BRACKEN FERN POISONING IN RUMINANTS

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ABSTRACT
The present article deals with the poisoning of bracken fern in ruminants. The accumulation of the active principle leads to generation of a wide array of toxic syndromes. The diagnosis and treatment of the animals affected by the toxicity of braken fern.

KEYWORDS: Bracken fern, Ruminants, Toxicity.

INTRODUCTION
Livestock form the major source of livelihood for the agricultural farmers in India. The rearing system is also not well developed in our country. The mechanised livestock farming has not yet been standardised. That’s why livestocks are mainly fed by grazing in the pasturelands. The accidental ingestion of the poisonous weeds and plants usually occurs in the field conditions. Pteridium aquilinum commonly known as bracken fern is a natural flora of the forest lands. Bracken fern is one of the five most successful plant organism of plant kingdom¹². The plant belongs to dennstaedtiaceae family, is classified as a toxic weed. Brakern fern contains a varying amount of toxic glycosides which is capable of producing toxicity and carcinogenesis. The toxicity of bracken fern in cattle occurs mainly due to the active principle called ptaquiloside, a nor-sesquiterpene glycoside which causes bleeding and damage to the bone marrow.

PREDISPOSING FACTORS
The toxicity of bracken fern depends on several factors such as
Part of the plant: Underground stems (rhizomes) are the most toxic part followed by younger green fronds and the mature fronds are the least toxic part of the plant.

All portions of the fern i.e. both green and in harvested hay are poisonous to livestock.

Draught season favours the toxicity as pasture feed is in scarcity at that time.

Age of the animal: The younger animals are mainly affected accidentally by the bracken fern poisoning as they have not yet been trained to avoid the fern during grazing.

TOXICITY

The poisoning of bracken fern in cattle and sheep has been reported after the cumulative effect of ingestion of plant for 2-4 weeks of time. The accumulation of the active principle leads to generation of a wide array of toxic syndromes. The effects of toxicity of bracken fern is manifested by following syndrome in cattle as:

**Acute Bracken fern toxicosis or Hemorrhagic Disease**

Acute poisoning of bracken fern occurs by accidental heavy ingestion of bracken fern in a shorter span of time. The toxicity develops due to the active ptaquiloside’s action over bone marrow\(^3\). The radiomimetic action of the compound will lead to the damage of bone marrow and thus hampering in the synthesis of erythrocytes and leucocytes. The pathological findings revealed thrombocytopenia and granulocytopenia\(^4\). Urinalysis reveals hematuria and proteinuria.

Affected animals have an increased susceptibility to infection because of reduction in leucocytes and also have susceptibility towards spontaneous haemorrhage. Lower doses of bracken fern for longer period are likely to produce carcinogenesis. The role of bracken fern as a carcinogen and ptaquiloside has been confirmed in large animals as cattle and also in laboratory animals as in rats, mice, guinea pigs etc. The carcinogens are also going to secrete in milk and urine\(^5\)-\(^7\).

**Enzootic Haematuria**

Bracken fern produces enzootic hematuria commonly in cattle and less commonly in the sheep\(^8\). This condition is world widely reported in cattle species. For the development of toxicity the animals require the exposure for a longer period of time and the concentration of the toxicant must have to be higher. The disease is mainly characterized by pyrexia, petechial haemorrhages in the mucosal surface and large amount of clotted blood. The coagulation time of blood increases along with the passage of red coloured urine. The main cause of enzootic hematuria is nor-sesquiterpene glycoside (ptaquiloside) which causing damage to bone marrow stem cells or blood forming cells and causing aplastic anaemia. The hematuria also develops because of thiaminase activity of bracken fern. The thiaminase enzyme is not completely destroyed in situ.
by streaming process. Along with thiaminase an anti thiamine factor is also present in bracken fern plant.

**Chronic bracken fern toxicity**

Chronic exposure to bracken fern plant leads to the development of neoplasia in urinary bladder and urogenital system. This carcinogenic effect is due to nor-sesquiterpene glycoside ptaquiloside.\(^9\)

The clinical signs include:

- Red water urine with clotted blood in urine
- Thickening of urinary bladder wall
- Death usually follows but it is a lingering in contrast to the sudden death which as in acute form

**Bright Blindness**

A less pronounced manifestation of ptaquiloside toxicity is known as bright blindness or day blindness. It is characterised by the hyper reflect activity of tapetum. The animal becomes blind and develops alert attitude. Histological examination reveals severe atrophy of the retinal rod cells and cone cells. In sheep, particularly the toxic effect of bracken fern is due to progressive degeneration of retina that leads to the aforesaid day/ bright blindness condition.\(^10\)

**DIAGNOSIS AND TREATMENT**

Diagnosis is based on

- History of exposure
- Clinical signs as hematuria, fever and supportive blood tests confirm a non-regenerative anaemia and thrombocytopenia.
- Post-mortem examination: Post mortem examination reveals generally pale carcass, poorly clotted blood and widespread Petechial haemorrhage throughout the body.

**PREVENTION AND TREATMENT**

- Removal of the animal from the source of infection i.e. poisonous plant
- Treatment should be done on symptomatic basis
- Antipyretics and multivitamins should be injected as early as possible.
- If possible then, go for platelet and blood transfusion.

**REFERENCES**


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