

## Polioencephalomalacia in goats: A case study

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### Abstract

In the month of October, twenty two goats of age group 2-8 years at farm showed sudden paralysis of hind limbs soon after grazing. Goats exhibited excitability, head pressing, circling movements, and muscular tremors with weakness of all extremities. Goats were unable to stand without support and stood either with right or left side of their body leaning against wall. On the basis of clinical examination, herd history, post mortem findings and differential diagnosis, the case was considered as a thiamine deficiency. Treatment with Thiamine (at 10-20 mg per kg body weight I/V or I/M), Dexamethasone (at 1-2 mg per kg body weight I/M), Procaine Penicillin (at 10000 IU per kg body weight I/M, Inj. DNS (Dextrose Normal Saline) 500 ml I/V and other supportive therapy was done. Out of twenty two affected goats, nine responded earlier and recovered completely in 3-4 doses of treatment. The twelve goats showed delayed recovery i.e. about after 7 days of therapy. One goat didn't respond well to treatment and died. Among the nine early responded goats, condition reoccurred in the two goats which died within 4-5 days. Thus in the present case, recovery has been achieved in less affected goats and those who have been treated at early stage of the disease, though recurrence has also been found in some goats.

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### Introduction

Thiamine (Vitamin B<sub>1</sub>) is naturally produced in the rumen of the goat by the ruminal microorganisms and so as a dietary supplement, is not required with proper functioning rumen (Brent and Bartley, 1984). However under certain conditions thiamine production may decrease and/or available thiamine may be destroyed leading to its deficiency. Thiamine deficiency or "Polioencephalomalacia (PEM)" or "Goat Polio" or "Cerebrocortical Necrosis", is a common metabolic disorder characterized by neuro-muscular alterations in the thiamine deficient of goats. This disorder may be acute or sub-acute in nature. Adult and young animals are equally at high risk for developing the disorder. Goat polio is usually seen in animals that are under high nutritional management condition such as feedlots, or animals on lush pasturage fed with highly concentrated rations (Dana *et al.*, 2010). Like all B vitamins, B<sub>1</sub> is a water soluble vitamin and is a major part of the several enzymes for carbohydrates and amino acid metabolism in the rumen as well as the nervous system and muscles. Thus, thiamine

deficiency causes nerve weakness and partial paralysis of the legs.

### Case History

In the month of October, twenty two goats of age group 2-8 years at farm showed sudden paralysis of hind limbs soon after grazing. In the first incidence, three goats showed the symptoms and after an interval of 3-4 days, the other goats in the group of 4-5 each, also started showing the same symptoms. Goats exhibited excitability, head pressing, circling movements, and muscular tremors with weakness of all extremities. Goats were unable to stand without support and stood either with right or left side of their body leaning against wall (Fig 1). Some of the goats were having blindness, became recumbent with opisthotonus posture and started paddling movements (Fig 2).

### Clinical Examination

On clinical examination temperature, respiratory rate and pulse rate were found in normal range. Rumen motility was decreased to 1 per 2 minutes. In later

stages, goats became anorectic and depressed. Corneal opacity was observed in the blind goat. In addition to above symptoms, some recumbent goats exhibited extensor rigidity i.e. kept limbs in extending position. Haematological values were within normal range. No blood parasites (*Trypanosome* spp.) were detected in blood smear.



Fig 1: Goat standing with left side of body leaning against wall



Fig 2: Goat having weakness of all extremities, blindness and opisthotonus posture

### Diagnosis

On the basis of symptoms, thiamine deficiency, tetanus, listeriosis, enterotoxemia, blood parasitic infestation and pregnancy toxemia were suspected. Diagnosis was based on clinical examination, herd history, feeding records, necropsy finding, differential diagnosis and response to treatment. In differential diagnosis, the related diseases affecting neuromuscular system were excluded. Absence of third eyelid flashing across and absence of lock-jaw ruled out tetanus. Absence of fever, presence of blindness and/or

extensor rigidity and absence of kidding ruled out listeriosis, enterotoxemia and pregnancy toxemia respectively. Negative blood smear report excluded any blood parasitic infestation. If case is considered as thiamine deficiency, the main cause may be either thiamine production failure and/or thiamine inactivity. Hence it could be due to sudden feed changes in diets, mouldy hay, stress, deworming with anthelmintics, and overdoses of some anticoccidial (amprolium) medications and high sulphur intake. An increase of *Clostridium* spp. in rumen after a high carbohydrate diet and/or intake of some types of ferns or weeds that inhibit processing of B<sub>1</sub> due to thiaminase induces thiamine deficiency (Wallace *et al.*, 2000).

Thiamine plays a major role in metabolism of carbohydrates in nervous system and muscles. Thus lack of thiamine induces a lower supply of carbohydrates to the neurons in the brain. As neurons require carbohydrates as an energy source necessary for nerve function, the depletion of carbohydrates causes alterations in the mechanism of action of the nervous system and ultimately neuronal death especially cortical region (Cushnie *et al.*, 1979). Hence damage to the brain cells may be responsible for origination of the symptoms.

According to the feeding records, all goats in herd were allowed to graze on lush green forage. Apart from grazing, goats were fed with concentrate mixture of maize and mustard cake and gram straw as roughage. Hence, the possible cause for generating symptoms might be lush pasture, concentrate (high carbohydrate) and intake of weeds and ferns during grazing. Another contributing factor might be the low levels of thiamine in the soil that drained out during rainy season. Thus inactivity and non-availability of thiamine were considered to be responsible for creating the condition. Symptoms shown by affected goats were similar as described by Pugh (1993) and Bulgin *et al.* (1996). At necropsy, yellowish dis-colouration of the dorsal cerebral cortex especially the occipital lobes was found, indicating necrosis. Necropsy findings were in accordance with (Tanwar, 1987). On the basis of clinical examination, herd history, necropsy findings, differential diagnosis and correlation with possible causes, this can be considered as a case of thiamine deficiency.

### Treatment

The deficiency of thiamine caused death of millions of brain cells, so quick treatment was imperative. All the affected goats were isolated from rest of the healthy herd and were provided treatment promptly. Treatment included the therapy for support of the neurological system, restoring natural microflora

balance of rumen, and supportive therapy for hydration and nutrition (Newsholme and O'Neill, 1985).

- Thiamine at 10-20 mg per kg body weight i.e. Inj. Tribivet (Thiamine hydrochloride 50 mg/ml) was given upto 10-15 ml intravenously depending upon severity and body weight BID for 2 doses and thereafter BID intramuscularly till recovery.
- Dexamethasone at 1-2 mg per kg body weight I/M- Inj. Dexona-vet 4-5 ml I/M SID for 5 days.
- Procaine Penicillin at 10000 IU per kg body weight I/M- Inj. Dicrysticin 4-5 ml I/M SID for 3 days, since this is the drug of choice for clostridia species.
- Inj. Intalylate 150 ml I/V for 5 days.
- Bolus Provisacc – Half bolus BID orally.
- Mineral mixture- Minfa Gold 15 grams orally.
- Feed comprising high roughage and low concentrate.
- To treat blindness (corneal opacity)
  - a. Inj. Gentamicin and Inj. Dexamethasone 0.25 ml each subconjunctivally on alternate days for 7 doses
  - b. Inj. Vitacept 2 ml I/M at 3 days interval for 6 doses was given

Treatment was continued further for 24 hours from complete recovery to prevent reoccurrence.

## Results and Discussion

Out of twenty two affected goats, nine responded earlier and recovered completely in 3-4 doses of treatment. The twelve, including blind goats showed delayed recovery i.e. about 7 days of therapy. Although the neurological symptoms of blind goats had been disappeared in a week but, the corneal opacity took another two consecutive weeks to recover. Out of delayed recovered goats, one showing knuckling of fetlock joint was treated by applying “Thomas splint” (Fig 3). One goat didn't respond well to treatment and the condition worsened from weakness to complete paralysis and ultimately death occurred after 8-9 days from onset of symptoms. Among the nine early responded goats condition reoccurred in the two goats after four days of recovery. The goats died within 4-5 days, although the same treatment was provided as before. Thus in the present case, recovery has been achieved with the given treatment to the affected goats. But some cases of reoccurrence of disease were also noticed.

In the present case death of one goat emphasized the need of early detection and treatment for polioencephalomalacia. Sometimes the only way to

make a quick diagnosis is through a response to treatment. Early polio cases often respond, at least partially if not completely to thiamine administration. Often some response occurs within a few hours of initial treatment. Most other cases respond slowly or not at all to indicated treatments. Because thiamine deficiency does cause brain necrosis however, time is important.



Fig 3: “Thomas splint” to treat fetlock knuckling

The longer treatment is delayed, the more likely irreversible brain damage may occur (Smith, 1979). Hence treatment should be started from the very first hour of symptoms appearance rather than waiting for laboratory findings. Apart from treatment, Polioencephalomalacia associated with thiamine deficiency in goat may be prevented by a strict feeding management. A supplementation of thiamine in the goat feed may decrease the chances of the occurrence the disease. Prevention should also include avoidance of sudden dietary changes to energy dense feedstuffs. Along with it the availability of sufficient and good quality of roughage, free access to trace minerals, thiamine supplementation in animal feed and monitoring animals feed and water for sulfates may prevent polioencephalomalacia (Rankins *et al.*, 2002).

## Conclusion

It can be concluded that treatment in case of thiamine deficiency should be start immediately after the very first hour of symptoms appearance rather than waiting for laboratory findings. The recovery has been achieved with the given treatment to the affected goats, however death of one goat emphasized the need of early detection and treatment for polioencephalomalacia. Further, the few cases of reoccurrence of disease were also noticed and this require carefully during treatment.

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