

## Case Report of Polioencephalomalacia due to high concentrate intake, high sulfur intake and fungi and bacterial contamination feed in dairy cattle

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**Abstract**—Polioencephalomalacia (PEM) is seen sporadically or as a herd outbreak. Cattle on high concentrate diets are higher risk. In this case report, PEM occurred due to high concentrate intake, and contamination concentrate to some bacterial and fungus producer sulfur that rotationally grazed dairy cattle consuming concentrate containing 1.08% sulfate. Clinical signs, autopsy findings, examination of diet concentrate and high concentrate sulfate levels confirmed the diagnosis of sulfur-induced Polioencephalomalacia. The incidence of disease reduced when the herd was switched to a low intake of concentrate level.

**Keyword:** Polioencephalomalacia, concentrate, sulfur, dairy cattle

### I. INTRODUCTION

Polioencephalomalacia (PEM) is an important neurologic disease of ruminants that is seen worldwide. The disease is seen sporadically or as a herd outbreak. Cattle on high concentrate diets are higher risk. Cattle fed ration with added sulfate to limit intake or with byproducts of corn or sugar cane processing are at higher risk. PEM has been associated with altered thiamine status, but more recently an association with high sulfur intake has been observed. A potential mechanism of thiamine inadequacy is the action of thiaminase on thiamine in the GI tract. In ruminants,

thiamine is produced by ruminal bacteria and protozoa under normal environmental conditions. Thiamine deficiency is related to overeating, acute impaction, grain engorgement, founder and grain overload. Acidosis can promote proliferation of thiaminase II producing bacteria (*C. sporogenes* and *Bacillus* sp.) This enzyme destroys thiamine, producing a thiamine analog that inhibits thiamine-dependent reactions of glycolysis and decarboxylations. It has been known that thiamin analogs in the presence of a cosubstrate are responsible for PEM development. PEM can also be caused by thiaminase I. Several drugs including promazines, levamisole, benzimidazoles act as a cofactor to thiaminase I. This enzyme is also present in different plants such as horsetail (*Equisetum arvense*), bracken fern (*Pteridium aquilinum*), prostrate pigweed (*Amaranthus blitoides*), small flowered mallow (*Malva parviflora*) (Main kikuyu grass (*Pennisetum clandestinum*), *Medicago sativa* and *Nardoo fern* (*Marsilea drummondii*). The excess of sulfur in diet is one of the related causes of sulfur-associated PEM (1). The major dietary sulfur sources are alfalfa hay, molasses, beet pulp, barley Malt sprouts, calcium sulphate, ammonium sulphate, sodium sulphate and grain-processing products (corn gluten meal, brewer's grain). The recommended maximum rate of sulfur on diet is 0.5% for cattle eating more than 40% forage. Younger beef cattle and lactating cows seem to be more susceptible to excess sulfur

intake; diets with high concentrations of sulfate has been reproduced PEM. It was demonstrated that the onset of clinical signs presents in animals with sulfur-related PEM coincided with excessive ruminal sulfide production. The sulfite produced during the reduction of sulfate to sulfide cleaves thiamine at the methylene bridge; thus, high sulfide levels could cause the brain lesions associated with PEM [1, 2].

## II. CASE REPORT:

### *Type of diet*

This case report was resulted from Holstein's dairy cattle farms in Esfahan province in Iran at June 2010. This farm was with the capacity of 54 (14 lactational) Holstein dairy cattle. Concentrate of formulation was according below table:

Table 1: The diets concentrate using in dairy herd

<i>Materials</i>	<i>Barley</i>	<i>Wheat bran</i>	<i>Cotton seed meal</i>	<i>Soybean</i>	<i>Corn</i>	<i>Supplement</i>	<i>Sodium bicarbonate</i>	<i>Salt</i>	<i>DCP</i>	<i>Total</i>
<i>Kg</i>	370	215	170	50	150	20	10	1	10	1000

Level of consumption was 10 kg/ cow in lactation cattle that dairyman was increased rate of concentrate from 100 kg (50 kg morning/ 50 kg evening) to 200 kg (100 kg morning/ 100 kg evening) after three week, because for milk yield has been increased. After 7 days from time-consume of concentrate 200 kg, lactation cows were illness and downer.

### *Clinical sing:*

All of lactational cows has been depression, ataxia, lethargy, hypermetric (39.2-39.5), sternal recumbency, muscular incoordination, nystagmus, paralysis of hind lame. Tongue was tonic and it wasn't problem in mastication. Ruminal motion was 1/min and fecal was normal. Rate of breath was 40/min and only one of case have 80/min. Nose, larynx and lung were normal. Rate of heart was between 72-84/min and only one of case have 96/min with sever nonrhythmic arrhythmia. All of them has vocalization for unable to standing. Clinical examination of the 2 sick females revealed tremors of the head and neck, and profuse slobbering. There were menace reflexes and the palpebral reflexes were slowed. Sample blood (10 ml) was collected from all cows and there were evaluated for CBC; levels of

Ca, P, Mg, Na and K. In the blood tables, blood parameters were normal and but there has monocytosis (12-21%). Examination of the herd showed that the majority of the mature females were sluggish and lethargic. The calves appeared unaffected. The sickest cow was slaughtered and total organs such as thoracic cavity, brain, spinal cord were exanimate. Although macroscopic lesions were not apparent in the brain tissues of some animals, histopathology typical of PEM was found in most cases: spongiosis in the neuropil and neuronal necrosis, haemorrhage, capillary hyperplasia, fibrinoid degeneration in arterioles, multifocal liquefaction necroses in the grey matter and abundance of gitter cells with vacuolar large cytoplasm. So, CSF was increased, the brain has an edema and it was flaccid. Clinical examination was observed B<sub>1</sub> deficiency due to high concentrate intake in total cows. After sampling of diet concentrate, the results of culture were indicated that more high in the rate of *C. sporogenes* and *Bacillus* spp bacterial and *Rhizopus oryzae*, *Mucor* spp, *Cladosporium* spp and yeast fungus. Also, diet concentrate samples were collected to test for high sulfur levels (1.08%). The final diagnosis in this outbreak was sulfur-induced PEM due to high concentrate intake and

contamination of diet to bacterial and fungus producing sulfur [1, 2, and 3].

These cows were treated with thiamine (Thiamine HCl; Vétoquinol, Lavaltrie, Quebec), 10 mg/kg bodyweight (BW), IM, each 3 hourly/one dose until to 3 time administration, and following treatment is dependent to animals reflex. Also, these were injected Ca, Mg, and P with fluid therapy. For reduction of cerebral edema were administrated of dexamethasone (Aburaihan Farm Co.) at a dosage of 0.1 mg/kg, IM, three days (In the non pregnant cows), and phenylbutason 20% (vetanyle 20% Aburaihan Farm Co.) (In the pregnant cow) [1, 2].

The cows herd was supplied with low concentrate. There were no more clinically ill animals or death losses, and the herd returned to a normal activity level [1].

This report demonstrates that high intake of concentrate and contamination concentrates to some bacterial and fungus derived to produce sulfur. Overall, sulfur was induced PEM. However, weekly or monthly concentrate evaluation of all sources a cattle herd may supply from can be a crucial monitor of herd health [2].

#### REFERENCE

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