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INFECTIOUS DISEASE

Meningoencephalitis in a Dog Due to *Trichosporon montevideense*

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Summary

Meningoencephalitis due to infection with *Trichosporon montevideense* was diagnosed in a 4-year-old dog with a brief clinical history of rapidly progressing neurological signs that culminated in a comatose state. No significant gross lesions were found at post-mortem examination. Microscopically, a few scattered areas of pyogranulomatous inflammation with a few small, non-pigmented fungal hyphae were found within the cerebrum surrounding the lateral ventricles. A *Trichosporon* sp. was identified through culture of the brain and species was determined via sequence analysis of the internal transcribed spacer region of the *Trichosporon* rRNA gene. DNA in-situ hybridization confirmed the diagnosis. This is the first reported case of *Trichosporon*-associated meningoencephalitis in a dog.

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The genus *Trichosporon* is comprised of 50 species of basidiomycetous, anamorphic, yeast-like organisms that thrive in temperate and tropical climates throughout the world and frequently inhabit soil, water and sometimes the faeces, skin, respiratory and gastrointestinal tracts of man and animals (Colombo *et al.*, 2011). Sixteen *Trichosporon* spp. are thought to be pathogenic in man, with most cases in people and non-human primates involving superficial infection of hair shafts (called 'white piedra'), interdigital skin or the nail bed (Alshahni *et al.*, 2009; Colombo *et al.*, 2011). Infrequently, *Trichosporon* spp. have been reported to cause urinary tract infections in man and cats (Sharman *et al.*, 2010; Colombo *et al.*, 2011). Invasive and systemic infection with *Trichosporon* spp. is rare, but has been reported in non-human primates and with increasing frequency in man (Vicek *et al.*, 1995; Ruan *et al.*, 2009; Colombo *et al.*, 2011). *Trichosporon montevideense* (initially categorized as *Endomycopsis montevidensis*) was first isolated in 1969 from wastewater at a water purification plant in Montevideo, Uruguay and has been found subsequently on human skin and within soil, water and human faeces (de Queiroz, 1973; Guého *et al.*, 1992; Alshahni *et al.*, 2009). *T. montevideense* is regarded as an emerging pathogen and has been isolated from a person with meningitis (Ruan *et al.*, 2009). This paper describes the first reported case of meningoencephalitis due to *Trichosporon* spp. in a dog.

A 4-year-old, neutered female Labrador retriever-cross presented with a 5-day history of intermittent vomiting and sudden development of blepharospasm, photophobia, fine muscle fasciculations and extensor rigidity. The dog had been full-body clipped 14 days prior to presentation and had an episode of diarrhoea 5 days after grooming. The dog lived outdoors in a rural area and had access to cattle, horses and pastures. The dog was treated with several courses of antibiotics, anthelmintics and 1.3 mg/kg prednisone over a 4-day period prior to referral. After initial

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clinical improvement, the dog had cyclical periods of depressed mentation and gradually lapsed into a comatose state. The dog was referred to Texas A&M University. On initial neurological examination, the dog was comatose and bradycardic (heart rate 60 beats per min) with intermittent paddling, pinpoint pupils with minimal response to light and no response to menace or corneal stimulation. Neurological disease was localized to the forebrain and brainstem. Severe brain swelling with herniation was suspected based on the clinical signs and a modified Glasgow coma score of 7 was assigned (Platt *et al.*, 2001). Within 2 h of treatment with diazepam, levetiracetam and mannitol, extensor tone normalized and the dog attempted to rise, began head pressing and vocalizing, but overall mentation remained obtunded. The dog exhibited anisocoria, a positive dazzle response and a weak gag response, but had an absent menace response bilaterally. Withdrawal reflexes and deep pain sensation were present in all limbs.

Differential diagnoses included intracranial neoplasia, infectious or inflammatory meningoencephalitis or neurotoxin exposure. Radiographs of the thorax and abdomen revealed no abnormalities apart from a mildly enlarged spleen. Magnetic resonance imaging (MRI) of the brain revealed subtentorial herniation of the caudal cerebellum, bilateral dilation of the lateral ventricles and multiple areas of hyperintensity in the paraventricular white matter on FLAIR (Fig. 1) and T2 weighted sequences, with greatest severity in the left cerebral hemisphere and non-contrast enhancement on the T1 sequence. Cerebrospinal fluid (CSF) was not obtained due to the risk of continued herniation and death. The MRI was thought to be most consistent with severe encephalitis, with infectious disease as the main differential. Despite aggressive treatment to control brain swelling, the clinical signs did not improve and the dog was humanely destroyed within 48 h after referral.

A post-mortem examination was conducted at Texas A&M University approximately 6 h after death. The dog was in good bodily condition. The left temporal muscle was mildly atrophic. Apart from mild dilation of both lateral ventricles, the brain was grossly normal. Rabies testing on a segment of fresh brainstem and cerebellum was negative.

Samples of the brain and other major organs were fixed in 10% neutral buffered formalin, processed routinely and embedded in paraffin wax. Sections were stained with haematoxylin and eosin (HE). Significant lesions were found only within a few areas of the cerebral parenchyma adjacent to the lateral ventricles (Fig. 2) and in small multifocal areas throughout the meninges. Scattered, multifocal areas of microcavitation in the paraventricular grey and white matter contained numerous epithelioid macrophages and foreign-body type giant cells that palisaded around central accumulations of degenerate neutrophils, necrotic and karyorrhectic cellular debris, extravasated erythrocytes and rare cross sections of 10 × 2 μm, non-pigmented fungal hyphae. Hyphal forms were positive on Gomori's methenamine silver staining and were characterized by infrequent septation, non-parallel walls and occasional 90° branching (Fig. 3). Multifocally, there was moderate to severe lymphoplasmacytic perivascular

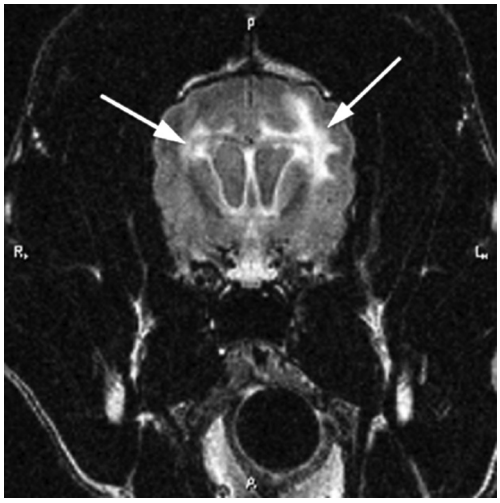

Fig. 1. Transverse MRI FLAIR sequence of the brain of the dog. There is marked focal hyperintensity (arrows) in the cerebral white matter tracts surrounding the lateral ventricles bilaterally, but with more severe changes evident on the left side.

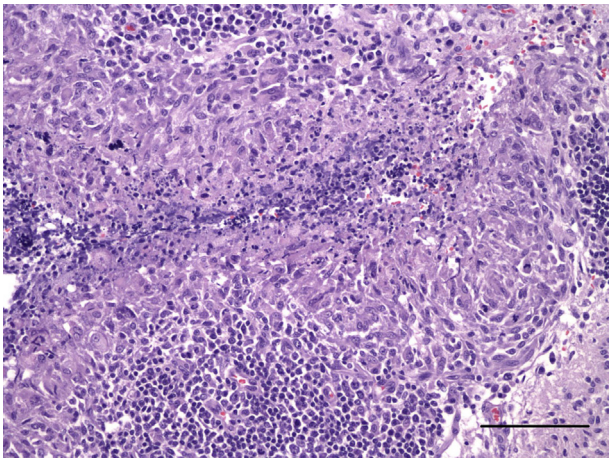

Fig. 2. Microscopical lesions in the cerebrum of the dog. Within the paraventricular parenchyma is a central, linear area of cavitation that is surrounded by numerous epithelioid macrophages with multiple perivascular cuffs of lymphocytes and plasma cells. HE. Bar, 75 μm.

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