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I,A. Schipper
North Dakota State University

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NebGuide

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Sweet Clover Poisoning

I. A. Schipper, D.V.M.
North Dakota State University
Department of Veterinary Science

Sweet clover poisoning is a problem of varying frequency and intensity in livestock wherever sweet clover grows. The toxic compound produced in sweet clover prevents normal blood clotting resulting in hemorrhages and associated symptoms.

The preliminary symptoms include stiffness, lameness, dull attitude, and swellings beneath the skin (hemotomas or blood clots) over all parts of the body but primarily at the hips, brisket or neck. The mucous membrances may be pale—indicating that anemia exists. Hemorrhage decreases the quantity of red blood cells available to transport oxygen to the body and carbon dioxide to the lungs. This results in varying degrees of respiratory stress, depending on the degree of red blood cell loss and the physical exertion of the animal. Blood may be present in the feces, urine or milk, come from nostrils or there may be extensive hemorrhage at parturition. Dicoumarin may pass the placental membrane and cause hemorrhage in the fetus. In some cases the dam may die without any visible signs.

The toxic factors associated with sweet clover poisoning may also interfere with reproduction and be responsible for fetal reabsorption, stillbirths, or neonatal deaths. The reproductive problems may exist without clinical signs of toxicosis in the dam.

What Is the Cause.

A substance referred to as coumarin is present in varying amounts in all sweet clover and is responsible for the characteristic odor associated with sweet clover. In sweet clover which is spoiled or damaged the coumarin is converted to a toxic substance called dicoumarin (bishydroxycoumarin). The dicoumarin will interfere with the metabolism and synthesis of vitamin K. Vitamin K is an essential to the liver synthesis of four components (prothrombin and factors VII, IX, X) necessary to the prevention of seepage of blood to the exterior of the circulatory system and to establish the clotting of blood expelled by injury or surgery.

Vitamin K_1 is found is green plants such as alfalfa. Vitamin K_2 is formed by the microflora of the digestive tract. These two sources are normally sufficient to provide the requirements of cattle. Menadione (vitamin K_3) is a synthetic compound that may be utilized as a feed supplement or injectable product to counteract vitamin K deficiency.

Not all moldy sweet clover is toxic and the absence of observable mold is not absolute assurance that dicoumarin is not present in the forage. Sweet clover poisoning occurs less frequently in silage than in hay and infrequently in pastured animals.

Toxic levels of dicoumarin in sweet clover forage may remain for 3 to 4 years of storage. Dicoumarin toxicity has never been observed in other clovers or alfalfa. If animals are fed sweet clover forage for three weeks with no signs of toxicity being exhibited the chances of toxicity are unlikely.

Host Animals.

Sweet clover toxicosis is usually a problem in the ruminant animal. The mature animal has a greater resistance to sweet clover toxicosis than the young animal. Newborn calves are usually deficient in vitamin K.

Though sweet clover poisoning may occur in any of our domestic animals, the more selective eating habits of horses and swine decreases the possibility of sweet clover poisoning in these species.

Prevention Based on Management.

Sweet clover that is spoiled or moldy should be fed with caution. The farmer or rancher may have the feed assayed to determine its dicoumarin content. Two to three pounds (minimum) of the sweet clover hay or silage is required for the analysis. It is imperative that the sample be representative, and include the moldiest sweet clover which can be found.

One method of avoiding toxicity is by planting only the low coumarin content sweet clovers *(Melilotus dentata)*.

Avoid contamination of pastures or hayfield with yellow *(M. olfficinalis)* or white *(M. albus)* which contains substantial levels of coumarin. Other preventive measures include stacking or baling sweet clover only when it is well cured and dry, and avoiding the use of large, tightly bound bales.

When sweet clover is suspected of being toxic it may be fed for 7 to 10 days and then replaced with alfalfa or other forage for an equal period of time. The intermittent feeding of alfalfa appears to be more effective in neutralizing the toxicity of sweet clover than the feeding of equal amounts of alfalfa simultaneously. Sweet clover should not be fed for at least two weeks before or during the calving period. Supplementation with vitamin K will aid in counteracting the effects of dicoumarin but is less practical than replacing the sweet clover forage with alfalfa. Adequate calcium supplementation will aid in the prevention of hemorrhage. Although calcium is not directly associated with dicoumarin toxicity it remains one of the essential elements in the blood clotting process. Alfalfa usually contains a high level of calcium.

Surgery of any kind should be avoided on animals consuming moldy or damaged sweet clover forage.

Treatment

Animals exhibiting the symptoms of sweet clover poisoning may be saved by direct blood transfusion from disease-free animals which have not consumed toxic sweet clover. Intramuscular administration of vitamin K (menadione) also will aid in counteracting the effects of dicoumarin. All animals involved should be removed from sweet clover and placed on high quality alfalfa which is high in vitamin K and in calcium.

Veterinary aid should be sought immediately upon suspicion of the presence of sweet clover poisoning as other diseases such as anaplasmosis may appear similar but require quite different approaches for control and treatment.

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LEO E. LUCAS, DIRECTOR