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Case Report

Thiamine Responsive Polioencephalomalacia in Goats

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Abstract

Three adult goats were referred to the Department of Veterinary Medicine with a history of anorexia, dullness, attacks of head tremors, star gazing, convulsions and general weakness since past few days. The clinical signs varied from goat to goat. One goat was reared in intensive system being fed high concentrate diet and remaining two were reared in loose housing system being fed or having easy access to kitchen wastes rich in carbohydrates. Clinical examination revealed normal physiological parameters in all the three. All the cases were tentatively diagnosed as Polioencephalomalacia on the basis of typical clinical signs, and hence were treated with thiamine (vitamin B1) injection (20mg; intramuscularly), repeated hourly for first three doses and then continued for three days (BID). Treatment showed drastic response ranging from 1 to three hours with complete abolition of nervous signs, head tremors, convulsions and star-gazing posture. The response to initial treatment in case of one case (reared on intensive system) was partial and recovery was gradual and showed full recovery on day 3. The deficiency of B1 and subsequent underlying pathogenesis caused by its deficiency can be reversed by providing the enzyme immediately in the injectable form.

Keywords: Polioencephalomalacia; Vitamin B1; Ruminal Acidosis

Introduction

Thiamine (vitamin B1) is a water-soluble vitamin, not stored in the body, required on daily basis for carbohydrate and energy metabolism, and essential for the functioning of the heart, muscles and nervous system [1]. Polioencephalomalacia (PEM) also referred to as thiamine (Vitamin B1) deficiency, Goat Polio and cerebrocortical necrosis may be acute or sub-acute in nature affecting both young animals and adults. Polioencephalomalacia is a common neurologic disease of ruminants showing clinical signs related to dysfunction of the cerebrum such as wandering, incoordination, head pressing, circling, cortical blindness, recumbency, nystagmus, seizure activity and death in extreme cases. Ruminal lactic acidosis (grain engorgement, grain overload, rumen overload, toxic indigestion, and lactic acidosis) develop in ruminants fed on fermentable carbohydrates such as grain, bread, sugar and potatoes, left over kitchen waste rich in carbohydrates resulting in the production of large quantities of D-lactic acid in the rumen and change in the rumen microflora including proliferation of thiaminase-producing bacteria and thus resulting thiamine deficiency [2]. Clinical diagnosis is difficult and suspected based on the neurologic signs, differential diagnoses, and response to thiamine (vitamin B1) administration [3].

Case History and Observations

Case 1 (Reared under intensive care)

The goat was reared in intensive system and was being fed high concentrate diet. The goat was showing anorexia and nervous signs including head tremors (head shaking), head pressing and if made

to walk showing unilateral circular movements for few seconds and then going into recumbency. The animal was showing typical clinical signs including elevated head with continuous regular shaking movements, opisthotonos, dorsomedial strabismus, and tucked up body with wide hypermetric gait. The goat was showing normal bilateral pupillary light and menace reflexes. Physiological parameters were in normal range and ruminal pH was slightly acidic (5.6)

Case 2

The goat was presented in recumbent condition, unable to stand even when supported with a history of anorexia, emaciation from past few days. On clinical examination physiological parameters were normal and ruminal pH was 5.8. The animal was showing typical clinical signs including elevated head with continuous regular shaking movements, dorsomedial strabismus, opisthotonos, and occasional chewing gum movements of lips along with grinding of the teeth. The goat was showing normal bilateral pupillary light and menace reflexs.

Case 3 (8 months old)

The case was having history of malnutrition, undergrowth and showing typical periodic paddling movements of front legs while going into recumbency. On clinical examination physiological parameters were normal and ruminal pH was 6.2. Clinical findings included ataxia, unable to walk properly and when forced to walk showing hypermetric gait followed by recumbency. The head was held high and body showing tucked up appearance as seen in tetanus. The bilateral pupillary light and menace reflexes were normal.

Diagnosis

On the basis of typical clinical signs and differential diagnosis from other diseases showing nervous signs, and prompt response to thiamine administration all three cases were diagnosed as suffering from polioencephalomalacia due to vitamin B1 (thiamin) deficiency.

Treatment

All the three cases were tentatively diagnosed as Polioencephalomalacia on the basis of typical clinical signs, and hence were treated with thiamine (vitamin B1) injection (20mg; intramuscularly), repeated hourly for first three doses and then continued for three days (BID). Treatment showed drastic response ranging from 1 to three hours with complete abolition of nervous signs, head tremors, convulsions and star-gazing posture. The response to initial treatment in case of one case (reared on intensive system) was partial and recovery was gradual and on day 3 recovered fully.

Discussion

All three goats affected with vitamin B1 deficiency or Polioencephalomalacia showed response to B1 therapy and recovered fully. Thiamine (vitamin B1) a water-soluble vitamin, is essential for carbohydrate and energy metabolism, and normal functioning of the heart, muscles and nervous system [1] and its deficiency leads to cerebrocortical necrosis (Polioencephalomalacia-PEM, Goat Polio) that may be acute or sub-acute in nature. It is a common neurologic disease of ruminants leading to dysfunction of the cerebrum clinically manifested as wandering, incoordination, head pressing, circling, cortical blindness, recumbency, nystagmus, seizure activity and death in extreme cases. Ruminants with functional rumen microbes synthesize thiamine sufficient to meet the daily demands, however, deficiency of it has been reported due to various conditions such as ruminal fermentative disorders, ruminal acidosis, ruminal microbial thiaminase production, intake of inactive thiamine analogs, and consumption of excessive sulfur either from feedstuffs or water [4,5]. Ruminal lactic acidosis (grain engorgement, grain overload, rumen overload, toxic indigestion, and lactic acidosis) develop in ruminants fed on fermentable carbohydrates such as grain, bread, sugar and potatoes, left over kitchen waste rich in carbohydrates resulting in change in the rumen microflora including proliferation of thiaminase-producing bacteria and thus resulting its deficiency [6-8]. Clinical diagnosis of Polioencephalomalacia is difficult and is mainly based on the neurologic signs, differential diagnoses from diseases showing similar signs, and response to thiamine (vitamin B1) administration. The deficiency of B1 and subsequent underlying pathogenesis caused by its deficiency can be reversed by providing the enzyme immediately in the injectable form.

Conclusion

Ruminants with healthy rumen synthesize thiamine and its deficiency can occur due to ruminal acidosis, ruminal microbial thiaminase production, intake of inactive thiamine analogs, and consumption of excessive sulfur either from feedstuffs or water that result in neurologic disease leading to dysfunction of the cerebrum that can be reversed initially with treatment with thiamine (vitamin B1) early in the course of disease when clinical manifestation start to appear.

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