

RESEARCH COMMUNICATION

CEREBRAL MYCOSIS IN A DOG CAUSED BY *CLADOSPORIUM TRICHOIDES* EMMONS 1952

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ABSTRACT

NEWSHOLME, S. J. & TYRER, MARGARET J., 1980. Cerebral mycosis in a dog caused by *Cladosporium trichoides* Emmons 1952. *Onderstepoort Journal of Veterinary Research*, 47, 47-49 (1980).

The fungus, *Cladosporium trichoides*, was isolated and cultured from a lesion in the cerebellum and from smaller lesions in the liver, kidney and spleen of a dog which had a history of behavioural changes, ataxia and collapse. Histopathological examination showed the cerebellar lesion to be a purulent granuloma which contained brown, septate hyphae and structures resembling conidia. The source of infection was not traced and no predisposing factors were apparent.

As far as is known, this is the first record of the condition in animals in southern Africa and the first report anywhere of this condition in the dog.

Résumé

MYCOSE CEREBRALE CHEZ UN CHIEN, CAUSÉE PAR *CLADOSPORIUM TRICHOIDES*
EMMONS 1952

Le fungus *Cladosporium trichoides* a été isolé et mis en culture à partir d'une lésion dans le cervelet et de lésions plus petites du foie, du rein et de la rate d'un chien ayant eu une histoire de changements de comportement, ataxia et collapsus. L'examen histopathologique a montré que la lésion cérébelleuse était un granulome purulent qui contenait des hyphes cloisonnées et des structures brunes ressemblant à des conidies. La source de l'infection ne fut pas trouvée et aucun facteur de prédisposition ne fut apparent.

Pour autant qu'on sache ceci est le premier rapport établi de cette condition chez les animaux en Afrique du Sud et le premier rapport de cette condition chez le chien n'importe où ailleurs.

INTRODUCTION

The isolation of a dematiaceous fungus from a human brain abscess has been reported by Binford, Thompson, Gorham & Emmons (1952). In the report, Emmons characterized and described this fungus and named it as a new species, *Cladosporium trichoides*.

Since 1952, occasional cases of cerebral abscesses in man caused by *Cladosporium trichoides* have been reported from different parts of the world and various aspects of collected cases have been discussed (Riley & Mann, 1960; Duque, 1961; Bennett, Bonner, Jennings & Lopez, 1972; Middleton, Jurgenson, Utz, Shadomy & Shadomy, 1976). Although it is rare, the condition is important, because response to medical or surgical treatment is usually poor and most cases have terminated fatally.

In all but 2 of the reported cases of human infection by *Cladosporium trichoides* (Emmons, Binford & Utz, 1970; Nsanzumuhire, Vollum & Poltera, 1974) there was cerebral involvement. Thus, *Cladosporium trichoides* appears to be truly neurotropic in man.

In South Africa, Watson & Lines (1957) described a human case of cerebral abscess in Natal. From the abscess they cultured a dematiaceous fungus which they considered to be a species of *Hormodendrum*, although their description of the fungus was very similar to that of *Cladosporium trichoides*. In a case reported by Manson (1958) in the Transvaal, no fungus could be isolated, but the morphology of a fungus present in the cerebral lesion closely resembled that of *Cladosporium trichoides*.

In animals, there are 2 reports of mycosis caused by *Cladosporium trichoides*, both in cats. Reed, Fox & Campbell (1974) isolated the fungus from renal lesions in a cat with leukaemia and Jang, Biberstein, Rinaldi,

Heness, Boorman & Taylor (1977) described two feline cases with brain abscesses from which *Cladosporium trichoides* was isolated.

This report describes a canine case in which *Cladosporium trichoides* was isolated from a lesion in the cerebellum and from smaller lesions in the liver, kidney and spleen. As far as is known, this is the first record of mycosis caused by *Cladosporium trichoides* in a dog.

CASE REPORT

Clinical history

A 9-year-old, spayed Corgi bitch was presented for examination at the surgery in Manzini, Swaziland, on 4 January 1979, following sudden collapse. The history preceding collapse was one of behavioural changes accompanied by head shaking and an altered facial expression. These features had first been noticed by the owner 2 months previously, but veterinary advice had not been sought, because the animal had continued to be bright and had a normal appetite. In addition, no otitis had been apparent on examination by the owner.

A clinical examination revealed a constant, rhythmic shaking of the head and neck and diminished corneal and pupillary reflexes. When the animal was encouraged to rise, ataxia of the hind limbs was apparent. Rectal temperature was 39,6 °C and mucous membranes were pallid. A smear of capillary blood was negative for *Babesia canis*.

A tentative diagnosis of meningitis was made and the bitch was hospitalized. Systemic treatment with chloramphenicol was instituted and signs regressed over the next few hours. The bitch was discharged with a supply of chloramphenicol capsules on 6 January but was readmitted the next day, following sudden collapse after light exertion. Examination showed the bitch to be semi-comatose with the heart in fibrillation. When the animal regained consciousness, incoordination, loss of balance and a constant lateral weaving of the body were noted. The bitch was bright and had a

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normal appetite, but severe incoordination persisted. Euthanasia was performed on 12 January after consultation with the owner.

Post-mortem examination

The carcass was in good condition, despite marked anaemia. The liver contained multiple, small (1–2 mm), cream-coloured lesions which revealed a milky fluid on incision. Similar, though larger (3–4 mm) lesions were present along the splenic margins and beneath the capsules of both kidneys. A large, 10 mm vegetation attached to the tricuspid valve was present. The brain, after being removed and bisected longitudinally, revealed a soft, grey, circular area (10 mm diameter) situated in the white matter at the base of the cerebellum. No other lesions were found in the brain. *Listeria monocytogenes* infection was suspected.

Laboratory findings

Gram-stained smears were prepared from the hepatic, splenic, renal and cerebellar lesions. Examination of all the smears revealed tangled masses of fungal hyphae, but no bacteria. Samples from the hepatic, splenic, renal and cerebellar lesions were plated out on sheep blood agar, MacConkey's agar and Sabouraud's agar. The cultures were incubated at 37 °C.

Sheep blood and MacConkey's agar plates failed to show any growth after 7 days. Dark grey colonies were visible on all the Sabouraud's agar plates after 7 days.

The culture plates together with the cerebellum, which was immersed in 10% buffered formalin, were submitted to the Veterinary Research Institute, Onderstepoort, for further examination.

Mycology

Colonies were replated from the original Sabouraud's agar cultures onto Sabouraud's dextrose agar, Czapek's agar and potato dextrose agar and stored at room temperature (20–30 °C). The development of fungal colonies was followed by intermittent examination of the plates.

On Sabouraud's dextrose agar, dark grey, velvety, round colonies became visible after 5 days, reaching a diameter of 1 cm after 2 weeks (Fig. 1). Microscopical examination of material from the colonies, mounted in 1% saline, showed brown, smooth walled, septate hyphae, 2 µm in diameter, and long, linear chains of conidia with a few branches (Fig. 2 and 3). The conidia were elliptical and measured 2,5–3,5 µm × 4–5 µm.

On Czapek's agar olive-green colonies with a narrow, white margin reached 0,25–0,5 cm diameter after 2 weeks.

Colonies reached 1 cm in diameter after 2 weeks on potato dextrose agar and were rich olive-green in colour with radial folds and a white margin.

Histological sections and cultures were examined by Dr D. B. Scott of the Department of Agricultural Science, University of Pretoria, who concluded that the fungus was *Cladosporium trichoides* Emmons 1952.

Histopathology

Blocks of formalin-fixed brain tissue containing the lesion were embedded in paraffin wax and sections cut at 5–7 µm thickness. Some of the sections were stained with haematoxylin and eosin (HE). In addition, to demonstrate the fungus, other sections were stained with the Periodic Acid-Schiff reaction (P.A.S.) (Pearse,

1961) and with Gomori's methenamine silver nitrate (G.M.S.) (Luna, 1968). Some sections were left unstained.

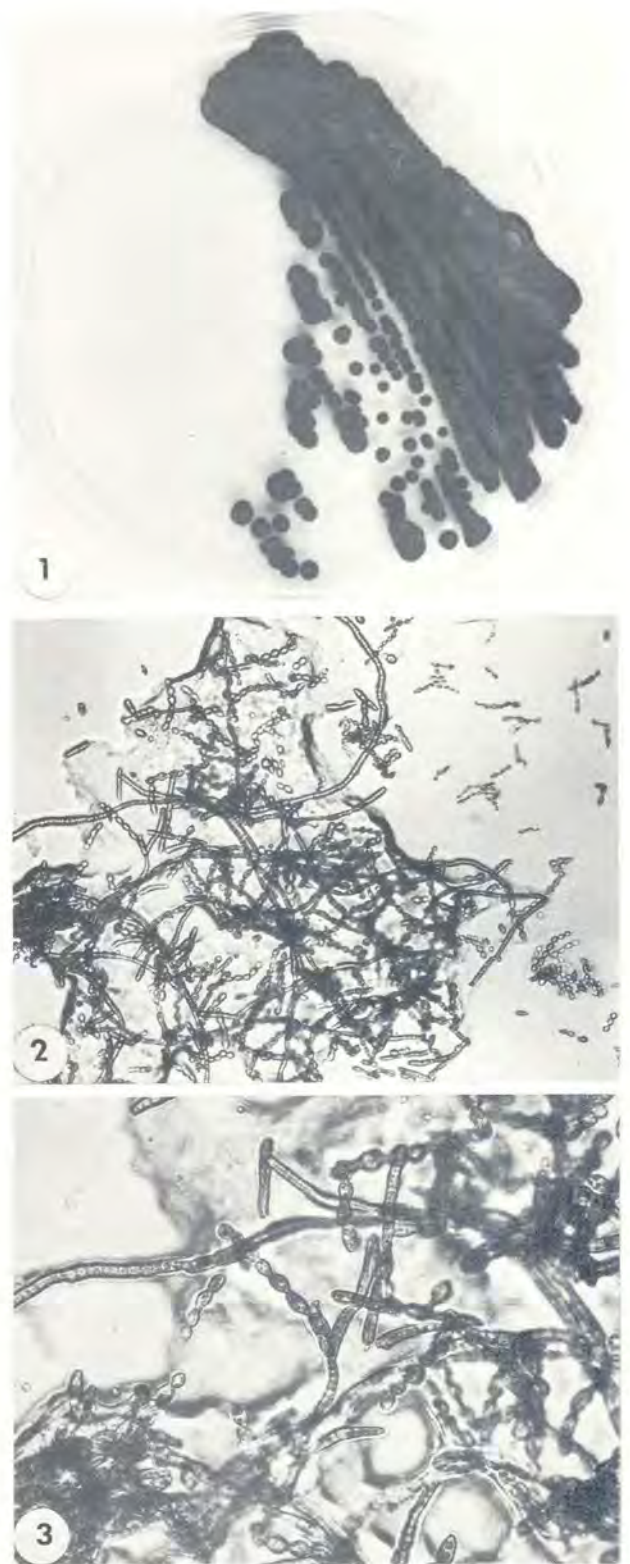


FIG. 1 Gross appearance of fungal colonies after 2 weeks on Sabouraud's agar. Liver isolate
 FIG. 2 Part of colony from the above culture, showing hyphae and linear chains of elliptical conidia; unstained ×500
 FIG. 3 The same colony showing hyphae with septa and linear chains of conidia; unstained ×800

The cerebellar lesion consisted of a central area containing neutrophils. These were interspersed with macrophages and surrounded by a thick layer containing astrocytes and some macrophages (Fig. 4 and 5). A few giant cells, situated amongst astrocytes in the inner part of the surrounding layer, were present.

Brown hyphae, 2 μm in diameter, apparent in HE sections, also showed up in the unstained sections (Fig. 6). Their number and distribution were shown to advantage in G.M.S. stained sections (Fig. 7) in which numerous hyphae were seen in the central area and some were present in the inner part of the surrounding layer. At a higher magnification, septa could be seen in the hyphae, and structures resembling conidia were discernible. Neutrophils were apposed very closely to some hyphae and the neutrophil cytoplasm was indented against the hyphal walls. This produced no demonstrable effect on hyphal integrity at these points (Fig. 8).

The perivascular spaces of blood vessels in a wide area outside the surrounding layer of the lesion were occupied by several layers of inflammatory cells. These were mostly neutrophils and macrophages with some plasma cells. The meningeal layers of the cerebellar folia were filled with similar cell populations.

DISCUSSION

The lesion in the cerebellum explains the history of ataxia in this dog. From its histological features this lesion should be referred to as a purulent granuloma, rather than as an abscess.

The close association between hyphae and inflammatory cells, together with the isolation of *Cladosporium trichoides* from the lesion, suggests that this fungus was the causative agent.

The source of infection is not clear but it was most probably the environment, since many members of the genus *Cladosporium* are known to be widely distributed plant saprophytes. However, little is known about the natural habitat of *Cladosporium trichoides* and there are only 2 reports of its isolation from nature. Klite, Kelley & Diercks (1965) isolated it from soil samples in Panama, and Dixon, Shadomy & Shadomy (1977) isolated a strain from tree stump bark in Virginia and established the neuropathogenicity of this strain in mice.

There are no obvious predisposing factors in this case. The bitch was old and, though it had a previous history of *B. canis* infection, it was active, alert and in good condition prior to the onset of nervous signs. No comment can be made on the aetiology of the vegetation on the tricuspid valve because, unfortunately, no histopathology or mycology was undertaken on this lesion. It is possible that this lesion caused cardiac dysfunction which could be considered as a possible aggravating factor. It is of interest that in the majority of human cases no predisposing factors were evident.

The isolation of the fungus from lesions in cerebellum, liver, spleen and kidneys suggests that haematogenous spread occurred.

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