specific clinical sign such as loss of milk production, anorexia, depression, reduced weight gain, and also in chronic case there is decrease growth rate and decreased feed conversion efficiency and in case of ruminants decrease ruminal motility [1].

Aflatoxins bind to macromolecules, especially nucleic acid and nucleoproteins. Their toxic effects include mutagenesis due to alkylation of DNA, carcinogenesis, teratogenesis, reduced protein synthesis and immunosuppression, reduced protein synthesis result in reduced production of essential metabolic enzymes and structural protein for growth. The liver is the principal organ that is affected. High dose of aflatoxins result in sever hepato-cellular necrosis; prolonged low dosages result in reduced growth rate and liver enlargement [1].

For diagnosis of aflatoxicosis, in addition to history and necropsy findings, microscopic examination of the liver should indicate the nature of hepatotoxin. The presence and level of aflatoxins in the feed should be determined in the urine or kidney or in milk of lactating animals if toxin intakes are high [1].

Aflatoxicosis can be treated by following detoxification, and by using adsorbent, hydrated sodium calcium aluminum silica, which absorbs the toxin, because they have high affinity for toxin. The recommended prevention of aflatoxicosis is inhibition of mold growth and treatment of grain with anhydrous ammonia for 10 - 14 days to reduce aflatoxin. Young, newly weaned, pregnant and lactating animals require special protection from suspected toxic feed [1].

4.2 Ergotism

Ergotism is a worldwide disease of farm animal resulting from continued ingestion of sclerotic of the parasitic fungus *Clavicepspurpurea*which replaces the grain or seed of rye and other small grains or forage plant, such as the bromes, blue grasses and rye glass. The hard, black, elongated sclerotia may contain varying qualities of ergot alkaloids, of which ergotamine and ergometrine and pharmacologically most important [9].

Ergot causes vasoconstriction by direct action on the muscle of the arteries or it is a potent initiator of smooth muscle contraction like uterus and arterioles and this leading to vasoconstriction and also repeated dosage injure vascular flow and eventually lead to complete stasis due to thrombosis, and then if the environment is cold predisposes the extremities to gangrene. In addition, ergot has potent oxytocic action and also causes stimulation of the central nervous system, followed by depression. Ergot alkaloids inhibit pituitary release of prolactin in many mammalian species, with failure of both mammary development in late gestation and delayed initiation of milk secretion, resulting in a galactia at parturition [15].

Cattle, pigs, sheeps and poultry are involved in sporadic outbreaks and most species are susceptible to ergotism. Diagnosis of ergot is based on finding the causative fungus (ergot sclerotia) in grain, hay, or pastures provided to livestock showing signs of ergotism. Ergot alkaloids may be extracted and detected in suspected ground grain meals. Identical sign and lesions of lameness, and sloughing of the hooves and tips of the ear and tail, are seen in fescue foot in cattle grazing in winter on tail fascue grass infected with an endophyte fungus, in which the ergot alkaloids ergovaline is considered a major toxic principle [2].

For ergotism replacement of contaminated feed, antibiotic and supplement feeding are recommended as a treatment and it can be controlled by an immediate change to an ergot free diet. Under pasture feeding, frequent grazing or topping of pastures prone to ergot ingestion during the summer or months reduces flower-head production and helps controls of this disease [2].

5. Farm Chemical Poisoning

With the increasing and indiscriminate use of chemicals in various agricultural and animal husbandry practices, the possibilities of toxicosis in animal have increased considerably. A large number of chemicals include pesticides, herbicides, fungicides and rodenticides are in use, which may be taken by animals either by accidental ingestion through weeds, grass, fodder forage of the field which these chemicals have been used [16].

Inaccordance to O'Brien (1970) poisoning of animal by agricultural chemicals has become a major sector in farm animal medicine. This is because of the multiplicity of the products used and difficulty of determining generic composition from trade name, or even there is difficulty of remembering the exact chemical formula of complicated organic compounds.

5.1 Organophosphorus compound poisoning

These compounds are insecticides and commonly used on stored grains, crops, and animals as well as in soil, and widely used in dips, sprays and dusting powders. They kill insects and ticks by interfering with the breakdown of chemical impulse and causing uncontrolled nervous stimulation [16].

Poisoning with organophosphatecompounds can occur in livestock as a result of contamination of feed stuff with granular insecticides as a rodenticides, using the wrong concentration or accidental ingestion, which causes a buildup of acetylcholine and causing uncontrolled nervous stimulation and showing a range of nervous signs, including excessive salivation and tear, constriction of the pupil, frequent urination and vomiting [2]. Severity and course of intoxication is influenced principally by the dosage and route of exposure. In acute poisoning, the principal clinical sign may be respiratory distress and collapse followed by death due to respiratory muscle paralysis.

Animals with acute organophosphate poisoning have nonspecific or no lesion; pulmonary oedema and congestion, hemorrhage and oedema of bowel are found. An important diagnostic aid is the cholinesterase activities in blood and brain. Three categories of drug are used to treat organophosphate poisoning; these are muscarinic blocking agents, cholinesterase reactivators, emitics, cathartics and absorbent for decrease further absorption. Atropine is one of its specific antidotes [2].

5.2 Chlorinated hydrocarbon poisoning

This poisoning could arise from dips, or sprays at the wrong concentration or accidental ingestion. These agents are stimulants of central nervous system and animals which are affected by these poisoning generally noted to be alert. Due to tissue residues and chronic toxicity, use of these chemical is drastically curtailed, only lindone and methoxychlor are approved for use on or around livestock [17].

Chlorinated hydrocarbons include Aldrin, Benzene Hexachloride, Chlordane, Dieldrin, Toxaphene, Methoxychlor. These insecticides are generally central nervous system stimulant. They produce a great variety of sign and the most obvious are neuromuscular tremor and convulsion [Miller, 1967:cited by 1] described that body temrature may be

very high and muscle fasciculation occurs, becoming visible in the facial region and extending backward until the whole body is involved.

Large dose of DDT and Methoxychlore causes progressive involvement leading to trembling or shivering followed by convulsion, and death. If death has occurred suddenly, there may be nothing more than cyanosis. Usually there is congestion of various organs (particularly, lunges, liver and kidney) and a balanched appearance of all organs if the body temperature was high before the death, the diagnosis of chlorinated hydrocarbon toxicity, use chemical analysis of brain, liver, kidney, fat and stomach or rumen content is necessary to confirm the poisoning [Ray, *et al.*, 1975 cited by [1].

These poisoning does not have known specific antidotes, but when exposure is by spraying, dipping or dusting, a thorough bathing without irritating the skin using detergents and copious quantities of cool water recommended. If exposure is by ingestion, gastric lavage and saline purgatives are indicated [Miller, 1967: cited by [1].

5.3 Arsenic poisoning

Arsenic poisoning in animal is caused by several different types of inorganic and organic arsenical compounds. Arsenic poisoning varies with factors such as oxidation state of arsenic, solubility, species of anima involved and duration of exposure. Therefore, the toxic effect is produced by phenyl arsenic feed additives and other inorganic and organic compounds must be distinguished (O'Mrien, 1970) [18].

Inorganic arsenic include tri-oxide, arsenic pento-oxide, sodium and potassium arsenate, sodium and potassium arsenite and lead or calcium arsenate are more soluble and therefore, more toxic than the pentavalents of arsenate compounds. The lethal oral dose of sodium arsenate in most species is between 1 and 1.25 mg per kg, cats may be more sensitive than other species. In anminal with recent exposure and no clinical sign, emisis should be indicated followed by activated charcoal with a cathartic [18].

Organic arsenic phenyl arsenic organic arsenicals are less toxic than inorganic compounds or aliphatic and other aromatic organic compounds. Aliphatic organic compounds (arsenicals) include cacodylic acid and acetorsonic acid. These are generally used as stimulants in large animals, but their use in lo longer common. Some aliphatic arsenical such as Mono Sodium Methane Arsenate and Disodium Methane Arsenate are still used as deforiant. Persistence of MSMA or DSMA in soil and their tendency to accumulate in plant is a potential for arsenic poisoning, especially in grazing animal.

For arsenic poisoning, there is no specific treatment, but the neurotoxic effect are usually reversible if the offending feed is withdrawn within 2-3 days of onset of ataxia. Once paralysis occurs, the nerve damage is irreversible. Blindness is also usually irreversible but, animals retain their appetite and weight gain good if competition for food is eliminated [19].

6. Conclusion and recommendations

Poisons in livestock are one of the major problems that need proper management and prevention rather than treatment. Toxic sources could arise from plants, fungal sources and chemicals. Plants in particular are of important in farm animals as most of the poisonous plant found mixed with pasture and if they are grazed upon or unintentionally incorporated into animal feed impose a devastating problem to animal causing death decreased production and reproduction problems. But under normal condition those poisonous plants have unpleasant flavor and animals usually does not consume them unless they become hungry due to different environmental factors such as drought and pasture scarcity. The recognition of poisonous plant in hay or forage is essential to the prevention of plant poisoning in animals. Fungal toxins are most of the time due to improper storage of grains and other animal feeds. The best way to support a diagnosis of a case of poisoning in animals is to confirm the presence of a toxic agent or its metabolites in the animal's body or environment. Treatment of disease caused by poisons are not effective so that prevention by destroying the source and as well as by denial to access of poisons sources. Hence, poisons of different sources are important to veterinary medicine:

- Poisons should be considered during diagnosis of usually acute clinical cases.
- Identification of plant types in a pasture is important before allowing grazing.
- Proper application of farm chemicals, proper storage of animal feeds will go long in prevention of farm chemical and mycotoxins.
- Treatment of poisonous cases should be as fast as possible and better if directed to neutralization of the toxins.
- A veterinarian, animal health workers and extension workers should know and adapt poisons plant in an area of working.

Acknowledgement

I would like to thanks Dr. Wubshet Asefa for his valuable comments and devotion of his precious time in correction of this manuscript.

7. Reference

1. Blood, D.C. Radostits, O.M. and Henderson, J.A. (2005). A Text Book of the Disease of a Cattel, Sheep, Pigs And Goat 9th Ed. Bailer Tindall.
2. Coste, R. and smith, A.J. (1994). The Tropical Agriculturalist. Macmillan Publisher Ltd.
3. Boyd, R. F. (1995). Basrc Medical Microbiology.Little Brown and company (Inc.)
4. Vankamper, K.R. (1969). Am, vet. Med. Assoc., **154**, 623.
5. O'Hara, P.J. and Fraster, A.J. (1973). N.Z. Vet, J., **56**, 256.
6. Pshigami, K. and Inoue, K. (1976). *Res. Bull. Obihirouniue.,* **10**, 45.
7. Burrowsa, G.E. and Way, J.L. (1979). Am *J.* vet.Res.**65**, 23-46
8. Aiello, S. Mays, M. (1998) the Merk Veterinary Manual, 8th Ed. Merk and Co. NIC. White House Station, N.J., U.S.A.
9. Stegelmeier, B.L., Jemes, L.F., Panter, K.E. and Molyneux, R.J. (1995). Tissue and Serum Concentration in Sheep Ingesting *AstragalusLentiginosus* (Locoweed). Vet. Hum. Toxico., **37**, 4.
10. Raiphs, M.H., Panter, K.E. and Jems, L.F. (1990). Feed Preference and Habituation of Sheep Poisoned by Locoweed J. Anim Sci. **68**: 1354-1362
11. Panter, K.E. and Hartery, W.J. (1989). Vet. Hum. Toxico., **31**, 42.
12. Clare, N.T. (1955). *Adv. Vet.sci.,* **2,** 182
13. Monlux, A.W. (1963). J.Am,vet.Med.Assoc., 142, 1989.
14. Prior, M.G. (1976). *Con. J. Camp. Med.*, **40**, 75.
15. Greatorex, *J.C* and Mantle, *P.G.(1974). J. Reperod.*, **37**, 33.
16. Chauhan, R.S. and Argarwals D.K (2006). Text Book of Quaternary Clinical and Laboratory Diagnosis.Jaypee Brothers Medical Publisher Ltd.
17. Di Pietro, *J.A.* and Halibrton, J.C. (1979). *J.Am,vet.Med.Assoc.*, **175,** 142.
18. O'Brien, J.J. (1970). Toxicological Aspects and Some Modern Anthelmintics, *Aust. Vet, J.*, **46**, 297.
19. Anon (1980). *J. Am.Vet.Med Assoc.*, **177**, 82.