

Case Report of Encephalopathy in Goats

Case Report

Gholami^{*1} M.R., Hablolvarid, M.H.,¹ Bazargani, T.² and Ezzi, A.¹

1. Pathology Dept., Razi Vaccine & Serum Research Institute, P.O.Box 11365-1558,
Tehran, Iran

2. Faculty of Veterinary Medicine, Tehran University, Tehran, Iran

Received 23 Aug 2002; accepted 3 Mar 2003

Polioencephalomalacia is none infectious neuro degenerative disease that affect sheep, goat, deer and cow. Polioencephalomalacia commonly occur in cattle fed ration rich in carbohydrates with little roughage that may changes microflora of rumen and impaired synthesize of thiamine (Thomson *et al* 2001, Boyd & Walton 1977). Polioencephalomalacia could represent a multifactoral metabolic disorder with multiple etiologies such as nutritional problem, high level of carbohydrate, water deprivation, salt intoxication, high level intake of sulfur, renal encephalopathy, amprolium and some plant such as Bracken fern and Horsetail. Probable mechanisms that may cause thiamine deficiency polioencephalomalacia in ruminant are disorder of absorption, synthesize and destruction of thiamine by thiaminase (Radostit *et al* 1994, Bakker *et al* 1980, Jeffrey *et al* 1994, Loew & Dunlop 1972, Olkowski 1992, Thornber *et al* 1979). Evidence (or theories) linking thiamine with the ruminant polioencephalomalacia disorder include clinical response to thiamine injection in some individual (Thomson *et al* 2001).

Case history

During 70 days 17of 115 sanan bread goats were affected and nine of them died. Clinical signs of few cases on primary stage of disease were reversed by injection of thiamine and general medicinal and nutritional cares but 6 cases were submitted in

* Author for correspondence. E-mail:dr_mr_Gholami@yahoo.com

Pathology department of Razi Institute. Clinical signs such as changes of behavior with elevation and deviation of head (star gazing) and tail cocked up over the back and forward rotation of ears were seen (Figure 1). Nibbling movements of lips, incoordination and ataxia, abduction of legs and hands, weakness and in end stage extreme emaciation and inability to move, sternal and lateral recumbence with extension of limbs, and some times deviation of head towards flank were also observed. In one case severe skin lesions on shoulder and another one blindness were seen (Figure 2).

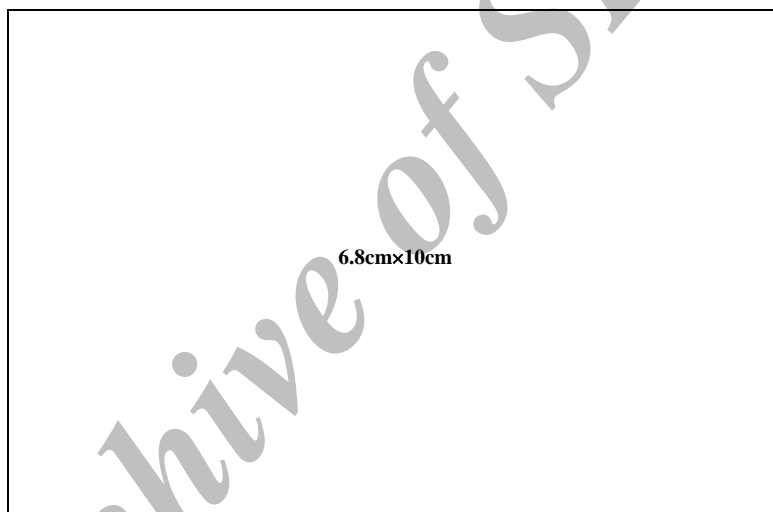


Figure 1. Elevation of head, neck and tail

Pathology

Postmortem finding of internal organs showed pneumonia of apical lob in one case. Samples of lung, liver, mesenteric lymph node, intestine, kidney and eye were taken and fixed in 10% formalin for histopathological examination. Different parts of brain and spinal cord were cut into slices and fixed in 10% formalin. Sections from paraffin embedded blocks were cut at 5 μ and stained by hematoxylin and eosin (H&E) and luxal fast blue. Bacterial examination of brain and blood was negative.

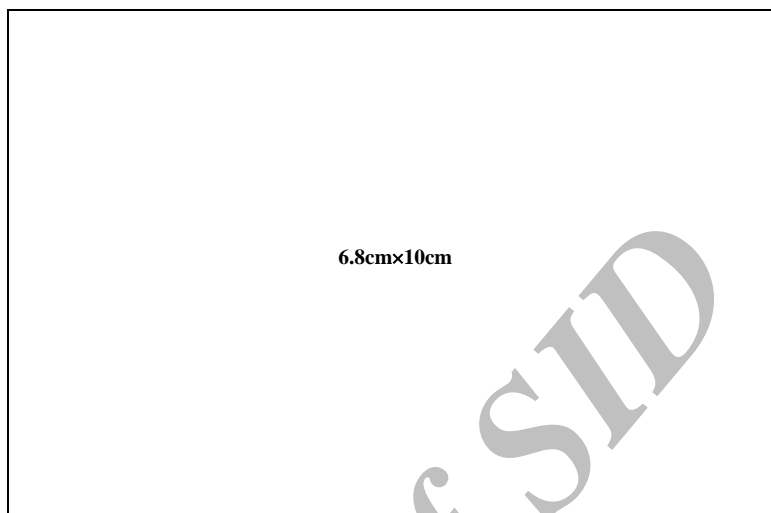


Figure 2. *Abduction of limb, sternal recumbence with extension of limbs and severe ulcer in shoulder*

In one case purulent bronchopneumonia was seen. The eyes of blind goat revealed mild uveitis and retinitis. Histopathological examination consistently revealed with lesions in the central nervous system. General lesions in brain are various forms of neuronal degeneration such as chromatolysis, pyknosis, necrosis and neuronal loss and astrogliosis reaction in some area, vacuolation of neuropil and increased perineuronal and periaxonal space. In cerebral cortex neuronal degeneration (hyperchromatic and shrunken) occasionally vacuolation of perikarya, gliosis and perineuronal satellitosis were seen. In some sections especially near central girus, severe increased perineuronal space was prominent. In cerebellum, hyaline eosinophilic degeneration of purkinje cells, severe and diffuse vacuolation of granulare layer and many purkinje cells were lost. In thalamus area neuronal degeneration and vacuolation of neutropil were seen. In some area most neuron was lost and network of fibers and strands with a few neuron remained. Such microscopic pictures were also seen in some parts of cerebellum and some others areas of brain. In medulla, pons and midbrain different stages of neuronal degeneration, perineuronal vacuolation and loss of neuron were prominent. The shapes of vacuoles in brain are different some of them are multiocular around the

degenerated and necrotic neurones; and the others maybe small bubble shapes. In the end stage of disease the vacuoles expanded, and neuron loss and different sizes of white holes remained (spongiosis). Figures 3a, b, c, and d show the different shapes of perineuronal vacuolation and spongiosis in different parts of brain (H&E×200).

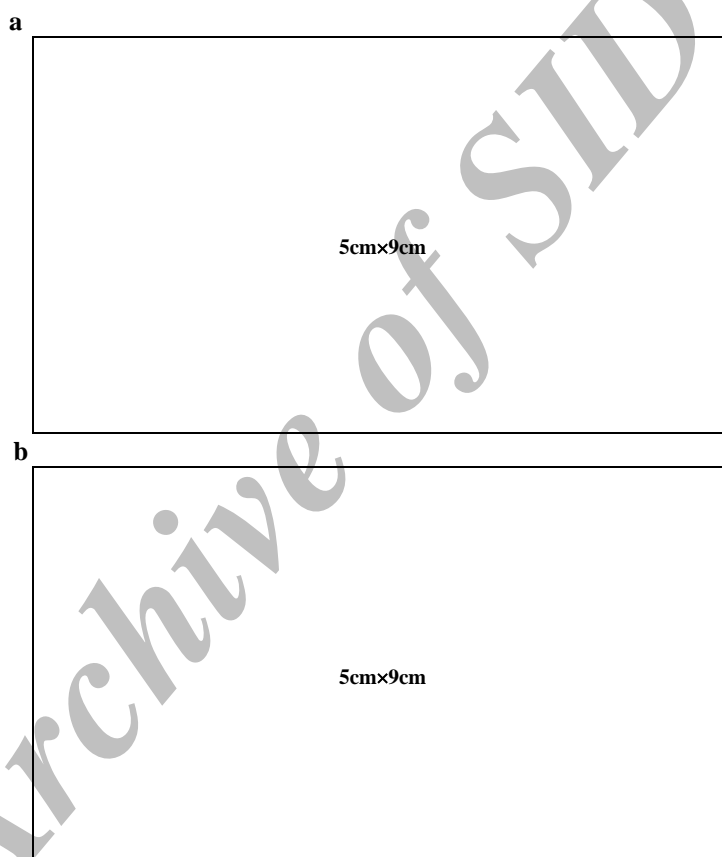


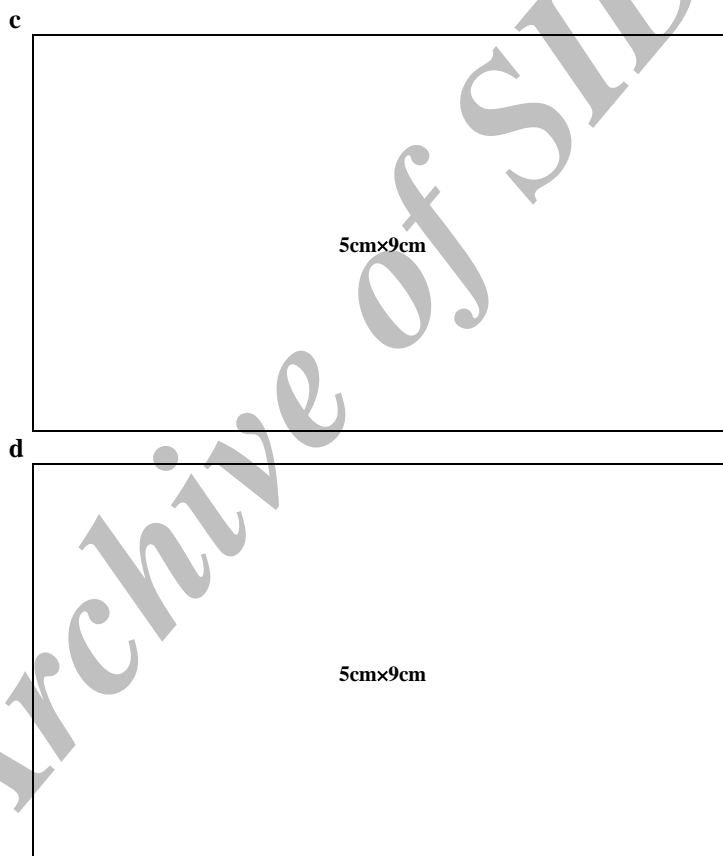
Figure 3. Different shapes of perineuronal vacuolation and spongiosis in different parts of brain (H&E×200)

In one case perivascular cuffing (P.V.C) with lymphocyt infiltration in brain in one artery was seen. Generally in white matter of brain different stages of axonal splitting and periaxonal vacuolation were prominent. The spinal cord lesions are

same as brain. In these cases there were some clinical and histopathological similarity with

Archive of SID

scrapie lesions. Samples of paraffin blocks and formalin fixed brain tissues and blood for scrapie and caprine arthritis encephalitis were sent to Weybridge laboratory in England and the negative result was confirmed. Examination for other central nervous system disorder such as listeriosis and toxoplasmosis were negative. Those reasons that mentioned and reversed of clinical sign in few cases on primary stage of disease, thiamine deficiency related polioencephalomalacia was suspected.



Acknowledgements

The authors would like to thanks F. Taleblo, A. Habibpour and M. Gholami for their skilled technical helps.

References

- Bakker, H.J., Dickson, J., Steele, P. and Nottle, M.C. (1980) Experimental induction of ovine polioencephalomalacia. *Veterinary Record* 107:464-466.
- Boyd, J.W., Walton, J.R. (1977). Cerebrocortical necrosis in ruminant; An attempt to identify the source of thiaminase in affected animals. *Journal of Comparative Pathology* 87:930-935.
- Jeffrey, M.D., Duff, J.P., Higgins, R.J., Simpson, V.R., Jakman, R., Jons, T.O., Mechie, S.C. and Liversey, C.T. (1994). Polioencephalomalacia associated with ingestion of ammonium sulfate by sheep and cattle. *Veterinary Record* 134:343-348.
- Loew, F.M., Dunlop, R.H. (1972). Induction of thiamine inadequacy and polioencephalomalacia in adult sheep with amprolium. *American Journal of Veterinary Research* 33:2195-2205.
- Markson, L.M., Giles, N. (1973). Cerebrocortical necrosis in a fallow deer (Dama dama). *Veterinary Record* 93:243-246.
- Olkowski, E. (1992). Role of thiamine status in sulphur induced polioencephalomalacia in sheep. *Research in Veterinary Science* 52:78-85.
- Radostits, O.M., Blood, D.C. and Gay, C.C. (1994). *Veterinary Medicine* (8th Edn.). Bailliere Tindall, London.
- Thomson, S., Donald, M., William, W. and James, F. (2001). *Special Veterinary Pathology*. Mosby, London.
- Thornber, E.J., Dunlop, R.H., Gawthorne, J.M. and Huxtable, C.R. (1979). Polioencephalomalacia (cerebrocortical necrosis) induced by experimental thiamine deficiency in lamb *Research in Veterinary Science* 26:378-380.