A Clinical report on Polioencephalomalacia in Goats

A Clinical Report Presented in Partial Satisfaction of the Requirements for the Degree of

Doctor of Veterinary Medicine (DVM)

By

.....

Sakib Shariar

Roll No.: 15/24

Reg. No.: 01439

Intern ID.: 22

Session: 2014-15

Faculty of Veterinary Medicine

Chattogram Veterinary and Animal Sciences University

Khulshi, Chattogram-4225, Bangladesh

A Clinical report on Polioencephalomalacia in Goats

A Clinical Report Submitted as per Approved Styles and Contents

Approved by:

.....

Dr. Tuli Dey

Assistant Professor

Department of Medicine and Surgery

Faculty of Veterinary Medicine

Chattogram Veterinary and Animal Sciences University

Khulshi, Chattogram-4225, Bangladesh

TABLE OF CONTENTS

List of figures

List of Abbreviations

	Abbreviations And Symbol	Elaboration
1	CVASU	Chattogram Veterinary and Animal Sciences University
2	Mg	Milligram
3	Kg	Kilogram
4	PEM	Polioencephalomalacia
5	pH	Potential of Hydrogen
6	Iv	Intravenous

Abstract

This study was planned to diagnose polioencephalomalacia and its response to thiamine treatment. In the month of October 2019 five goats were brought to Upazilla Livestock Office and Veterinary Hospital, Sadar, Mymensingh with the neurological signs including staggering gait, incoordination, torticollis, nystagmus and apparent blindness. The disease was diagnosed as polioencephalomalacia on the basis of clinical examination, herd history and differential diagnosis.Treatment was provided with thiamine @10mg/kg body weight intravenously, dexamethasone @1mg/kg body weight intramuscularly, dextrose saline intravenously and other supportive treatment daily until recovery. One of the goats died and four goats completely recovered within two weeks. Immediate treatment of polioencephalomalacia cases with thiamine was essential for better recovery.

Keywords: Polioencephalomalacia, incoordination, torticollis, thiamine**.**

Chapter-1

Introduction

Livestock is an essential part of the economy of Bangladesh. There are many well established livestock farms as well as backyard farming of livestock that play an important role in poverty elevation. Especially small ruminants help many poor people in maintaining their livelihood. Polioencephalomalacia in ruminants may cause a huge economical loss for the farmers.

Polioencephalomalacia (PEM), which soften the grey matter of brain, is an important neurological disease process that could affect many species of ruminants and contributes to substantial economic loss to livestock industry (De Sant'Ana*et al.*,2010). The disease causes necrosis of the cerebral cortex of brain and also called as cerebrocortical necrosis (Roberts and Boyd, 1974).

The disease is non-infectious in nature and responsible for neurological disorder of ruminants (Debasis and Ghosh, 2009). It is mostly 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). The disease occurs due to disturbance in thiamine absorption and metabolism (Rachid*et al.*, 2011).

Thiamine (Vitamin B1) 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. Like all B vitamins, B1 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 paralysisof the legs. This disorder may be acute or sub-acute in nature. Animals of all ages could be affected but young animals appeared to be more vulnerable.

Studies have been done on diagnosis of polioencephalomalacia in goats and its response to thiamine treatment (Ramella and Hill, 1986; Rachid et all. 2011). But few studies were documented on polioencephalomalacia in goat in Bangladesh. Here, the study was conducted to diagnose polioencephalomalacia in goat and its response to thiamine treatment at Upazilla Livestock Office and Veterinary Hospital, Sadar, Mymensingh, Bangladesh.

Objectives:

1. Diagnosis of polioencephalomalacia in goat.
2. Assessment of thiamine treatment for polioencephalomalacia in goat.

Chapter-2

Materials and methods

2.1 Case History:

In the month of October 2019, five goats of 1year to 1.5years of age were presented in Upazilla Livestock Office and Veterinary Hospital, Sadar, Mymensingh with the owner's complaint of lameness and blindness. Goats were unable to stand without support and stood either with right or left side of their body. Some of the goats were having blindness problem, became recumbent with opisthotonus posture and started paddling movements.

2.2 Clinical Examination:

On clinical examination body temperature was found normal in two goats and subnormal in other three goats. Respiratory rate was normal. Rumen motility was taken using fist method and pH was measured using pH roll. Rumen motility was 1-2 per minute and rumen pH was slightly acidic. Appetite was normal but later become anorexic and depressed. Corneal opacity was observed in case of blind goats. Clinical signs included staggering gait, incoordination, torticollis, nystagmus and apparent blindness. Defecation and micturation was normal. Feeding history of the goats were taken which were mainly concentrate(rice) and grass.

2.3 Diagnosis:

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

Therefore, based on clinical examination, herd history, differential diagnosis and correlation with possible causes, this can be considered as a case of thiamine deficiency.

2.4 Treatment:

The treatment protocol followed in this case was thiamine @10mg/kg body weight intravenously, dexamethasone @1mg/kg body weight intramuscularly, dextrose saline and other supportive treatment until recovery. In case of blindness ciprofloxacin along with dexamethasone eye drops were used for 5 days.

Figures

Figure 1: Lateral recumbancy

Figure 2: Blindness and Nystagmus

Figure 3: Torticollis

Figure 4: IV administration of Thiamine and Dextrose

Chapter-3

Results and Discussions

After diagnosis treatment was started immediately and continued up to recovery. Within 7-9 days 4 goats(80%) started recovering and 1 died(20%).Within day 14 all the 4 goats with the signs of PEM recovered completely.

The death of one goat found in this study may be due to delayed treatment. Which implies that treatment should be given as soon as possible upon early diagnosis of PEM for better recovery. Response to treatment may sometimes be a quick way to diagnose the disease. Early polio cases often respond, at least partially if not completely to thiamine administration. Response to thiamine treatment may differ from individual to individual based on the level of thiamine deficiency. Some may response within a few hours of initial treatment while other cases may respond slowly or not at all to indicated treatments. Because the deficiency thiamine causes brain necrosis and time plays an important role here. The longer the 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.

Thiamine deficiency may occur due to two main reasons, either failure of thiamine production and/or thiamine inactivity. It could be due to sudden changes offeed in diets as the ration of the goats contained rice in it which might be a predominant cause. One study found that after a high carbohydrate diet an increase of *Clostridium spp.* in rumen and/or intake of some types of ferns or weeds that inhibit processing of B1 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). Lower levels of thiamine would promote a lower supply of carbohydrates to the nerve cells, leading to central nervous disorders polioencephalomalacia and death, (De Sant'Ana*et al., loc. cit*).Hence damage to the brain cells may be responsible for origination of the symptoms.

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).

Chapter-4

Conclusions

Treatment in case of thiamine deficiency should be started immediately after the very first hour of symptoms appearance rather than waiting for laboratory findings. Response to treatment may be considered as a quick tool to diagnose the disease.The death of one goat in this study strongly emphasize the importance of early diagnosis and treatment for polioencephalomalacia. Treatment with thiamine intravenously showed affective response in recovery of the PEM affected patients. Polioencephalomalacia can be prevented by increasing the availability of sufficient and good quality roughage, reducing high amount of carbohydrate in feed.

Acknowledgements

All praises are due to the Almighty, the creator and supreme authority of the universe, have enabled the author to accomplish this work.

The author would like to express his deep sense of gratitude and thanks to his supervisor **Dr. Tuli Dey**, Assistant Professor, Department of Medicine and Surgery, Chattogram Veterinary and Animal Sciences University, whose encouragement and support helped for the completion of the report.

The author also wishes to give his gratitude to **Professor Dr. Abdul Ahad,** Dean, Faculty of Veterinary Medicine, **Professor Dr. A. K. M. Saifuddin,** Director of External affairs, Chattogram Veterinary and Animal Sciences University.

The Author.

References

Brent BE and Bartley EE (1984): Thiamin and niacin in the rumen. *Journal of Animal Science*, 59(3): 813-822.

Cushnie GH, Richardson AJ, Lawson WJ and Sharman GA (1979): Cerebrocortical necrosis in ruminants:Effect of thiaminase type 1-producing *Clostridiumsporogenes*in lambs. *Veterinary Record*, 105(21):480-482.

Dana GA, Peter DC, Peter RD, Katherine EQ, Philip TR, Jagdev MS, Roger KWS and Tracee T (2010): Polioencephalomalacia: Introduction (cerebrocortical necrosis). *The Merck Veterinarian Manual.Whitehouse Station, NJ: Merck and Co., Inc*. 10th ed. Pp 1174-1177.

Debasis, J. and Ghosh, M. (2009): Management of polioencephalomalacia in a buffalo heifer – A case report. *North eastVeterinarian*, 11 (2): 11-13.

De Sant'Ana, Fabiano, J.F. and Barros, C.S.L. (2010): Polioencephalomalacia in ruminants in Brazil.*Braz J Vet Pathology*., 3:70.

Rachid, M.A., Filho, E.F., Carvalho, A.U., Vasconwelos, A.C. and Ferreira, P.M. (2011):Polioencephalomalacia in cattle. *Asian J. Anim. Vet. Adv*.,6(2): 126-131.

Rammell, C.G. and Hill, J.H. (1986): A review of thiamine deficiency and its diagnosis, especially in Ruminants. *New ZealandVeterinary Journal.*,34:202.

Rankins DC, Ruffin DC and Pugh DG (2002): Feedingand Nutrition. Sheep and goat medicine. *D. G. Pugh ed. Philadelphia. W. B. Saunders.* Pp 19-60.

Roberts, G.W. and Boyd, J.W. (1974):Cerebrocortical necrosis in ruminants. Occurrence of thiaminase in the gut of normal and affected animals and its effect on thiamine status. *J Comp. Pathol*., 84:365.

Smith MC (1979): Polioencephalomalacia in goats. *Journal of the American Veterinary MedicalAssociation*, 174(12): 1328-1332.

Wallace RJ, Wallace SJ and McKain N (2000): Proteolytic activity of ruminaldigesta during the feeding cycle in sheep receiving grass hay/concentrate or maize silage/concentrate diets. *Letters in Applied Microbiology*, 30(4): 317-319.