Iraqi Journal of Veterinary Sciences, Vol. 23, Supplement I, 2009 (119-122)
Proceedings of the 5th Scientific Conference, College of Veterinary Medicine, University of Mosul

The occurrence of thiamine-responsive polioencephalomalacia in dromedary breeding camels in Libya: preliminary investigation of diagnosis

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Abstract

In this study, thiamine-responsive polioencephalomalacia (PEM) has been detected in sporadic cases of 8 adult dromedary breeding camels at different farms around Tripoli. Clinical cases showed variable signs including disorientation, aimless walking with a high stepping gait due to blindness, anorexia, opisthotonus or head retraction (star gazing), muscle tremor, convulsion and recumbency. Post mortem was carried on one recumbent camel, with particular attention to examine brain tissue for evidence of the disease gross and histopathological lesions which shows positive results of PEM. The result of the study of clinical cases treated by parenteral injection of thiamine preparation showed a good response within 6 hours after initial treatment. The immediate response to the specific treatment was diagnostic for PEM and can differentiate it from other neurological conditions based on the animal's response to injection of thiamine beside specific clinical findings and necropsy lesions in the brain of sacrificed camel.

Keywords: Cerebrocortical necrosis, Camel, Polioencephalomalacia, Thiamine deficiency.
Available online at http://www.vetmedmosul.org/ijvs

حدوث مرض تلين الدماغ المستجيب للعلاج بالثايمين في الجمال في ليبيا: استقصاء اولي للتشخيص

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قسم الامراض الباطنية، كلية الطب البيطري، جامعة الفاتح، طرابلس، الجماهيرية العربية الليبية الشعبية الاشتراكية العظمى

الخلاصة

في هذه الدراسة تبين أن مرض تلين الدماغ المستجيب للعلاج بالثيامين قد سجل حدوثه في حالات فردية والتي إشتملت على وجود المرض في 8 جمال. لوحظت العلامات المميزة للمرض والتي شملت خلل في الرؤيا و العمى، المشي بدون هدف مع خطوات ترنحية، إرتجاف عضلات الرأس و إرتفاع الرأس نحو الأعلى (وقفة مراقب النجوم) مع رقاد ثم موت الحيوان إذا لم يعالج بالعلاج المحدد وبسرعة. تم اجراء الفحص التشريحي المرضي لحالة واحدة حيث تم التركيز على فحص انسجة الدماغ لغرض معرفة التغيرات المرضية العيانية والنسجية والتي اوضحت نتائج موجبة للاصابة بتلين الدماغ. إن نتائج البحث بينت أن علاج الحيوانات المريضة بحقن الثيامين أدت إلى إستجابة جيدة و سريعة بعد 6 ساعات من أول حقنة علاجية بالوريد. إن الإستجابة لمعظم الحالات المرضية للعلاج الخاص و المحدد و الذي يمكن أن يستعمل لتشخيص المرض إضافة إلى تفريقه عن الحالات المرضية العصبية المشابهة للمرض. اعتمادا على الاستجابة للعلاج بالثيامين اضافة الى الاعراض المرضية المميزة مع نتائج التشريح المرضي لدماغ الحيوان المضحى به والذي ساعد في تاكيد حصول المرض.

Introduction

Polioencephalomalacia (PEM), also called cerebrocortical necrosis, has been reported in dromedary camel (1,2), Llama (3,4), Alpacas (5), and in other ruminants such as cattle (6,7) sheep and goats (8-10). PEM is considered as metabolic neuropathogenic condition of ruminants (11,12). The disease is characterized by a disturbance of CNS that results from necrosis of the gray matter in ruminants (9,13), Llama (4) and Alpacas (5).

Affected camels may show signs of blindness, disorientation, loss of appetite, aimless wandering, tremors, helding the head high and the animal is reluctant to move followed by recumbency with paddling movement and death (5,14).

The naturally occurring disease is frequently associated with a deficiency of thiamine (vitamin B1) which was proposed to be attributed to dietary changes such as feeding of concentrates feeds rich in carbohydrates or high grain diet causing acidosis in camels and other ruminants (7,15-17) which encourage the growth of thiaminase bacteria that produce thiaminase type I and type II enzymes in the rumen and intestine causing destruction of all gut thiamine with progressive thiamine inadequacy in ruminants (13).

The disease has been also associated with ingestion of thiaminase I containing plants (16) that destroy any ingested or synthesized B1 in the rumen. Some studies showed that treatment with some anthelmintics (as amprolium) may have anti-thiamine activity causing PEM in camel and other ruminants (1,6,18).

On post mortem examination, gross lesions of the brain are characteristics, there is cerebral edema with soft swollen flattened gyri, the gray matter was necrotic and hemorrhagic in llama and other ruminants (4,19).

Under field condition, presumptive diagnosis of PEM cases in camels may depend on the pattern of clinical findings mentioned above which may be suggestive for the disease. However, in camels other diseases such as lead poisoning, focal symmetrical encephalomalacia, hepatic encephalopathy, or head trauma may show similar clinical signs (5). Laboratory confirmation of thiamine deficiency can be based on histopathology, blood biochemistry including erythrocyte transketolase levels, blood pyruvate and lactate levels (4,20).

The purpose of the present investigation was to study some of the epidemiological aspects of the disease in camels, to evaluate treatment with vitamin B1 as a tool for rapid diagnosis of polioencephalomalacia under conditions of normal farm management in the absence of laboratory facilities. Also using the response to treatment, specific signs and necropsy findings to differentiate PEM from other neurological diseases showing similar nervous signs.

Materials and methods

Clinical cases

During the period from 2005-2008, sporadic naturally occurring cases of 8 adult dromedary breeding camels with variable degree of clinical signs of blindness, anorexia, aimless wandering, disorientation, tremors and convulsion, head pressing with opisthotonus (Figure 1) and recumbency have been detected in different areas around Tripoli. Cortical blindness was evident through absence of menace reflex, persistence of palpebral reflexes with a pupil's response to light. The feeding regimen of the affected animals was variable but mostly ranging from concentrates to a pasture feeding.

Figure 1: Camel affected with PEM showing backward retraction of the head and recumbency.

Treatment and post mortem examination

The affected animals were treated intravenously with 10-20 mg/kg body weight of thiamine hydrochloride (vitamin B1) initially, and then it is followed by intramuscular injection of the same dose every 12 hours for a total of 4 occasions. One of camel was sacrificed and necropsy was performed for evidence of abnormal gross and histopathological lesions (Figure 3 and 4) of PEM or the lesions specific to other similar diseases conditions.

Results and discussion

Thiamine responsive PEM in ruminants is a neurological metabolic disorder (21). It has been reported in camels in different countries around the world (3-4).

In this study, the occurrence of disease cases in camel reported here was sporadic. Although it may occur in outbreak form with morbidity rate up to 100% in certain circumstances (15), but in most instances the condition is sporadic in camels (3,5,22,23). All affected camels showing clinical signs of PEM respond to treatment regimen within six hours after the first intravenous injection of thiamine.

This was clear through improvement of affected animals, regaining their normal appetite and disappearance of the specific clinical signs of the disease (Figure 2) particularly cortical blindness with gradual return of vision. This was in consistent with the findings of others (3,5,24).

Figure 2: Return of the animal's head to normal position and in standing position after treatment with thiamine.

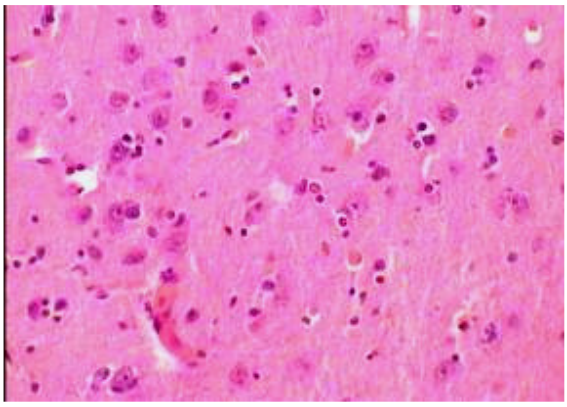

Figure 3: Cerebrum, gliosis and moderate neurophagia. Blood vessels are congested with slight endothelial swelling.

The naturally occurring polioencephalomalacia in camels and other ruminants is frequently associated with thiamine deficiency (1,2). This was supported by the recovery of spontaneously affected camels after thiamine therapy (2,3,5).

Early treatment of clinical cases of PEM in camels with overdosing of parenterally injected thiamine was the most effective form of treatment (2). Because thiamine is water soluble, it is quickly eliminated from the body through kidneys and, therefore there is little risk of overdosing (25). It was found that the mortality among PEM affected animals increase if treatment with thiamine is delayed for more than a few hours after the onset of signs (13). Mortality of untreated ruminants may be more than 50% (16).

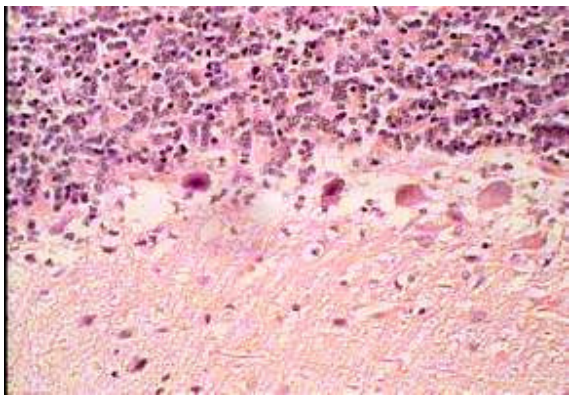

Figure 4: Cerebullum, Purkinji cell degeneration and necrosis. Degenerated cells are shrinkage and deeply eosinophic. Dead cells are resorbed partially or completely leaving empty spaces.

The inciting cause of occurrence of PEM in animals on pasture is unexplainable. However, it was reported that release of thiaminase is increased during diets changes from concentrates to pasture (26).

This study offers a method for early diagnosis of the disease through rapid response to parenteral treatment with no need to wait for the results of laboratory tests which takes time to confirm the tentative diagnosis since early treatment is necessary for ruminants with PEM (2,13) and the prognosis is considered favorable (27). Successful treatment of affected camels was attributed to early and aggressive therapy (3). It was reported that if there is no treatment on time, most animals with PEM may have a permanent brain damage within 3 days (12) and euthanasia may be recommended (19). Secondly, response to treatment with thiamine beside clinical signs of PEM is diagnostic (3,24,28) and quick, as the response to treatment was evident within 6 hours. Third, this method of diagnosis is of great help in excluding other serious diseases showing similar nervous signs in ruminants and camels as mentioned by others (5,12,13,16).

Laboratory tests which may be used to confirm clinical diagnosis in ruminants are based upon blood chemistry to determine the depressed level of blood thiamine and RBCs transketolase activity and rising of pyruvate and lactate levels (29, 30). Rumen and faecal samples may be tested for thiamine activity. However, these tests are not routinely available (19).

At necropsy, the post mortem gross findings observed in this study were limited to the brain particularly cerebral cortex. It shows diffuse cerebral edematous swelling

(5,10,31) and necrosis of cortical gray matter (4). Such lesions may be used as an additional proof for confirmation. Edema and necrosis of cerebral cortex could be explained by the high and specific requirement of the cortex for oxidative metabolism of glucose in which thiamine is essential cofactor in oxidative decarboxylation of some intermediate compounds in carbohydrate metabolism in the brain (20,13). So deficiency of thiamine will inhibit the thiamine-dependent reactions of glycolysis and tri-carboxylic acid cycle (32). Resulting in impairment of energy production in the neurons and cellular osmotic gradients is not maintained with subsequent neuronal swelling (19). This is followed by neuronal degeneration. In this study, the histological changes of the cerebrum (Figure 3) were gliosis and moderate neurophagia. Blood vessels are congested with slight endothelial swelling (4). Lesions of the cerebellum (Figure 4) are also present (13) with purkinji cell degeneration and necrosis. Degenerated cells are shrinkage and deeply eosinophilic. Dead cells are resorbed partially or completely leaving empty spaces.

The study was concluded that thiamine deficiency as the cause of PEM was the most probable and is supported by the recovery of naturally-occurring disease in affected camels following thiamine therapy, specific clinical signs and necropsy findings. Death may occur if affected animal is not treated with thiamine.

Acknowledgement

We acknowledged the department of pathology for their help in reading the results of gross pathology and histopathology.

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