معهد بحوث صحة الحيوان ـ الدقي . ج . م . ع.
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وباء البوليوانسفالومالاشيا في الأغنام
واستجابته للعلاج بالثيامين

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أجريت دراسة على حالات حملان وأغنام أصيبت با ال CCN واستخدمت كافة الطرق العملية للتشخيص وجرب العلاج بالثيامين عن طريق اعطاء الخميرة المجففه والمطحونه بنسبة ٢ - ٣٪ حيث إستجابت هذه الحالات للعلاج .

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AN OUTBREAK OF POLIOENCEPHALOMALACI AND ITS RESPONSE TO THIAMINE TREATMENT IN SHEEP

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(Received at 30/8/1980)

SUMMARY

A nervous condition involving mainly lambs and some ewes associated with cerebrocortical necrosis was discribed. Collected specimens were subjected to various laboratory investigations which proved negative for isolation of any causative agent or toxin.

A comparison was made with similar nervous diseases described in the literature.

The response to thiamine therapy of clinical cases proved the diagnosis.

The prophylactic effect of minced commercial dried yeast in 2-3% of the ration could be adviced.

INTRODUCTION

Polioencephalomalacia (PEM) known also as cerebrocortical necrosis (CCN)of sheep is a non infectious non contagious disease of sheep and cattle and was described in the fifties by JENSEN, et al. (1956), TERLECI and MARKSON (1959), YAMAGIVA and TAJIMA (1952).

The disease got a practical importance in this country in sheep as it occured in our local breeds in Egypt.

The aim of this report is to throw some lights - on the basis of some clinical and laboratory observation- on this disease in Egypt.

MATERIAL AND METHODS

At the North Tahrir Breeding Organization lot of deaths occured. In this area there were 3 flocks consisting of about 6 thousands heads. The disease appeared only in one flock of about 1100 animals of which 700 were lambs. The first cases were observed at the 2nd half of march, (1976). The main clinical signs were those denoting affection of CNS, which included disclination to move,incoordination,recumbency with periodic paddling of legs, foaming with complete absecence of eye preservation reflex and full dilatation of pupils.

The temperature was usually normal and the affected animal died after 2-3 days. Post mortum findings were not suggestive of any particular disease. No particular lesions could be observed by the naked eye. Specimens were taken from all internal organs and were subjected to various laboratory investigations including bacteriological, mycological, virological and chemical examinations which all proved negative results.

Histopathological examinations revealed characterestic pathological changes in the brain of animals affected with the disease and multiple degenerative necrotic processes in the cerebral cortex. By the end of March 48 lambs, between 3-8 months age and 7 ewes,died. Nine lambs were slaughtered in different stages during the manifistation of the clinical signs.

Specimens were taken from the respective cases for further laboratory investigations.

Treatment:

On the base of these clinical findings and the results of laboratory examination the disease is diagnosed as cerebral cortical necrosis (CCN). Thiamine therapy was suggested as that described by FENWICK (1967) and DALY (1968). The drug was given in 10% solution at a rate of 400 mg for young sheep and up to 600 mg for adults partly by the intravenous route and partly intramuscularly. If requried, this dosage was repreated several times of 8-24 hrs. Trials was made firstly on 45 isolated cases which were unable to move with slight incoordination of muscles. Partial response was observed in about ten of them. The other five cases regained their heath and with a full vitality after about 5 hours. Minced commercial dried yeast in a percentage of 3-4% of the total rations was also offered as preventive measure which proved to be of great benefit in reducing and then stopping the appearance of new clincal cases in the flock.

DISCUSSION

Although polioencephalomalacia is a condition of increasing importance, only few epizootalogical studies of the condition in sheep have been undertaken, that of HARTELY and KARTER (1959) in New Zealand, and SPENCE et al. (1961); in Great Britain and GABBEDY and RICHARDS (1977), in Australia. The reached diagnosis in this report which is based on the described clinical signs and P.M. findings simulates that reported by PASS (1968) and DIRKSEN and HOFMANN (1974) who stated that no infective agents or toxins have been isolated from clinical cases and the conditions has never been transmitted from affected to unaffected animals. As a differential diagnosis hypomagnoesamic tetany, enterotoxaemia, lead poisoning, gid, meningitis and encephalitis, Listeriosis, Colisepsis were taken in consideration.

The cause of the disease is not fully understood, however, the most important findings is the focal necrosis dissiminated throughout the cerebral cortex. Diagnosis being frequently made on clinical grounds and based on response to thiamine therapy (GABBEDY and RICHARD, 1977). DAVIS et al. (1965) described a similar disease recommended that affected animals could be treated successfully with thiamine which supported our findings. LEWIS et al. (1967) produced lesions in lambs similar to CCN by feeding a thiamine deficient diet. More recently, it was suggested that thiaminase producing organisms, Cl. sporogenes (SHREEVE and EDWIN, 1974) and Bacillus species (MORGAN and LAWSON, 1974) might play a definite role in the development of the condition. The present outbreak simulated that menitoned by SPENCE et al. (1961) who stated that the disease involving primary lambs and occured during the period from April to July.

PASS (1968), agreed with our view of feeding a vitamine supplement containing thiamine to the entire flock, once the condition has been established in the farm.

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