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Popular Article

Bracken Fern Poisoning in Animals

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Abstract
The most prevalent poisonous plant species found in temperate and subtropical climates is bracken fern. Although young plants are said to be more toxic than older ones, plant material as a whole is typically toxic. Various active principles present in it have the potential to cause disease conditions in animals. The fully characterized toxic factors of ferns include thiaminase, quercetin, aplastic anemia factor, and hematuria factor. Among these, quercetin is carcinogenic; long-term ingestion of bracken fern has been linked to bladder tumours. Bone marrow tissue is depressed by the fern poison. Most impacted is the thrombocytic count. Hemorrhages, which are typically located on the outside of the heart and occasionally in the kidney but more frequently in the intestines, are the most common findings in cases of simple fern poisoning.
Keywords: Haematuria, thiaminase, ptaquiloside

Introduction

One of the top five vascular plants in the world, bracken fern (*Pteridium aquilinum*) is distributed all over the world. With upright deciduous fronds that stay green until they are killed by frost or drought, bracken fern is a perennial plant. Primarily dispersing via dense rhizome networks, it has the ability to take over plant communities, particularly those that have been burned or disturbed. Though it can be found in many different places, bracken fern is most frequently found in open, semi-shaded woodlands that have good drainage.

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Fig.1. Bracken fern

Cattle, sheep, and horses are all poisoned by bracken fern; sheep are less susceptible. Bracken possesses a thiaminase inhibitor that causes horses to become thiamine deficient, a condition that can be treated by administering thiamine. According to research, bracken fern can cause cancer as well. Milk from cows fed bracken fern could be harmful to humans.

Toxic principles

There are four main toxic principles i.e. thiaminase, aplastic anemia factor, hematuria factor and quercetin. Thiaminase produces thiamine deficiency in non-ruminants. Aplastic anemia factor (Ptaquiloside) a norsesquiterpene glycoside produces bone marrow suppression and anemia, is also a carcinogen. Hematuria factor causes enzootic hematuria with hemorrhages. Quercetin is a co- carcinogen. The fern's toxicity increases with age.

Clinical signs

Horses shows bracken staggers which occurs after consumption of fern for 1-2 months along with emaciation, lethargy, anorexia, incoordination and staggering gait. Animal stand with feet well spread and back arched. Recumbency followed by muscle tremors, cardiac irregularity, clonic spasms and opisthotonos. Colic, hemoglobinuria, hyperthermia, severe anemia, tachycardia and hemolytic crises may be seen. In pigs it is rare and less distinct signs occurs like anorexia, weight loss and resemble heart failure in terminal phase. Ruminants in acute or subacute poisoning shows bone marrow suppression. It occurs at 7-8 weeks after first access to the fern. Bleeding from body orifices, hyphema, anorexia, pyrexia (106-110°F), pale mucus membrane or with petechiae and clots of blood in feces i.e. acute hemorrhagic syndrome occurs.

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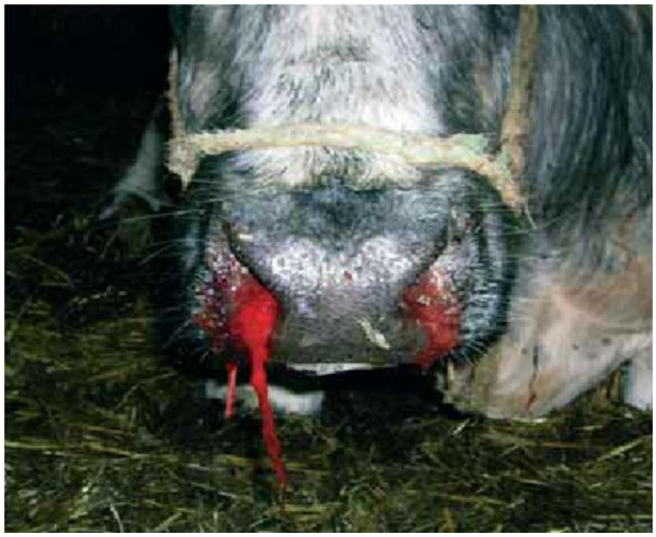

Fig.2. Epistaxis in a cow suffering from coagulation disorders due to acute bracken fern poisoning

In calves, a laryngic form with depression and excessive mucous discharge from nostrils, edema of throat with difficult breathing. Enzootic hematuria (most common) oucrs in chronic cases which is characterized by intermittent hematuria and ultimate death due to anemia. Sheep shows bright blindness i.e. permanent blindness due to progressive retinal atrophy.

Necropsy findings

Haemorrhage is the only constant feature that can be found despite the wide variations in other features. Horse shows no specific lesions. Acute poisoning in cattle shows multiple hemorrhages throughout the carcass, hemorrhagic and necrotic ulcers in the GI tract.

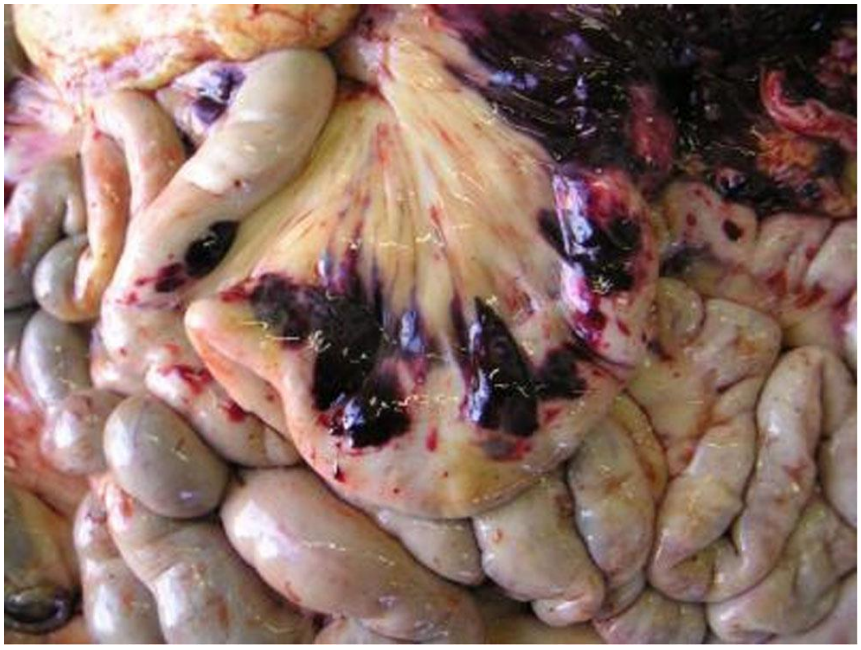

Fig.3. Hemorrhages in the mesentery of a cow with bracken fern poisoning

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In chronic poisoning, bladder mucosa contains small hemorrhages, dilated vessels, vascular, fibrous, or epithelial neoplasms and others neoplasms in upper GIT. In sheep, histologically severe atrophy of retinal rods and cones, outer nuclear layer is most pronounced in the tapetal portion of retina.

Diagnosis

Evidence of ingestion combined with relevant clinical and necropsy findings is typically used to make the diagnosis. The poison from ferns depresses bone marrow tissue, which lowers the concentration of various cell types in the blood mainly thrombocytes. Urinalysis reveals hematuria, proteinuria. Ophthalmoscopic examination of sheep with bright blindness shows narrowing of arteries and veins and a pale tapetum nigrum with fine cracks and spots of grey color. Blood thiamine level in non-ruminants (8.5µg/dl) decreases to 2.5µg/dl. Blood pyruvate level (2mg/dl) increases to 8.5mg/dl. Fall in erythrocyte *transketolase* activity occurs.

Differential diagnosis

In non-ruminants with other plant poisoning inducing thiamine deficiency- e.g. horse tail (*Equisetum arvense*) and turnip (*Beta vulgaris*). In cattle, acute hemorrhagic syndrome should be differentiated from acute septicemia, anthrax, mycotoxins and sweet clover poisoning. Chronic enzootic hematuria should be differentiated from babesiosis.

Treatment

If a timely diagnosis is made, horses with thiamine deficiency caused by bracken fern respond very well to treatment. It is advised to inject a 5 mg/kg thiamine solution intravenously (IV) every three hours for a few days. While oral supplementation may be necessary for an extra 1-2 weeks, some patients have found success with a SC injection of 100–200 mg per day for 6 days. Animals that have been similarly exposed but have not yet displayed symptoms should also receive thiamine treatment because symptoms can appear days or weeks after an animal has been removed from the bracken fern source. Antibiotics and sulpha medications were employed to stop bacterial invasion. DL-butyl alcohol stimulated bone marrow cells in an unique way. While transfusions of blood or even platelets might be appropriate, large volumes (2–4 L blood) are needed to treat cattle efficiently.

Prevention

Preventive care now receives greater attention. The main defence against the poisoning may be to refrain from giving bracken fern to animals. Eliminate all bracken fern from pastures and hayfields. Do not use hay from bracken infested meadows for feeding or bedding. Reduce the size of large infestation of bracken fern by pulling or mowing the fronds at least twice a

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year. Herbicide treatment using glyphosate.

Conclusion

There is a bone marrow toxin in Fern. This results in a decreased immunity against infection. If enough fern is consumed, death from simple fern poisoning with a high fever and hemorrhages will occur. For the treatment of fern poisoning, butyl alcohol injections combined with antibiotics or sulpha medications have shown excellent results. By reducing the likelihood that young animals will consume large amounts of young, growing fern in a short period of time, the risk of bracken fern poisoning may be reduced. The removal of all bracken is the only viable option; however, paddocks that are almost exclusively composed of young fern should be handled carefully when using the currently available techniques, such as rolling and slashing.

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