MELIOIDOSIS WITH ACUTE MENINGOENCEPHALOMYELITIS IN A HORSE

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SUMMARY: A case of acute meningoencephalomyelitis caused by infection with *Pseudomonas pseudomallei* is described. Clinically there was inability to stand, opisthotonus, facial paralysis and nystagmus, rapidly progressing to violent struggling.

Gross examination revealed malacia and haemorrhage in the medulla oblongata and adjacent spinal cord. Microscopically there were disseminated focal neutrophilic accumulations in affected areas, perivascular cuffing with mononuclear cells and lymphocytes and marked oedema. Intracellular bacteria were identified in sections stained by the Giemsa method.

Introduction

Infection of horses with *Pseudomonas pseudomallei* appears to occur infrequently. A recent report on equine melioidosis in France, however, concerns 180 clinically recognised cases of which 44% were serologically positive and 4% died (Desbrosse *et al* 1978). Colic was the most frequently observed abnormality in these horses.

Other reports of equine melioidosis by Bourrier (1978) in France, Baharsefat and Amjadi (1970) in Iran, Stanton *et al* (1927) and Davie and Wells (1952) in Malaysia, described clinical signs and lesions mostly involving the lungs or lymphatics of the legs. Laws and Hall (1963) described one horse which died within 24 hours of first being noticed sick; no gross lesions were seen but a suppurative meningoencephalitis of the medulla and pons was observed histologically.

A further equine case of melioidosis with dramatic and fulminating nervous signs and pronounced gross and microscopic lesions of meningoencephalomyelitis is described in this report.

History and Clinical Findings

A 15-month-old crossbred colt was first observed sick by its owner at 0700 hours on 25 February 1979 when it was in lateral recumbency and unable to stand. Later in the day the owner observed that the colt was "struggling violently". The sudden onset of clinical signs was emphasised by the fact that on the previous evening the animal was observed to be galloping and apparently normal. Approximately 2 months previously a laceration along the trachea in the mid-ventral neck region was reported to have healed quickly following suturing and injection of long-acting penicillin.§

Clinical examination at 0900 hours on 25 February revealed a rectal temperature of 39.6°C, a pulse rate of 75/min. and respiratory rate of approximately 50/min. The mucous membranes and conjunctivae appeared normal. Opisthotonus to the left in the region of the second and third cervical vertebrae was observed, and there was facial paralysis on the left side. Nystagmus and apparent protrusion of the eyeballs were also apparent.

During examination the colt exhibited spasmodic generalised contractions of extensor muscles and a type of paddling motion. It was treated at this time with chloramphenicol succinate of rolitetracycline,¶ isopyrin and phenylubutazone,** as well as hyoscine-n-butylbromide and dipyrone††.

As no remission of signs occurred following treatment, the animal, at the owner's request, was euthanatised at 1600 hours. The head only was submitted for pathological and bacteriological examination.

Pathological Findings

There was a heavy infestation of ear mites *(Psoroptes cuniculi)* associated with large plugs of ceruminous exudate which almost occluded the external auditory canals on both sides. Thickening of the leptomeninges associated with loss of

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§Duplocillin injection, Pitman-Moore/Ethnor, 1-5 Khartoum Rd, North Ryde, New South Wales, 2113.
¶Protercicline, Arnold's of Reading Pty Ltd, 10/42 Barry Pde, Bayswater, Victoria, 3153.
**Tomanol, Bayer Australia, 47-67 Wilson St, Botany, New South Wales, 2019.
††Buscopan Compsoitum V, Boehringer Ingleheim Pty Ltd, 50 Broughton Rd, Artarmon, New South Wales, 2064.

translucency — especially around the brain stem — was apparent. Several pronounced areas of meningeal thickening up to 8 mm in diameter and which appeared to be suppurative foci, were observed in fissures of the cerebral hemispheres.

Transverse incisions of the brain and contiguous spinal cord revealed extensive bilateral malacia and associated disseminated focal haemorrhages. These changes were most prominent throughout the small portion of spinal cord examined and the medulla oblongata (Figure 1); they probably also extended further into the adjacent cervical cord, which unfortunately was not submitted for examination.

Representative blocks of tissue from the lesion and other parts of the brain were fixed in 10% buffered neutral formalin, embedded in paraffin, and sections cut at 6 μm were stained with haematoxylin and eosin (H & E) and by the Gram and Giemsa methods.

Microscopic examination of the thickened cerebral meninges revealed oedema and only slight infiltration of mononuclear cells, lymphocytes and scattered neutrophils. In the most severely affected tissue (anterior spinal cord and medulla at obex) normal parenchyma was replaced in many locations by large accumulations of neutrophils and fewer peripheral histiocytes. Encapsulation of these accumulations was not apparent. There was pronounced cuffing of larger blood vessels in affected areas with large mononuclear cells, lymphocytes and occasional neutrophils (Figure 2). Damage to smaller vessels in and adjacent to neutrophilic accumulations was indicated by marked oedema and numerous focal haemorrhages. Swelling of endothelial cells was often apparent but distinct vasculitis was not observed. Swollen axons were often present in vacuolated areas (Figure 3). In brain parenchyma away from these lesions there was slight gliosis and indications of neuronal degeneration.

Bacteria were not identified in sections stained with H & E, or by the Gram method, but in Giemsa-stained sections — especially at the periphery of neutrophilic accumulations — bacilli were seen to be located both extracellularly and within macrophages (Figure 4). Oedema of the meninges of the medulla was confirmed and was accompanied by a slight, but diffuse lymphocytic infilatration.

Qualitatively similar, but much less pronounced lesions to those described, extended into the central and cranial parts of the medulla. Extension of lesions into the pons and cerebellum appeared to be minimal as the only change observed in these locations was slight perivascular cuffing.

Bacteriological Findings

Samples of medulla and midbrain were examined. *P. pseudomallei* and *Streptococcus equisimilis*, the latter in low numbers, were isolated from both samples. In addition, *Escherichia coli* was isolated from the medulla and *P. fluorescens* from the midbrain.

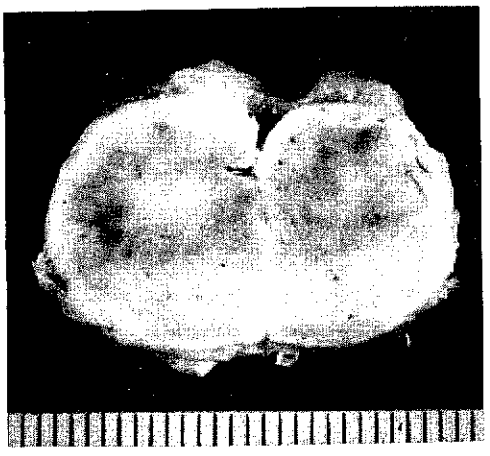

Figure 1. Transverse section through the spinal cord near its junction with the medulla oblongata. Note extensive discoloration of bilateral malacic areas and focal haemorrhages. The scale is in millimetre.

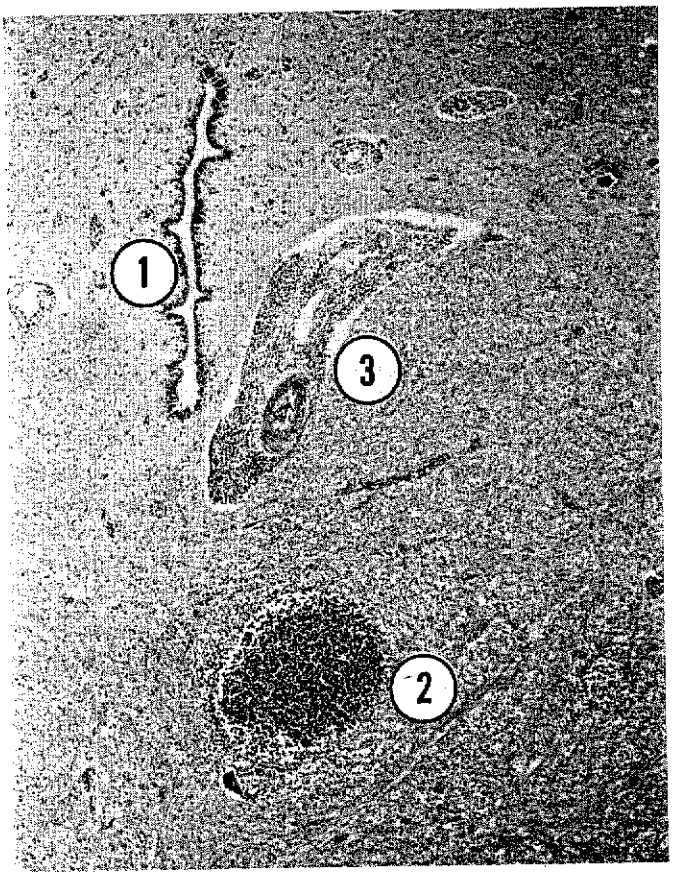

Figure 2. Photomicrograph of section from the spinal cord at the same level as Figure 1. Note central canal (1), large non-encapsulated accumulation of neutrophils (2), and prominent perivascular cuff (3). (Haematoxylin and eosin x 36).

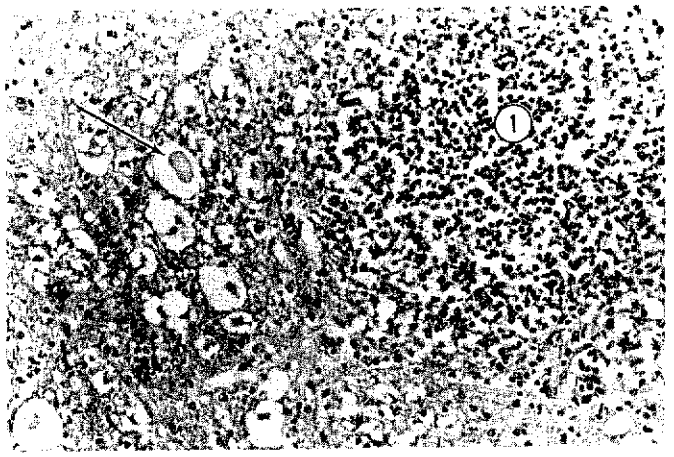

Figure 3. Vacuolated parenchyma adjacent to a focus of predominantly neutrophilic infiltration (1) in the spinal cord. Note swollen axon (arrow). (Haematoxylin and eosin x 150).

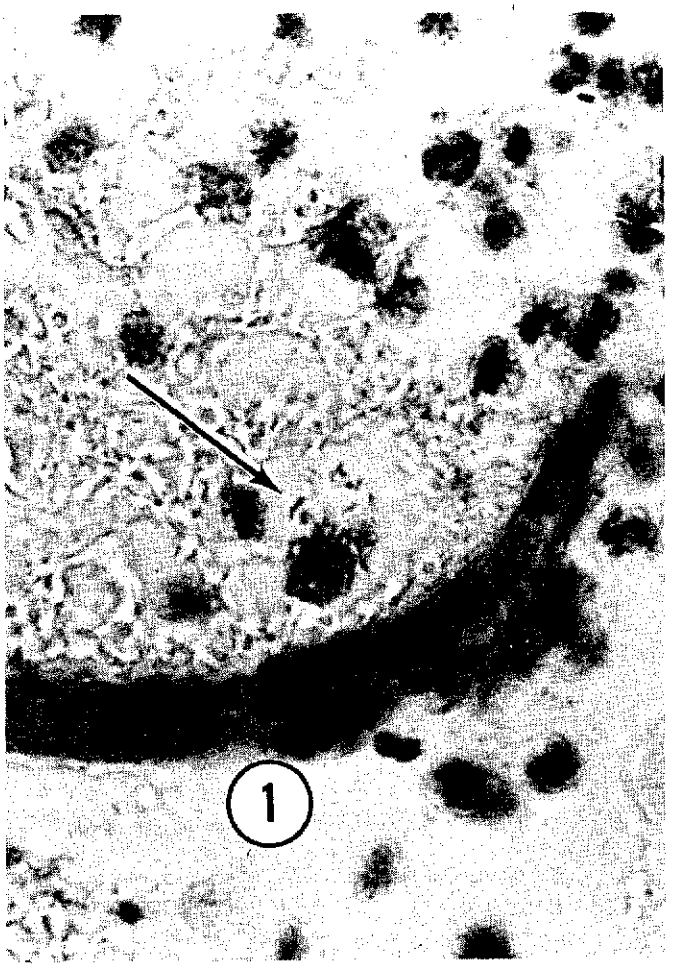

Figure 4. Photomicrograph of periphery of a malacic area. Note rod-shaped organisms (arrow) which appear to be confined within the cytoplasm of a macrophage adjacent to a capillary (1). (Giemsa x 2020).

Discussion

Melioidosis continues to be a problem in domestic and wild animals and man in northern Queensland; additionally, recent reports describe its occurrence in a cockatoo (Thomas *et al* 1978) and a galah (Thomas *et al* 1980). About 4 to 5 human cases are diagnosed annually in this region (L. Ashdown, personal communication).

Although the sudden and dramatic onset of nervous signs in this colt suggests primary central nervous system (CNS) involvement, lack of a complete necropsy examination prevents us from excluding the possiblity of CNS localisation of *P. pseudomallei* subsequent to its establishment (most probably as an abscess) elsewhere in the body.

From their consideration of 116 cases of melioidosis of domestic animals, Laws and Hall (1963) concluded that this infection is most often a chronic disease except when the CNS is infected. They described CNS lesions essentially similar to those seen in the present case in experimentally infected sheep, and in 2 naturally infected cows, a horse and a goat. Macroscopic CNS lesions were observed in only one of their animals, and that case — involving an aged dairy cow — was later described in some detail (Laws and Mahoney 1964). The lesion seen in the affected cow consisted of a small necrotic area in the right anterior aspect of the medulla oblongata, with fibrin on the overlying leptomeninges. Gross lesions in the present equine case were more extensive and haemorrhagic, and readily recognised at necropsy.

The present case appears to be only the second equine case in which CNS involvement was confirmed, but as in the first case and other domestic animals (Laws and Hall 1963), the organism appeared to have predilection for the brain stem and spinal cord. Adequate description of the pathology of melioidosis of the CNS in man appears to be lacking. A primary human case of melioidotic meningitis with positive serology, isolation of *P. pseudomallei* from the CSF, and subsequent death, occurred recently in this area (L. Ashdown, personal communication).

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