



## Polioencephalomalacia Caused by Thiamine Deficiency in Sheep of Basrah Province, Iraq

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**P**OLIOENCEPHALOMALACIA caused by thiamine deficiency in sheep of Basrah province have been diagnosed. This current research was conducted on 44 local sheep breeds 1-3 years old and of both sexes reared in Basrah province. Ten clinically normal local sheep breeds were selected as a controls. Diseased sheep show various clinical manifestations, such as anorexia, head pressing and muscular tremors, opisthotonus and nystagmus, incoordination and ataxia, Furthermore, circling with lateral deviation of the head (41%), recumbency and inability to stand and some sheep exhibit blindness (18.1%). Moreover, significant increase in respiratory and heart rate of diseased animals in comparison with control animals, while rumen motility was decreased significantly in affected animals compared to control group. Considerable changes were detected in biochemical parameters of affected sheep in comparison with controls. The gross pathological features and histopathological examination of dying or slaughtered sheep show pale discoloration of the dorsal cerebral cortex, especially the occipital lobes, which might indicate a necrosis. However, gross lesions might also represent flattening of cerebral gyri, hemorrhages, necrosis to cavitations in the gray matter of the occipital and temporal regions. The histopathological examinations revealed gliosis as well to vacuolative changes of some neurons. The white matter of brain showed also gliosis with wallerian degeneration of some neuronal axons and severe vaculation of neuronal cell body and central chromatolysis of some neurons. thiamine estimated by simple spectrophotometric method. It is concluded that thiamine deficiency is an important disease which could affect domesticated animals and result in economic losses, Therefore, good care and good management will be the ideal way to control the disease.

**Keywords:** Thiamine deficiency, Polioencephalomalacia, Sheep, Basrah, Iraq.

### Introduction

It has been documented for a long time that there is a relationship between a nutrient deficiency and visually measurable health [1]. The B group of vitamin comprises essential nutrients that support the metabolism of carbohydrate, besides enhancing the immune system functions, and promoting cell growth, since Riboflavin (or vitamin B2), Niacin (or vitamin B3), and Pantothenic acid (vitamin B5) will roll with thiamine (vitamin B1) as essential coenzymes for energy metabolism [2].

Thiamine is a water-soluble vitamin that is not stored in the body and needs to be replaced with daily diet. It has significant role to play in carbohydrate and energy metabolism, as the thiamine is converted to aneurine pyrophosphohate carboxylate which is critical in carbohydrate build up or break down. It is also required for the functioning of the heart, muscles, and nervous system, since tissues such as the brain and the heart are particularly compromised in thiamine deficiency [3].

Although ruminal microorganisms will

synthesize thiamine and other types of feeds, deficiencies can develop in ruminants for different reasons some of these, conditions are the ruminal fermentative disorders, which will aid in ruminal microbial thiaminase production, subsequently inactivates thiamine analogs. Moreover, consuming excessive sulfur either from foodstuffs or water leads to thiamine deficiency in ruminants [4]. It has been documented that, thiamine deficiency decreases the energy available to the brain, which results in a type of brain degeneration called "Polioencephalomalacia" or PEM [5].

To the best knowledge of the researchers, reports concerning thiamine deficiency in local sheep breeds of Basrah province, Iraq, are very limited and little information is provided. Therefore, the aims of this current study were includes, to clinical evaluation of diseased sheep with hypothiaminosis, to exploring the main clinical manifestations of diseased animals, to evaluate the hematological changes, Besides estimation of thiamine, troponine I and creatine kinase myocardial band in diseased local sheep breeds. And to assess the gross post-mortem examinations and histopathological changes of recently died and /or slaughtered animals.

## **Materials and Methods**

### *Animals and study design*

The study involved 44 local sheep breeds of 1-3 years old and of both sexes reared in Basrah province, Iraq. Animals showed signs of weakness and manifestations of nervousness. Ten clinically normal local sheep breeds were allocated as controls. Comprehensive clinical examinations were carried out of all animals. Complete coprological and hematological examinations were performed to exclude the endoparasitic and blood parasitic infestations, using the flotation and sedimentation methods and the Giemsa stain blood examinations[6].

### *Collection of samples and Hematological examinations*

Ten milliliters of blood (10 ml) were drawn from individual animals via the jugular vein and from these 2.5 milliliters of blood were mixed with EDTA to evaluate total erythrocyte count (RBCs), hemoglobin concentration (Hb), packed cell volume (PCV), and Total leukocyte count (TLC), (Analysis was done by, Hematology analyzer from Genex, USA).

### *Biochemical analysis*

*Evaluation of thiamine*, -It's done according to the procedure of [7].

### *Evaluation of troponin I and Creatine kinase-myocardial band (CpK-MB)*

Serum was evaluated for Troponin I (cTnI) concentration measured by AFIAS-6 (AFIAS-automated fluorescent immunoassay system) from, boditech. Moreover, Creatine kinase-myocardial band (CpK-MB) was analyzed by (spectrophotometer using commercial kits), (Roche Diagnostics, Indianapolis, GMBH, Germany).

### *Grosspost-mortemandHistopathologyexaminations*

Animals which died or were slaughtered with the owner's approval were subjected to post-mortem examinations and laboratory histopathological evaluations. The tissue samples were collected from different parts of the brain. The collected samples were fixed at 10% neutral buffered formalin solution for 72 hrs, then trimmed to suitable sizes and washed, then dehydrated and cleared in xylol. Finally, it was embedded in paraffin wax, and sectioned at 4-5  $\mu$  thickness, stained with hematoxyline and eosin, and examined under a light microscope[8].

*Statistical analysis*, It was done using (SPSS) student *t*-test [9]

## **Results**

Diseased local sheep breeds exhibited various clinical manifestations, such as Anorexia, and head pressing (88.6%), and muscular tremors (75%). Moreover, diseased sheep showed signs of opisthotonus and nystagmus, incoordination and ataxia in 66% of diseased sheep, while, 41% of diseased sheep showed circling with lateral deviation of the head, recumbency and inability to stand (36.3%), However, 18.1% of the diseased sheep showed blindness, Furthermore, 13.6% of diseased sheep had abnormal heart murmurs detected on auscultation of the chest (Table 1).

Moreover, a significant increase ( $P < 0.05$ ) was detected in respiratory and heart rate of diseased animals in comparison with controls. However, ruminal motility was decreased significantly ( $P < 0.05$ ) in diseased animals compared to controls (Table 2). Results of hematological parameters refer to a non-significant change in TRBc, Hb and PCV, However, no significant changes were detected in total leukocyte count in diseased animals compared to controls (Table 3).

**TABLE 1. Clinical manifestations of diseased local sheep breeds**

Clinical manifestations	Number of affected animals	%
Anorexia and head pressing	39	88.6
Muscular tremors	33	75
Opisthotonus , Nystagmus	30	66
Incoordination and ataxia	30	66
Circling with lateral deviation of the head	18	41
Recumbency and Unable to stand	16	36.3
Blindness	8	18.1
Abnormal heart sounds (murmurs)	6	13.6

**Fig. 1. Recumbency and inability to stand of the head****Fig. 2. Lateral deviation of the head.****TABLE 2. Clinical parameters of diseased sheep and controls**

Parameters	Controls n=10	Diseased sheep n=44
Body temperature C	38.5± 0.83	38.4± 0.47
Respiratory rate / min	22.7± 5.66	48.6± 7.75*
Heart rate / min	70 ±4.65	91.6± 18.74*
Ruminal contractions / 5 min	4.1± 1.32	2.45± 1.44 *

Values are mean ± standard error of mean. \* (P<0.05).

**TABLE 3. Hematological parameters of diseased sheep and controls**

Parameters	Controls n= 10	Diseased lambs n=44
RBC ×10 <sup>6</sup>	7.91±1.33	7.95±0.36
Hb g/dl	13.21 ± 1.79	13.5±0.4
PCV %	32.55 ± 4.73	33.91±1.08
TLC ×10 <sup>3</sup>	12.45±1.72	12.84±0.75

Values are mean ± standard error of mean. \*\* (P<0.05).

In addition, significant changes were detected in biochemical parameters of diseased sheep in comparison with controls (Table 4).

The results of gross pathological features and histopathological examination of dying or slaughtered sheep showed pale discoloration of the dorsal cerebral cortex, especially the occipital lobes (Fig. 3), which might indicate a necrosis.

Gross lesions might also represent flattening of cerebral gyri, hemorrhages, necrosis to

cavitations in the gray matter of the occipital and temporal regions (Fig. 4).

On the other hand, the histopathological examinations revealed gliosis as well to vacuolative changes of some neurons (Fig. 5, 6). Sections of the white matter of brain showed also gliosis with wallerian degeneration of neuronal axons, but, central chromatolysis of some neurons in gray matter were also detected (Fig. 7,8).

**TABLE 4. Biochemical analysis of diseased sheep and controls**

Biochemical parameters	Controls n=10	Diseased sheep n=44
Thiamine nmol/L	118 ± 4.12 nmol/L	47 ± 2.44 nmol/L*
Troponin (cTnI ng/ml)	0.21 ± 0.05	8.23 ± 1.4*
Creatine kinase (CpK-MB) U/l	57 ± 22	91 ± 36.4*

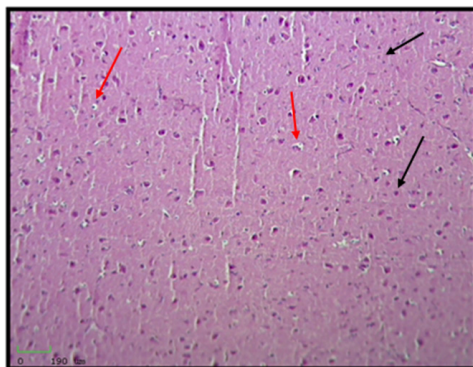
Values are mean ± standard error of mean. \* (P<0.05).



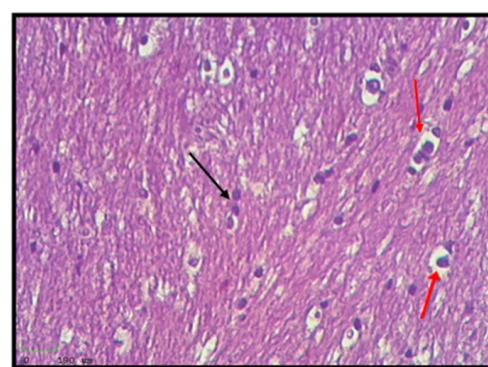
**Fig. 3. Pale discoloration of the dorsal cerebral cortex, flattening of cerebral gyri and hemorrhage.**



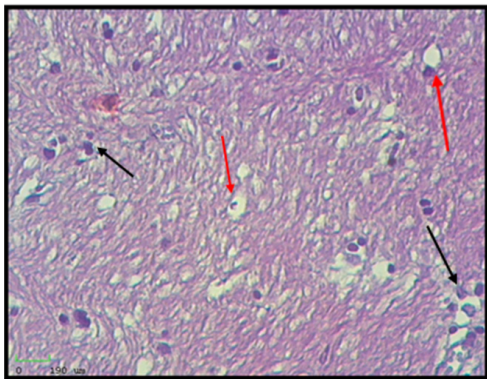
**Fig. 4. Flattening of cerebral gyri, hemorrhages, necrosis to cavitations in the gray matter of the occipital and temporal region.**



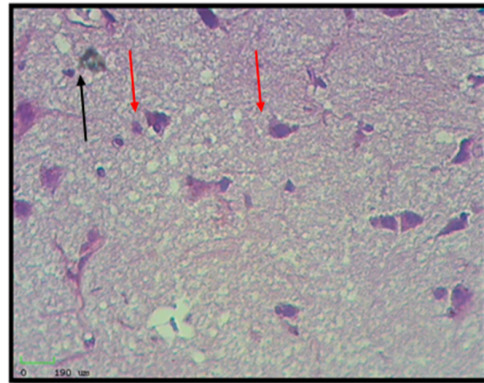
**Fig. 5. Histopathological section of gray matter of brain showing gliosis (black arrow), as well to vacuolative changes of some neurons (red arrow). H&E stain. 10X.**



**Fig. 6. Histopathological section of brain showing gliosis (black arrows), as well to severe vacuolation of neuronal cell body (red arrows). H&E stain. 40X.**



**Fig.7. Histopathological section of white matter of brain showing gliosis (black arrows), as well to wallerian degeneration of some neuronal axons (red arrows). H&E stain. 40X.**



**Fig. 8. Histopathological section of gray matter of brain showing gliosis (black arrow), as well to central chromatolysis of some neurons (red arrow). H&E stain. 40X.**

### **Discussion**

On the basis of clinical examinations, animal history, necropsy findings, differential diagnosis and correlation with possible causes, the current clinical cases can be considered as a clinical case of thiamine deficiency.

Thiamine plays a major role in metabolism of carbohydrates in the nervous system and muscles. Therefore, lack of thiamine induces a lower supply of carbohydrates to the neurons in the brain [3]. Since neurons need a lot of carbohydrates as an energy source necessary for nerve function [5], the depletion of carbohydrate sources might cause alterations in the mechanism of action of all nervous system parts, but final neuronal death, especially cortical region might suspect [9]. Thereby, damage to the brain cells may be responsible for the demonstration of the symptoms [10].

In the current study, there are different possibilities for the causes of thiamine deficiency. According to the history of indoor feeding system, all affected sheep were fed on concentrated mixture of carbohydrates and the intake of weeds and ferns during grazing. Another contributing factor might be low levels of thiamine in the soil that is drained out during the rainy season. Thus, inactivity and non availability of thiamine are considered to be responsible for creating the condition, Sulfur is needed for to synthesize important sulfur containing amino acids and their contribution to the synthesis of different hormones, enzymes, and structural proteins. The ruminant diet, particularly that of cattle, can be

over-concentrated with sulfur. In ruminants, the same rumen microbes that generate thiamine molecules reduce sulfur into toxic sulfides. Among the sulfide toxins is hydrogen sulfide, a gas compound that will compete with oxygen to bind to red blood cells and ultimately enter the brain to disrupt neural activity [11].

Mechanism of sulfur-induced polioencephalomalacia had been proposed in [12].

Although Sulfur-induced polioencephalomalacia has been recognized in the last 30 years, the role that sulfur plays in polioencephalomalacia is still not clearly defined [10]. It has been proposed that lesion development has an association with the inhalation of eructed  $H_2S$  from the rumen. For example, when excess sulfur is ingested, a relatively high concentration of sulfide is generated due to sulfur reduction by rumen microbes. Some sulfide from the fluid phase will be released into the rumen gas cap as  $H_2S$ . Formalization of  $H_2S$  from the sulfide ion depends mostly on the pH. As rumen pH drops, the  $H_2S$  in the rumen gas cap rises. As ruminants inhale more than 70-80% of the eructed gas, it has been suggested that most of the eructed  $H_2S$  gas might be absorbed into the pulmonary blood system through inhalation of eructed gas, and some inhaled  $H_2S$  may access the brain without going through hepatic detoxification and this leads toxic damage [11]. Sulfide in the brain tissue will convert into sulfate via the mitochondrial sulfide oxidation process [5]. Tissues with a high oxygen demand, such as the brain, are more sensitive to disruption of oxidative metabolism by sulfide, which is the primary mechanism for sulfide toxicity.

Furthermore, consuming contaminated water that is always present in Basrah province, as most water here have high levels of sodium chloride and sulfur, which indicates the decreased absorption of thiamine from ruminal mucosa. Further, thiaminase produced in those ruminants will distract the thiamine [9,12,13].

In the present study, symptoms shown by diseased animals were similar to what were described by [14,15]. Most of the signs present were indicative of nervousness. Name et al and DiNicolantonio et al. [16,17] mentioned that, thiamine deficiency can cause two very dissimilar disorders - a dry and wet beriberi. Dry beriberi involves neurological complications, whilst, wet beriberi has the involvement of cardiovascular complications which include, enlargement of the heart which might culminate to a heart failure, therefore the blood brain barrier is interrupted and cerebral hypoperfusion occurs resulting in manifestations of nervousness which was detected in the present study. On the other hand Khan and Garg [18], added, that acute decompensated heart failure and hyperdynamic circulation were detected clinically in thiamine deficient patients. Moreover, cardiac beriberi might be mistaken in clinical practice because of the absence of classically described symptoms, such as edema and or anasarca. It has been reported that these patients have ongoing myocardial damage with troponin rise.

It had been proved that thiamine is a key chemical in glucose metabolism and for that reason, its deficiency is extremely menacing to neurological activity. Domestic animals like cattle, sheep, goats, and other ruminants diagnosed with polioencephalomalacia or pre polioencephalomalacia will show signs of opisthotonus, cortical blindness, disoriented movement, and will be terminated by death if left untreated. [2]

The principal function of thiamine in all body cells is as the coenzyme cocarboxylase or Thiamine pyrophosphate or thiamine diphosphate. Thiamine pyrophosphate is a cofactor that exists in all living systems, in which it catalyzes several biochemical reactions. Thiamine pyrophosphate participates in the decarboxylation of keto acids and is also a coenzyme of transketolase. These enzymes play significant roles, especially in the

metabolism of carbohydrates and independent specific roles in neurophysiology [4,19,20]. Therefore, thiamine deficiency causes a wide range of clinical appearances from anorexia to polioencephalomalacia because of the numerous functions of thiamine, which has been observed in the current study.

It has been documented that glucose metabolism is controlled by thiamine, However, the overconsumption of glucose can also cause thiamine inadequacy, Should there be a sudden increase of glucose in the body, thiamine will be deleted so that it is unavailable when the next round of glucose needs to be metabolized [11,13].

In recent studies, sulfur intake with high concretions might be the important reason for polioencephalomalacias [21].

It has been mentioned that, the results of the gross and histopathological appearance of thiamine deficient carcasses vary according to the intensity and duration of disease, but most of the necropsy findings in the current study are in accordance with [22]. At necropsy, pale or yellowish discoloration of the dorsal cerebral cortex occurs, especially in the occipital lobes, indicating necrosis [1]. Furthermore, Jortner, [23]. described a clear gross evidence of edema in the brain as cerebellar protrusion into the occipital foramen and in more advanced cases the telencephalic cortex exhibits more characteristic changes such as flattening of gyri, softening with gelatinous or depressed areas, and cavitations filled with yellow liquid, hemorrhagic foci in the meningeal and sub-cortical regions are also observed. In addition, Loneragan and Gould [24] added, that the histopathological appearance is characterized by segmentar laminar necrosis of cortical telencephalic neurons (red neurons). This lesion is characterized by shrunk and eosinophilic cytoplasm, chromatolysis and nuclear picnosis. This type of neuronal change should not be mistaken by the black or dark-blue, so called "dark neurons," that are artifacts due to excessive post-mortem manipulation of the brain. Edema is a common change and comprises augmentation of the perineuronal and perivascular spaces with multiple vacuoles in the neuropil (spongiosis).

In the current study, evaluation of thiamine was shown to be in line with the results of many researches [14,25], Moreover, troponin

showed slight significant increase in diseased sheep compared to controls. It was proved that, troponins could be released in response to any myocardial harm of different origin and the increased levels can occur in a lot of conditions such as ischemic damage which could explain the slightly increased values in this study [26]. Furthermore, it was shown that Creatine phosphokinase is the most specific biochemical indicator used for the diagnosis of acute myocardial problems, especially myocardial infarction, However, the degree and the duration of Creatine phosphokinase elevation in serum approximates the range of an acute myocardial infarction [27]. This result might prove that thiamine cause non acute type of heart problem.

### **Conclusions**

It is concluded that thiamine deficiency is an important disease which could affect domesticated animals and result in economic losses. Therefore, good care and good management will be the ideal way to control the disease.

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### *Conflict of interest*

No conflict in interest

### *Funding statements*

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## تلين سنجابية الدماغ الناتج عن نقص الثيامين في ضأن محافظة البصرة , العراق

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تشخيص تلين سنجابية الدماغ في ضأن محافظة البصرة , العراق . اذ اجري البحث على اربع وأربعين من الضأن المحلية تراوحت اعمارها بين 1-3 سنوات ومن كلا الجنسين . تم اختيار عشرة من الضأن المحلية السوية سريريا كمجموعة ضبط . اظهرت الحيوانات المريضة علامات سريرية مختلفة تمثلت بفقدان الشهية والضغط على الرأس مع ارتجاف العضلات فضلا عن ذلك فقد اظهرت الضأن المريضة علامات الشنج والرأه, واللاتساق الحركي , علاوة على ذلك فقد اظهرت 41% من الضأن المريضة علامات الدوران مع انحراف الرأس الى الجانب , الاستلقاء وعدم المقدرة على الوقوف . ومع ذلك فإن 18,1% من الحيوانات المريضة اظهرت علامات العمى . لوحظ تزايد في معدلات تردد التنفس وضربات القلب في الضأن المريضة بالمقارنة مع مجموعة الضبط . اوضحت في حين تناقصت تقلصات الكرش في الحيوانات المريضة بالمقارنة مع مجموعة الضبط بالإضافة الى ذلك تم اكتشاف تغيرات واضحة في المعايير الكيموحيوية للضأن المريضة بالمقارنة مع مجموعة الضبط . اوضحت نتائج التشريح المرضي والنسجي للحيوانات النافقة او المذبوحة بأن هناك شحوب للقشرة الدماغية الظهريه وبخاصة الفص القذالي والتي قد تشير الى وجود نخر ومع ذلك فإن الافات الجسيمة قد تمثل أيضاً تسطح الجير الدماغية والنزيف ونخر والنزيف الى التجويف في المادة الرمادية في المناطق القذالية والزمانية , علاوة على ذلك كشفت فحوصات التشريح المرضي النسجي عن وجود الدباق وكذلك التغيرات المفاجئة لبعض الخلايا العصبية فضلا عن ذلك اظهرت بعض مقاطع المادة البيضاء وجود الدباق مع وجود التنكس الالوي لبعض المحاور العصبية في حين اظهرت مقاطع اخرى تفجي الخلايا العصبية وانحلال تصبغها .