Pathological changes in calves that died from experimental water intoxication

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ABSTRACT

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The pathology of calves that died from experimental water intoxication was investigated. Oedema of the brain and urinary bladder, and renal damage were significant pathological findings in these calves. The findings were attributed to positive water balance in calves suffering from water intoxication.

Keywords: Brain, calves, oedema, pathological changes, renal damage, urinary bladder, water intoxication

INTRODUCTION

Most authors have reported that no significant gross lesions occur in calves that die from water intoxication (Wright 1961; Kirkbride & Frey 1967). However, Gibson, Counter & Barnes (1976) described post-mortem lesions in a naturally occurring case of the disease. They described the carcase to be in poor body condition with severe ruminal tympany. The lungs were congested, with slight interlobular oedema. Brain oedema, especially in the white matter, was evident. In an attempt to better understand water intoxication, the sequelae of calves suffering from the disease were investigated to determine whether other pathological changes occur in cases of the disease. Such findings would be important for confirmatory diagnosis of water intoxication.

MATERIALS AND METHODS

Experimental design

A total of ten two-week-old male calves (five Friesians and five Ayrshires) were used for the experiment. They were randomly divided into two groups of five each. One group was used as a test group while the other served as a control. Each animal was ear-tagged and given an identification number according to its respective group.

Feeding and management of the two groups of calves

Cow milk was fed to the calves in the test group at 10% live body mass (Einsminger 1976). Milk was provided to each calf by bucket in two equal portions, one in the morning and one in the evening. The diet of each calf was also supplemented with 1,5 kg of early weaner calf pellets (Unga Feeds Ltd, Kenya), given daily in three portions. Hay was provided *ad libitum*. The calves were given no water other than that contained in the milk.

At the age of 3 months, the calves were weaned and allowed to drink water *ad libitum* from a bucket.

Calves in the control group received the same treatment as those in the test group did, except that water was provided *ad libitum* throughout the whole experimental period. Salt licks were provided *ad libitum* in plastic buckets. The water was also provided in plastic buckets, and replaced three times a day to minimize contamination.

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Induction of water intoxication

Water intoxication was induced twice in the test calves, first when they were weaned at the age of three months and again one month after weaning. The disease was induced by allowing the calves to take water *ad libitum* at these times. Before weaning, the calves did not receive any water other than that contained in the milk. After weaning, the calves received only 3 ℓ of water per day.

After the first induction of the disease, the calves that were terminally ill were treated as recommended by Harwood (1976), with some modification. They were given a 10% saline-dextrose solution intravenously at a dosage rate of 100 mℓ/total body mass, and this dosage was repeated after 24 h. A 5% saline solution was also provided *ad libitum* for oral consumption.

After the second induction of the disease, no treatment was offered even to the terminally ill calves so as to determine the sequela of water intoxication.

Post-mortem examination

All calves that died from the disease were examined for gross lesions, and sections of the kidney, liver, urinary bladder and cardiac muscle were taken for histopathological examination.

The brains were fixed in formalin for 7 d before being sectioned for histological examination. The sections were routinely embedded, mounted on glass slides and stained with haematoxylin and eosin (HE).

RESULTS

After the first induction, four out of five animals showed all the signs of water intoxication, with varying degrees of severity (Hannan 1965; Kirkbride & Frey 1967; Harwood 1976). The signs included hyperesthesia, muscular tremors, nystagmus, lethargy and haemoglobinuria (Fig. 1). Other signs were blindness, salivation, colic, cardiac arrhythmias, and oedema of the eyelids. However, after re-induction of the disease, all animals suffered from mild to severe cases of water intoxication, and two of them died.

Complete post-mortem examinations were performed on all the calves that died of water intoxication. Grossly, approximately 100 mℓ of a straw-coloured fluid was present in both the abdominal and thoracic cavities. The fat in the coronary grooves of the heart appeared oedematous and jelly-like, indicating serous atrophy of fat. The urinary bladder contained about 300 mℓ of red urine. The kidneys had a dark red appearance. The sulci on the surface of the brain were flattened, indicating a degree of oedema.

On microscopic examination, the histological sections revealed oedema and haemorrhage of the brain parenchyma (Fig. 2). Cerebral veins and venules were congested and filled the Virchow-Robin spaces. There was also mild dilatation and eosinophilic fluid in the Virchow-Robin spaces (Fig. 3). The kidney tissues showed marked multifocal lymphocytic infiltration of the cortical interstitium (Fig. 4A and 4B) and fibrosis and oedema around the arterioles (Fig. 5). The renal cortex had areas of haemorrhage and congested capillaries (Fig. 6). The epithelial cells of the kidney tubules had fine red granules. There was severe oedema and desquamation of the stratified mucosa of the urinary bladder (Fig. 7). Oedema was also present in the muscular layer of the urinary bladder (Fig. 8).

DISCUSSION

In this study, water intoxication was induced by the method of Kirkbride & Frey (1967) with some modifications. The calves in the test group were

FIG. 1 Sternal recumbency in a calf suffering from a severe case of water intoxication. The calf was also showing hyperesthesia, muscle tremors and haemoglobinuria

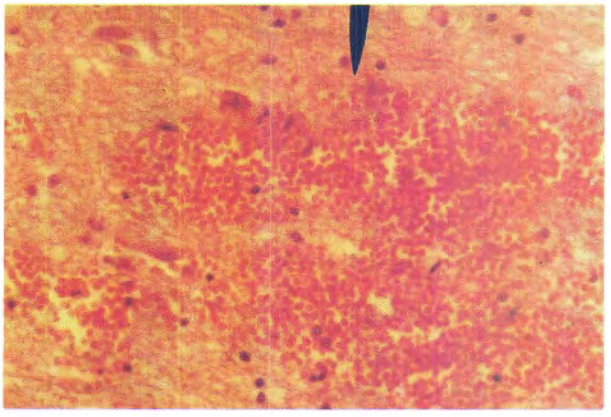

FIG. 2 A histological section of the brain of a calf that died of water intoxication, showing areas of haemorrhages (HE x 400)

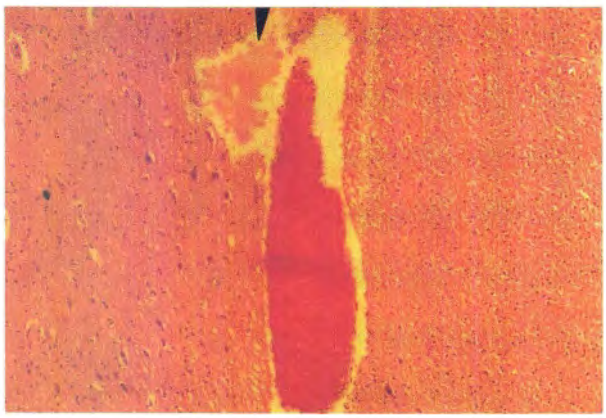

FIG. 3 A histological section of the brain of a calf that died of water intoxication, showing venous congestion and oedematous fluid (arrow)
(HE x 100)

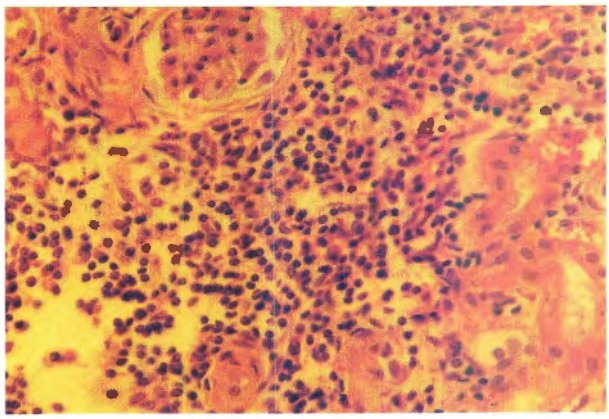

FIG. 4B The same histological section as Fig. 4A at a higher magnification
(HE x 400)

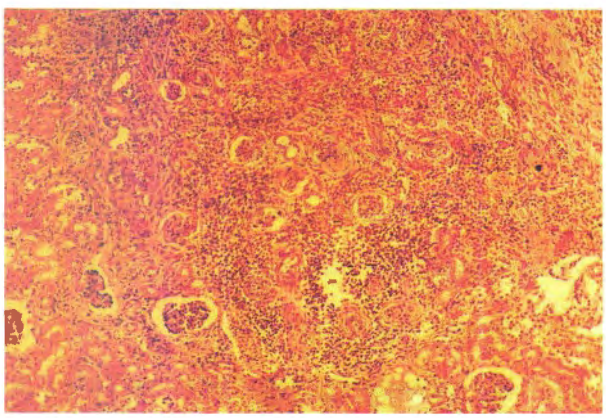

FIG. 4A A histological section of the kidney of a calf that died of water intoxication, showing lymphocytic infiltration
(HE x 100)

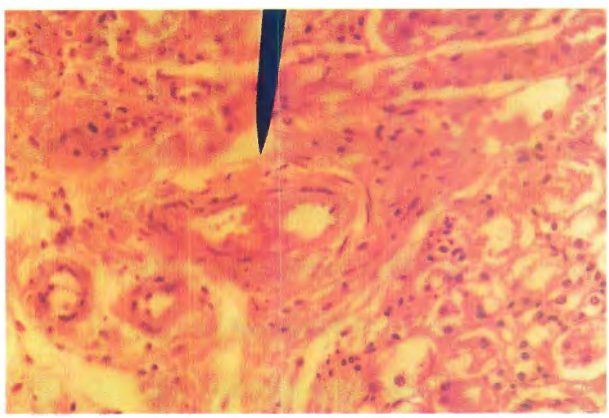

FIG. 5 A histological section of the kidney of a calf that died of water intoxication, showing fibrous tissue formation around the capillaries
(HE x 400)

deprived of both salts and water because naturally occuring cases of the disease in Kenyan farms have been associated with lack of both salts and water in the feed of the calves.

The findings in this study are in agreement with those of Gibson *et al.* (1976). However, additional findings were also recorded in the current study. These include histological data of the brain, kidney and the urinary bladder. Gibson *et al.* (1976) most probably did not carry out a histological examination of the animals as they were dealing with a naturally occurring water intoxication in the field. The experimental set-up in the current study was such that facilities for histological work were available.

In the current study, post-mortem examination was carried out on animals that died after the re-induction of water intoxication and therefore the changes seen in the kidney had most probably resulted from acute damage that occurred after the first induction of the disease. The presence of lymphocytic infiltration, together with fibrous tissue formation around the blood vessels, indicate long-standing damage that had already started healing.

The histopathological findings observed in this study may be explained on the basis of water metabolism in the body (Blood & Radostits 1989). The calves were in a state of positive water balance. The excess water circulating in the body lowered the osmotic pressure of the blood. This led to increased absorption of water down a concentration gradient into the cells. In unorganized tissues such as blood, the cells became turgid and ruptured, causing haemolysis. This is evident in the current study as shown by the presence of haemoglobinuria in the clinical signs, and venous congestion at post-mortem. In organized

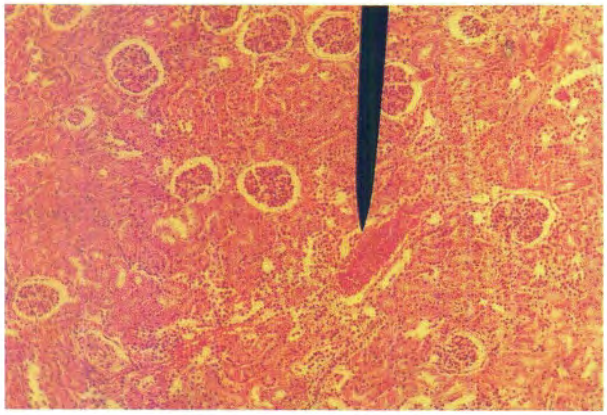

FIG. 6 A histological section of the kidney of a calf, showing capillary congestion in the cortex
(HE x 100)

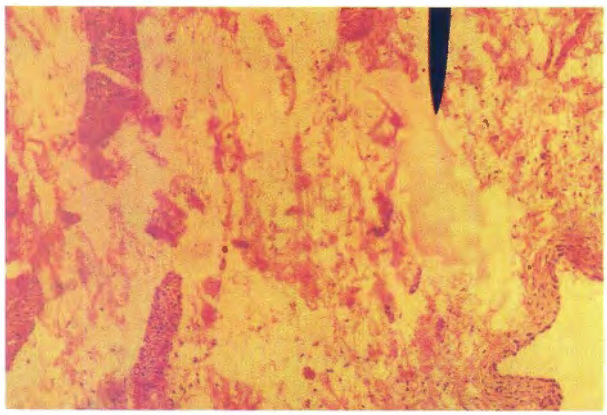

FIG. 7 A histological section of the urinary bladder of a calf that died of water intoxication, showing areas of oedema beneath the stratified mucosa
(HE x 40)

tissues, excess water caused oedema. In the current study, oedema was present in the brain (Fig. 3) and is postulated to be the cause of nervous signs. Oedema was also present in both the kidney and the urinary blood (Fig. 6 and 7).

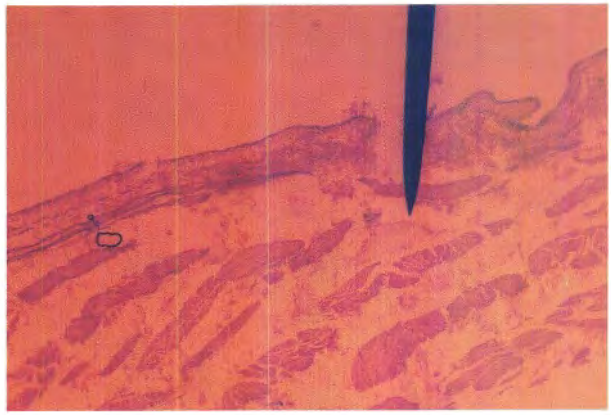

FIG. 8 A histological section of the urinary bladder of a calf that died of water intoxication, showing oedema of the tunica muscularis
(HE x 40)

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Rachel N. Gitau typed the manuscript.

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