

Cyanide Poisoning in Ruminants

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Prussic acid, cyanide, or hydrocyanic acid are all terms relating to the same toxic substance. Hydrogen cyanide was first isolated from a blue dye (Prussian blue) and because of its acidic nature it became known by the common name “prussic acid.” Cyanide is one of the most rapidly acting toxins that affect cattle.

Cause

The primary cause of cyanide poisoning in ruminants is the ingestion of plants containing compounds called “cyanogenic glycosides.” These cyanogenic glycosides and the enzymes that convert them to free cyanide reside in different locations within the plant cells. When plant cells are crushed, chewed, wilted, frozen, chopped or otherwise ruptured, the cyanogenic glycosides and the enzymes can physically come together and rapidly form free cyanide. As ruminants consume these plant materials, hydrogen cyanide gas is liberated in the rumen and rapidly absorbed into the bloodstream. Ruminants are very susceptible to cyanide poisoning because the rumen microflora contain enzymes that, in the presence of water, are also capable of converting cyanogenic glycosides in plants to free cyanide gas. Cyanide ultimately prevents hemoglobin in red blood cells from releasing its oxygen to the tissues and the animal subsequently dies from lack of oxygen.

The cyanogenic potential of plants is affected by the species and variety of the plant, weather, soil fertility and stage of plant growth. Cyanide poisoning of livestock is commonly associated with johnsongrass, sorghum-sudangrass, and other forage sorghums. Choke-cherry or wild cherry, elderberry, and arrow grass are less frequent causes. Young plants,

new shoots, and regrowth of plants after cutting often contain the highest levels of cyanogenic glycosides. The risk of poisoning decreases as forages mature. Leaf blades are higher risk than leaf sheaths or stems, upper leaves are higher risk than older leaves, and seed heads are considered low risk. Application of herbicides such as 2,4-D have been shown to increase the cyanogenic potential of plants. Drying plants decreases the cyanogenic potential over time so hay is rarely hazardous if adequately cured. Ensiling plants will significantly reduce the cyanogenic glycoside content.

Clinical Signs

Cyanide is one of the most potent toxins in nature. If large quantities of cyanide are absorbed rapidly enough, the body’s detoxification mechanisms are overwhelmed and the animal soon dies. Affected animals rarely survive more than 1-2 hours after consuming lethal quantities of cyanogenic plants and usually die within 5-15 minutes of developing clinical signs of poisoning. Signs may include rapid labored breathing, irregular pulse, frothing at the mouth, dilated pupils, muscle tremors, and staggering. The mucous membranes are bright red in color due to oxygen saturation of the hemoglobin.

Diagnosis and Treatment

Cyanide is rapidly lost from animal tissues unless collected within a few hours of death and promptly frozen. Liver, muscle (heart [ventricular myocardium] preferred), whole blood, and rumen contents should be collected and frozen in air-tight containers before shipment to a laboratory capable of performing cyanide analysis. Perhaps most important

in the diagnosis of cyanide poisoning is to identify plants in the area accessible to the animals and determine if they are likely to contain cyanogenic glycosides. Cyanide concentration determinations in suspect plants can be performed if samples are frozen immediately or sent on ice overnight to a diagnostic laboratory. Treatment can be attempted if affected animals are discovered quickly, but often animals are found dead. Contact a veterinarian immediately if cyanide poisoning is suspected. The intravenous administration of sodium thiosulfate by a veterinarian is an effective treatment for cyanide poisoning. The dose can be repeated after a few minutes if the animal does not respond. Most animals that live after treatment will recover.

Prevention

The risk from potentially dangerous forages may be reduced by following these management practices:

Graze sorghum, sorghum crosses, or johnsongrass plants only when they are at least 18-24 inches tall. Young rapidly growing plants or regrowth have the highest concentrations of cyanogenic glycosides, especially in the newest leaves and tender tips. Do not graze plants with young tillers.

Do not graze plants during drought periods when growth is severely reduced or the plant is wilted or twisted. Drought increases the chance for cyanide because slowed growth and the inability of the plant to mature favors the formation of cyanogenic compounds in the leaves. Do not graze sorghums after drought until growth has resumed for 4-5 days after rainfall.

Do not graze potentially hazardous forages when frost is likely (including at night).

Frost allows conversion to hydrogen cyanide within the plant. Do not graze for two weeks after a non-killing (>28 degrees) frost. It is best not to allow ruminants to graze after a light frost as this is an extremely dangerous time and it may be several weeks before the cyanide potential subsides. Do not graze after a killing frost until plant material is completely dry and brown (the toxin is usually dissipated within 72 hours).

Do not allow access to wild cherry leaves.

After storms or before turnout to a new pasture, always check for and remove fallen cherry tree limbs.

If high cyanide is suspected in forages, do not feed as green chop.

If cut for hay, allow to dry completely so the cyanide will volatilize before baling. Allow slow and thorough drying because toxicity can be retained in cool or moist weather. Delay feeding silage 6 to 8 weeks following ensiling.

Forage species and varieties may be selected for low cyanide potential.

There are wide differences among plant varieties. Some of the sudangrasses, such as piper, are low in cyanide.

Test any suspect forages before allowing animal access.

A rapid field test is available that can provide on-site results. Contact your county Agricultural Extension agent for further information.

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Issued 4-2014