NON-SUPPURATIVE MENINGOENCEPHALITIS IN DROMEDARY CAMELS IN SAUDI ARABIA

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ABSTRACT

Histological evidence of a non-suppurative meningoencephalitis was detected in 5 adult camels (*Camelus dromedarius*), brought for postmortem examination with a clinical history of nervous signs. The inflammatory reaction was dominated by intense lymphocytic infiltration and perivascular cuffing, microgliosis, and degeneration and necrosis of some individual neurons in the cerebral and cerebellar cortex. Severe vascular changes characterised by diffuse capillary congestion and focal haemorrhages in the white matter were frequently observed. Leptomeningitis was further indicated by fibrinous exudation and inflammatory cellular infiltration of lymphocytes, macrophages and glial cells. The causative agent was not yet determined. However, the purely lymphocytic nature of the inflammatory reaction is highly suggestive of viral infection.

Keywords: Dromedary camel, encephalitis, meningoencephalitis, Saudi Arabia

Infectious diseases of the nervous system are not adequately studied in camels. However, the majority of clinical cases with nervous manifestations observed by camel owners were mostly attributed to rabies virus infections (El Mardi and Ali, 2001; Ali et al, 2004). Listeric encephalitis which is very common in sheep and goat, was only reported in llamas (Butt et al, 1991; Hamir and Moser, 1998) but not in dromedary or bactrian camels. A sudden onset of an ambiguous disease with neurological disturbances was recently observed in some individual camels in the eastern province of the Kingdom of Saudi Arabia. However, none of the affected cases was diagnosed because of the rapid death of animals before reaching the nearest veterinary clinic. The following report provides gross and histopathological evidence of the occurrence of a non-suppurative meningoencephalitis highly suggestive of a viral infection in all affected cases.

Materials and Methods

The materials of this report comprised 5 dead bodies of dromedary camels that were submitted for postmortem examination. The dead camels were brought at weekly intervals from the same location in the Saudi desert (Al-Ahsa, Eastern Province) during the period from April to June 2007. One animal was a male and the others were females and their age ranged from 5 to 8 years. The attached clinical history in each case indicated the occurrence of neurological disturbances characterised by ataxia, incoordinated staggering gait and occasional nervous excitation. However, profuse salivation, aggressive behaviour, paralysis or muscular spasms were not reported. Death occurred after a short period of recumbency as indicated in the clinical history. The camel owners also indicated the occurrence of previous mortalities from similar neurological disturbances. Detailed postmortem examination of individual animals was performed by standard necropsy procedures. The head was removed by decapitation from the atlantooccipital joint and the intact brain and meninges were first exposed by opening the skull using an electric saw. Lateral incisions were made on each side from the foramen magnum to the median surfaces of the orbital fossa together with a transverse incision behind the orbits. The dissected bones were then lifted out and the exposed dura mater was carefully examined, incised and removed from the dorsal part of the brain. The whole brain was then taken out as one unit for visual examination of gross lesions. The two cerebral hemispheres were separated by sectioning through the longitudinal fissure. Cross sections were also made on the junctional areas of the occipital and parietal lobes of the left and right hemispheres and similar sections were further made on the frontal lobe with serial sections across the cerebellum, midbrain and medulla oblongata. Small pieces of the cerebrum, cerebellum, midbrain and medulla oblongata were then fixed in 10% buffered formalin solution for histopathological

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processing and staining with haematoxylin and eosin (H&E) by the paraffin wax method described by Drury and Wallington (1980).

Results

The gross and microscopic lesions in the brain of 5 camels were strikingly similar to each other. The gross changes in all animals were mainly dominated by moderate to severe congestion of meningeal blood vessels together with the presence of numerous tiny haemorrhages on the brain substance, particularly, in the white matter. Other pathological abnormalities as cerebral abscesses, parasitic cysts or traumatic, neoplastic or congenital lesions were not encountered in either of these cases. No significant gross lesions were found in other visceral organs.

On the other hand, the histological examination of the brain, revealed typical features of an acute and/or subacute non-suppurative lymphocytic meningoencephalitis characterised by diffuse infiltration of lymphocytes with prominent lymphocytic perivascular cuffing all around blood vessels and capillaries (Figs 1 and 2). The intense lymphocytic infiltration was accompanied by diffuse microgliosis, focal malacia of the white matter and individual cell necrosis and degeneration of scattered neurons in the cerebral and cerebellar cortex. In addition, wide spread capillary congestion and focal haemorrhages were consistently observed in the white matter of the cerebrum and cerebellum of all affected animals (Fig 3). Tiny haemorrhages of extremely small size were also present at the periphery of the granular layer of the cerebellar cortex adjacent to Purkinje cells (Fig 4). Leptomeningitis was further evident by fibrinous exudation and inflammatory infiltration of mainly lymphocytes with some other mononuclear cells such as macrophages and microglial cells in the meningeal spaces (Fig 5). Neutrophils and other polymorphonuclear leukocytes were not detected in the inflammatory reaction of all examined sections and there was no evidence of the presence of intracytoplasmic or intranuclear inclusion bodies in the neurons or glial cells.

Discussion

The present study demonstrated the occurrence of non-suppurative meningoencephalitis in 5 adult camels with a clinical history of nervous signs. Nevertheless, the aetiological agent of such conditions was not yet determined but the purely lymphocytic nature of the inflammatory reaction was highly suggestive of a viral infection. Non-suppurative or lymphocytic inflammation of the central nervous system is the major finding of a wide range of specific viral infections including rabies virus (Lyssavirus), Borna disease virus (BDV), flavivirus, togavirus and many others (Storts and Montgomery, 2001; Radostits *et al*, 2007).

Rabies virus was found to be the most common cause of non-suppurative encephalitis in domesticated and wild animals as well as in human beings all over the continents. The incidence of rabies in camels has previously been reported in many locations in Asia, Africa and in the Arabian Peninsula (Afzal et al, 1993; Atta et al, 1993; El Mardi and Ali, 2001, Ali et al, 2004). It is well observed that rabies produces the most severe type of non-suppurative meningoencephalitis with very prominent lymphocytic perivascular cuffing and frequent presence of eosinophilic inclusion bodies (Negri bodies) in the cytoplasm of largesized neurons (Storts and Montgomery, 2001). These intracytoplasmic inclusions have long been considered highly pathognomonic, despite the fact they are not always detectable in all cases. In the present situation, rabies might be a possible diagnosis in all of the 5 cases because of predominantly purely lymphocytic and severe nature of the inflammatory infiltration. However, the absence of intracytoplasmic inclusions in the neurons of all affected animals together with the lack of typical signs characteristic of rabies previously reported for rabid camels such as profuse salivation, aggressive behaviour, attacking objects and biting their body (El Mardi and Ali, 2001; Ali et al, 2004) might throw some doubt in the presently described cases in which none of these characteristic signs were indicated in the attached clinical history. Borna disease is another very common viral infection causing nonsuppurative encephalitis in domestic animals. The disease primarily occurs in horses and sheep, although many other animals including cats, cattle, human and non-human primates were also found to be susceptible to natural or experimental infections (Kao et al, 1993; Lungren and Ludwig 1993; Hornig et al, 2003). The incidence of Borna disease has previously been reported in llamas (Lama lama) and alpacas (Lama pacos) (Rott and Becht, 1995) but not in the dromedary or bactrian camels. The possibility of infection with the Borna disease virus should not be ruled out in the present situation due to the previous occurrence of the disease in related camelids and the wide spectrum of host susceptibility of the virus in various animal species. Furthermore, the possible involvement of many arthropod-borne



Fig 1. Diffuse infiltration of lymphocytes with prominent lymphocytic perivascular cuffing on the molecular layer of the cerebral cortex (H&E ['] 100).



Fig 4. Tiny haemorrhages at the peripheral zone of the granular layer of the cerebellar cortex (H&E ´ 400).



Fig 2. Wide-spread capillary congestion involving various parts of the cerebellar cortex. Also note lymphocytic perivascular cuffing and tiny haemorrhages (H&E ´ 40).



Fig 3. Multiple areas of scattered haemorrhages on the multiform layer of the cerebral cortex (H&E ' 40).



Fig 5. Fibrinous exudation and cellular infiltration on the meningeal spaces (H&E ′ 40).

viruses of a similar epidemiological impact such as flaviviruses and togaviruses including Eastern, Western and Venezuelan equine encephalitis viruses should also be considered in connection with camel encephalitis. Therefore, the use of highly specific laboratory techniques such as immunohistochemical methods, serological tests (e.g. ELISA) and molecular characterisations are essentially needed for further diagnosis of similar cases of suspected viral encephalitis in camel populations in the vast desert of the Kingdom of Saudi Arabia.

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