Trakia Journal of Sciences, Vol. 3, No.5, pp 65-69, 2005
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ISSN 1312-1723

*Original Contribution*

# CLINICAL AND MORPHOLOGIC STUDIES OF NEONATAL ENZOOTIC ATAXIA IN THE GOAT KIDS
# II. PATHOMORPHOLOGIC STUDIES

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**ABSTRACT**

The present article describes the pathomorphologic studies of five newborn goats. All of them had shown clinical evidences of ataxia first day after birth. The animals came from different farms located in same village. Pathoanatomy established liquefactive necrosis in the white brain matter of the right hemisphere. In addition, one of them showed inborn dysplasia of the cerebellum, another opacity of the cornea. The basic pathohystologic lesions in the animals were deficiency of myelin and vacuolation of the white matter of the brain and the spinal cord. Other findings were defective formation of the cerebellum, degenerative-necrobiotic changes in the neurons of the ventral horns of the spinal cords and the degenerative-inflammatory changes in the articulations.

**Key words:** Neuropathology, Enzootic ataxia, Copper deficiency, Prenatal development, goat kids.

## INTRODUCTION

Copper is one of the most important elements for the development of the embryo and the foetus Insufficiency of this in the pregnant animals inevitably leads to its deficiency in the foetuses (1). Therefore neonatal enzootic ataxia of the newborn invariably results from grazing pregnant animals in pasture areas poor in copper (2, 3, 4). Environmental copper deficiency is due to the presence of molybdenum and sulphates in the soil, which consequently affects the plants. The rumens of these herbivores contain sulphide-generating bacteria that can form thiomolybdate complexes, which naturally have strong affinity for copper ions. Plants with little copper content serve no useful purpose to the animals since these thiomolybdate complexes easily form insoluble super-complexes with this little copper, thereby rendering it unabsorbable (5, 6, 7). Consequent upon this scenario, embryonic and foetal copper deficiency results with the attendant embryonic death or disturbances in the growth of the newborn, pathological changes in the central nervous system, the skeleton and disturbances in metabolism (8).

Studies in Australia in 1937 established that the enzootic neonatal ataxia in lambs resulted from the pregnant sheep grazing in copper-poor pastures (3). The disease was described in lambs in England in 1952 (2).

The changes in the white matter of the nervous tissue in enzootic ataxia are due to low-level synthesis and degeneration of myelin (9). In neuropathology they are described as deficiency of myelin, vacuolation, and cavitation of the white brain matter of cerebral hemispheres, the brainstem and spinal cord. Very often the described finding is also a chromatolysis and hyalinization of the neurons of the ventral horns of the spinal cord (10, 11, 12, 13). Such decreased synthesis of myelin and deficiency of myelin are seen not only in lambs and kid goats but also in deer and pigs with deficiency of copper (14, 15, 16). The neuropathologic lesions in the cerebellum associated with this

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disease are seen more rarely with lambs, but are usually found in kid goats (16a). They are characterized by a distinct thinning of the granular layer and a decrease in the number of cells in it, as well as a loss of the Purkinje cells. Aetiologically, the neural degeneration is caused by copper deficiency (11, 16a, 17, 18).

The neurological disturbances connected with copper deficiency in lambs have been described as 'swayback' (swaying, swinging caused by congenital deficiency of copper), enzootic ataxia (ataxia with a delayed beginning) and cerebral oedema (10, 12, 15, 16).

In one of our previous reports we gave the result from the clinical and para-clinical studies of enzootic ataxia in kid goats (in press). As follow up, we present here a description of results from the pathoanatomic and pathohystologic case studies.

## MATERIALS AND METHODS

After euthanasia we performed pathoanatomical examinations of five out of a total of twelve kid goats. The animals were from different farms of the same village. All of them had shown clinical signs of ataxia during the first days after birth. We took materials for histological examination from different sections of the brain (hemispheres, stem, cerebellum) and spinal cord (lumbar area). The samples were fixed by immersion in 10% neutral formalin and embedded in paraffin using ethyl alcohol. Sections were stained with haematoxylin and eosin (H/E). For the bacteriological examination we took samples from articulation content from two kid goats showing signs of arthritis. The samples were tested using the particular conventional bacteriological methods.

## RESULTS

Pathoanatomy on one of the kid goats showed a necrotic focus in the subcortical region of the white matter in the right hemisphere of the brain. The lesion was about three centimetres in diameter and was located dorsally to the side ventricle. The necrotic tissue was slightly sunken and well outlined with its dark grey colour against the white brain matter (**Figure 1**). Similarly, another kid goat showed an inborn decrease in the size of the cerebellum. Further sectioning helped us establish that the organ lacked basic structure (dysplasia). The cut surface showed only longitudinal convolutions and the typical arbor vitae structures were missing (**Figure 2**). Clinical studies in another case showed unilateral blindness of the left eye and dim cornea. The same animal showed a depigmented line of hair round the eyes (**Figure 3**).

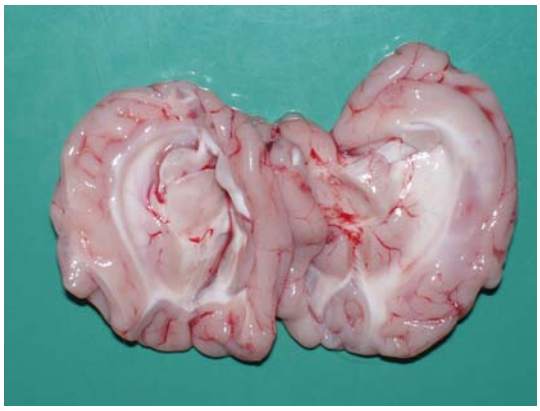

***Figure 1***. *Necrosis in the white matter of the right cerebral hemisphere.*

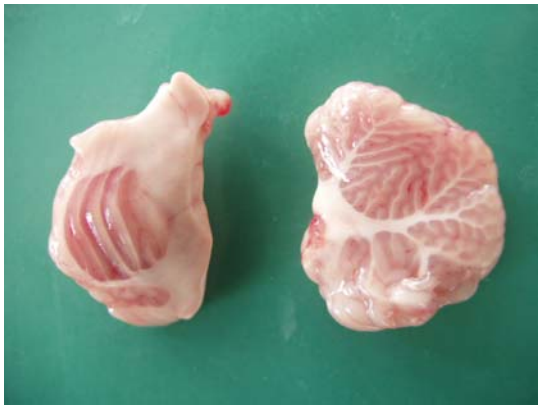

***Figure 2.*** *Longitudinal convolutions on the cut surface and lack of typical arbor vitae structures for cerebellum (dysplasia of the cerebellum)-left in comparison with normal-right.*

Clinical examination of two of the animals showed a bilateral oedema of the carpal articulations (**Figure 4**). After sectioning we found degenerative-necrobiotic changes that had affected the articulation surfaces. The cartilage tissue was damaged in different degrees with a thick content in the articulation cavity (**Figure 5**).

There were histological changes in the nervous tissue with localisation in the brain and spinal cord. There were various extents of liquefactive necrosis in the white brain matter of the hemispheres of the studied animals. The vacuoles that had formed varied in size and showed porous appearance of the brain tissue. Several neighbouring vacuoles merged around and formed multi-chamber cavities (**Figure 6**).

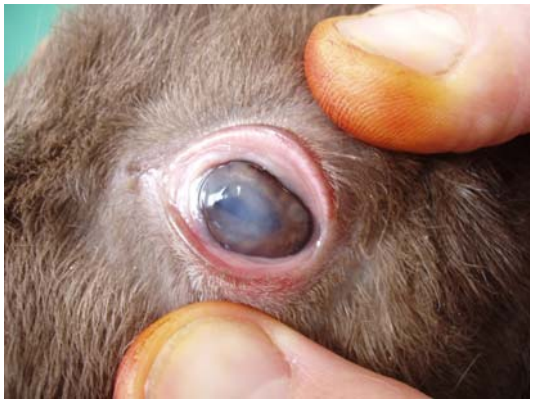

***Figure 3.*** *Unilateral blindness, dimness of the cornea and depigmentation of the hair cover round the eye.*

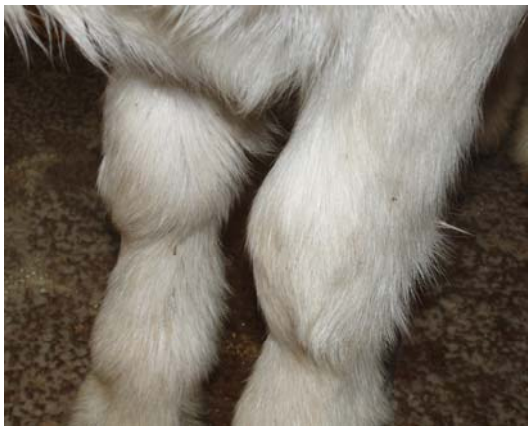

***Figure 4.*** *Bilateral tumefaction of carpal articulation*

***Figure 5****. Lesion of degenerative-necrobiotic type in the articulations after section.*

We also found poor myelin sheaths of the nerve fibres in the white brain matter of spinal cord (**Figure 7**). The accompanying lesions in almost all the cases, were degenerative-necrobiotic changes in the ganglion cells of the ventral horns. In several of the cases, the affected neurons were difficult to distinguish in the environment due to lysis of the chromatophilic matter and the nucleus (**Figure 8**). In other neurons the chromatophilic matter was densely condensed. This pycnosis of the neurons was the reason for forming the bright zone round them (**Figure 8**).

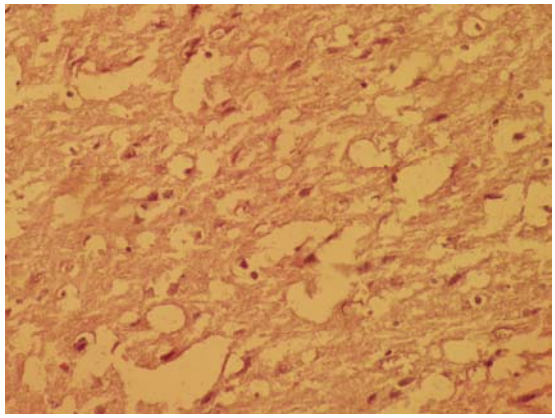

***Figure 6 .****Liquefactive necrosis, spongoid vacuolation ,and multi-chamber cavities in the white substance of cerebral hemisphere. Haematoxylin and eosin stain, x260.*

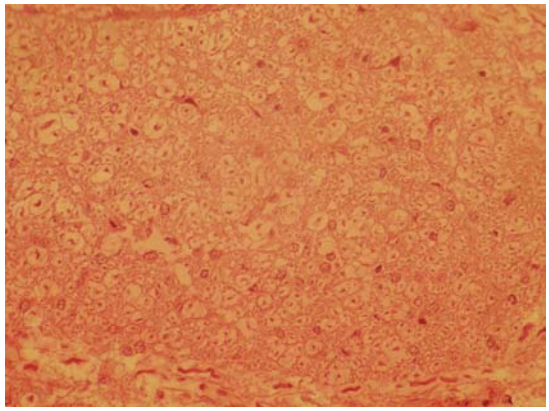

***Figure 7****. Deficit of myelin in nerve fibres in the white substance in lumbar segment of spinal cord. Haematoxylin and eosin stain, x260.*

Histological findings in animals with inborn dysplasia of the cerebellum revealed disordered layers of the cortex with different widths. At some other places the Purkinje cells were missing. At the same time there were separate centre formations with a typical structure of the cortex, in the white brain matter.

Bacteriology showed *E. coli* in samples of the articulation substance from animals with oedema of the carpal articulations.

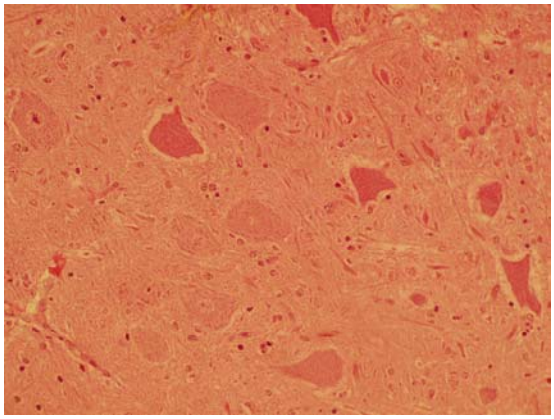

***Figure 8***. *High-level neuronal chromatolysis and pycnosis of the neurons in white matter in lumbar segment of spinal cord. One normal neuron is evident. Haematoxylin and eosin stain, x300.*

## DISCUSSION

The results from the pathomorphological examination expand and support the picture of clinical and paraclinical signs characterizing the enzootic ataxia with kids, described in our previous report (in press). The established pathoanatomical and histopathological lesions are logical explanations of the clinically observed neurological disturbances. The pathoanatomically established lesions, such as liquefactive necrosis in the white brain matter and defective formation of the cerebellum in our examinations, are in line with the described cases of enzootic ataxia in lambs and kid goats (10, 11, 12, 19). The established lesions during the histopathological examination-aplasia of myelin and spongy vacuolation of the white brain matter of the big hemispheres and the spinal cord, are in conformity with the described disease both with the lambs and the kid goats (10, 11, 12, 14, 15). We also accept that the decreased synthesis of myelin in combination with the instability and the predisposition to degeneration of the already formed myelin in some regions of the brain and spinal cord, lead to the observed structural changes in the white cerebral matter (9, 16a, 19). The reason for the decreased synthesis of myelin is the inhibited activity of the respiratory enzyme cytochrome oxidase that has copper ion. The observed posterior paralysis and ataxia in this case could be associated with the established degenerative-necrobiotic damage of the neurons in the ventral horns of the spinal cord; a situation that has been noted by a number of other researchers (13, 20, 21). The neurons in this type contain high amounts of cytochrome oxidase in their mitochondria. That is the reason why they are the most vulnerable when there is copper deficiency (11, 16a). The growth of the axons and the condition of their casing are highly dependent on the functional activity of the perikaryon in relation to the protein synthesis (22). That is the reason why the axons are the first to be damaged (11), and as a result degenerative-necrobiotic changes occur in the cellular body as well (19, 23, 24, 25). The established cases of arthritis affecting the carpal articulations with two of the kids which we examined, are very close to those described in the deer with a state of copper deficiency (14). We also accept that the original reason for the established arthritis is the degenerative changes affecting the articulation surface, as a result of copper deficiency. We can support this statement with the results of the microbiological examination, where *E.coli* was isolated in one of the two animals showing signs of arthritis. This gives us the good reason to think that the interference of the bacteriological agent in this case is of secondary importance.

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