Lead - An Emerging threat to livestock

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Lead is a common cause of poisoning of domestic animals throughout the world. Lead is a heavy, low melting, bluish-gray metal that occurs naturally in the Earth's crust. However, it is rarely found naturally as a metal. It is usually found combined with two or more other elements to form lead compounds. Lead is a naturally occurring element that people have used almost since the beginning of civilization. Human activities have spread lead widely throughout the environment-the air, water, soil, plants, animals and man-made constructions. Because lead is spread so widely throughout the environment, it can now be found in everyone's bodies; most people have lead levels that are orders of magnitude greater than that of ancient times (Flegal and Smith., 1992, 1995). Lead poisoning affects cattle of all ages, but is most common in calves (Blakley BR and Brockman RP. 1976), (Botts RP. 1977). The highest incidence has been reported in dairy cattle (Botts RP. 1977). It has been estimated that 150,000 cattle worldwide are exposed annually to toxic levels of lead and that at least 20,000 acute deaths occur (Bratton GR and Zmudzki J. 1984).

Cattle have relatively indiscriminate eating habits. They will readily drink crankcase oil, lick machinery grease and chew batteries (Blakley BR., 1984). In ruminants there is a tendency for metallic lead particles to settle in the reticulum; poisoning results from the gradual conversion of lead particles to soluble lead acetate due to the acidity of the forestomachs (Blakley BR., 1976). Young calves are more susceptible to lead poisoning because of their innate curiosity, their active calcium absorption mechanism and the fact that milk and milk-replacer diets promote lead absorption (Zmudzki J *et al* 1986 a),

Once lead falls onto soil, it sticks strongly to soil particles and remains in the upper layer of soil. That is why past uses of lead such as lead in gasoline, house paint and pesticides are so important in the amount of lead found in soil. Small amounts of lead may enter rivers, lakes and streams when soil particles are moved by rainwater. Cattle are the most susceptible livestock, with calves the most likely victims. However, lead poisoning can occur in all domestic animals including horses, birds/ poultry and dogs. Pigs are the least susceptible. Lead poisoning is most common among calves because they are curious feeders, and both milk and milk substitutes increase the amount of lead absorbed by calves. Sucking animals can also receive lead in their milk.

Permissible limits in various media

The permissible limits of lead in (a) ambient air:0.75 mg/m3 for sensitive areas (bird sanctuaries);1.0 mg/m3 for residential areas;1.5 mg/ m3 for industrial areas (CPCB,1995-96), (b) Water:0.05 mg/L for drinking water (c) Effluents: 0.10 mg/L for discharge of industrial effluents in inland surface water. These are generally in line with international standards.

Major sources of lead on the farm

Livestock may find lead in rubbish dumps and around farm buildings and machinery.

In cattle, many cases are associated with seeding and harvesting activities when used oil and battery disposal from machinery is handled improperly. Other sources of lead include paint, linoleum, grease, lead weights, lead shot, and contaminated foliage growing near smelters or along roadsides. Lead poisoning is also encountered in urban environments, and renovation of old houses that have been painted with lead-based paint has been associated with lead poisoning in small animals and children.

Animals may find sump oil and other sources of lead attractive as lead compounds can have a sweet taste. Contributing factors may include boredom from confinement, and depraved appetites

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from malnutrition and phosphorus deficiency. However, predisposing factors are not essential. Lead shot may be a source of poisoning of domestic poultry and wild birds. It is most often seen in water fowl, such as ducks and geese, which swallow lead shot and fisherman's sinkers from the bottom of lakes and ponds.

Sources in the Home

* Lead-Based Paint-Present on many surfaces in homes not recently rebuilt or remodeled
* Lead Pipes-More common in older homes
* Lead Solder -On pipes and water heaters
* Enameled or Ceramic Pots and Dishware Improper glazing can leech lead into foods
* Paper Wrappings-Holiday paper and party decorations (10g/kg).
* Food Packages-Polythene plastic bags, flour bags(20mg/kg),cardboard boxes with dyes (50mg/kg).
* Candy Packaging-Candy bar wrappers(7g/kg), Colored sports trading cards packaged with gum(88mg/kg).

Lethal levels of lead in animals:

1. Cattle-Intakes of greater then 6 mg/kg body weight can lead to chronic poisoning and intakes greater than 10 mg/kg BW may cause acute lead poisoning.
2. Sheep-Generally occurs only in lambs and symptoms of poisoning appear at intakes greater than 4.5 mg/kg BW.
3. Pigs, Goats and Rabbits-More resistant than sheep or cows. Very minor signs of poisoning occur at intakes of 60 mg/kg BW. This is equal to blood concentrations of 130 micrograms per dl.
4. Horses-Respiratory "roaring" occurs at intakes of 6.4 mg/kg BW. Signs of anemia occur at intakes of 7.4 mg/kg.
5. Birds-Poultry can withstand dietary intakes of 100 mg/kg feed with no symptoms. Levels of 500 mg/kg induced serious poisoning.

Dogs and Cats-Nervous symptoms of poisoning appear at intakes of 5 mg/kg BW/day.

Pathogenesis

Lead is a well known to inhibit the biosynthesis of heme, and consequently of hemoglobin and to decrease the life span of circulating red blood cells (Potula, 1996).

Absorbed lead enters the blood and soft tissues and eventually redistributes to the bone. The degree of absorption and retention is influenced by dietary factors such as calcium or iron levels. In ruminants, particulate lead lodged in the reticulum slowly dissolves and releases significant quantities of lead. Lead has a profound effect on sulfhydryl-containing enzymes, the thiol content of erythrocytes, antioxidant defenses, and tissues rich in mitochondria, which is reflected in the clinical syndrome. In addition to the cerebellar hemorrhage and edema associated with capillary damage, lead is also irritating, immunosuppressive, gametotoxic, teratogenic, nephrotoxic, and toxic to the hematopoietic system. (The Merck's manual)

Even a small amount of lead can kill cattle. Cattle will readily drink crankcase oil, lick grease from machinery and chew on lead plumbing and batteries. The lead in these materials settles in the stomachs of cattle, where stomach acids gradually change the lead into poisonous salts. Lead causes anemia when it combines with red blood cells and bone marrow. It damages the small blood vessels, causing bleeding, and deprives the nerves, the brain and other organs of oxygen. Lead severely damages the kidney and liver. It also causes sterility, fetal death and abortion. Cattle that eat lead will likely die. A single battery left in a field can poison 10 to 20 calves. All animals with access to a source of lead are at risk. When one or two animals in a herd die or show signs of poisoning, other animals in the herd may also be suffering from lead poisoning. These animals may appear healthy, but be growing poorly as a result of subclinical lead poisoning.

Clinical findings

Although clinical signs of poisoning normally precede death, most animals are simply found down or dead on the ground.

1. Acute lead poisoning: Animals show signs like colic, staggering gait, rolling eyes, slobbering, muscle spasms, blindness, uncoordinated attempts to climb obstacles, excessive response to external stimuli, head pressing and convulsions. This may be followed by death.
2. Sub acute lead poisoning: Animals with subacute poisoning may display dullness, loss of appetite, abdominal pain and diarrhoea.
3. Chronic poisoning: Animals show wasting, loss of appetite, anaemia, constipation and recumbency. Paralysis and death may occur.

Sometimes acute attacks may occur during the course of chronic poisoning. Horses show dyspnea. A combination of gastro-intestinal and nervous signs may also occur. The gastro-intestinal effects may

produce either constipation or diarrhoea.

It is often difficult to distinguish lead poisoning from other diseases that affect the nervous system of cattle. A correct diagnosis is extremely important for identifying the problem and preventing a recurrence of the disease.

Lesions

Animals that die from acute lead poisoning may have few observable gross lesions. Oil or flakes of paint or battery may be evident in the GI tract. The caustic action of lead salts causes gastroenteritis. In the nervous system, edema, congestion of the cerebral cortex, and flattening of the cortical gyri are present. Histologically, endothelial swelling, laminar cortical necrosis, and edema of the white matter may be evident. Tubular necrosis and degeneration and intranuclear acid-fast inclusion bodies may be seen in the kidneys. Osteoporosis has been described in lambs. Placentitis and accumulation of lead in the fetus may result in abortion. (Osweiler,G.D. *et al* 1985).

Differential diagnosis

1 Nutritional deficiencies - Thiamine and Vit. A deficiency.
2 Metabolic factors deficiencies: Ketosis, Hypoglycaemia.
3 Bacterial infection: Clostridium group of organisms, Listeriosis.
4 Viral Infection: Infectious Bovine Rhinotracheitis, Bovine Malignant catarrh.
5 Poisons: Mercury, Organochlorins, Plant poisons.

Prevention

The growing recognition of lead's dangerous effects has led to a worldwide initiative to reduce lead content of gasoline (Lovei, 1998). In India, Pb levels have been reduced from 0.56 g/L in gasoline (pre 1986) to 0.013 g/L (in year 2000)(CPCB, 1997). The following practices should be done to reduce the incidence of lead poisoning in livestock.

1. Sound nutrition and consistent, regular feeding will minimize pica or abnormal feeding behavior in livestock.
2. Refuse should always be dumped at a single, isolated, fenced-off location, and preferably buried; pastures are unsuitable sites for refuse.
3. Used batteries and crankcase oil should be stored and disposed of safely, without spillage and confined to areas where animals have no access.
4. Vehicle service and machinery storage areas should be separate from areas used by livestock.
5. Holding of animals in the yard should be minimized, because yards tend to be multi-purpose areas with high risk for contamination.
6. Only lead-free paints should be used on surfaces and fixtures to which livestock has access.
7. Because most poisonings occur following a change of location or management procedure, all pastures and holding areas should be inspected carefully before introducing animals to them.

Treatment

Studies have shown that the consumption of certain nutrients in the diet including minerals such as Ca, P, Fe and Zn and vitamins such as vitamin C, E and thiamine can reduce absorption of dietary lead in children (Sonawane,1999).

There is ample evidence that vitamins, essential minerals and trace elements play a preventive role in reducing lead poisoning in humans (Calabrese, 1978) and animals (Flora et al, 1982; Flora & Tandon, 1990).

Treatment for acute lead poisoning is seldom effective. The disease has usually progressed too far to be treated once clinical signs are seen. Treatment only stops or lessens the clinical signs of lead poisoning and must be begun early if an animal is to be saved.

1. Calcium - disodium EDTA: - For large animals, slow I/V injection of 6.6% solution @ 70 mg/kg/day divided in 2 - 3 doses for 3-5 days. The injection should be repeated after a gap of two days.
2. Emetics, Saline purgatives and sedatives or hypnotics.

Specimens for laboratory examination:

1. Clotted blood and faeces should be submitted from live animals.
2. From dead animals submit liver and kidney, unpreserved. Preserved liver/kidney, EDTA blood and a blood smear may also assist differential diagnosis.
3. In addition, supply samples of any suspected sources of lead.

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H5N1 fears on smuggled chickens - China (Hong Kong)

Smuggled chickens may have been the cause of the latest outbreak of the bird flu virus. The suspicions arose after the farms that supplied the 3 stalls at the center of the H5N1 outbreak cleared preliminary checks. Customs officials said that they are treating reports of smuggling seriously. Officers also said they would be stepping up intelligence-gathering operations to prevent underground poultry shipments. AFCD assistant director of inspection and quarantine Thomas Sit Hon-chung said that the invoices show the chickens that tested positive came from 3 local and 6 mainland registered farms. But checks at the 3 local farms showed no problems, and mainland inspection and quarantine authorities have also confirmed there are no problems with the 6 farms on their side. Sit said more than 70 samples taken from farms, wholesale, and retail markets all tested negative for H5N1. More tests will be conducted at local and mainland farms and the results will be out in a few days. The scare erupted on Saturday [7 Jun 2008] after chickens at the Po On Road market in Sham Shui Po tested positive. All 2700 chickens in the market's 9 stalls were culled.

Indonesia quits offering prompt notice of H5N1 cases

Indonesia's health minister said that the government has stopped the practice of promptly notifying global health officials each time it confirms a human H5N1 avian influenza case or death, a move some say will likely hamper efforts to monitor the world's pandemic risk level. Health Minister Siti Fadilah Supari today confirmed that a 15-year-old girl from Jakarta tested positive for H5N1 avian influenza on May 13 and died the next day, according to a report from the Associated Press (AP). Indonesia's National Committee for Avian Influenza Control and Pandemic Influenza had previously listed the case as confirmed on its Web site, but the information is apparently no longer listed. The WHO, which typically confirms cases when it receives notifications from health ministries or test results from its collaborating laboratories, has not yet confirmed Indonesia's most recent case and has not commented on Supari's decision to stop sending out H5N1 case notifications. The WHO's last confirmed an Indonesian H5N1 case, in a 3-year old boy who died on Apr 23. Indonesia has been hit hardest of any country by the H5N1 virus. According to the WHO's most recent count, the country has had 133 cases and 108 deaths. The country's refusal to share timely reports of human H5N1 cases is the latest in a series of controversies that began when Indonesia stopped sharing its H5N1 isolates in early 2007 to protest what it views as a lack of access to affordable H5N1 therapies and vaccines. The WHO has held several meetings to resolve the virus sharing issues, but so far no agreements have been reached.
Source: http://www.cidrap.umn.edu/cidrap/content/influenza/avianflu/news/jun0508indonesia-br.html