Lead Poisoning in Cattle and Food Chain Implications

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Synopsis: Decades after the United States outlawed use of lead (Pb) in paint and gasoline, Pb poisoning remains a clinically relevant problem in domestic animals. Lead exposure in cattle and other livestock is particularly problematic, since products from these animals enter the human food chain. It behoves the veterinarian to be familiar with not only clinical signs and diagnosis of Pb poisoning, but to work to minimize the risk to consumers of animal products.

Learning Objectives:

- Know common sources of Pb for livestock.
- Know how to work-up a case of Pb poisoning in livestock.
    - What biological and environmental samples to take for analysis and why.
    - Client communication and herd assessment.
- Know what the food-chain implications are for Pb contaminated livestock.
    - Working with state regulators.
    - Benefits and drawback of quarantine and retesting.

Sources of Pb: Lead poisoning is the most commonly reported metal toxicosis in domestic animals, and cattle are the domestic species most commonly affected.[1,2,3,4,5,6,7] Lead poisoning occurs most often on pasture due to the indiscriminate eating habits and natural curiosity of cattle, particularly calves, but Pb can also contaminate batches of feed for confined cattle.[3,4,8] Lead poisoning in beef and dairy cattle directly cause economic losses due to mortality and treatment costs.[9]

Lead is ubiquitous in the environment and has no known biological role.[2,10] Some environmental Pb is from geochemical sources, but most comes from anthropogenic sources include mining, smelting, battery production and recycling, and other commercial uses. Lead paint and automotive batteries are the most common sources for cattle. As point sources, paint and batteries are associated with short-term exposure.[1,2] However, Pb paint removal has been associated with widespread contamination of forage, and batteries have been baled into feed, causing chronic Pb exposure to large numbers of animals.[7,12,13] Lead ammunition used by sports enthusiasts and is a potential source of Pb for cattle and contaminates game meat.

Toxicokinetics: Lead is poorly absorbed in the gastrointestinal tract of the mature animals, at a rate of only 1-3%, but calves can absorb up to 50% of dietary Pb. Absorbed Pb is bound to circulating erythrocytes. Blood Pb concentrations rise quickly, then decline and plateau as Pb is distributed to soft tissues.[14,15] Lead is distributed to liver, kidneys, and other soft tissues, but Pb also crosses the blood-brain barrier and placenta.[16] Lead is redistributed over time to bone; Pb in bone is relatively inert.[15] Bone accounts for 90% of the body's store of Pb in adult people, and 75% in children.[17]

Elimination of Pb is different in ruminants compared to monogastrics due in part to the retention of Pb particles in the reticulum and rumen.[15] Blood Pb concentrations at any given time are dependent upon absorption of gastrointestinal Pb and mobilization from bone and other tissues.[15] The ranges for blood Pb half-life in cattle varies markedly in the literature. Half-lives for experimental cattle given intravenous Pb acetate ranged from 6 to 19 days, but field exposures are associated with half-lives as long as 2507 days.[15,18,19] The differences in half-life in field exposed cattle was not dependent on the source of Pb, sex, or age of the animal, though the half-life was shorter in lactating cattle.[8,15] Lead can be detected in the blood of Pb-exposed cattle for at least 2 years.[15]

Diagnosis: Clinical signs of Pb poisoning include seizures or unexpected death in calves or blindness and bruxism in mature cattle. Antemortem diagnosis is based on blood Pb analysis (note that serum and plasma are inappropriate samples) and postmortem diagnosis is based on analysis of liver or kidney. When diagnosed, the source of the Pb must be identified to end exposure of prevent further exposure of herd-mates. Suspected sources are collected during a careful field investigation and could include feed, paint, grease, soil, and water.

Food chain effects: Although acute and subacute clinical Pb poisonings in cattle are most commonly reported, chronic, subclinical toxicosis is more likely to impact the human food chain.[4,14,20,21] Cattle that do not have overt clinical signs of Pb poisoning are usually not tested, but in herds where Pb poisoning has been diagnosed, 7 to 100% of asymptomatic cattle can have elevated blood Pb concentrations.[9,19] Clinical Pb poisoning from ingestion of contaminated cattle products is unlikely.[3,12] However, subclinical Pb exposure in children is associated with life-long impacts including intellectual and cognitive deficits, aggressive behaviour, renal impairment, hypertension, and cataracts.[12,22] Subclinical poisoning in adults is associated with miscarriages, stillbirths, and other fertility problems.[22]

Several authors report that milk Pb concentrations have a mathematical relationship with blood Pb concentrations.[7,16,23,24] Though Pb excretion into milk is limited, a milk Pb concentration > 2.00 mg/L was reported in a cow experimentally dosed with Pb oxide.[25] Lead contamination of milk has been documented years after cessation of exposure.[24] Lead sequestration in the rumen and bone probably contributes to the long-term potential for milk contamination.

There is limited information on Pb concentration in muscle and organ meat from exposed livestock. Studies documenting Pb concentrations in edible tissues from livestock with known exposure are more rare still. One study found that Pb concentrations in liver and kidney correlated with blood Pb concentrations. Lead in skeletal muscle did not correlate with the blood, but Pb was detected in skeletal muscle of a cow with a blood Pb concentration of 4.57 µg/dL, which is below the 5.00 µg/dL upper limit for slaughter cattle in some US states.[26] Recent studies of hunter killed game have found Pb contamination of game meat due to ammunition fragmentation.

Conclusions: Diagnosis of Pb poisoning in cattle is based on clinical signs and analysis of whole blood from live animals or liver or kidney postmortem. Identification of the source of contamination is critical to the management of Pb poisoning in livestock and to minimizing the risk to the human food chain. Lead elimination is unpredictable and can be prolonged in ruminants, both due to Pb particle sequestration in the rumen and, in chronic exposures, storage of Pb in the bony matrix. Lead is variably excreted in milk, and excretion can continue over several lactations. Residues in skeletal muscle are usually negligible, but are not predictable based on antemortem testing. Bone and organ meats from animals with known Pb exposure are best discarded.

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