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RESEARCH NOTE

CEREBRAL BABESIOSIS IN A NEW-BORN CALF

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ABSTRACT

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A case of intra-uterine transmission of *Babesia bovis* is reported. The calf was born normally but showed signs of intravascular haemolysis and nervous involvement 24 h after birth. It died shortly afterwards from cerebral babesiosis. The dam was not clinically affected.

Résumé

BABÉSIOSE CÉRÉBRALE DANS UN VEAU NOUVEAU-NÉ

Les auteurs constatent la transmission intra-utérine de Babesia bovis. *Le veau, né en bonne santé, a manifesté 24 h plus tard des signes d'hémolyse intravasculaire et des symptômes nerveux. Peu après il est mort de babésiose cérébrale, alors que sa mère n'a manifesté aucuns signes cliniques de la maladie.*

INTRODUCTION

Babesiosis, caused by intra-uterine transmission of various *Babesia* spp., has already been reported in the literature (Neitz, 1956). The disease is rarely found in cattle, Enigk (1942) and Neitz (1956) having reported only 4 such cases in their reviews. Two of these were caused apparently by *Babesia bigemina*, and 2 by *Babesia bovis*.

This note is a report on a case of cerebral babesiosis in a calf which became infected with *B. bovis in utero*.

HISTORY AND CLINICAL SIGNS

The calf, a male Brown Swiss, was born at Cwaka College, Kwazulu on 12 September 1972. Birth was slightly protracted but otherwise normal, and no noticeable signs of disease were observed in either the calf or the dam. One day after birth, however, the calf refused to suckle and an examination revealed ataxia, haemoglobinuria and yellowish mucous membranes. No subsequent clinical observations were made and the calf was found dead in its pen shortly afterwards.

PATHOLOGICAL FINDINGS

Autopsy

An autopsy was performed within a few hours of the death of the animal. The major macroscopic lesions were haemoglobinuria, giving the urine a port-red discoloration, *tumor hepatis*, *tumor splenis* and brain congestion.

Bacteriological examination

Escherichia coli was isolated from the gut of the animal but not from other organs.

Examination of blood and brain smears

A blood smear made from the tip of the tail and stained with Giemsa's stain revealed parasites in approximately 0,05% of the red blood cells. Most of the parasites were typical single or paired forms of *B. bovis*, but some organisms appeared to be rounded off, due possibly to post mortem degeneration. The parasitized cells often formed aggregations with up to 10 cells cohering or in close proximity to one another. A brain smear was made from the cerebral cortex and stained in the same way as the blood smear. It revealed massive accumulations of parasitized red blood cells in the capillaries with a resultant marked distension of these vessels (Fig. 1). The percentage infection of the red blood cells in the capillaries exceeded 90%, with many of the cells harbouring 2, 3 or even 4 parasites. Some apparently extracellular babesias were also seen. The parasites in the brain capillaries appeared to be somewhat larger than those seen in the blood smear, and many were rounded.

Histo-pathology

Pieces of cerebellum, midbrain, hippocampus and cerebral cortex were collected and fixed in 10% formalin. These were subsequently embedded in paraffin wax, sectioned at 4–6 μm and stained with haematoxylin-eosin (HE) stain, Giemsa's stain, haematoxylin-phloxine (HP) stain, the periodic acid-Schiff (PAS) reaction and PAS reaction combined with Mallory's phosphotungstic acid haematoxylin (MPAH) stain, and Schmorl's stain.

Severe capillary congestion was seen in all sections but was most pronounced in the granular and molecular layers of the cerebellum and grey matter of the cerebral cortex. These vessels were distended with erythrocytes, almost all of which were parasitized (Fig. 2). An examination of larger vessels within the brain and meninges, which were also congested, did not show such a high percentage of parasitized cells, but those present were more abundant along the vessel walls where they had a pavementing effect (Fig. 3).

Small, acute, perivascular haemorrhages were observed in the granular, Purkinje cell, the molecular layers and white tracts of the cerebellum in the midbrain (Fig. 4), the hippocampus and the white

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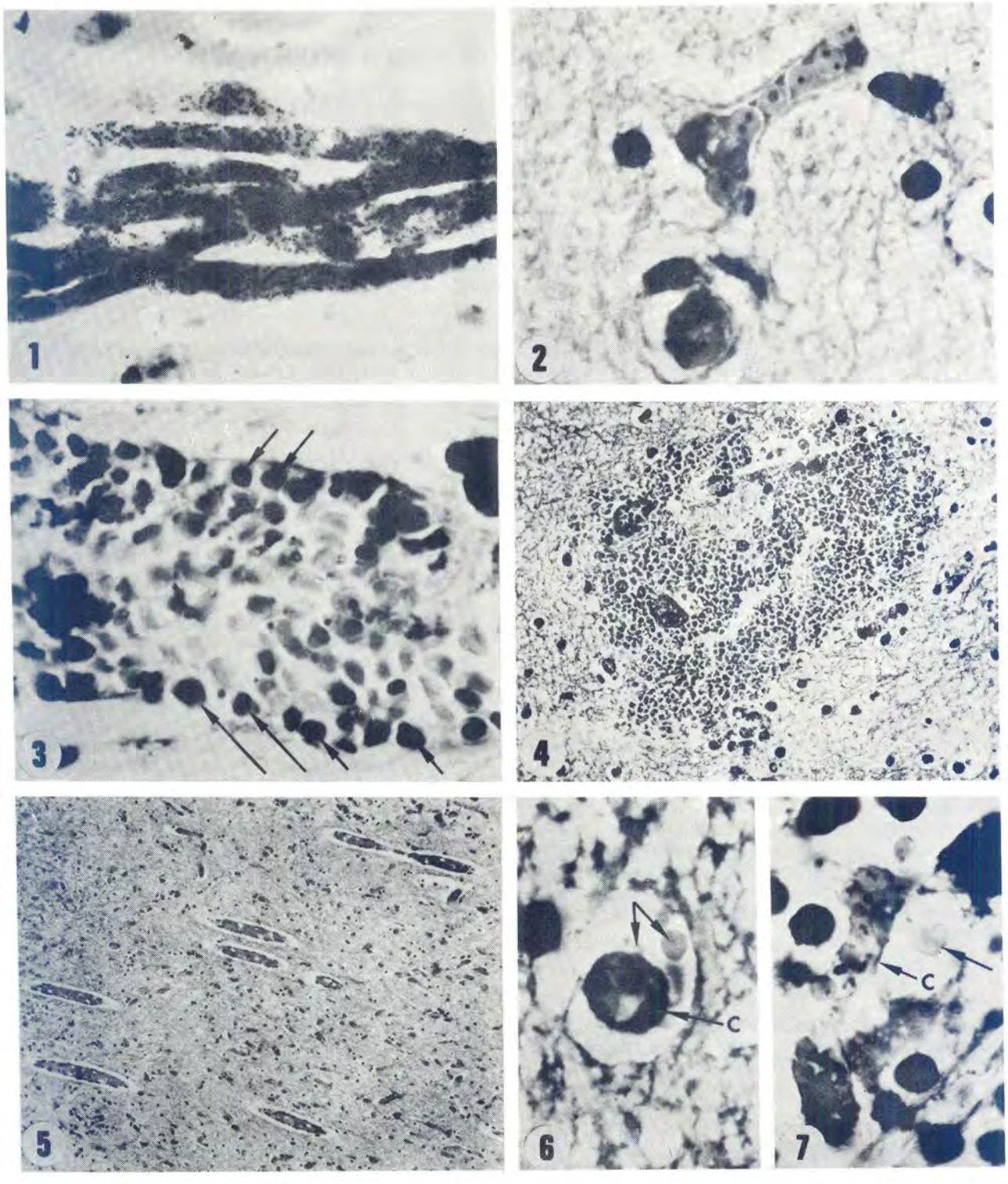

FIG. 1 Brain smear, showing severely distended capillaries. The parasites appear as numerous dark staining specks. Giemsa ×600

FIG. 2 Cross and longitudinal sections through 2 capillaries in the midbrain. Note high infection rate of erythrocytes. HE ×1 200

FIG. 3 Longitudinal section of larger blood vessel in midbrain, with majority of infected erythrocytes (arrows) located along the vessel wall. Giemsa ×1 200

FIG. 4 Section through small haemorrhage in midbrain. Note the necrotic capillary (arrow). HE ×600

FIG. 5. Section of midbrain showing clear spaces around bloodvessels. HE ×300

FIG. 6 Cross section of capillary (C) in cerebellum with a clear space surrounding it. Note spherical droplets in this space (arrows). HE ×1 200

FIG. 7 Longitudinal section of capillary (C) in cerebellum. Droplet with fine processes is present in the surrounding clear space (arrow). HE ×1 200

matter of the cerebral cortex near the junction with the peripheral grey matter. A few capillaries within these haemorrhages were necrotic.

Most of the blood vessels were surrounded by a clear space (Fig. 5) which, in some cases, contained spherical to teardrop-shaped, pink-staining droplets, ranging from 1–15 μm in diameter. Some were smooth in outline (Fig. 6) while others had fine processes (Fig. 7). They were stained by the PAS reaction, PAS reaction plus MPAH stain and HP stain, but not by Schmorl's stain for lipofuschin. In the cerebellum, these droplets were seen in large numbers around capillaries in the molecular and Purkinje cell layers and the Purkinje cell layer was almost uniformly separated from the molecular layer. Only a few droplets were seen in the white tracts of the cerebellum. In the cerebrum they were most prevalent in the grey matter.

Discussion

This is the first reported case of cerebral babesiosis due to infection with *B. bovis*. It is noticeable (De Vos, unpublished observations, 1973) that this organism shows a marked predilection for capillaries of the brain with resultant congestion of this organ and, in some cases, clinical signs indicating nervous involvement in the later stages of the disease. This predilection for capillaries has also been reported for *Babesia argentina* (Rees, 1934; Callow & MacGavin, 1963; Seneviratna, 1963; Uilenberg, 1965; Wright, 1972a,b) and *Babesia berbera* (Tchernomoretz, 1943), but not for *B. bigemina* (Rees, 1934; Callow & Johnston, 1963). Although still considered by many to be different species, it has been shown that *B. bovis, B. argentina* and *B. berbera* are in fact closely related (Goldman & Rosenberg, 1974), and may even be synonymous (Riek, 1968).

Callow & McGavin (1963) described the neuropathology of 4 cases of cerebral babesiosis due to *B. argentina* infections. They reported severe capillary congestion which was most severe in the cerebellum and molecular layer of the cerebral cortex. In one case, they found numerous perivascular haemorrhages in the grey and white matter of the medulla oblongata and midbrain. The findings in the present case were virtually the same except that the medulla oblongata was not examined, and haemorrhages were found in the granular, Purkinje cell and molecular layers, and white tracts of the cerebellum.

The same authors also reported oedema appearing as wide spaces around blood vessels and neurons and the separation of fibres in the white matter. Similar spaces were seen in this calf, and there was also a separation along the Purkinje cell layer which appeared to be related to oedema rather than artefacts. The eosinophilic droplets in the spaces were similar to those described by Pienaar, Basson & Van der Merwe (1966) in a study of the neuropathology changes in bovine heartwater. The staining reaction indicated that these globules were mucoprotein in character. Droplets with fine projections were also seen by these authors and were interpreted as being within the glial processes, or sucker feet, of astrocytes attached to blood vessels. According to them, this constitutes strong evidence of intracellular oedema or brain swelling. They also observed increased eosinophilic cytoplasm in astrocytes in the white matter.

The absence of any clinical signs of the disease in the dam at birth effectively excludes the possibility of a massive infection of the calf through contact with infected maternal blood at birth. Infection acquired from ticks can similarly be excluded, as the prepatent period of infection with *B. bovis* infection after tick infestation is not less than 5 days (Potgieter, personal communication, 1973). The severity of the symptoms seen 24 h after birth, therefore, undoubtedly indicates that there was already an infection at birth, even though the calf appeared to be clinically normal at the time.

Hall (1960, 1963) demonstrated a sterile, passive immunity in new-born calves of immune dams, not shared by the offspring of susceptible animals. He suggested that this immunity was probably transferred from the mother to the offspring via the colostrum. The fatal case of intra-uterine-transmitted babesiosis described here seems to indicate that calves of infected, and therefore immune, dams may well be highly susceptible before and at birth. This gives weight to Hall's suggestion of a colostral transfer of immunity.

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