Clinical Indicators of Thiamine deficiency Induced Polioencephalomalacia (PEM) associated with coccidiosis in goat

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Abstract
Polioencephalomalacia (PEM), also known as cerebrocortical necrosis, is an important neurologic disease that affects ruminants. The correlation between between coccidia infection, feeding of ruminants with brewed products was examined and suspectedly associated with thiamine deficiency induced PEM. Thiamine deficiency have been well recognized as major etiological factors in PEM. The mechanism of thiamine deficiency associated PEM has been well elucidated. The role of coccidiosis in PEM pathogenesis is linked to the protozoan excessive utilization of thiamine in reproduction and the feeding of ruminants with high molases suppresses the activity of rumen microbes and hence the synthesis of thiamine. The development of clinical PEM is believed to be dependent of thiamine deficiency. This suggests a possible metabolic relationship involvement. Systemic failure to synthesize metabolically requisite levels of Thiamine in the rumen has been suspected in this report. There is increasing evidence of the importance of Thiamine deficiency, host parasite relationship and feeding regime in goats and pathogenesis of Thiamine deficiency induced PEM. This article examined the clinical inter-relationships between the factors mentioned above and PEM, and discuss the potential role of each in the pathogenesis PEM in goats.

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1. Introduction

Polioencephalomalacia (PEM), softening of grey matter, is an important neurological disease process that can affect many species of ruminants and contributes to substantial economic loss to livestock industry (De Santa et al., 2010; Robert et al., 1974). Animals of all ages can be affected but young animals appear to be more vulnerable (Niles et al., 2002; Rachid et al., 2011). Several risk factors such as thiamine deficiency, S toxicity, lead toxicity, and water deprivation-sodium ion toxicity have been implicated in the development of PEM.

All these factors produce similar brain lesions (Niles et al., 2002; Gould, 1998). Regardless of the suspected cause of PEM, affected animals frequently respond to thiamine administration (Olkowski, 1997). For this reason, it is commonly believed that thiamine deficiency is a major...
metabolic factor involved in the pathogenesis of PEM. However, the biochemical mechanisms of lesion development are still vague.

It has been suggested that the inhalation and absorption of eructated hydrogen sulfide (H2S) gas generated from the rumen and the combine presence of massive coccidia infection that utilises thiamine highly for reproduction and feeding of ruminants with high molasses that will eventually lower rumen PH and inhibit rumen microbial activity are major risk factors leading to PEM (Niles et al., 2002). However, to date, there is no convincing evidence to support the theory that the concentration of inhaled H2S from the rumen is high enough to induce PEM lesions. Furthermore, cattle affected by S-induced PEM frequently respond to thiamine treatment (Beke et al., 1991), and thiamine supplementation decreased the incidence and severity of S-induced PEM (Rousseaux et al., 1991). Hence the combine presence of massive coccidia oocysts and high carbohydrate diet can be clinically suggestive to be causes of thiamine deficiency in the case presented in this article. The objective of this article is to provide an overview of thiamine deficiency, massive coccidia oocysts and high carbohydrate diet associated PEM, and to discuss the potential role that each play in thiamine interaction in the pathogenesis of PEM in ruminants.

1.1 Objective of Report
This report examines the clinical correlation of massive coccidia infection, diet and PEM in a goat and draws a conclusion on the possibility of associating the risk factors and the disease.

1.2 Justification of Report
Clinical cases repeat themselves but in some cases you rarely see similar occurrences, this report is clinically pertinent and scientifically justifiable to report because of the case history and the clinical picture observed in the patient.

2. Case presentation
Doe of two and half years old kidded thrice with the recent being about two months ago. Prevailing clinical signs include, torticoli, unsteady gait (Plate 1), anorexia, lethargy, bilateral exophthalmos, opisthotonus, slight muscle spasm, easily pulled and rough hair coat. Animal is on intensive management system fed with grass and waste from brewery (molasses). All vital parameters on the day of presentation and subsequent days were within normal values, capillary refill time(CRT) less than 2secs and body condition score 2/5.

2.1 Diagnostic plan
Cervical radiograph, faecal analysis, blood sample in sterile heparinise bottle for haemoparasite screening, serum assay for thiamine using high performance liquid chromatography (HPLC) was recommended and complete blood count do be carried out.

2.2 Laboratory results
Right lateral cervical radiograph didn’t show any sign of trauma (Plate 2), no haemoparasite observe from blood screened, massive coccidia oocysts (+++++) was observe from faecal egg analysis. Red cell was normocytic normochromic, and haematological values fall within normal range.

2.3 Treatment and Management
Base on clinical assessment and prevailing condition of the animal, the following treatment was prescribed: Toltrazuril 25mg/kg body weight per-os X 3/7, Dexamethasone inj 5mg/kg i/m stat. Vitamin B-complex inj 1/m 40mg/kgX5/7 (Dosage in this case was raised inorder to provide Thiamine as fortified thiamine was not readily available), 50% Destrose(150ml) i/v stat.

3. Discussion
Thiamine deficiency induced PEM has been reported in cattle, sheep, horses, dogs (Rammell et al., 1986), goats (Sakhaee et al., 2010), camels (Milad et al., 2009), and cats (Palus et al., 2010). Thiamine deficiency in ruminants has been associated with several factors such as an impairment of microbial thiamine synthesis, thiamine destroying activity of bacterial thiaminase, along with other dietary factors involved in thiamine destroying activity in the rumen (Brent et al., 1984). Bacterial thiaminase has been considered the main factor leading to thiamine deficiency in ruminants. Two types of thiaminase (Type I and II) are produced by different types of ruminant bacteria. Both types have a destructive effect on thiamine in the rumen. Thiaminase type I catalyzes the nucleophilic displacement of the thiazole moiety of thiamine by another base known as a co-substrate and generates thiamine analogues that inhibit thiamine dependent reactions. Thiaminase type I requires a co-factor to accomplish its thiamine destroying activity (Cebra et al., 2004). Some medications such as promazines and levamisole along with substrates produced during fermentation appear to act as cofactor to thiaminase type I (Cebra et al., 2004). Thiaminase type I is also present in plants such as bracken fern, horsetail and nar do ferns (Rachid et al., 2011). Animals exposed to these plants have subsequently developed PEM (Ramos et al., 2005). Thiaminase type II splits thiamine by catalyzing the hydrolysis process and thereby may reduce the amount of thiamine absorbed from rumen (Murata 1982).
Several outbreaks of PEM in sheep and cattle with high thiaminase activity in the rumen have been reported (Robert et al., 1974; Edwin et al., 1970). Amprolium, a potent coccidiostat and thiamine analogue, is believed to be another major factor associated with PEM. It inhibits the conversion of free-base thiamine to thiamine pyrophosphate (TPP), thereby depriving tissues (especially brain) of TPP (Cebra et al., 2004; Loew and Dunlop, 1972). (Thornber et al., 1979) induced PEM in lambs by feeding a thiamine free diet with high levels of amprolium (280 mg/kg of BW). As well, oral administration of amprolium leads to a reduction of blood and tissue thiamine levels and subsequent development of PEM in calves (Kasahara et al., 1989).

In the management of the massive coccidiosis encountered in this case, toltrazuril was employed because it is sulfa-free. However, clinical and histopathological lesions indicative of thiamine deficiency have been produced in pre-ruminant lambs by feeding a thiamine free artificial milk diet (Thornber et al., 1980).

Other factors, such as production of inactive or poorly absorbed forms of thiamine in the rumen, or inhibition of phosphorylation and absorption may also contribute to functional thiamine deficiency (TPP deficiency), subsequently leading to malacic lesions and clinically massive presence of coccidia oocysts couple with dietary feeding of ruminants with molasses to ruminants can potentiate Thiamine induced PEM. Cebra et al., (2004) which in this case is the likely cause of the disorder.

**Figure 1:** Torticolis on day of presentation

**Figure 2:** Cervical Radiograph

**Conclusion**

Polioencephalomalacia and thiamine deficiency are associated, in whatsoever way the deficiency occur the sequelae may present as PEM. Base on clinical assessment of the case reported, massive/heavy coccidiosis infection that utilises thiamine immensely for reproduction, feeding of ruminants with brewery byproduct (Molases) that inhibits the activity of the rumen microbes that synthesise Vitamin B inclusive of thiamine and the clinical signs of torticolis, unsteady gait, anorexia, lethargy, bilateral exophthalmos, opisthotonus, slight muscle spasm, we conclude that the PEM in this case is associated coccidiosis in goat.

**Recommendations**

Ruminants should not be fed brewed by-products that have the potential to inhibit the function of the rumen microflora. To limit coccidiosis, animals should be treated prophylactically using sulfa free anticoccidia drugs e.g toltrazuril. Suspected cases similar to the one reported in this article should be reported to the veterinarian immediately to minimise mortality and complications.

**Case Highlights**

Polioencephalomalacia can occur in goats, thiamine deficiency is a risk factor to PEM and is a major cause of the disease. Massive/heavy coccidiosis infection feeding of ruminants with Molases are associated with PEM because both causes thiamine deficiency. Clinical signs of torticolis, unsteady gait, anorexia, lethargy, bilateral exophthalmos,
opisthotonus, slight muscle spasm, are observed in goats that suffer from PEM.

Limitations

Laboratory analyses of blood thiamine level was very expensive and samples collected has to be analyse within 72 hours of collection. The same analysis is very costly and the laboratory that could run the assay was not close to our location that is why the thiamine assay was not carried out.

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Author’s Contribution and Competing Interests

We declare there is no competing interest.

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