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DIAGNOSIS OF POLIOENCEPHALOMALACIA IN DROMEDARY CAMELS (*CAMELUS DROMEDARIUS*) FROM AL-HIADYIA

Ali Hussein Aldujaily[1]*, Shatha Atta Abeed[2] and Nadia Abdul Hadee Abdul Ameer[3]

[1*]Veterinary College, Kufa University, Iraq.
[2]Al-Forat Al-Awsat Technical University 31003, Iraq.
[3]Al-Forat Al-Awsat Technical University 31003, Iraq.

Abstract

The present study aimed to describe clinical, hematological and pathological findings of polioencephalomalacia (PEM) in dromedary camels (*Camelus dromedarius*) from Al-Hiadyia in the desert region of Al- Najaf, Iraq. The clinical signs included blindness, lethargy incoordination, ataxia, extension of the limbs, hyperesthesia, no menace reflexes, paddling movements, opisthotonos and recumbency. Also, hematological and biochemical parameters results were normal. On the other hand, postmortem was carried on two recumbent camel, with particular attention to examine brain tissue for evidence of the disease. Major macroscopic changes included congestion of cerebral vessels, edema, and herniation of the cerebellum. The most observed microscopic lesions, between two assessed cases, were laminar and segmental neuronal necrosis at different regions of the brain, spongiosis, nuclear pyknosis and red nucleus neurons. Clinical disease, gross and histopathological lesions which shows positive results of polioencephalomalacia. There are very few reports on incidence and/or prevalence of PEM in Iraq. However, this is the first study in younger Camels are reported in Al-Hiadyia at Al- Najaf desert.

Key words : Camel, Polioencephalomalacia, hematological, biochemical, cerebrocortical necrosis.

Introduction

Polioencephalomalacia" (PEM), also known as 'Cerebrocortical Necrosis' (CCN), is a non-infectious neurological disorder of ruminants that is seen sporadically worldwide characterized by descriptive lesions that occur in the gray matter of the brain (Radostits *et al.,* 2007; Milad & Ridha, (2009). The disease is causing acute blindness, ataxia, anorexia, and potentially progressing to recumbency, death and contributes substantial economic loss to animals' industry (Amat *et al.,* 2013).

Polioencephalomalacia occurs due to disturbance in thiamine absorption and metabolism (Rachid *et al.,* 2011). In ruminants, thiamine is produced by ruminal bacteria and protozoa under normal environmental conditions. Thiamine deficiency is related to overeating, acute impaction, grain engorgement, founder and grain overload (Nema *et al.,* 2014). Bacterial thiaminase has been considered the main factor causing thiamine deficiency in ruminants (Cebra and Cebra, 2004). Thiaminase is also present in plants such as bracken fern, horse tail and Nardoo ferns (Ramos *et al.,* 2005; Rachid *et al.,* 2011). Sulfur toxicity is one of the major causes of PEM and toxicity depends on the dose, duration, and bioavailability of the sulfur ingested (Kiupel *et al.,* 2003).

The aim of study was to identify the polioencephalomalacia in dromedary camel through clinical, hematological and histopathological confirmed Necrotizing meningoencephalitis that have not been previously reported as affected breeds in an Al- Najaf desert.

Material and Methods

The study was conducted at faculty of Veterinary medicine during the month of October 2018 to January 2019. The affected camels were between 4 -10 month-old. A total of 25 blood samples were collected among which 15 samples were taken as control group.

Sample blood (10 ml) was collected from all camels and there were evaluated for CBC. Packed cell volume

***Author for correspondence :** E-mail : alih.aldujaily@uokufa.edu.iq

(PCV) was measured using micro hematocrit centrifuge according to (Kerr, 2002). The Hb was converted into cyanmethemoglobins by using drabkins reagent and measured by spectrophotometer (NCCS). Red blood cells and white blood cells counts were evaluated by using the hemocytometer method according to (Coles, 1986).

Moreover, blood specimens were estimated for ESR using Westegren tubes, blood withdrawn to mark (0) and the tubes stand vertically on the rake (Maghsoodi *et al.,* 2005). The ESR values were recorded in mm after 24 hrs. A general guideline for estimating platelet numbers on a blood smear is to determine the average number of platelets in 10 oil immersion fields using a 100 objective and multiply the average by 15×10^3 to obtain the estimated number of platelets per microliter (Weiss and Wardrop 2010).

The blood centrifuged for 5-10 minutes at 3000 rounds (Coles, 1986). The separated sera were used directly for the measurement of iron and copper. The serum iron and copper were measured by atomic absorption spectrophotometers.

Two camels euthanized, after necropsy, brain samples were fixed in 10 % neutral buffered formalin and then submitted to the pathology laboratory. After fixation, tissue samples of parietal cortex, occipital cortex, and medulla were embedded in paraffin wax, and sections (5–6 µm) were cut and stained with hematoxylin and eosin (H&E).

Data were analyzed using SPSS version 21. The least significant differences test (LSD) were used to determine differences among groups. Data were subjected to analysis of variance statistically using one-way ANOVA.

Results

In the present study clinical signs observed based on their commonality were ataxia, anorexia, blindness, lethargy, incoordination, extension of the limbs, hyperesthesia and periodic tonic-clonic convulsions, no menace reflexes and the palpebral reflexes were slowed, paddling movements, teeth grinding, opisthotonos, strabismus, star gazing and recumbency.

Hematological and biochemical values of camels affected with PEM and control group is presented in (Table 1).

Among various hematological parameters evaluated from 25 camels, camel with PEM showed statistically non-significant differences in mean values of Hb, TEC, platelet count, PCV, TLC and ESR, as were as, there are no significant differences in iron and copper in camels with polio encephalomalacia compared to healthy ones.

However, major macroscopic changes included congestion of cerebral vessels, edema, and herniation of the cerebellum. Histopathological examination revealed severe cerebrocortical laminar neuronal necrosis and perineuronal vacuolation with activation of endothelial cells in the parietal and occipital lobes.

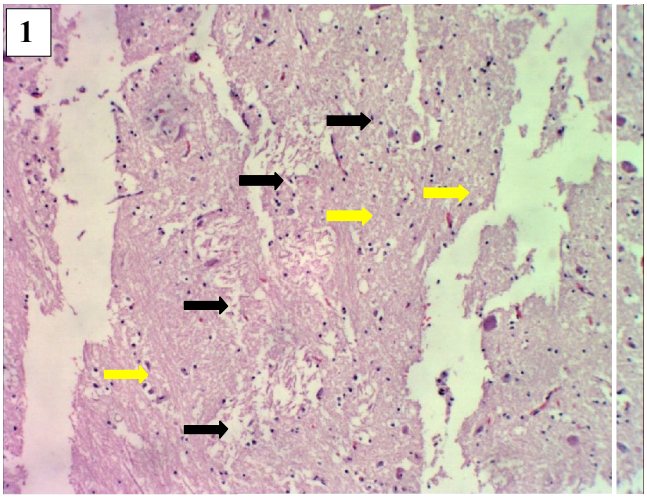

Fig. 1: Photomicrograph of cerebrum of Camel treated with methadine.

In Fig. (1) necrosis of neurons forming a space (black arrows) within glial cells (yellow arrows) in cerebral parenchyma.

In Fig. (2) observation of vacuoles due to presence of neurons necrosis. Note the necrotic neurons vacuoles (black arrows) within glial cells in cerebral parenchyma. Cerebral fibrosis (yellow arrows) due to severe lesion of neurons was observed. H&E. 1&2: 100x.

The lesions were widespread in cerebral cortex with polioencephalomalacia being obvious (Fig. 1). There was bilateral symmetrical status spongiosus of the white matter of the brain, microcavitations and bilateral laminar necrosis (Fig. 2).

Table 1: Hematological and biochemical parameters for normal and infected camels; ranges and means± SE.

Parameters	Group1with polio No.10	Group 2 without polio No. 15
RBCS (×10 6/µL)	9500-1215010691±200.7A	9825-1210010724±244.1A
HB(g/dL)	10.2-12.811.8±0.1A	11.8-12.512.1±0.1A
PCV (%)	28-3229.9±0.2A	30-3130.4±0.1A
Platelet (×10 3/µL)	109-282174.9±12.8A	104-243167.8±13.5A
ESR (mm) /8 h	6-127.6±0.4A	6-118.4±0.4A
WBC /µ L	9600-1160010416±172A	9150-1220010020±263A
Iron µmol/L	9.45-15.2512.03±0.59	9.12-17.7912.9±0.72
Copper µmol/L	6.92-15.1010.6±0.76	7.53-11.779.92±0.36

Different letters horizontally refer to the presence of significant ($P<0.05$) differences.

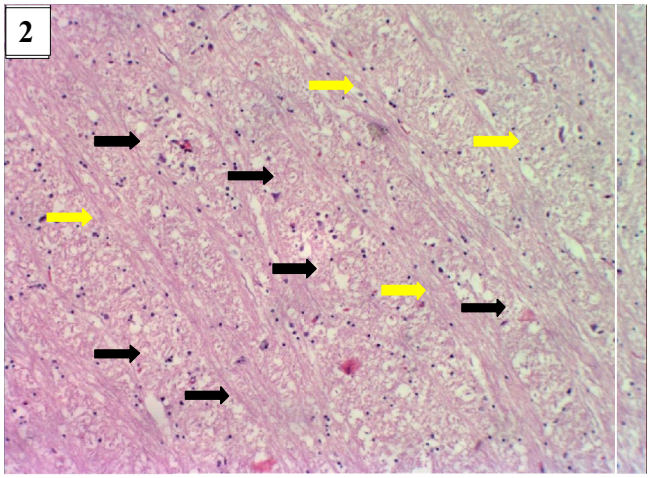

Fig. 2: Photomicrograph of cerebrum of Camel treated with methadine.

Discussion

The clinical signs of polioencephalomalacia in camels include altered mentation, blindness, ataxia, circling, muscle fasciculations, opisthotonos, recumbency and seizures these signs agreed with (Kiupel *et al.,* 2003 and Himsworth, 2008). However, 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. Hence damage to the brain cells may be responsible for origination of the symptoms (Mohanambal, 2017).

On the other hand, hematological and biochemical values examination tests were performed on twenty-five camels are presented in Table (1) revealed no significant differences ($P<0.05$) between two groups these results agreed with (Marks *et al.,* 2011; Moon *et al.,* 2013; Mahajan *et al.,* (2013); Anuradha *et al.,* (2014).

Moreover, the cut surface of the cerebral cortex showed areas of yellowish coloration with a decrease in consistency, suggesting malacia. Histopathoplogical examination in Fig. 1 and 2 revealed severe cerebrocortical laminar neuronal necrosis and perinueuronal vacuolation these results agreed with (Kiupel *et al.,* 2003 and Himsworth, 2008).

Based on the clinical, hematological and histopathological findings, these diseases are PEM. The incidence of occurrence was more in the month of (October-January). This could be due to change in the feed from dry roughage to lush green pasture, Also, that one of the contributing factors for thiamine deficiency after rainy season was due to the low levels of thiamine in the soil due to draining out by the rain. This might be the reason of high occurrence of polioencephalomalacia during September to December (Wallace *et al.,* 2000; Anuradha *et al.,* 2014).

Finally, affected animals should be given a high dose of thiamine (10-20 mg/kg) I/V every 6 hours due to the thiamine is water soluble and excreted quickly from the body (Niles *et al.,* 2002; Cebra and Cebra, 2004).

Conclusion

Our study indicated the major clinical findings in PEM positive camels were presence of nystagmus, circling movement, ataxia, recumbency, head-pressing, convulsion, opisthotonos and star gazing. Camels positive for PEM showed normal hematological and biochemical values. Thus, it is concluded that histopathology has diagnostic significance in Camels with PEM.

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