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Extended Drought Raises Potential Health Issues

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The extended drought being experienced across most of Kansas has reacquainted livestock producers and veterinarians with many drought-related problems. Not only has the drought caused a shortage of pasture and hay, but has threatened livestock with other problems that do not occur every year.

Feed-related problems

In many cases, grasses and desirable forbs (weeds) either did not get enough rain to initiate spring growth, or grew for a short period and then either died out or did not regrow due to the heat and lack of rainfall. In some pastures, drought-tolerant weedy species are the predominant green plants available. In some cases, these plants are toxic to livestock. Good pasture and cattle management calls for knowing toxic plant species common in area pastures. Livestock producers should recognize when pastures are getting short enough that cattle may be forced to consider consuming undesirable plants. They should supplement the cattle with hay or other feedstuffs, move the cattle to pastures where available forages do not pose a toxicity threat, or move cattle to a drylot situation where they can be fed. Cattle will eat toxic plants, especially when starved.

Nitrate toxicity always becomes a potential problem during dry periods but may be a bigger problem this year than most. Many corn and milo fields did not receive enough moisture to fill ears or heads with grain, and some farmers want to harvest the standing forage and make hay out of it. Insist that they test for nitrate levels before harvesting because many fields will be too high in nitrate to consider using for feed. Test the more stunted plants in the field to determine the highest nitrate levels present. Remind producers that nitrate does not dissipate from hay like prussic acid does. Also, test pigweed or Johnsongrass that might be in the field because it often contains higher levels of nitrate than corn or sorghum varieties. Forage sorghums/haygrazer should be tested before being grazed or hayed. Prussic acid (HCN) also can be a problem, especially following a rain on drought-stressed forages. The prussic acid will build up in any new green growth following the rain (new leaves, suckers, tillers, etc.) and can be extremely toxic until the new growth matures. Regrowth should be tested until it reaches maturity or at least a week after the first killing frost.

Vitamin A deficiency is frequently a hidden problem that surfaces after cattle have gone without consuming green forage for an extended period of time and when the vitamin has not been included in feed or mineral supplements. Vitamin A deficiency can lead to retained placentas when cows calve next spring, as well as increased problems with mastitis. Deficiencies also can lead to improper fetal development and calf growth and set the stage for decreased resistance to calf scours and pneumonia.

Water-related problems

When cattle are watering from surface water sources, problems may begin to occur as the water source is beginning to dry up. Non-volatile contents in the water tend to concentrate as the water level recedes, causing an increase in such problems as increased nitrate and sulfate toxicity, increased hardness/salinity, increased coliform-related diseases, decreased water consumption, and ultimately, decreased intake of whatever feedstuffs are available. Also, problems such as cyanobacterial (blue-green algae) toxicity tends to increase as the water becomes stagnant and ponds heat up. This is compounded if chemical fertilizer or manure runoff enriches the ponds.

Options

Early weaning of calves and extensive culling of cows are both management decisions that producers should consider or implement. Depending on whether early weaned calves are shipped directly to market or the feedlot or held and preconditioned before being shipped, the higher protein and more concentrated energy needs of these lighter weight ani-

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mals must be considered and provided. If producers normally market calves through a value-added or other preconditioning program and they have sufficient time and feed resources to meet the needs of those programs, they should be encouraged to continue to participate. Some producers have waited so long hoping that it would rain that they now have no choice but to wean the calves on the truck on the way to the auction market. Cow culling criteria should include open cows, old cows, or cows in poor body condition score that need extra supplementation to make it through the winter, cows that will be late calving next spring and not match the bulk of the herd's calving dates, oddball-looking cows that do not match well phenotypically with the rest of the herd, and ornery cows (those with disposition problems). Pregnancy checking is imperative this year, and with many herds needs to be done within 45 days after the bulls are removed so that open cows can be marketed as soon as possible to reduce the number of head that have to be fed from now until green grass arrives next spring.

It has been a tough summer on cattle producers and veterinarians, alike. Hopefully it will begin raining again in time for pastures to regrow, ponds to refill, and producers not to have to depopulate or buy super-expensive feed to make it through the upcoming fall and winter. Encourage them to manage well based on resources and options.

Equine Motor Neuron Disease in Kansas

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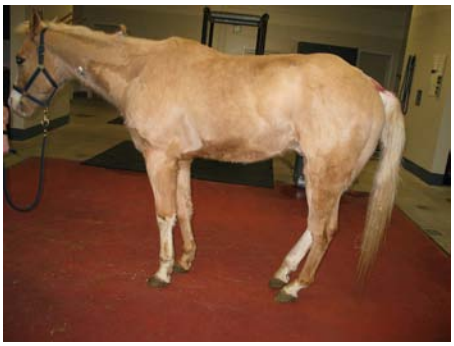
Clinical Sciences

Equine motor neuron disease (EMND) is a neurodegenerative disorder of the somatic lower motor neurons of horses. The disease was first described approximately 20 years ago with a relative geographic predisposition[1]. Since that initial report, laboratory investigations by Divers and colleagues have been able to determine risk factors for the development EMND so that disease onset can be prevented[2;3].

Horses with acquired EMND are 2 years of age or older. There is frequently an acute onset of trembling, almost constant shifting of weight in the rear legs when standing, sweating, and preferred recumbency. Muscle wasting is noticeable, and owners frequently comment that weight loss was apparent before onset of muscle trembling. Funduscopic examination has revealed brown streaking discoloration in up to 40% of the reported cases[4]. The tail head is often carried in an elevated position, whereas the head and neck are in abnormally low position (Figure 1, top). In a smaller percentage of cases, trembling and constant shifting of weight may either not occur or not be observed and the predominant clinical sign may be muscle wasting. Although the clinical signs are characteristic for EMND, other neuromuscular disorders, particularly chronic myopathies, may look clinically similar. A biopsy of the sacrocaudalis dorsalis medialis (tail head) muscle or spinal accessory nerve can be useful in confirming the diagnosis (Figure 1, bottom). Approximately 40% of horses with EMND continue to deteriorate and are euthanized within 4 weeks of onset of signs; approximately 40% have marked improvement in clinical signs within 4 to 6 weeks after either relocation to another stable or administration of antioxidants, and approximately 20% remain permanently and noticeably atrophied.



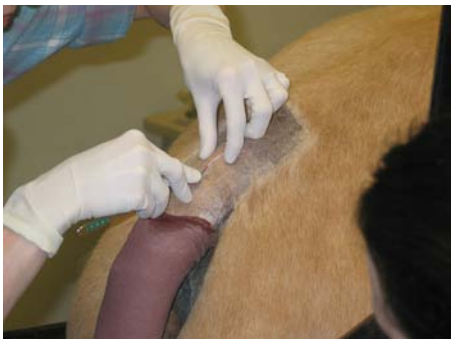


Figure 1: The top figure shows an example of low head and elevated tail head carriage that are characteristic clinical findings in horses suffering from EMND. The bottom figure demonstrates the region where tail head biopsy can be performed.

Clinical Findings

- Muscle fasciculations
- Symmetric sweating
- Weight loss/muscle atrophy
- Tendency for recumbency
- Camped under appearance
- Elevated tail head

Laboratory Findings

The only consistently abnormal laboratory finding on routine hematologic analysis is a mild to moderate elevation in muscle enzyme activity: creatine kinase (CK) and aspartate aminotransferase (AST)1. Plasma vitamin E values are typically low (<1 ug/ml)[5], selenium is often normal, vitamin A may be slightly low and serum ferritin has been speculated to be high in horses with EMND[6]. Reports have clearly demonstrated that horses suffering from EMND do not universally have low serum vitamin E status; therefore, diagnosis should not be based on serum vitamin E status alone[6]. EMND affected individuals have been reported to have increased spinal cord copper concentration when compared with controls, while spinal cord vitamin E is consistently low. Some investigations have examined glucose absorption tests that revealed some EMND affected patients to have reduced intestinal glucose absorption.

Pathology

Pathologic lesions are generally limited to the lower motor neuron system. Loss of approximately 30% of motor neurons occurs before clinical signs are obvious.

Muscles with a predominance of type I fibers are more severely atrophied than are those with mostly type II fibers. Lipofuscin deposition is consistently observed in the endothelial capillaries in the spinal cord and occasionally in the pigmented retinal epithelium[4]. Abnormal lipopigment deposition is sometimes found in the liver and gut and has been referred to as brown bowel disorder.

Epidemiology

Horses with EMND have frequently been housed at the same location for at least 18 months.

There is no gender predilection; quarter horses and thoroughbreds are overrepresented in the original patient population[7]. There is minimal or no pasture for the great majority of cases. In cases that have both EMND and normal pasture access, infiltrative bowel disease (malabsorption) should be considered and therefore investigated. Many EMND cases have been fed grass hay (no alfalfa). Mineral supplements, especially selenium, have been reported EMND horses when compared with off-farm healthy horses. The disease was initially reported in the northeastern United States, but it has been reported in most states and many countries of the world.

EMND in Kansas

Among patients that have been diagnosed in the KS/NE region, horses often have a clinical history that is consistent with the findings of previous investigations. Table 1 outlines the cases that have been evaluated at KSU in the past 10 years. Among those cases that were suspected to suffer from EMND and had serum vitamin E levels examined 4/7 (57%) of these patients had vitamin E levels below the reference range, while 6/6 (100%) of the horses that had tail head muscle biopsy performed were confirmed to have disease based on muscle histopathology. Patients that had clinical signs consistent with the disease, but did not have muscle biopsy and/or vitamin E levels examined and showed a positive response to d-alpha tocopherol supplementation are listed as being presumed to be positive for the disease.

Clinical Management

- Green forage
- Alfalfa hay supplementation
- Natural d-alpha tocopherol (vitamin E) 10,000 U daily

Summary

Epidemiological, pathological, laboratory, and experimental studies all support the hypothesis that EMND occurs after prolonged vitamin E deficiency. Horses with limited access to green forage for prolonged periods should be examined for serum vitamin E status. Those suspected of clinical disease should have a tail head muscle biopsy performed. In confirmed cases, nutritional modification should be implemented in combination with the supplementation of d-alpha tocopherol (natural vitamin E) at a dose of approximately 10,000 U natural vitamin E/day.

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Table 1. Kansas State University EMND Cases 2000-2011

Case	Signalment	Pres. Complaint	Vit. E	Biopsy	Diagnosis
1	4 YO QH Geld.	Liver / muscle dz.	0.65 µg/mL	Anguloid atrophy	EMND
2	4 YO QH Geld.	Neurologic dz.	ND	Anguloid atrophy	EMND
3	5 YO QH Geld.	Weakness	3.68 µg/mL	ND	Presumed
4	12 YO QH Geld.	Colic / weakness	2.38 µg/mL	ND	Presumed
5	12 YO Donkey mare	Recumbency	ND	Anguloid atrophy	EMND
6	7 YO Paint mare	Colic / weakness	0.65 µg/mL	ND	EMND
7	12 YO Morgan Geld.	Colic / weakness	2.72 µg/mL	Anguloid atrophy	EMND
8	5 YO Paint stallion	Neurologic disease	1.92 µg/mL	Anguloid atrophy	EMND
9	22 YO QH Geld.	Weakness	1.23 µg/mL	ND	EMND
10	12 Pain stallion	Muscle fasciulation	ND	Anguloid atrophy	EMND

Xylitol Toxicosis in a Dog

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A six-year-old neutered male golden retriever/standard poodle dog was presented to his veterinarian with a 48-hour history of vomiting and diarrhea that began shortly after ingestion of an unknown amount of xylitol sweetener. A blood sample was submitted for a complete blood count and serum for a chemistry panel (Tables 1 and 2). Symptomatic treatment was initiated, but the dog did not respond to therapy and died approximately 45 minutes after presentation. A necropsy conducted by the referring veterinarian revealed multifocal hemorrhages in the subcutaneous tissue of the ventral trunk, the serosa of the intestines, the lungs, and the right kidney. There was free blood in the abdominal cavity, and the stomach contained mucus and melena. Microscopically, there was diffuse, massive hepatocellular necrosis with loss of hepatocytes. There was acute hemorrhage in the heart, lungs, small and large intestines, and the mesentery.

A diagnosis of acute, massive hepatocellular necrosis due to xylitol toxicity was made based on history, clinical pathology (hypoglycemia and marked increase in hepatic enzymes) and histopathologic findings (acute hepatic necrosis).

Xylitol, a pentose sugar alcohol, is used as a reduced-calorie sweetener in baked goods, chewing gums, and candies for humans. Accidental and experimental xylitol induced hepatocellular necrosis has been reported in dogs. Xylitol is metabolized in the liver and causes ATP depletion Hepatocellular injury and necrosis are proposed to be secondary to ATP depletion and increased production of reactive oxygen products, resulting in oxidative membrane damage.

Elevated concentrations of alanine aminotransferase, alkaline phosphatase and total bilirubin in this case indicate severe hepatocellular injury and biliary stasis. Multifocal hemorrhages in multiple organs suggest lack of production of blood clotting factors secondary to severe hepatocellular damage. The platelet counts were also below normal, most likely due to platelet consumption.

In one experimental study, the circulating insulin concentrations in dogs were at least five times higher after either oral or intravenous administration of xylitol (1.0 g/kg), when compared to glucose administration at the same dose rate and route. Therefore, xylitol is a potent stimulator of insulin secretion in dogs and causes hypoglycemia. In the present case, the dog was hypoglycemic similar to previous reports of xylitol toxicity. The serum potassium level was within the normal range in the present case, although a mild hypokalemia was reported previously because of elevated insulin concentrations.

The elevated hematocrit was likely secondary to dehydration. Elevated BUN and creatinine concentrations possibly indicate prerenal azotemia. The hyperphosphatemia could be due to release of inorganic phosphates after hepatocellular necrosis as reported previously, or due to decreased glomerular filtration rate secondary to dehydration.

Hepatoxic mushrooms (*Amanita sp*), blue green algae, aflatoxins and idiosyncratic drug reactions can cause similar liver lesions and are differential diagnoses in the absence of a definitive history.

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Table 1. CBC Results

Test Requested	Results	Reference range	Units
Hematocrit	64 (high)	36-60	%
Hemoglobin	20.6 (high)	12.1-20.3	g/dL
Platelet count	160 (low)	170-400	103/µL

The WBC, RBC, MCV, MCH, MCHC and differential leukocyte counts were normal.

Table 2. Serum Chemistry Results

Test Requested	Results	Reference Range	Units
Alanine aminotransferase	22862 (high)	12-118	IU/L
Alkaline phosphatase	364 (high)	5-131	IU/L
Total Bilirubin	2.3 (high)	0.1-0.3	mg/dL
Glucose	31 (low)	70-138	mg/dL
BUN	43 (high)	6-31	mg/dL
Creatinine	2.1 (high)	0.5-1.6	mg/dL
Phosphorus	9.3 (high)	2.5-8.0	mg/dL
Chloride	99 (low)	102-120	mEq/L

Serum concentrations of calcium, sodium, potassium, total protein, cholesterol albumin, globulin and albumin/globulin ratio were in the normal range.

No Evidence of Terrorism in Minnesota Anthrax Case

The Minnesota Department of Health investigated an "extremely rare" case of inhaled anthrax last month. Officials said there was no threat to the public. The patient, who was not identified, was hospitalized in Minnesota after traveling through Montana, Wyoming, and the Dakotas, where anthrax spores occur in the environment.

The infectious agent probably came from natural sources, and there was no evidence of a link to terrorism, according to a health department statement. Nevertheless, the Federal Bureau of Investigation (FBI) joined in the investigation because anthrax can be used as a bioterrorism agent.

The case most likely occurred when the individual was exposed to soil or animal remains infected with anthrax, a bacterial spore that can cause potentially fatal illness. State officials did not give details because there was no risk to the public and because of privacy concerns.

The state epidemiologist, noted that the patient was treated, but that anthrax is not spread from person to person. The patient was hospitalized after developing fever and pneumonia. During routine tests, the lab discovered an unusual grown and sent it to the health deparment for further testing. It was confirmed as anthrax, and the FBI was contacted as "standard protocol."

Government officials have treated anthrax exposure as a possible act of terrorism since anthrax-laced letters began appearing in the U.S. mail following the September 11, 2001, terrorist attacks. That year, 22 people developed anthrax infections and five died after handling or receiving letters containing anthrax spores. In spite of intensive investigation, no one was charged in that case.

In the Minnesota case, investigators found no evidence suggesting it was a criminal or terrorist act. The FBI is no longer actively investigating the incident. Health officials said such cases are "extremely rare," although in the past anthrax was known as "wool sorter's disease" because people would breathe in spores from infected animals. Anthrax can infect cattle, sheep, and other animals, and can live in soil for years.

Inhaled anthrax is far more dangerous than a more common type of anthrax infection, which infects the skin. The illness can start with flu-like symptoms, such as cough, fever, and muscle aches, and last several days before seeming to disappear. When it returns, it can cause lung problems, breathing difficulty, and shock. Without treatment, it is fatal in up to 90 per cent of cases, according to the health department.

Minnesota health officials later said the man had a prior chronic lung condition that may have made him more susceptible to infection with anthrax.

New Tick-Borne Illness Infests Midwest

Deer ticks carrying a newly identified bacterium are infecting residents of the Midwestern United States with a disease called ehrlichiosis, and experts say it will likely appear in other areas of the country. The still unnamed bacterium, which causes fever, body aches and fatigue, has been identified in 25 people in Minnesota and Wisconsin but has probably infected many more, researchers said in a new study. "So far we have tested thousands of patients from around the United States, and we have only found it in the blood of patients from Wisconsin and Minnesota," said lead researcher Dr. Bobbi Pritt, director of the Clinical Parasitology and Virology Laboratories at the Mayo Clinic in Rochester, Minn. "We have also found it in ticks. Specifically, in the ticks called *Ixodes scapularis*, also known as the deer tick," she said. "That's the same little tick that transmits a number of really serious diseases – Lyme disease, anaplasmosis, and babesiosis," she added. "Ehrlichiosis is an emerging disease. Its range is expanding throughout the United States," she said. The bacterium, first identified in 2009, was initially confined to a small area in Minnesota, but now is seen throughout the state and in Wisconsin, Pritt explained. Ehrlichiosis kills white blood cells, causing a feverish illness. Severe cases can involve the lungs, kidneys, and brain, and require hospitalization, but rarely result in death, the researchers said in their report, published August 4 in the New England Journal of Medicine.

DHS Infectious Disease Report
August 5, 2011

KSVDL: New BVDV Testing Procedures

KSVDL is no longer offering the Enfer Tissue Tag™ System. We are now pooling ear notch tissue for BVDV PCR testing in groups of up to five animals for $21 per pool. If a positive pool is found, the submitting veterinarian will be contacted by KSVDL before further testing is initiated to find the positive animal(s) contained in the pool. The follow-up PCR to identify the positive animal(s) will be $21 per tissue. If you have questions about BVDV testing please contact Dr. Dick Oberst, 785-532-4411, or Dr. Gregg Hanzlicek, 785-532-4853.

Continuing Education

September 24, 2011
Natural disasters…What about the animals?

November 5-6, 2011
Annual equine reproduction conference for horse breeders and farm personnel: From egg to foal

January 6, 2012
Annual small ruminant conference (featuring Dr. Lionel Dawson)

January 6, 2012
Bull evaluation and management clinic

February 2012
Annual conference on animal diagnostics and field applications

For the most complete, up-to-date conference information visit our website at: *www.vet.ksu.edu* and click on Continuing Education, or contact: Megan Kilgore at 785-532-4528 or *meganlk@vet.ksu.edu*

Upcoming Events

September 9-18
Kansas State Fair, Hutchinson, Kan.

September 12-14
Grasslands in a Global Context international symposium, Manhattan, Kan.

September 22
K-State Beef Stocker Field Day, Beef Stocker Unit— Manhattan, Kan.

October 25
American Royal National 4-H Meat Judging and Identification Contest, Manhattan, Kan.

November 4-6
K-State Sheep and Meat Goat Conference Manhattan, Kan.

December 3
K-State Junior Beef Day, Manhattan, Kan.

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