Acute Lead Poisoning in Beef Cattle: Insurance Liability Claims, Clinical, Pathologic, Toxicologic and Epidemiologic Findings

Terry M. Wilson, *D.V.M., Ph.D.*
William Ring, *B.A.*
James Roof, *B.A.*
Animal Disease Diagnostic Laboratory
The Pennsylvania State University
University Park, PA 16802 (Wilson)
R. D. #4, Mount Equity Road
Muncy, PA 17756 (Ring) and the
State Veterinary Diagnostic Laboratory
Department of Agriculture,
Bureau of Animal Industry
P. O. Box 1430
Harrisburg, PA 17105 (Roof)

Introduction

Lead Poisoning in cattle is possibly one of the most commonly diagnosed intoxications (1-11). Lead toxicosis in cattle may present as a neurological or intestinal syndrome and may be subacute, acute, or chronic. Sources of lead are variable and include oils, paints, lead batteries, and lead shot (6-9). Frequently the source of lead is undetermined (3-5).

In this unique report, we describe an acute outbreak of lead poisoning (ALP) in cattle which was traced to a discarded tractor battery. In addition, insurance liability claims are discussed and presented.

Case Report

Approximately 6 adult beef cows became acutely ill and exhibited ataxia and excessive salivation. Animals, usually afebrile, also exhibited maniacal excitement and odontoprisis. Only adult cows appeared affected while young nursing calves appeared clinically normal. Head pressing was also observed and animals that became recumbent would exhibit muscular spasms and occasionally opisthotonus. Within 2 days of exhibiting anorexia, 2 mature cows were found dead. Within 13 days, 29 mature cows and 1 mature bull died. Six cows survived while 5 nursing calves died from malnutrition. During the epizootic, the cattle were fed local-grown hay and corn silage from a recently opened silo.

Gross and histopathologic findings—No significant gross lesions were observed in one pregnant 3-year-old cow that died acutely. Coronal sectioning of the fresh brain was unrevealing. Specimens for histopathology were fixed in 10% buffered formalin and stained with H & E. No significant lesions were observed in the lung, myocardium, liver, adrenal, spleen, pancreas, small and large intestine. Significant pathologic lesions, primarily in the cerebral cortex, included endothelial proliferation, neuronal degeneration and focal spongiosis. Focal degeneration of proximal renal tubular epithelial cells was also observed microscopically.

Toxicologic findings—Kidney, liver, and rumen contents from the necropsied cow and silage were submitted for lead analysis (12). The liver contained 45 ppm lead while the rumen contents and silage contained 140 ppm and 175 ppm lead, respectively.

Epidemiologic findings—Based on the negative necropsy results from the one acutely dead cow and the clinical symptomatology in other cows, the Veterinary Diagnostic Laboratory indicated to the field veterinarian and relevant personnel that a possible poison might be involved and suggested heavy metals, pesticides, fungicides, herbicides, and any other related commonly used farm products. The laboratory pathologist suggested a thorough environmental search to include the feed and physical facilities (13,14). In addition, it was suggested that a thorough review of farm management practices and procedures be conducted. Within 24 hours after the first death, a thorough search was made of the relevant farm environment and feed and small black plastic fragments, believed to be plates from a tractor battery case, were found in the silage. In addition, thick pliable solder fragments were also located in the silage (Fig. 1). Toxicological analysis of this material indicated it to be pure lead (12).

Upon finding the suspect tractor battery fragments, the complete background of this toxic epizootic began to unfold. The owner of this beef farm, being an elderly widow, was unable to harvest the field corn to prepare silage. Four neighbors, to include a distant relative, identified in this

Figure 1. Black plastic like material and U-shaped solder bar believed to be parts of the chopped up discarded tractor battery. Chemical analysis determined these fragments to be pure lead.

report as helpers 1, 2, 3, and 4, volunteered, as a good neighborly act, to harvest the field corn for the owner. On a Saturday morning these 4 helpers, using the owner's equipment and without any consideration for remuneration, began harvesting the field corn and filling the 16′ x 45′ steel automatic top unloading silo.

The harvesting operation was planned so that helper 1 would remain in the field and cut and chop the field corn leaving the filled self-unloading wagons on the edge of the field for helpers 2 and 3 to shuttle back and forth to the farm (Fig 2). Helper 4 remained, through the morning, back at the farm to assist with the unloading. During mid-morning, helper 1 experienced battery problems with his tractor and replaced the battery, leaving the discarded old battery on the edge of the field. During the afternoon, helper 4 had to leave for work which enabled helper 1 to outpace helpers 2 and 3. Helper 1, having placed several loaded wagons on the edge of the field to await the shuttle helpers, picked up the discarded tractor battery, 2 gas cans, and a funnel and placed them on the conveyor belt in the unloading chute (Fig 2). At no time was any discussion regarding the battery entertained by the 3 helpers.

It was assumed that this wagon, with the battery, possibly covered by corn chop, was taken back to the farm by either helper 2 or 3 and blown into the silo. In fact, workers 2 and 3 recalled at one point in the afternoon, blowing along with the silage, a collection of large stones into the silo. Very likely, this noise was the tractor battery being fragmented by the blower blades. On the following Monday, no work being conducted on Sunday, 6 to 7 more wagon loads were blown into the silo by helpers 1, 2, and 3 to top it off.

Approximately 5 weeks later the silo, which automatically unloaded into a chute and then onto a conveyor belt into a feeding trough, was opened. Five weeks after opening the silo, the cattle became anorectic and 2 died acutely. This situation rapidly commenced into a toxic epizootic (Fig 3).

Figure 2. Illustration of corn harvesting operation depicting helpers 1, 2 and 3 and the discarded tractor battery, gas can and funnel on unloading belt. The tractor battery was eventually chopped and blown into the silo initiating an acute epizootic of lead poisoning.

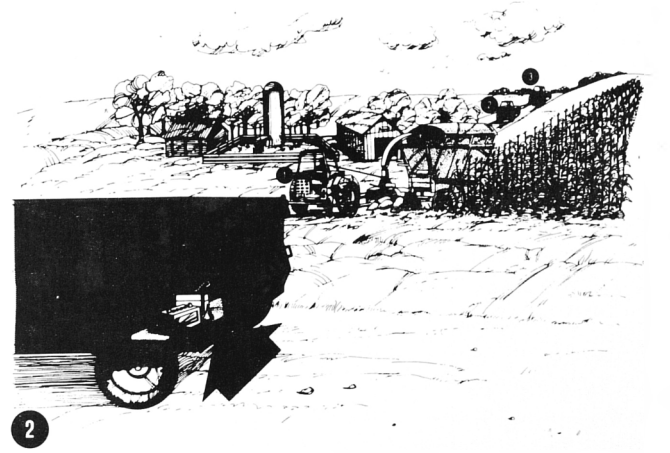
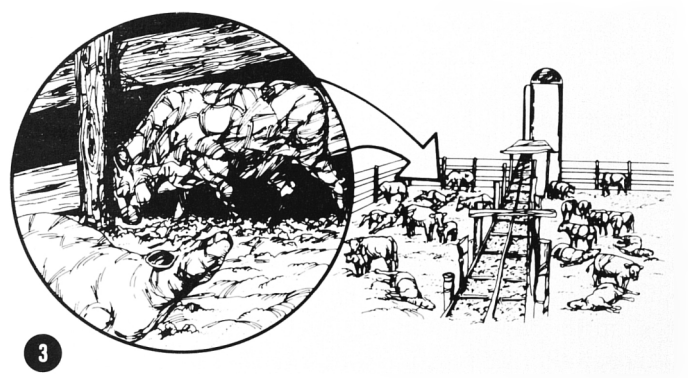


Figure 3. Illustration of farm pen several days after initial deaths due to lead poisoning showing adult cattle head pressing and in various stages of recumbency. Young stock appear clinically normal because they could not reach into the feed trough.



Insurance Aspects

Reporting of Claim and Investigation

Helpers 1 and 2 approached their insurance company two days after the first cow died to inquire about the possible coverage offered by their insurance policies. Helper 3 also contacted his insurance agent to review the matter. Prior to any consideration and settlement of any claims, the insurance agent reported the claim to the company insuring helpers 1 and 2. The company responded the same date with an adjuster. The adjuster indicated to helpers 1 and 2 that a full investigation was required, that a review of the veterinary diagnostic laboratory reports, toxicology and analytical records, and statements of all of those involved, as well as a full list of all dead animals, was going to be

required. The insurance adjuster received and reviewed the above information, had discussions with the farm veterinarian, relevant diagnostic laboratory staff and staff at the Animal Poison Control Center, College of Veterinary Medicine at University of Illinois and took signed statements of those people involved. In addition, the insurance adjuster inspected the farm premises, observed several cattle near death and based on verbal and written information from the above sources, lead poisoning, due to a discarded tractor battery chopped up in the corn silage, was determined to be the source of the intoxicant.

Determination of Liability

At this point, the adjuster had to determine if the liability policies that his company provided for helpers 1 and 2 permitted coverage for this situation. Helper 1 was a farmer by vocation and had a farm owner's liability policy. To have coverage, the incident had to be sudden and accidental, not associated with any commercial endeavor and the policyholder had to be legally responsible for the damages.

Helper 2 worked at a manufacturing plant and owned his own home. He had a homeowner's policy which provide personal liability for the homeowner and his family. Some basic liability restrictions applied to this policy as the farm owner's policy. Helper 3 also worked at a local manufacturing plant, owned his own home and also had a homeowner's policy.

It was concluded from the investigation and the information developed in the investigation, that helpers 1, 2 and 3 were in fact liable and could be held legally responsible for the damages that occurred.

Coordination of Coverages and Percentages of Liability

The insurance adjuster for company 1, then contacted the company hereafter called company 2, who insured helper 3 and explained to them how he had investigated this matter, his conclusions and if in fact, they would concur with this. Through a long discussion with company 2 and an exchange of correspondence with them and applying the Comparative Negligence Laws as applicable in this state, it was finally agreed that helper 1 would be assessed 50% of the liability, helper 2 and 3—25% each. Part of the reasoning for this was that helper 1 was the person that actually changed the battery in the field and placed it in the silage wagon and did neglect to tell either helpers 2 or 3 hauling the wagons of his actions. Helpers 2 and 3 shared in the responsibility to ascertain that only ground up corn and stalks went into the silage and since it was not possible to determine which of the helpers, 2 or 3, actually transported the wagon and battery back to the barn; the liability was divided equally among the two. Company 2 also agreed that since the adjuster for company 1 had already done 75% of the work on this claim, that they would accept his findings, his amount of damages, etc. and verbally agreed that they would honor a subrogation in the amount of 25% against their insured. This allowed the first adjuster to work the claim the entire way through to its conclusion.

Damages

The dead cattle were sent to the local rendering plant and an authenticated list of these cows prepared by the farmer was obtained by the insurance adjuster and verified by the rendering plant truck driver. This list included the approximate weight, age, and date of death of all animals. Based on local current beef prices, realizing that this was a brood cow operation, a price of .40$/lb was established for the mature dead cattle. The total weight of mature dead cows, as ascertained from the above list, was 30,850. The 5 dead calves were valued at .60$/lb with the total weight estimated to be 450 pounds. One dead bull weighed 1,400 pounds and was valued at .55$/lb. The total monetary value for the dead stock was estimated to be $13,380.00 (Table 1).

TABLE 1. Monetary Value of Dead Animals Disposed of in an Acute Lead Poisoning Outbreak.

29 Dead Cows or 30,850 Total Pounds Cows
30,850 lbs
× .40 $/lb
$12,340.00 Value of Cows
5 Calves @ 90 lb = 450 lbs
405 lbs
× .60 $/lb
$270.00 Value
1 Bull @ 1,400 lbs
1,400 lbs
× .55 $/lb
$770.00 Value of Bull
TOTAL ANIMAL VALUE
$12,340.00 Cow Value
270.00 Calf Value
+ 770.00 Bull Value
$13,380.00 Total Animal Value

When it was determined that a discarded tractor battery had been chopped and blown into the silo within the last few loads of topping off the silo, the immediate concern was the possible lead contamination of the remaining silage. Despite discussions between the farmer, insurance adjustor, and numerous authorities on silage storage, fermentation and heavy metal toxicology, the lead-free status of the remaining silage could not be guaranteed. It was, therefore, deemed appropriate to empty the silo and haul the discarded silage to a municipal landfill. Landfill authorities represented by the Department of Environmental Resources required the toxicology report indicating the lead level in the silage before permission was given to use the landfill.

Based on information from the Forage Testing Laboratory at Pennsylvania State University, the farmer and insurance adjuster were able to estimate the monetary value of the discarded silage. The value/ton of silage was

estimated by multiplying the price of shelled corn by a factor of 5 added to $2.50. Discarded silage was estimated to be 137.903 tons at an estimated value of $2,240.92 (Table II). Labor to empty the silo and to thoroughly clean the silo, around the silo, the feed bunker, elevators, augers and unloaders was estimated to require 162 hours valued at $5.00/hour. The vehicular costs to haul the discarded silage to the municipal landfill for disposal was valued at $5.00/ton. Total costs for labor and hauling were estimated to be $1,499.50 (Table III).

The final claim by the farmer and total claim and expenses are indicated (Tabel IV, V).

TABLE 2. Monetary Value of Discarded Corn Silage Due to Possible Lead Contamination.

Price of Bu. Shelled Corn × 5 + $2.50 = Value/Ton
$2.75 × 5 = $13.75 + $2.50 = $16.25/Ton
137.903 Tons Silage
× $16.25
$2,240.92 Silage Value

TABLE 3. Labor Costs For Farm Clean Up and Silage Removal and Truck Hauling Services to Municipal Landfill.

LABOR COSTS
162 Total Hrs
× 5 $/Hr
$810.00 Total Labor Costs
HAULING COSTS
137.903 Tons Hauled
× 5 $/Ton Truck Fee
$689.52 Total Hauling Costs
TOTAL COSTS
$ 810.00 Labor
+ 689.52 Truck
$1,499.52 Total Costs

TABLE 4. Claim By Farmer To Insurance Company After Lead Poisoning Outbreak In Cattle.

$ 2,240.92	Silage
13,380.00	Cattle
123.00	Vet. Bills
$15,743.92	TOTAL CLAIM

TABLE 5. Total Farmer Claim and Related Expenses In A Lead Poisoning Outbreak.

$15,743.92	Farmer
1,499.52	Labor/Hauling
1,379.65	Landfill
$18,623.09	CLAIMS $ EXPENSES

Settlement

Following the establishing of the damages as previously outlined and also indicated in the various tables attached to the rear of this report, the adjustor for company 1 then went to the owner of this herd and did pay her a total amount of damages that are indicated.

Upon verification of this, he did send all of his documents to company 2, who did respond within a week to their share of the claim and thus concluding the settlement and the claim from the insurance aspects.

TABLE 6. Financial Liability Assessment Among Farm Helpers In Case of Acute Bovine Lead Toxicosis.

Helper 1 - 50%	$ 9,311.55
Helper 2 - 25%	4,655.77
Helper 3 - 25%	4,655.77
	$18,623.09

Discussion

Lead poisoning is a major cause of morbidity and mortality in cattle in North American (1-11). Lead sources may be variable but usually the ingestion of oil and paint substances are most common (2,7). Lead shot pellets (8,11) and batteries (2,4,6,7) have also been documented as sources for lead poisoning; however, in approximately 25-50% of reported cases, the source was never determined (3-5).

The syndrome is usually subacute or acute and variable clinical signs include ataxia, blindness, circling, head pressing, excessive salivation, muscle twitching, and hyperexcitability. An abdominal form of lead toxicosis may occur and include anorexia and diarrhea (2, 3). More common neurological signs include blindness, excessive salivation, muscle twitching, and hyperirritability. Morbidity rates are very variable and possibly reflect the source and amount of lead consumed, the age and health and nutrition status of the animals involved. Lead poisoning occurs throughout the year but more cases seem to occur in the spring and summer when cattle are moved to pasture and allowed to forage. During that period of the year, the abrupt diet change may precipitate pica among the cattle (4).

Necropsy lesions in subacute or acute cases of bovine lead toxicosis may be neglible or inconsistent; however, there may be some cerebral edema and yellow discoloration of the occipital and parietal cortex. Histopathological lesions are very variable and in subacute and acute cases, cerebral edema, cortical neuronal necrosis, and endothelial proliferation may be prominent (15-17).

Chemical analysis for lead in the kidney, liver, blood, and rumen contents and feed are considered important parameters in the establishment of a diagnosis of lead poisoning in cattle (2,5). While lead levels in the kidneys and liver may be variable and include low background levels (11, 18-20), values in excess of 10 ppm wet weight basis suggest a

diagnosis of lead poisoning (2). In some cases of bovine lead poisoning, lead levels in the liver and kidney have been well below 10 ppm rendering it imperative that a confirmed diagnosis of lead poisoning in cattle be based on history, clinical signs, necropsy and histopathological examination and toxicological analysis (5). Lead poisoning in cattle must be differentiated from polioencephalomalacia (BPE), infectious embolic meningoencephalitis (IEME), pasturellosis and various pesticide intoxications (10,18,19). The differential diagnosis of lead poisoning, polioencephalomalacia, and infectious embolic meningoencephalitis in cattle has been well documented and the clinical signs, gross pathological lesions and central nervous system pathological lesions tabulated for ready comparison (Table VII, VIII, IX) (16).

TABLE 7. Some Clinical Differential Features of Bovine Polioencephalomalacia, Infectious Embolic Meningoencephalitis, and Acute Lead Poisoning In Feedlot Cattle[16].

Observation	BPE	IEME	ALP
Premonitory Signs	Separation from the group	Stiffness, Knuckled fetlocks	—
Blindness	+	±	+
Convulsions	+	+	+
Opisthotonus	+	±	±
Early Diarrhea	±	—	±
Fever	—	+	±
Sudden Death	—	+	+
CSF Pleocytosis	—	+	—
CSF Protein Elevation	— until later	+	— until later
CSF Pressure Elevation	±	+	+
Ruminal Protozoan Mobility	+	+	—
Blood Lead	—	—	+
Proteinuria	—	—	±
Dye Retention Test	—	—	+
Response to early thiamin therapy	+	—	—
Response to early antibiotic therapy	—	+	—
Response to lead chelators	—	—	±

In this case report, the acute onset of an afebrile neurological disease characterized by anorexia, excessive salivation, head pressing, ataxia, and hyperexcitability strongly suggested an unspecified poisoning. Necropsy of one acutely dead cow, to include gross sectioning and examination of the unfixed brain, revealed no significant lesions and supported the diagnosis of an unknown intoxicant. A confirmed diagnosis of lead poisoning was based on the history, clinical symptomalogy, histopathological brain lesions and lead levels in the liver, rumen content, and silage. Histopathological brain lesions in our case were consistent with lead poisoning while lesions commonly associated with BPE and IEME were absent.

Because of the close cooperation and frequent communication between the Veterinary Diagnostic Laboratory and the farmer and her veterinarian, relayed

TABLE 8. Gross Pathologic Lesions of Bovine Polioencephalomalacia, Infectious Embolic Meningoencephalitis, and Acute Lead Poisoning [16].

Observation	BPE	IEME	ALP
Cortical necrosis	+	—	—
Cerebral swelling	±	—	+
Bilateral posterior collicular necrosis	±	—	—
Superficial and deep brain and spinal cord infarcts	—	+	—
Meningeal congestion	±	+	+
Fibrinous serositis	—	±	—
Pneumonia	±	±	—
Retropharyngeal and generalized lymphadenitis	—	+	—
Muscular, renal, and myocardial infarcts	—	+	—
Pale swollen kidneys	—	—	+
Pale swollen liver	—	—	±
Subepicardial subendocardial hemorrhage	+	+	+
Abomasitis	—	—	±

TABLE 9. Location and Nature of CNS Lesions in Bovine Polioencephalomalacia, Infectious Embolic Meningoencephalitis, and Acute Lead Poisoning[16]

Location	BPE	IEME	ALP
Cerebral cortex	Diffuse laminar cortical necrosis, ischemic type. Principally occipital and parietal areas	Focal septic infarcts at the junction of the cortex and the Underlying white matter	Focal laminar cortical necrosis or scattered neuronal degeneration, ischemic type
Basal ganglia	Scattered neuronal necrosis, occasional large foci	Variable occurrence of septic infarcts	None
Thalamus	Scattered neuronal necrosis, occasional large foci in the lateral geniculate bodies	Variable occurrence of septic infarcts	None
Mesencephalon	Bilateral necrosis of posterior collicular neclei	Variable occurance of septic infarcts	None
Cerebellum	Scattered Purkinje's cell necrosis, hemorrhage	Variable occurance of septic infarcts	None
Medulla	Scattered neuronal necrosis vestibular nuclei	Variable occurance of septic infarcts	None
Spinal Cord	None	Variable occurance of septic infarcts	None

information strongly suggesting a possible poisoning prompted an immediate farm search which uncovered the battery fragments in the silage. Additionally, because one of the first cows to die was promptly made available for necropsy at the Veterinary Diagnostic Laboratory; autopsy, histopathological and toxicological results were available within 4 days which assisted immeasurably in completing the farm search and early confirmation of the diagnosis. Due to the prompt team action by various animal health officials coordinated through the Veterinary Diagnostic Laboratory, insurance liability claims were presented, evaluated, assigned, and finalized in a professional, equitable manner.

Summary

A tractor battery, inadvertently chopped into corn silage, caused an acute neurological syndrome in 30 adult beef cattle. Within 14 days, 30 animals died. Necropsy, histopathologic and toxicologic studies determined lead to be the intoxicant. Epidemiological information revealed that a discarded tractor battery was the lead source, and based on this and other pertinent information, insurance liability responsibilities were ascertained.

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