Yellow Buckeye (*Aescultus octandra* Marsh) Toxicity in Calves

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Introduction

Yellow Buckeye is a species of horse chestnut tree *(Fig 1)* that is found in the mountains and woodlands from Pennsylvania to Iowa and southward. It is the only naturally occurring species of horse chestnut in Southwestern Virginia. The tree can be recognized by its leaves which are opposite palmately compound with 5-7 leaflets *(Fig 2)* and in Spring by its yellow flowers *(Fig 3).* The fruit consists of 1 to 3 seeds enclosed in a pale brown, smooth to slightly pitted capsule that splits on 2 to 3 lines. The seeds or nuts are large, glossy brown, and have a distinct paler scar, hence the name buckeye *(Fig 4).*

Buckeyes are widely recognized by livestock owners as a potential threat to their livestock. Very little evidence exists in the literature to support this belief.

Reports are available on the toxic effects to livestock caused by ingestion of red buckeye, *Aesculus pavia* L.,[2] Ohio buckeye, *Aesculus glabra,* Willd,[3] California buckeye, *Aesculus californica* (Spach) Nutt,[4] and common horse chestnut, *Aesculus hippocastanam* L.[5] Yellow buckeye is listed in the North Carolina Experiment Station Bulletin of 1953[6] as one of the ten most troublesome poisonous plants in that state. However, no evidence is given for that distinction.

Reported signs of toxicity include sluggishness, motor paralysis, incoordination, and muscle twitching from red buckeye;[2] and a peculiar gait of the forelimbs as if the animal "were walking on hot pavement" from Ohio buckeye.[3] No signs associated with yellow buckeye toxicity have been reported.

In the Fall of 1981, at least 33 animals including 14 cows and heifers, 18 calves and 1 mature bull were treated by the Virginia-Maryland Regional College of Veterinary Medicine Ambulatory Health Service clinicians for suspected poisoning by yellow buckeye nuts. These animals ranged in age from three months to nine years of age. Affected animals showed hyperesthesia. They were often recumbent. Some could stand with assistance. Animals that were less severely affected moved relatively normally until they were excited. Excitement caused mild to severe gait abnormalities. These ranged from mild myoclonus of the neck muscles with hypermetria of the forelimbs to tonoclonic spasms of all skeletal muscles causing tumbling and falling resulting in lateral recumbency. Vision and cranial nerve functions remained normal. Pulse and respiratory rates were elevated consistent with the animal's level of activity and temperatures were normal. Chips of buckeye nuts were recognized in the manure of one severely affected cow.

All of the animals were on pasture when clinical signs were first observed. Most, but not all, were reported to have access to buckeye nuts. Various treatments were used on affected animals including tetanus antitoxin, calcium gluconate and magnesium[a] intravenously, magnesium oxide orally, mineral oil and activated charcoal[b] orally, tranquilizers[c] and sedatives[d] systemically. Most of the mildly affected animals recovered regardless of treatment while those more severely affected died despite treatment. Secondary complications including bloat, pulmonary aspiration and musculoskeletal damage often contributed to death. Animals that remained recumbent for more than two days usually died.

Three animals that were examined *post mortem* had whole and masticated nuts in the forestomachs. Lesions on gross examination were limited to the kidney where

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[a] *Cal-Dextro II, Fort Dodge Laboratories, Inc., Fort Dodge, Iowa 50501*

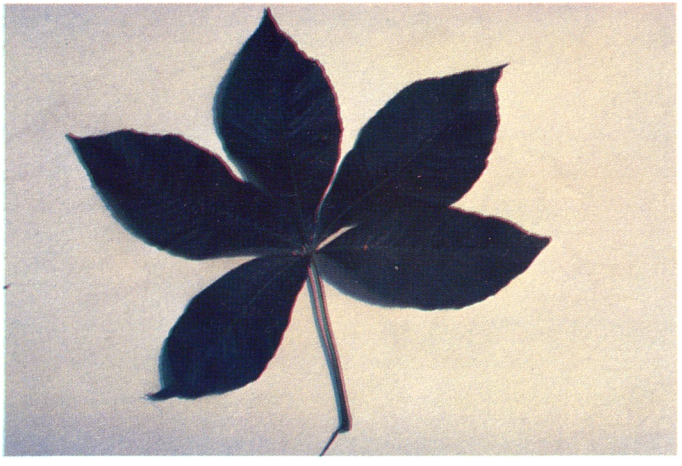
[b] *Charcoal Activated U.S.P., Humco Laboratory, Texarkana, Texas 75501*

[c] *Acepromazine Maleate Inj., Med-Tech Inc., Elwood, Kansas 66024*

[d] *Rompun, Haver-Lockart, Bayvet Division of Miles Laboratories, Inc., Shawnee, Kansas 66201*

Fig 1 Mature yellow buckeye tree (*Aesculus octandra* Marsh) in a pasture in Southwestern Virginia.

Fig 2 Typical leaf from a yellow buckeye tree is palmately compound with 5-7 leaflets.



congestion and obvious streaking of the renal cortex were noted. Histopathological lesions included moderate hepatic congestion, thickening of glomerular tufts and presence of a proteinaceous material in the distal renal tubules and collecting ducts.

In May 1982, a clinical trial was designed to determine if the signs shown by field cases could be recreated by dosing calves orally with yellow buckeye nuts.

Fig 3 Yellow flowers which bloom in the Spring give this species of buckeye its name.

Fig 4 Yellow buckeye nuts are shown with their dried and split capsules. The distinct paler scar on the nut gives it the appearance of a buck's eye.

Materials and Methods

Three calves weighing between 182 kg and 205 kg were obtained. The two treatment calves were given buckeye nuts orally at a dose of 1% bodyweight (calf 1) and ½% body weight (calf 2). The third calf was an untreated control.

Yellow buckeye nuts that had been obtained from the previous fall were used. These were from a farm where calves had died of suspected buckeye toxicity. The nuts were removed from the capsules and were ground into a coarse meal. The calculated weight of meal was added to enough water to produce a slurry that could be delivered through a stomach tube.

Calves were examined once daily for three days before dosing and at four hour intervals after dosing until the calves were euthanatized. Blood, serum, and urine were colected at each of the pre-and post dosing examination times. The

control calf was examined and samples were obtained once daily until the end of the trial when this calf was also euthanatized. All calves were submitted for necropsy.

Results

Calf 1 showed signs of hyperesthesia beginning 16 hours after dosing. An anxious facial expression and extension of the head and neck were observed. Myoclonus was present spontaneously and could be induced by touch or noise (hand clap) stimulation. A hypermetric gait of the hind limbs similar to equine stringhalt was observed. The forelimb gait remained normal. Pupillary light reflexes and vision were normal. Bilateral medio-dorsal strabismus was first noticed at this time.

By 20 hours the calf was in lateral recumbency and could not rise on to its sternum. The head and neck were hyperextended in positional opisthotonus and all four legs were held in rigid extension. Tonoclonic spasms were triggered by light, sound, or touch. Pulse and respiratory rates were slightly elevated. Body temperature remained normal. Rumen activity remained normal throughout the experiment.

Twenty-four hours post dosing, opisthotonos and hyperesthesia were more pronounced and nystagmus became evident. By 28 hours spontaneous tonoclonic spasms were occurring irregularly every 10-30 seconds and euthanasia was performed.

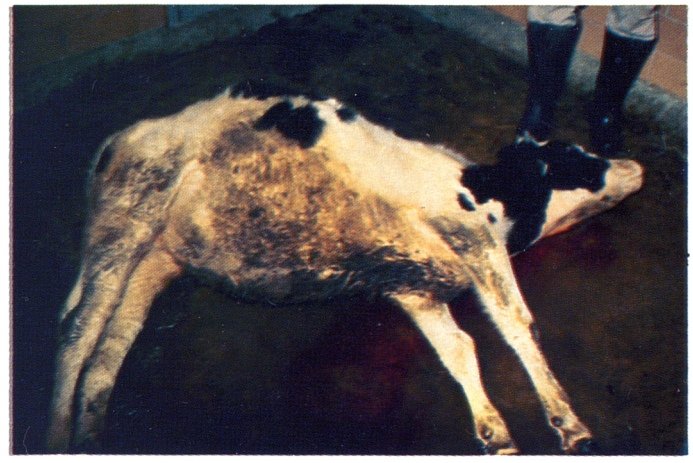
Because of the rapidity and severity of the effects on the first calf, calf 2 was given ½ the original dose. At 16 hours post dosing, muscle twitching of the triceps and flank accompanied by a stiff legged gait were first observed. These signs became more pronounced over the next twelve hours. By 28 hours the calf was lying in sternal recumbency and wouldn't rise without help. Bilateral-medio-dorsal strabismus was evident. Restraint to collect blood samples triggered a tonoclonic seizure. The seizure was characterized by worsening of the strabismus, extensor rigidity of the hind limbs, and a stiff, high stepping gait of the fore limbs that progressively lifted the fore quarters off the ground until the calf lost balance and fell stiffly to the ground. After resting in lateral recumbency for a short period of time, the calf was able to rise again. Three of these seizures were observed within one hour.

At 32 hours the calf was in sternal recumbency and could not get up. Each attempt to rise ended when the hind legs snapped into rigid extension causing the calf to flip over head first on to its side. Medio-dorsal strabismus continued with normal pupillary and menace reflexes. Spontaneous tonoclonic spasms of the head and body continued. Rumen contractions were strong and the calf continued to eat hay. Drinking became more difficult as spasms were initiated each time the calf's nose contacted the water.

By 36 hours the calf was in lateral recumbency and couldn't sit up. This posture was maintained for the next 16 hours. Muscle twitching, spontaneous tonoclonic spasms, and positional opisthotonus were present during this time period *(Fig 5)*. Values for temperature, pulse and respiratory rate were within normal limits except for a period of time that the calf lay outside in the sunlight with no protection when these values all increased. By 54 hours the calf was able to maintain sternal recumbency and improvement was obvious. Ten hours later the calf rose stiffly when prodded and was able to walk unassisted. This calf and the control were euthanatized and were submitted for necropsy.

Hematology from calf 1 showed a left shift from the time of dosing until euthanasia. A neutrophilia and monocytosis were present from 20 hours post dosing until euthanasia. Calf 2 had a left shift from 20 hours until 36 hours post dosing. The control calf had a decreasing PCV and RBC count over the time of the experiment and a neutropenia was present for the last four days.

Fig 5 Experimental calf 2 in lateral recumbency showing positional opisthotomus and extensor rigidity caused by tonoclonic spasms. The blood on the floor is from the calf's left horn which was broken during a seizure.



Serum analysis included calcium, phosphorus, magnesium, total protein, albumin, sodium, chloride, potassium, bicarbonate, glucose, urea (BUN) and serum glutamic oxaloacetic transaminase (SGOT). Calf 1 had hyperglycemia reaching a value of 197 g/dl and a decreasing BUN dropping to a level of 6.4 g/dl from 20 hours until euthanasia at 28 hours post dosing. Calf 2 had hyperglycemia reaching a value of 150.0 g/dl from 28 to 44 hours and a decreasing BUN from 4 to 52 hours post dosing dropping to a level of 5.1 g/dl. Potassium levels were decreased mildly and transiently from 36 to 40 hours. No abnormal serum values were exhibited by the control calf.

Urinalysis showed glycosuria in calf 1 from 24 hours until euthanasia. Proteinuria was present from 8 hours post dosing until euthanasia. This reached a level of 3+ at 16 hours, then dropped to 1+ before euthanasia. Glycosuria with trace ketonuria developed in calf 2 between 40 and 44 hours. Proteinuria was also present in this calf from 8 to 24

hours reaching a peak at 24 hours. Urinalysis values for the control calf were within normal limits throughout the experiment.

Necropsies were done on all calves. Other than lesions relating to trauma while recumbent, the two treated calves did not have noticeable gross lesions. Histologic lesions consisted of multifocal alveolar hemorrhage in calf 1. There were also hemosiderin-laden macrophages in the spleen of this animal, suggestive of prior hemolysis. There were slight degenerative changes in the livers of both treated calves consisting of centrilobular vacuolization, cytoplasmic swelling and individualization of hepatocytes and occasional focal necrosis. No histologic kidney lesions were noted in the treated calves. No gross nor histologic lesions were noted in the control calf.

Discussion:

Hyperesthesia, myoclonus, and recumbency similar to that seen in field cases were induced in calves by oral dosing with mature yellow buckeye nuts. Severity of clinical signs in this experiment appeared to be dose related. This may help explain the range of signs exhibited by naturally affected animals. Obviously, more work is necessary before a definite correlation can be established. Of the clinical signs observed in the two treatment calves, only the strabismus which was exhibited by both calves had not been seen previously in field cases. Sluggishness and motor paralysis reported as characteristic for red buckeye toxicity[2] were not seen in the field or experimental cases in this report.[3]

Clinical pathologic alterations including left shift, neutrophilia, monocytosis, hyperglycemia, glycosuria, ketonuria and trace proteinuria could be attributed to endogenous corticosteroid release related to stress. The low BUN levels and the occasional 3+ proteinuria may be meaningful. Low BUN may result from decreased hepatic blood flow, a decrease in functional hepatic tissue or an enzyme abnormality. Proteinuria could result from transient glomerular leakage. Hematologic abnormalities in the control calf were thought to be caused by internal parasites.

Gross pathological lesions were unremarkable. The significance of multifocal alveolar hemorrhage in the lungs of the most severely affected calf is not known. Histopathologic liver changes were mild but may be significant when considered along with BUN levels which suggested a decrease in hepatic function.

Further studies, including toxicology, are underway to characterize the toxic principle(s) so that the pathogenesis of yellow buckeye toxicity can be determined. Hopefully this will lead to establishment of rational treatment.

It is important that clinicians and practitioners working in areas where yellow buckeye grows be aware that this tree poses a threat to cattle and be able to recognize signs of toxicity. Cases are seen in the Fall and early Winter especially following severe windstorms. Incidence can be expected to change from year to year depending on fluctuations in nut production. Prevention of this problem should be easily accomplished but the problem continued due to owner's reluctance to remove trees and to expansion of pastures into wooded areas.

Diagnosis is made on clinical signs. Hyperesthesia, myoclonus, and recumbency exhibited by pastured cattle especially in the fall in areas where yellow buckeye grows should prompt consideration of buckeye toxicity. Since there are no reliable diagnostic tests for this condition, diagnosis must depend on ruling out other conditions that present similar clinical signs. Hypomagnesemic tetany presents signs very similar to buckeye toxicity, but can be ruled out by evaluation of serum magnesium levels. All field cases which were examined and the two experimental calves in this report had normal magnesium, calcium, and phosphorus levels. Cattle with polio-encephalomalacia show forelimb gait abnormalities, strabismus, nystagmus, recumbency, tonoclonic convulsions and opisthotonos, but are also typically blind.[7] None of the calves in this report were blind. The early stages of tetanus could be mistaken for buckeye toxicity, but tetanus is more rapidly progressive to severe tetanic seizures and death.

Treatment of cattle with suspected buckeye toxicity should be directed at preventing absorption of the toxic principal(s) and hastening removal of buckeye nuts from the gastrointestinal tract. In mild cases this may be accomplished by oral dosing with mineral oil and activated charcoal. Rumenotomy or rumen lavage[8] may be indicated in more severely affected or valuable animals. Tranquilizers seem to reduce the intensity of muscular spasms. However, animals for which this treatment is indicated usually have a poor prognosis. Since most mildly affected animals recover with time, good nursing care and the provision of feed and water to recumbent animals may be the most important treatment considerations. Prevention of bloat, pulmonary aspiration, and musculoskeletal trauma are extremely crucial.

Summary

Yellow Buckeye *(Aesculus octandra* Marsh) nuts were incriminated as a possible cause of naturally occurring toxicity in cattle. Clinical signs included hyperesthesia, myoclonus, recumbency, tonoclonic spasms and occasionally death. Clinical pathologic and necropsy findings, on field cases were not remarkable.

A clinical trial was conducted to determine if typical clinical signs could be produced in calves by oral dosing with buckeye nuts. Two calves were dosed with ground nuts; one at a dose of 1% body weight and the other at ½%. Clinical signs similar to those seen in field cases were produced in both calves. More severe signs were exhibited by the calf that received the higher dose. Clinical pathologic, gross pathologic and histopathologic results were reported.

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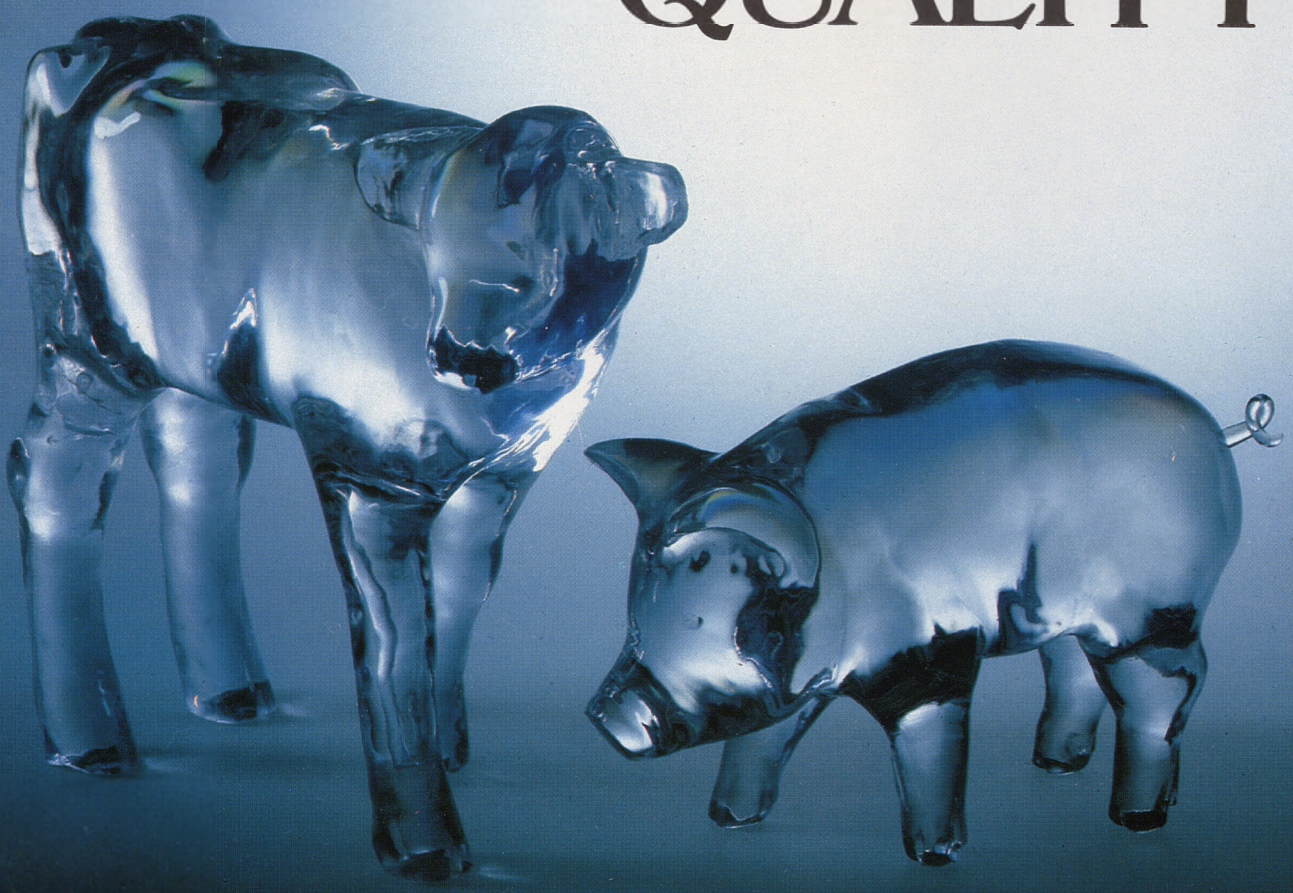

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