



## Case Report

# *Gurltia paralyans* infection in a domestic cat in the São Paulo state, southeastern Brazil

Rafael Rocha Mello Emboaba da Costa<sup>1</sup>, Júlio Edward Hough Monteiro<sup>2</sup>,  
Claudia de Souza Silva<sup>3</sup>, Fernanda Ramalho Ramos<sup>3</sup>, Bethânia Almeida Gouveia<sup>3</sup>,  
Paulo Henrique Leal Bertolo<sup>4</sup>, Rosemeri de Oliveira Vasconcelos<sup>4\*</sup>

<sup>1</sup>Graduação em Medicina Veterinária, Faculdade de Ciências Agrárias e Veterinárias (FCAV),  
Universidade Estadual Paulista (UNESP), Campus de Jaboticabal, SP, Brasil.

<sup>2</sup>Laboratório Novolabvet, Ribeirão Preto, SP, Brasil.

<sup>3</sup>Programa de Residência em Área Profissional da Saúde – Medicina Veterinária e Saúde, FCAV-UNESP, Campus de Jaboticabal, SP, Brasil.

<sup>4</sup>Departamento de Patologia, Reprodução e Saúde Única, FCAV-UNESP, Campus de Jaboticabal, SP, Brasil.

\*Corresponding author: [rosemeri.vasconcelos@unesp.br](mailto:rosemeri.vasconcelos@unesp.br)

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## Abstract

*Gurltia paralyans* is a nematode first described in 1933 in Chile, causing a syndrome called feline crural parasitic paraplegia. Insects, mollusks, frogs, lizards, and rodents are paratenic hosts of this nematode, and cats probably become infected by ingesting them. This report aims to discuss the main anatomopathological findings of gurltiosis in a cat submitted to necropsy in a laboratory in Ribeirão Preto city, São Paulo state, southeastern Brazil, being the first case reported in this state. The main necroscopic findings were extramedullary reddened areas below the leptomeninges in the cervical and lumbar segments of the spinal cord. The histopathological examination showed marked thickening of the leptomeninges in the lumbar segment of the spinal cord, with marked neovascularization and fibrosis associated with eosinophilic and mononuclear inflammatory infiltration, as well as the presence of intravascular nematodes. The diagnosis of this lesion was chronic segmental meningomyelitis associated with intralésional parasites. Although uncommon, feline gurltiosis is a neglected parasitic disease that should be included as a differential diagnosis of paraparesis in domestic cats.

**Keywords:** chronic myelopathy, feline diseases, gurltiosis.

## Introduction

*Gurltia paralyans* is a nematode (order Strongylida; family Protostrongylidae; subfamily Angiostrongylidae) and was first described in 1933, in Chile, as the cause of a syndrome called feline parasitic crural paraplegia (18). Since then, this disease has been described in Argentina, Uruguay, Colombia, Brazil, and the Canary Islands (6, 3, 14, 10, 15, 8, 16). Its cycle is unknown, but it is known to have a heteroxenic cycle, as do other metastrongylids, using some paratenic hosts such as lizards, frogs, mollusks, and earthworms for the development of the larval forms (4). *Gurltia paralyans* can be found in the veins of the leptomeninges and the spinal cord of felines. The clinical signs described for

this infection are progressive paraparesis, paraplegia, urinary incontinence, and tail paralysis (5, 10).

The definitive diagnosis of this disease was only possible at necropsy (10), but there are recent reports of molecular and serological diagnoses of this parasitosis (7,12). Two reports in the species *Leopardus tigrinus* and *Leopardus wieddi* also indicate the parasite's presence in spinal cord segments (13,1). During the necropsy of parasitized domestic cats, reddening of the leptomeninges and changes secondary to paraplegia, such as muscular atrophy of the pelvic limbs and tail, are observed (10).

The main histopathological findings include adult forms of the parasites in the meningeal venules and the white matter, associated with congestion, phlebo-sclerosis, intraluminal papillary projections, and with thrombi in different

stages of the organization (5, 10, 9, 15, 11, 4). The medullary parenchyma shows multifocal to diffuse hemorrhages, foci of liquefactive necrosis associated with Gitter cells, and varying degrees of intensity of Wallerian degeneration. The inflammatory infiltrate is chronic, predominating lymphocytes and eosinophils (15, 4). However, mononuclear inflammatory infiltrate in the choroid plexus (2) and perivascular neutrophilic infiltrate in the subarachnoid space and the neuropil (15) are also observed. In the liver, hepatic periportal degeneration was associated with a neutrophilic and mononuclear infiltrate. Feline gurltiosis is related to hyaline deposits in the kidneys, thickening of Bowman's capsule, and an interstitial inflammatory infiltrate composed of neutrophils and eosinophils (17).

### Case description

An approximately 2-year-old feline, who had been rescued from the street 6 months ago and already had pelvic limb paralysis, was referred for computerized tomography (CT) scan to determine the cause of the paralysis. After the CT, the animal presented other neurological signs in the anesthetic return, such as nystagmus, incoordination, and hyperreflexia. The animal was then taken to a private intensive care clinic, where it died and was sent for necropsic examination in a laboratory in Ribeirão Preto city, São Paulo state, southeastern Brazil. During necropsy, the macroscopic findings were a reddish area, similar to an intradural extramedullary clot, located in the cervical region, between the C1 to C2 vertebrae, an extramedullary reddish area in the lumbar region (L3 to L6) below the leptomeninges, which measured about 2.0 x 0.5 cm, similar to the lesion observed in the cervical region, besides a reddish area of approximately 0.8 cm in diameter in the region of the pons and cerebellum (Fig. 1).

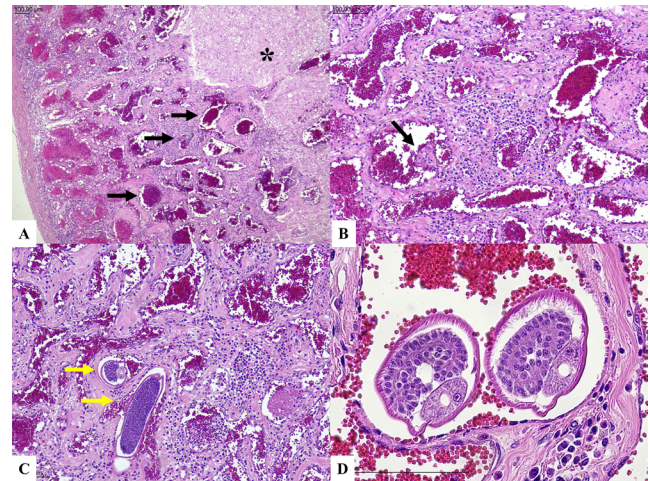


**Figure 1.** Segment of a feline's lumbar region of the spinal cord with pelvic limb paralysis, showing a hemorrhagic area below the leptomeninges, similar to a crucic clot (locally extensive hemorrhage).

Histopathological evaluation of the lumbar segment of the spinal cord showed severe thickening of the leptomeninges due to marked neovascularization and fibrosis (Fig. 2). This change was associated with a marked inflammatory infiltrate composed of eosinophils, lymphocytes, and plasma cells. The leptomeningeal vessels were tortuous and sometimes showed papillary projections into the vascular lumen. The adult forms of intralumenal nematodes were found in intravascular compartments of the leptomeninges on transverse and longitudinal sections. Furthermore, the proliferative vascular lesion in the meninges caused compression of the adjacent medullary white matter, which resulted in foci of degeneration and necrosis of axons with the formation of axonal spheres (Wallerian degeneration). The lesion was diagnosed as chronic segmental meningomyelitis associated with intralumenal parasites. In the brain, only mild satellitosis was noted. There was a locally extensive hemorrhage in the brainstem and cerebellum.

### Discussion

The current hypothesis of the pathogenesis of feline gurlitiasis is the association of the presence of the parasite with the formation of thrombi in leptomeningeal vessels,



**Figure 2.** Photomicrographs of a feline's lumbar spinal cord region with pelvic limb paralysis. A- Note severe thickening of the leptomeninges due to marked neovascularization (arrows), inflammation, and fibrosis. This reaction leads to compression of the adjacent medullary white matter (asterisk). (4x obj.). B- Neoformed tortuous vessels in the leptomeninges, sometimes with intraluminal papillary projections (arrowhead). (Obj. of 10x). C- Intravascular nematodes in transverse and longitudinal sections (yellow arrows) associated with marked fibrosis and inflammatory infiltrate in the interstitium (Obj. of 10x). D- Detail of intravascular parasites in a transverse section (Obj. at 40x). Hematoxylin and Eosin.

resulting in vascular obstruction with intense venous stasis and consequent increase in pressure and vascular dilation. The stimulus generated results in the occurrence of varicose veins (8). Also, the mechanical compression exerted by the parasite on the white matter of the spinal cord and initial portions of the spinal nerves results in progressive paraparesis, the main clinical sign associated with gurltiosis. In the present report, we have a chronic infection, chronic meningomyelitis, Wallerian degeneration, malacia, and fibrosis of the nervous tissue, in addition to the proliferation of tortuous veins and sometimes varicose veins, according to the literature. In addition to the lesions, the presence of the parasite within vessels allowed us to affirm feline gurlithiasis.

The literature does not describe hemorrhagic lesions in the pons and cerebellum (4). However, the type of injury caused by the parasite leading to vascular fragility and consequent thrombosis could result in hemorrhagic foci in distant sites.

The tomography performed on the feline was not elucidative for diagnosing spinal cord injury. Necropsy followed by microscopic analysis was the diagnostic method for identifying this type of parasitosis, as described by other authors (5,10, 9, 15, 11, 4). CSF serology and molecular techniques have recently proven effective in diagnosing this parasite (7). The variceal lesion type is rarely seen in veterinary medicine, especially in domestic cats. Because of this, recognition of this lesion can be complex for the veterinary pathologist.

The association of case reports diagnosed in Brazil's South (15) and Northeast regions (8), with this first report from the southeast region, indicates a possible distribution of the parasite in the national territory. Therefore, although uncommon, this disease should be included in the differential diagnosis of feline neurological diseases with paraparesis.

### Conflict of Interest

The authors declare no competing interests.

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