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Winter-Associated Toxicities in Livestock: Causes, Clinical Signs and Preventive Measures

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INTRODUCTION

Livestock are at greater risk of toxicity during the winter season because of changes in feed availability and environmental conditions. The shortage of forage may force animals to consume unfamiliar, less palatable, or potentially poisonous plants. Cold temperatures and frost can also influence plant physiology and increase the concentration or activity of certain toxic substances. In addition, poor ventilation, physiological stress, increased use of chemicals and greater dependence on stored feeds may contribute to different types of toxicity, including plant poisoning and toxicity caused by chemicals, air pollutants, salt, heavy metals and mycotoxins [1].

1. Plant Toxicities in Winter

Cyanogenic Plants

Plants such as sorghum, Johnson grass, linseed and bamboo shoots may contain cyanogenic compounds that release hydrogen cyanide, particularly under stressful conditions such as frost. Cyanide interferes with cytochrome oxidase in the mitochondrial electron transport chain, resulting in cellular hypoxia. Affected animals may develop rapid breathing, seizures and characteristic bright-red blood.

Treatment: Sodium nitrite @ 20 mg/kg body weight, administered slowly by IV route over 3–4 minutes, followed by sodium thiosulfate @ 500–660 mg/kg IV.

Prevention: Frost-damaged forage should be removed or avoided.

Nitrate-Accumulating Plants

Plants such as amaranthus, pigweed and beet tops can accumulate high levels of nitrate, particularly under conditions of heavy fertilization and limited sunlight. In the animal body, nitrate is converted to nitrite, which interferes with the oxygen-carrying capacity of blood and may result in methemoglobinemia. Affected animals commonly show difficulty in breathing, weakness and chocolate-brown coloured blood.

Treatment: Methylene blue @ 8–10 mg/kg body weight, administered slowly by IV route.

Prevention: Forage should be tested for nitrate concentration before feeding, particularly when there is a suspected risk of nitrate accumulation.

Oxalate-Rich Plants

Plants containing high levels of oxalates, including spinach, beet leaves, dock and sorrel, can cause toxicity when consumed in large quantities. Oxalates bind with calcium in the body and may result in hypocalcaemia and, in severe cases, kidney damage. Clinical signs may include weakness, muscle tremors and reduced appetite, while severe cases can progress to renal failure.

Treatment: Calcium borogluconate (20–25%) @ 250–500 ml, administered slowly by IV route, as indicated by the severity of hypocalcaemia.

***Lantana camara* Toxicity**

Lantana camara is an important poisonous plant affecting grazing livestock. It contains pentacyclic triterpenoids, mainly lantadenes, which cause liver damage and interfere with bile excretion. This may result in secondary photosensitization, particularly in areas of unpigmented skin. Affected animals may show anorexia, constipation, jaundice, photosensitization and other signs of liver dysfunction.

Treatment: Activated charcoal @ 1–4 g/kg body weight orally and saline purgatives such as magnesium sulfate @ 0.5–1 g/kg orally may be used as supportive measures.

Bracken Fern Toxicity

Bracken fern (*Pteridium aquilinum*) can cause toxicity in livestock when consumed repeatedly over a prolonged period. It contains ptaquiloside and other toxic compounds that may cause bone marrow suppression, thiamine deficiency and carcinogenic effects. Clinical signs depend on the amount and duration of intake and may include weight loss, anaemia, haemorrhages and incoordination. In severe or chronic cases, urinary bladder tumours and acute haemorrhagic syndromes may occur.

Treatment: Thiamine (vitamin B_1) @ 5–10 mg/kg body weight by IV or IM route may be administered in cases associated with thiamine deficiency. Supportive treatment and antibiotics may be used when indicated.

2. Pesticide Toxicities

Pesticides and rodenticides are widely used for crop protection, pest control and treatment of stored grains. During cold weather, the breakdown of some chemicals may be slower, allowing them to remain in the environment for longer periods and increasing the possibility of accidental exposure or poisoning in livestock [2].

Organophosphate and Carbamate Insecticides

Organophosphate and carbamate insecticides produce toxicity mainly by inhibiting acetylcholinesterase, resulting in excessive accumulation of acetylcholine at nerve endings. Affected animals commonly show increased salivation, lacrimation, urination and diarrhoea (SLUD signs). Other signs may include constricted pupils, muscle fasciculations, respiratory distress and weakness.

Treatment: Atropine @ 0.2–0.5 mg/kg IV or IM may be administered. Pralidoxime (2-PAM) @ 20–50 mg/kg IV may be given at 8–12-hour intervals when indicated. Activated charcoal @ 1–4 g/kg body weight orally may be used to reduce further absorption in suitable cases.

Prevention: Pesticides should be stored away from feed and water sources and their use inside poorly ventilated animal sheds should be avoided.

Pyrethroid Toxicity

Pyrethroids are commonly used insecticides and their persistence may increase under cold conditions. Accidental exposure can result in neurological signs, particularly muscle tremors and excessive salivation.

Treatment: Diazepam @ 0.1–0.5 mg/kg IV, administered slowly, may be used to control severe tremors or seizures. Activated charcoal @ 1–4 g/kg body weight orally may be considered following recent ingestion.

Rodenticide Toxicity

Anticoagulant rodenticides such as warfarin and brodifacoum interfere with normal blood clotting and can result in prolonged bleeding. Clinical signs may include weakness, anaemia, external or internal bleeding and widespread haemorrhages.

Treatment: Vitamin K_1 @ 0.5–2.5 mg/kg body weight may be administered, with treatment continued for several weeks depending on the specific rodenticide and clinical response.

Bromethalin is a neurotoxic rodenticide that interferes with energy metabolism in the central nervous system and may cause cerebral oedema. Affected animals may develop neurological signs such as tremors, weakness, incoordination and progressive paralysis.

Treatment of Bromethalin Toxicity: Activated charcoal @ 1–4 g/kg body weight orally may be given to reduce absorption. Mannitol 20% @ 0.5–1 g/kg IV, administered slowly, may be used to help manage cerebral oedema. Anticonvulsants such as diazepam @ 0.1–0.5 mg/kg IV, administered slowly, may be used to control seizures.

3. Weedicide (Herbicide) Toxicities

Paraquat Toxicity

Paraquat is a highly toxic herbicide and accidental ingestion can cause serious damage to the respiratory system. Exposure may generate free radicals and result in severe oxidative injury to lung tissue, which can progress to pulmonary fibrosis. Cold stress may further aggravate respiratory damage. Affected animals may show difficulty in breathing, weakness and cyanosis.

Treatment: Fuller's earth @ 0.5–1 g/kg body weight orally may be administered following recent ingestion. Antioxidants and other supportive measures may also be used.

Prevention: Livestock should be prevented from entering recently sprayed fields until they are considered safe for grazing.

Glyphosate Toxicity

Glyphosate is a commonly used herbicide. Accidental ingestion generally causes irritation of the gastrointestinal tract and may result in signs such as salivation, vomiting, diarrhoea and abdominal discomfort. Most cases are relatively mild, although severe exposure may occasionally affect the kidneys.

Treatment: Activated charcoal @ 1–4 g/kg body weight orally and appropriate fluid therapy may be used according to the severity of poisoning.

Prevention: Glyphosate should be stored and handled properly and animals should not be allowed access to treated areas or improperly stored herbicide.

2,4-D Herbicide Toxicity

2,4-D herbicide can produce gastrointestinal and neuromuscular disturbances following excessive exposure. It may interfere with cellular metabolism and affect cell membrane function. Clinical signs may include ruminal atony, muscle rigidity, weakness, incoordination and ataxia.

Treatment: Activated charcoal @ 1–4 g/kg body weight orally may be administered. Sodium bicarbonate 5% or 8.4% @ 1–5 mEq/kg IV, given slowly, may be used when indicated. Methocarbamol @ 44–220 mg/kg IV, administered slowly and carefully, may help control muscle rigidity and tremors.

Prevention: Animals should be kept away from treated fields until the spray has dried and the recommended re-entry period has passed.

4. Chemical Fertilizer Toxicities

Chemical fertilizer poisoning can become a concern during winter because of changes in feeding behaviour and management. Reduced availability of forage may encourage animals to consume spilled fertilizer. Reduced water intake and changes in rumen microbial activity may further influence the severity of toxicity.

Nitrogen-containing fertilizers, particularly urea and ammonium nitrate, are important sources of poisoning in livestock [3].

Urea Fertilizer Toxicity

Urea is rapidly converted into ammonia in the rumen. When large quantities of urea are consumed, ammonia production increases rapidly and the rumen pH may rise above 7.5–8.0. Under these alkaline conditions, a greater proportion of ammonia remains in its toxic, un-ionized form and can be absorbed into the bloodstream. Clinical signs usually develop rapidly and may include excessive salivation, ruminal bloat, muscle tremors, incoordination and convulsions.

Treatment of Urea Toxicity: Vinegar diluted with cold water may be administered orally to reduce ruminal pH, along with rumen lavage where appropriate. Treatment should be carried out carefully according to the animal's condition.

Prevention: Urea should be mixed thoroughly with the feed and should never be offered in concentrated form. Animals should not be given urea-containing feed when they are excessively hungry.

Ammonium Nitrate Fertilizer Toxicity

After ingestion, rumen microorganisms convert nitrate into nitrite. Nitrite is absorbed into the bloodstream and converts haemoglobin iron from the ferrous (Fe^{2+}) to the ferric (Fe^{3+}) state, resulting in the formation of methemoglobin. Since methemoglobin cannot carry oxygen effectively, affected animals develop tissue hypoxia. The blood may appear characteristically chocolate brown. Clinical signs may include weakness, rapid breathing, tremors and respiratory distress.

Treatment: Methylene blue @ 8–10 mg/kg body weight, administered slowly by IV route.

5. Air Pollutant and Gas Toxicities

Carbon Monoxide (CO) Toxicity

The risk of carbon monoxide poisoning increases during winter because coal, wood and other fuels may be burned inside animal sheds for heating. Poorly ventilated sheds allow this colourless and odourless gas to accumulate. Carbon monoxide binds strongly with haemoglobin and forms carboxyhaemoglobin, reducing the oxygen supply to tissues. Affected animals may show incoordination, staggering, sudden collapse and, in severe cases, death. The mucous membranes may have a characteristic bright or cherry-red appearance [4].

Treatment: Immediate administration of 100% oxygen is recommended.

Prevention: Animal sheds should be properly ventilated and adequate ventilation should be maintained whenever heating devices are used. Dry bedding should also be provided.

Ammonia Toxicity

Ammonia can accumulate inside poorly ventilated animal sheds due to the bacterial decomposition of urine and manure. Since the gas tends to accumulate near the floor, animals may be exposed to high concentrations for prolonged periods. Inhalation causes irritation and damage to the respiratory mucosa, reducing the normal defence mechanisms of the respiratory tract. Prolonged exposure may result in coughing, conjunctivitis and an increased risk of secondary respiratory infections such as pneumonia.

Treatment: Supportive treatment should be provided according to the severity of respiratory signs. Antibiotics may be used when secondary bacterial infection is suspected. Meloxicam @ 0.5 mg/kg SC or IM may be used for inflammation and associated discomfort when indicated.

Prevention: Proper ventilation, regular removal of manure and urine and the use of dry bedding can help reduce ammonia accumulation.

Sulfur Dioxide and Nitrogen Oxide Toxicity

Sulfur dioxide and nitrogen oxides may be released during the combustion of fossil fuels, particularly when heating equipment is used in

poorly ventilated sheds. When inhaled, these gases react with moisture in the respiratory tract and produce irritating compounds. Exposure can cause coughing, nasal and respiratory irritation and difficulty in breathing. Severe or prolonged exposure may result in significant lung damage and pulmonary oedema.

Treatment: Oxygen therapy and supportive respiratory care are important. Methylprednisolone @ 1–2 mg/kg IV or IM may be used when clinically indicated. Albuterol @ 0.025–0.05 mg/kg by nebulization may be used as a bronchodilator.

Prevention: Adequate ventilation should be maintained in animal sheds, particularly when heaters or fuel-burning equipment are being used.

6. Coal Emissions-Related Toxicities

Burning coal releases a mixture of harmful pollutants, including carbon monoxide, sulfur dioxide, nitrogen dioxide and fine particulate matter (PM2.5). During winter, exposure may increase when coal is burned inside poorly ventilated animal sheds. Inhalation of these pollutants can irritate and inflame the respiratory tract, reduce the normal defence mechanisms of the lungs and increase the risk of respiratory infections, including pneumonia [5].

Treatment: Oxygen therapy and supportive treatment may be provided. Methylprednisolone @ 1–2 mg/kg IV or IM may be used when indicated, while albuterol @ 0.025–0.05 mg/kg by nebulization may help relieve bronchoconstriction.

Prevention: Indoor burning of coal should be avoided. Animal sheds should have adequate ventilation and good airflow.

7. Salt Toxicity

Salt poisoning, or sodium ion toxicity, may occur when livestock have limited access to water during winter. Frozen water sources, dehydration and the consumption of excessive amounts of salt or highly concentrated feed can increase the risk. Excess sodium draws water out of cells initially and, when water is provided too rapidly during correction, rapid movement of water into brain cells may contribute to cerebral oedema. Affected animals may show neurological signs such as excessive thirst, weakness, incoordination, blindness, circling and seizures. Severe cases may be fatal [6].

Treatment: Rehydration should be slow and carefully controlled, with the rate adjusted according to the animal's condition and degree of dehydration. Mannitol 20% @ 0.5–1 g/kg IV, administered slowly, may be considered for the management of cerebral oedema when clinically indicated.

Prevention: Animals should have regular access to clean, unfrozen water during winter. Sudden access to large quantities of water should be avoided in severely dehydrated animals.

8. Heavy Metal Toxicities

Lead Toxicity

Lead poisoning may occur when livestock are exposed to lead-containing materials, including discarded batteries, contaminated feed or other sources. During winter, burning of waste materials for heating and keeping vehicles running inside poorly ventilated sheds may increase the risk of exposure. Lead affects both the nervous and digestive systems. Clinical signs may include loss of appetite, ruminal stasis, abdominal discomfort, blindness, muscle tremors and severe convulsions [7].

Treatment: Calcium disodium EDTA (Ca-EDTA) @ 50–75 mg/kg/day IV, administered slowly, for 3–5 days may be used as a chelating agent. Thiamine @ 2–5 mg/kg may also be administered slowly as supportive therapy.

Cadmium Toxicity

Cadmium exposure in livestock may occur through contaminated feed, soil and prolonged use of phosphate fertilizers containing cadmium. Plants can absorb cadmium from contaminated soil, allowing it to enter the livestock food chain. Chronic exposure can

interfere with mineral metabolism, particularly iron utilization and may cause anaemia and damage to the kidneys. Renal tubular dysfunction can become severe and may be irreversible with prolonged exposure.

Treatment: There is no specific antidote for chronic cadmium poisoning. Treatment is mainly supportive and should focus on removing the source of exposure and managing the affected animal according to clinical signs.

Mercury Toxicity

Mercury poisoning may occur following exposure to mercury-containing fungicides, industrial waste and contaminated feed or water. Mercury can affect the nervous system and kidneys and prolonged or severe exposure may result in serious organ damage. Clinical signs may include weakness, tremors, incoordination, blindness and impaired kidney function.

Treatment: Chelating agents such as dimercaptosuccinic acid (DMSA) @ 10 mg/kg body weight orally every 8–12 hours may be used under veterinary supervision.

Zinc Toxicity

Zinc poisoning may occur when animals repeatedly lick or chew galvanized wires, pipes, containers or other zinc-containing materials. Excessive zinc intake can cause gastrointestinal irritation and may interfere with normal red blood cell function. Affected animals may develop severe diarrhoea, weakness, haemolysis and jaundice.

Treatment: Calcium disodium EDTA (Ca-EDTA) @ 50–75 mg/kg/day IV, administered slowly for 3–5 days, may be used as a chelating agent. Supportive treatment should be provided according to the clinical condition.

9. Enterotoxemia

Enterotoxemia caused by *Clostridium perfringens*, particularly type D, can occur when animals are suddenly shifted to high-energy diets or when there is inadequate roughage in the ration. During winter, reduced forage availability and stress associated with cold weather may increase the risk. Affected animals may develop diarrhoea, abdominal distension, neurological signs such as opisthotonus and, in severe cases, sudden death.

Treatment: Clostridial antitoxin may be useful when administered early, along with fluid therapy and other supportive treatment.

Prevention: Regular vaccination, proper dietary management and avoidance of sudden changes to the ration are important preventive measures.

10. Mycotoxin Toxicities

Mycotoxin contamination of stored feed can become a problem during winter when feed is stored under conditions that favour mould growth. High moisture, inadequate drying and poor ventilation can promote fungal growth in feed and fodder. Important mycotoxins include aflatoxin, which mainly affects the liver; ochratoxin, which primarily affects the kidneys; fumonisin, which may cause respiratory and other systemic problems; and ergot alkaloids, which cause vasoconstriction and may lead to tissue damage and gangrene. Animals exposed to mycotoxins may show reduced feed intake, poor growth, reduced milk production, jaundice, weakness and lameness, depending on the type and level of toxin involved [8].

Treatment: Removal of the contaminated feed is the most important step. Supportive treatment, including suitable hepatoprotective measures, may be provided according to the clinical signs. Activated charcoal @ 1–4 g/kg body weight orally may be used in selected cases to reduce toxin absorption.

Prevention: Mouldy or visibly contaminated feed should be removed and not offered to animals. Proper drying, storage and regular inspection of feed can help reduce mycotoxin contamination.

CONCLUSION

Winter can increase the risk of different types of toxicity in livestock because of changes in feed availability, storage conditions, water intake and housing management. As a general preventive measure, feed should be properly stored and checked for mould and high-risk feeds should be tested for nitrates and mycotoxins. Animals should have continuous access to clean, unfrozen water and sudden changes in the ration should be avoided. Livestock sheds should be adequately ventilated and cleaned regularly and burning coal, wood or other fuels inside poorly ventilated sheds should be avoided. Pesticides, fertilizers and other chemicals should be stored safely and kept away from feed, water and bedding. Animals should be provided with a balanced ration, gradually introduced to changes in winter feeding and prevented from grazing areas containing known poisonous plants or contaminated feed. Regular vaccination against enterotoxemia, appropriate mineral supplementation and careful use of urea and chemical fertilizers can further help in reducing the risk of toxicity during winter.

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