THE SCIENCE WORLD
A Monthly e Magazine
ISSN:2583-2212
Nov, 2023 3(11), 2951-2957

Popular Article

Lead Poisoning in Domestic Animals

Dr. Kamal Sarma[1], Dr. Jonali Devi[2] and Dr. J.S. Sasan[3]
[1] Professor, [3] Assistant Professor, Division of Veterinary Anatomy, [2] Professor & Head, Division of Veterinary Physiology & Biochemistry
Faculty of Veterinary Sciences & A.H., S.K. University of Agricultural Sciences & Technology of Jammu
R.S. Pura, Jammu-181102 (J&K)
https://doi.org/10.5281/zenodo.10204641

Increased amounts of heavy metal lead in the body produce lead poisoning, which is a type of metal poisoning. Lead disrupts a variety of biological functions and is harmful to a variety of organs and tissues. The brain is the organ that is most vulnerable to lead poisoning. Lead interferes with the neurological system's development, making it harmful to calves, resulting in potentially lasting learning and behaviour issues, including aggressiveness. Abdominal pain, confusion, headaches, anaemia, irritability, and, in severe cases, seizures, coma, and death are among the symptoms.

Published: **24.11.2023**

Contaminated air, water, soil, food, and consumer products are all potential sources of lead exposure. Adults are frequently poisoned by lead as a result of occupational exposure. Lead paint, which is found in many homes, especially older ones, is one of the most serious hazards to calves. As a result, calves in older dwellings with flaking paint or lead dust from mobile window frames with lead paint are more vulnerable.

Classification

Lead poisoning can be acute (from a single, brief exposure) or chronic (from repeated low-level exposure over time), with the latter being far more common.

Lead can be found in a range of compounds and in diverse forms in the environment. The characteristics of poisoning varies depending on whether the agent is an organic (carbon-containing) or inorganic (non-carbon-containing) chemical. Organic lead poisoning is now extremely rare because organic lead compounds are no longer utilised as gasoline additives in most countries, but they are still employed in industrial settings. Organic lead compounds, which are easily absorbed through the skin and respiratory tract, primarily impact the central nervous system.

The peripheral nervous system (particularly motor nerves) and the central nervous system are both affected by lead. Adults are more affected by peripheral nervous system impacts, while young calves are more affected by central nervous system effects. The axons of nerve cells deteriorate and lose their myelin coverings when exposed to lead. Lead exposure in young calves has been linked to learning disabilities.

Animal and human lead poisoning is a big concern around the world. Poisoning in animal populations could act as a sentinel for determining the amount of environmental contamination and lead-related human health issues.

Official Website
www.thescienceworld.net
thescienceworldmagazine@gmail.com
Published: **24.11.2023**

Lead poisoning is most common in dogs and cattle in field conditions. Reduced accessibility, more selective eating habits, or decreased sensitivity restrict lead poisoning in other animals. When old oil and battery disposal from machinery is handled inappropriately, many occurrences in cattle are related with sowing and harvesting activities. The frequency of lead poisoning cases linked to oil consumption has decreased in recent years, thanks to the removal of tetraethyl lead from gasoline in several nations. Paint, linoleum, grease, lead weights, lead shot, and contaminated flora growing near smelters or along roadsides are all sources of lead. It is critical to determine the source of lead poisoning in order to prevent future cases. Small animals and children are also exposed to lead poisoning in urban surroundings and during the refurbishment of historic houses that have been painted with lead-based paint. Cats have been found to consume lead-contaminated dust when grooming. Non-target scavenger animals may develop toxicoses as a result of improper disposal of lead-poisoned animal carcasses. Scavenging by endangered animals like the condor creates special issues.

Pathogenesis

Lead is absorbed into the bloodstream and soft tissues before being redistributed to the bone. Dietary variables such as calcium and iron levels have an impact on absorption and retention. Particulate lead deposited in the reticulum of ruminants progressively degrades and releases considerable amounts of lead. Sulfhydryl-containing enzymes, erythrocyte thiol concentration, antioxidant defences, and tissues rich in mitochondria are all affected by lead, as seen by the clinical illness. Lead is irritating, immunosuppressive, gametotoxic, teratogenic, nephrotoxic, and toxic to the haematological system, in addition to the cerebral haemorrhage and edoema associated with capillary injury.

Clinical Findings

Young animals are particularly susceptible to acute lead poisoning. The GI and neurological systems are the most significant clinical indications. Ataxia, blindness, salivation, spastic twitching of eyelids, jaw champing, bruxism, muscle tremors, and convulsions are indications that develop in cattle within 24–48 hours after exposure.

Calf with lead toxicity showing blind eyes gazing at sky

Official Website
www.thescienceworld.net
thescienceworldmagazine@gmail.com
Published: 24.11.2023

Anorexia, rumen stasis, colic, dullness, and temporary constipation are common symptoms of subacute lead poisoning in sheep and older cattle, and are typically followed by diarrhoea, blindness, head pushing, bruxism, hyperesthesia, and incoordination. At high levels of exposure, inhaling corrosive elemental mercury vapours, which causes severe dyspnea and reduced respiratory function, is frequently lethal. If exposure is not extensive, neurologic symptoms may occur. Because of its corrosive nature, inorganic mercury causes GI symptoms such as colic, anorexia, stomatitis, pharyngitis, vomiting, diarrhoea, shock, dyspnea, and dehydration. At high levels of exposure, death generally occurs within hours. Survivors may have dermatitis, keratinization of the skin, anuria, polydypsia, hematuria, or melena.

Chronic lead poisoning in cattle can cause a condition that shares many characteristics with acute or subacute lead poisoning. Chronic exposure can result in neurologic signs such as CNS depression or excitement similar to that seen in organic mercury poisoning. Clinical signs can take days to appear depending on the level of exposure to organic mercury compounds like methylmercury. Aspiration pneumonia is commonly caused by a loss of swallowing reflexes. Infertility can be caused by embryotoxicity and poor semen quality. Organic mercury exposure is highly harmful to the neurological systems of young, developing animals, resulting in cerebellar ataxia and death.

In dogs, GI disorders such as anorexia, colic, emesis, and diarrhoea or constipation are the most common symptoms. Anxiety, frantic barking, jaw champing, salivation, blindness, ataxia, muscle spasms, opisthotonos, and convulsions are some of the symptoms that can occur. Some dogs may show CNS depression rather than CNS excitation. Weight loss, depression, weakness, colic, diarrhoea, laryngeal or pharyngeal paralysis (roaring), and dysphagia are common symptoms of lead poisoning in horses, and aspiration pneumonia is a common complication.

The most noticeable signs in birds are anorexia, ataxia, loss of condition, wing and leg weakness, and anaemia.

Lesions:

Acute lead poisoning can leave animals with minimal visible gross lesions. In the GI tract, oil, paint flakes, or battery shards may be visible. Gastroenteritis is caused by the caustic action of lead salts. Edema, cerebral cortex congestion, and cortical gyri flattening can all be found in the neurological system. Endothelial swelling, laminar cortical necrosis, and white matter edoema may be visible histologically. The kidneys may show tubular necrosis and degeneration, as well as intranuclear acid-fast inclusion bodies. Lambs have been found to have osteoporosis. Abortion may occur as a result of placentitis and the buildup of lead in the foetus.

Official Website
www.thescienceworld.net
thescienceworldmagazine@gmail.com
Published: **24.11.2023**

Diagnosis

Lead concentrations in different tissues can be used to assess excessive buildup and represent the level or duration of exposure, severity, prognosis, and treatment success. In most species, lead concentrations of 0.35 ppm in the blood, 10 ppm in the liver, or 10 ppm in the kidney cortex are consistent with a diagnosis of lead poisoning. Blood lead values of >0.05–0.10 ppm in food-producing animals are considered a notifiable disease in several countries. Before a cargo for food consumption is allowed, it must be inspected or cleared by a regulation veterinary officer or biosecurity inspector.

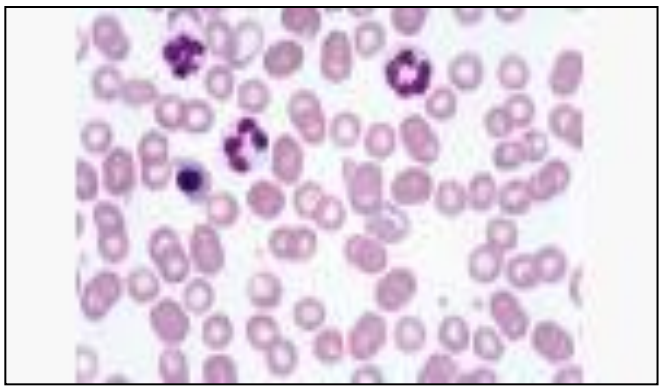

Fig. Basophilic stippling, dog blood smear

Anemia, anisocytosis, poikilocytosis, polychromasia, basophilic stippling, metarubricytosis, and hypochromia are hematologic abnormalities that may be symptomatic but not conclusive of lead poisoning. Levels of -aminolevulinic acid in the blood or urine, as well as free erythrocyte protoporphyrin, are sensitive indicators of lead exposure but may not be accurate indicators of clinical disease. A radiologic examination may be helpful in determining the extent of lead poisoning. Other disorders that induce neurological or GI issues can be confused with lead poisoning. Polioencephalomalacia, nervous coccidiosis, tetanus, hypovitaminosis A, hypomagnesemic tetany, nervous acetonemia, organochlorine insecticide poisoning, arsenic or mercury poisoning, brain abscess or neoplasia, rabies, listeriosis, and *Haemophilus* infections are all diseases that can affect cattle.

In dogs, rabies, distemper, and hepatitis may appear similar to lead poisoning.

Treatment

Treatment may not be successful if tissue damage is substantial, particularly in the nervous system. Calcium disodium edetate (Ca-EDTA) is given IV or SC (110 mg/kg/day) divided bid for three days in animals, then repeated two days later. In dogs, a comparable dose divided qid in 5 percent dextrose is given SC for 2–5 days. If clinical indications remain after a 1-week rest period,

Official Website
www.thescienceworld.net
thescienceworldmagazine@gmail.com
Published: 24.11.2023

a 5-day therapy may be required. There is presently no commercially marketed veterinary product that contains Ca-EDTA.

Thiamine (2–4 mg/kg/day, SC) decreases lead tissue deposition and alleviates clinical symptoms. The most favourable response appears to be a combination of Ca-EDTA and thiamine therapy.

Dogs can be given 110 mg/kg/day of D-penicillamine by mouth for two weeks. However, this medication has been linked to unpleasant side effects such emesis and anorexia. The use of D-penicillamine in animals is not suggested. Succimer (DMSA, or meso 2,3-dimercaptosuccinic acid) is a chelating drug that has been shown to be effective in dogs (10 mg/kg, PO, tid for 10 days) and birds. DMSA has been linked to fewer negative side effects than Ca-EDTA.

To eliminate lead from the GI tract, cathartics such as magnesium sulphate (400 mg/kg, PO) or a rumenotomy may be used. Surgery to remove particulate lead material from the reticulum after a battery intake is rarely successful in cattle. To control convulsions, barbiturates or tranquillizers may be prescribed. Chelation therapy combined with antioxidant therapy may help to reduce the oxidative damage caused by acute lead poisoning. In addition to DMSA, antioxidants such as n-acetylcysteine (50 mg/kg/day, PO) have been employed.

Lead mobilization at parturition, lead excretion in milk, and long withdrawal durations in food-producing animals have sparked substantial debate about the reasons for treatment from both a public health and an animal management standpoint. Lead in the blood of cattle consuming particle lead has a half-life of more than 9 weeks. Blood lead concentrations should be monitored on a regular basis to estimate withdrawal durations, which might be over a year. All possibly exposed cattle in a herd of cattle with confirmed instances of lead poisoning should be assessed. A small but considerable percentage of asymptomatic cattle may have lead amounts in tissues that exceed food safety regulations.

Treatment options may be ineffectual since the neurologic and renal damage is irreversible. As a result, the chances of making a full recovery are slim. Significant mercury deposition in edible tissues and dramatic effects on reproduction limit treatment possibilities in food-producing animals. It is advised that euthanasia and disposal be carried out in consultation with regulatory officials. Activated charcoal (1–3 g/kg) and sodium thiosulfate (0.5–1 g/kg) administered orally bind mercury and inhibit absorption. Antioxidants like vitamin E and selenium may help to prevent oxidative damage. If treatment is initiated soon after exposure, before the nephrotoxic effects become severe, chelation therapy may be beneficial. Dimercaprol (3 mg/kg body wt, IM, every 4 hr for 2 days, followed by qid treatment on day 3 and bid treatment for 10 days) is a lipid-soluble

Official Website
www.thescienceworld.net
thescienceworldmagazine@gmail.com
Published: 24.11.2023

chelator that may be useful. 2.3-dimercaptosuccinic acid (10 mg/kg, PO, tid for 10 days) has been shown to help dogs with organic mercury toxicity. Penicillamine (50–100 mg/kg/day, PO, for 2 weeks) may help to diminish clinical symptoms once the GI tract has been decontaminated for mercury.

Official Website
www.thescienceworld.net
thescienceworldmagazine@gmail.com
Published: **24.11.2023**