

The dangerous elevation of lead levels in the bodies of domestic animals: a review

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Abstract

Recent lead consumption and the potential for a worsening sickness are both indicated by elevated lead levels in the blood and feces. Analyzing the blood and feces of a living animal may help with a more accurate diagnosis and prognosis for lead poisoning. As it can be challenging to express, clinically and histologically, the difference between lead poisoning and the numerous diseases that affect the central nervous system, it is advised to perform a chemical analysis for lead in animals that have had access to this material or are exhibiting symptoms related to the cerebral region. Dangerous concentrations have not been determined, and there is currently no consensus regarding the outcomes of the published studies. Furthermore, due to the dependence of each mineral element on the species, age, and interactions between minerals in the metabolic process, it is challenging to estimate a dangerous dose of a mineral element as well as the physiological causes of mineral poisoning, changes that take place during poisoning, and changes that occur during poisoning. Validating a diagnosis of poisoning necessitates knowledge of toxic



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dosages, physiological changes that occur during poisoning, symptoms, and the mineral concentration in tissues from poisoned animals. However, these factors are not well understood. The physiological consequences, symptoms, diagnostic procedures, and therapeutic options of lead poisoning in equines, bovines, ovines, and swine were reviewed.

Keywords: lead poisoning, mineral elements, lead levels, chemical analysis.

Introduction

Environmental lead exposure is widespread (1). Natural and anthropogenic processes, such as coal and mineral oil combustion, smelters, mining, alloy processing units, and paint businesses, are all common lead sources (2). Interest in the study of toxic compounds and their repercussions for the biological system has been sparked by the ever-increasing levels of environmental contaminants caused by rising urbanization, industrialization, and scientific and technological progress (1, 2). Lead is a toxic metal that has been shown to induce oxidative stress, membrane disturbances, vacuolation,

and degeneration in the brains of mammals that are exposed to even low levels (1, 2). Animals can consume toxic levels of minerals in a variety of ways, including through improperly balanced mineral supplements and/or complete feed, consumption of plants with high mineral concentrations, and application of fertilizers, herbicides, insecticides, and fungicides on pasture. Pollutants can build up due to several causes, including the breakdown of urban and industrial wastes, leaks, and unintentional spills (2). It is also widely recognized as a very toxic substance that can lead to tissue damage and oxidative stress (3). Both acute and chronic lead (Pb) toxicity have

the potential to generate several detrimental systemic consequences at the cellular level (3), eventually leading to various illnesses. The purpose of this review was to look into how metals alone can cause toxicity in the animal body and how this manifests in organs body such as kidneys, heart, liver, pancreas, and spleen and els. The purpose of this review is to draw attention to the physiological effects, symptoms, diagnosis, and treatment of lead poisoning that is deemed dangerous for animal intake.

Background

Exposure to chemical substances found in the environment, whether they are manmade or naturally occurring, has been linked to metabolic diseases (2, 3, 4). Metals play crucial roles in the body,

but too much of one can be harmful (5, 6). Contaminated air, water, and food can cause metals to accumulate in key organ tissues, leading to chronic toxicity (7, 8). Heavy metal pollution of water and air is a worldwide problem (9, 10, and 11). Acute and chronic lead (Pb) toxicity can both generate a wide range of harmful systemic effects at the cellular level, leading to a variety of illnesses (12, 13). Construction materials, weaponry, batteries, alloys, paint, and fuel all include lead, making it a pervasive part of our everyday lives (13). Lead exposure is responsible for over 19 % of annual deaths in the United States. One of the most serious unresolved risks for humans and animals is lead, which enters the environment through industrial waste and produces contamination. Large animals like swine, cattle, sheep, and others have a tolerance

for lead; thus, they will not show any adverse effects from eating feeds containing lead (13). Lead pollution in the air is mostly caused by tetraethyl lead in gasoline, which poses a threat to human health through inhalation. In addition, lead exposure is heightened when people use food and beauty products. It is highly bioavailable, meaning it can build up in organisms and eventually lead to toxicity or even death (14). Lead (Pb) is very hazardous to animals and humans despite serving biological functions in the bodies of animals (14). One of the most toxic minerals to animals, it is found all over the world and is increasingly concentrated due to industrial contamination (15).

The consequences and dangers of lead poisoning on domesticated farm animals

Lead pollution, caused by the byproducts of industrial operations, is one of the most pressing issues that have not been adequately addressed, even though it endangers human and animal life (15). It can be absorbed and stored in the body by inhalation, skin contact, and ingestion. Tetraethyl lead, which is found in gas, is toxic to humans and the main source of lead pollution in the environment (14). In addition, people are exposed to extra lead when they eat and use cosmetics. Because of its high bioavailability, lead can build up to dangerous levels in living creatures, potentially causing their demise. Lead (Pb) has biological functions in animal bodies, but it is also highly toxic to humans and other animals (14). As a result of industrial pollution, one of the minerals most harmful to animals is now

found in increasingly higher concentrations around the world (15).

The most preliminary signs of lead poisoning found included weakness in the muscles (16), ataxia, stomach pain, and convulsions are all symptoms of subacute lead poisoning, the most frequent form of lead poisoning in sheep. Clinical symptoms of lead poisoning in cattle include sadness, loss of appetite, staggering or circular gait, twitching muscles, gnashing teeth, ruminal stasis, constipation, and death within 72 to 96 hours (16). The most common kind of lead poisoning in sheep is subacute lead poisoning, which can cause symptoms like weakening in the muscles, ataxia, stomach pain, and convulsions. Reproductive abnormalities like anestrus, prolonged parturition intervals, and abortion have been linked to subacute poisoning (16, 17).

Sever signs of lead poisoning are when animals consume high amounts of lead at once, they become poisoned, most commonly after 5 days. It is also stated that between 1 and 3 days after ingesting a toxic dose of lead, clinical symptoms can appear in cattle that have been acutely poisoned (17, 18). These symptoms include a staggering gait caused by the unfavorable effect of contaminated forage; muscle twitching primarily in the head, ear, and neck (19); gnashing of teeth (20); progressive incoordination of the hind limbs; and, after recumbency, ataxia (21, 22). Mild to severe recumbency abdominal discomfort convulsions, and a loss of appetite are all possible outcomes of acute lead poisoning in horses(23).

Lead toxicity, long-term

breathing; muscle weakness; stiff joints;
 colic; and even death.

Animals become poisoned by lead when

Assessing blood lead levels

they consume levels of the metal in their
 diet that are consistently too high.

Ruminants, pigs, and horses have a
 normal lead blood content of 0.05 to

Symptoms of chronic lead poisoning in

0.25 ppm (26, 27). Lead levels above 1

cattle include opaque hair, thickened

ppm in circulation can be fatal to

phalange epiphyses, mild anemia (24),

animals. Swine currently have a lead

severe depression, paresis of the

poisoning concentration of 1.2 ppm.

hypoglossal nerve, incoordination,

Prior research ¹ investigating the

ataxia, muscular twitching, opisthotonus,

relationship between ⁴ blood lead levels

convulsion coma, respiratory failure, and

and lead excretion ² in milk found a

death (25). Sheep with chronic lead

significant correlation between the two

poisoning have hind limb paralysis, gait

(28). The milk lead excretion was

problems, bone loss, limited range of

considerably higher in lactating cows

motion in their joints, spontaneous

with blood lead levels over 0.20 µg/ml

abortions, and temporary sterility.

than in those with blood lead levels

Horses may already show symptoms

between non-detectable and 0.20 µg/ml.

such as brittle, dry hair; loss of appetite;

A significant correlation was found

paralysis of the pharynx; regurgitation of

between these two parameters with

food and water via the nostrils; difficulty

blood lead levels above 0.20 µg/ml,

indicating that the excretion of lead through milk increases with an increase in blood lead levels above 0.20 $\mu\text{g/ml}$ (28).

First-Stage Therapy

Lead accumulated in the tissues of poisoned animals can be removed with edetate calcium disodium (CaEDTA). Rapid injection of two doses of 110 mg CaEDTA/Kg PV with a six-hour gap twice a day for three to five days will provide intravenous CaEDTA for 12 hours at a dose of 110 to 220 mg CaEDTA/Kg PV (29, 30). Vitamin B1 (thiamine chloride), by chelating lead in poisoned animals' tissues, decreases lead deposition (especially in the kidney, liver, and central and peripheral nervous systems) and enhances lead elimination (through urine and bile) from the bodies of poisoned animals (30). Therapeutic

doses of thiamine are 25 mg/kg of body weight given subcutaneously twice daily for calves and 75 mg/kg of body weight given subcutaneously twice daily for sheep (31). Lead sulfate is formed when taken orally with 200–300 gm of magnesium sulfate; the resulting precipitate is then passed into the feces. Within a short time after consuming lead-rich materials or food, symptoms of toxicity will manifest (32 and 33). Usually, there was no way to pinpoint exactly where the lead was coming from, but in one case (32), it turned out to be an old paint container being used to treat the water. Lead poisoning was diagnosed in some cases where there was clinical evidence. Leaded gasoline is no longer available, and the use of lead-based paints in homes and barns has been drastically cut back. Lead poisoning is still the leading cause of poisoning in

cattle and sheep, even though most cases are avoidable (32). Lead is an extremely dangerous substance via inhalation and can be ingested with water and meals. In addition to the skin and muscles, it also builds up in the liver, heart, and kidneys. Lead poisoning causes problems with the neurological system and the blood cells. Lead causes mutations, causes cancer, and harms embryos (34). In addition, a study conducted in the lab on the analysis of the muscles and organs revealed that the kidneys and liver had the highest concentrations of the examined heavy metals. If the lead and cadmium levels observed in muscle tissue are considered normal, the study reveals the following lead and cadmium concentrations in the heart (1.19%), liver (1.71%), and kidneys (1.07%), and cadmium concentrations in the liver (1.17%) and kidneys (greater than

(1.21%) (35, 36, 37). Furthermore, the effects of environmental lead on the bone, mouth, and teeth, including osteoporosis and dental caries, have also been shown to be caused by an increase in osteoclast production, bone resorption, oxidative stress, changes in the level of vitamin D in the plasma, disruption of mineralization, and a delay in fracture healing (38). More research is needed to understand the relationship between lead and canine infectious illnesses. It's possible that, much like in humans, viral diseases can cause the rapid and potentially harmful release of lead stored in bone. Whether dogs, like cattle, actively seek out and consume lead compounds or whether they unintentionally consume lead through chewing on lead-painted furniture or paint stir sticks is unclear. Zoo animals and birds, as well as those used in

scientific experiments, are at risk from lead exposure.

In conclusion, the analysis and prospects of lead poisoning may be improved by analyzing the blood and excrement of the living animal. Lead in the blood and feces at high levels suggests recent lead consumption and possible disease progression. Given the conditions, the vet would likely elect to treat any animals that were exposed to lead and then, in cases where blood and feces tests were feasible, continue treatment for any animals that were found to have eaten lead. Chemical testing for lead is advised in animals with access to this material or displaying cerebral symptoms due to the difficulties in distinguishing lead poisoning from various disorders involving the central

nervous system on a clinical and histological level. There is conflicting evidence on what constitutes a toxic dose of a mineral, and our understanding of the physiological mechanisms underlying mineral poisoning is limited at best. This is likely because it is challenging to describe the physiological mechanisms, the changes involved in the process of poisoning, and the difficulty of establishing a toxic dose of a mineral element.

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