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Case Report

Canine Toxoplasma Gondii and Distemper Virus Co-Infection in a Dog with Neuro-ophthalmologic Signs from Córdoba, Argentina

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Abstract

Toxoplasmosis is caused by the globally distributed parasitic intracellular protozoan Toxoplasma gondii (phylum Apicomplexa, family Sarcocystidae). Domestic cats and other felids are the definitive hosts. All non feline animals, including dogs, are intermediate hosts. In many animal species, the infection is typically subclinical, though toxoplasmosis can be fatal, even in pets. The disease is often associated with immunosuppressive conditions-for example, infection by the Canine Distemper Virus (CDV) or immunosuppressive drugs-which conditions predispose an animal towards a more unrestrained multiplication of the protozoon. The aim of this case report was therefore to describe the clinical course and successful response to therapy of a dog with a coinfection of CDV and toxoplasmosis exhibiting neuroöftalmologic signs.

Background

Toxoplasmosis is caused by the globally distributed parasitic intracellular protozoan Toxoplasma gondii (phylum Apicomplexa, family Sarcocystidae). Domestic cats and other felids are the definitive hosts. All non feline animals, including dogs, are intermediate hosts [1]. In many animal species, the infection is typically subclinical, though toxoplasmosis can be fatal, even in pets [1-5]. At the present time, primary clinical toxoplasmosis in dogs is considered rare, but can involve multiple organs. The disease is often associated with immunosuppressive conditions-for example, infection by the Canine Distemper Virus (CDV) [6-8] or immunosuppressive drugs-which conditions predispose an animal towards a more unrestrained multiplication of the protozoon. CDV is a highly contagious virus that occurs worldwide and may cause a fatal disease in carnivores. A member of the genus Morbillivirus belonging to the family Paramyxoviridae, the virus can infect epithelial tissues of the respiratory, gastrointestinal, or nervous systems [9]. At an early stage, CDV infection manifests a great diversity of signs in dogs such as respiratory, gastrointestinal, and cutaneous symptoms. Either in a sequence or even only weeks later, severe neurological injuries occur and in some cases may be the only overt symptom presented by that time in the absence of any previous or concomitant signs [10]. Therefore, dogs infected with CDV are more susceptible to develop secondary infections by T. gondii, the coinfectant thus being considered a neurotropic opportunist protozoan [9]. Neurologic and neuromuscular signs described in patients coinfected with toxoplasmosis and CDV are seizures, cranial-nerve deficits, tremors, ataxia, and paresis or paralysis along with encephalomyelitis behavioral changes, dysphagia, and upper- and lower–motor-neuron signs [1,6,11]. The type and severity of clinical illness depend on the degree and localization of the tissue injury, in which cell necrosis as well

as focal hemorrhagia and malacia is caused by the intracellular growth of T. gondii [9,12,13]. Reports of successful treatment of neurologic toxoplasmosis associated with CDV are few, as most cases have been diagnosed on necropsy [6]. The aim of this case report was therefore to describe the clinical course and successful response to therapy of a dog with a coinfection of CDV and toxoplasmosis exhibiting neuro-ophthalmologic signs.

Case presentation

Orson is a male 4-year-old Shar Pei crossbred dog. He was confiscated from his home because of animal abuse. The Catholic University of Córdoba became his legal custodian. He was allowed to walk around the university campus and returned to his kennel to rest and feed. We need to note that cows, goats, sheep, and feral cats are kept on the university campus with the last of these being trapped, sterilized, and released. The dog was dewormed and is left under observation.

At one point Orson did not want to go on his daily walk. He was observed listless with bilateral conjunctivitis and a mild cough along with anorexia and fever (39.8°C). With a presumptive diagnosis of a viral respiratory disease (i.e., CDV, parainfluenza), a complete blood work-up, was performed. A mild anemia was found along with a normal leucogram and hepatic-enzyme, blood-urea-nitrogen, and creatinine levels. We decided to start a 7 day treatment with enrofloxacin (10 mg/kg every 24 h), prednisolone 0.5 mg/kg every 24 h, and a topical treatment with tobramycin eye drops every 12 h. Although the dog responded completely to the treatment, two weeks after finishing it, he began to exhibit a neuro-ophthalmologic syndrome, described below. His level of consciousness was obtunded. He had abnormal proprioception and ataxia with a wide-based stance, a swaying gait, and an increased stride length; suggesting a brainstem or forebrain lesión [14]. He also developed a right head tilt, abnormal vertical positional nystagmus, decreased facial response on the right side, and a leaning and falling to the right side; suggesting a forebrain and/or central vestibular lesion on that side. He had a diminished ability to perform voluntary movements with the four limbs (tetraparesis), with pleurothotonus, suggesting an upper motor neuron syndrome [14]. The eyeballs were of normal size and position. He had bilateral mydriasis and in the examination room, he could not navigate through obstacles. The menace response was bilaterally negative. Direct and indirect photopupillary and the dazzle reflexes were negative, as well as photochromic reflexes for retinal rhodopsin and optic nerve melanopsin, indicating that the patient was blind [14]. The intraocular pressure was normal in both eyes (left eye: 19 mmHg; right eye: 17 mmHg). Examination of the fundus revealed retinal atrophy due to retinal thinning and a slight increase in hyperreflectivity [15,16]. Neurologic and ophthalmic signs suggested a multifocal disease that involved several areas of the central nervous system.

Investigations

Differential diagnosis

In view of the patient's history, the place where he lived, and the neurologic signs, a process compatible with diffuse central-nervous disease, was suspected. The differential diagnoses included: A viral-type infectious, parasitic, or neoplastic process. We decided at that time, to perform a serological analysis for the detection of the most common agents observed locally. Serum IgM and IgG titers for CDV, T. gondii and Neospora caninum were measured by indirect immunofluorescence assay (IFA). The results confirmed a positive IgM for T. gondii, positive IgG for T. gondii (IFA titer: 1/256) and positive IgG for CDV (IFA titer: 1/800), with the N. Caninum titers being negative. Two months later T. gondii titers were repeated, resulting in a negative IgM and a two-fold-increasing of IgG (IFA titer: 1/512). A diagnosis of clinical CDV and T. gondii coinfection was made-based on the high IgG titers of CDV9 and positive IgM titers along with IgG seroconversion of toxoplasma antibodies-along with an exclusion of neosporosis, but with a beneficial clinical response to anti-Toxoplasma drugs [2].

Treatment

At the time when the presence of elevated IgM and IgG titers against T. gondii were detected to be positive, we decided to start a treatment with an anti-Toxoplasma protocol consisting of clindamycin (10 mg/kg every 12 h) and sulfonamide (15 mg/kg, per os, every 12 h) for 30 days [2]. An anti-inflammatory dose of prednisolone (0.5 mg/kg per os, every 24 h) was also administered for a week.

Outcome and follow-up

The dog's neurologic signs improved in the first two weeks of treatment, although a mild ataxia and blindness persisted. For that reason, we decided to continue with clindamycin for 4 more weeks. Although by the end of that treatment the dog recovered completely from the neurologic syndrome, his vision remained reduced even at 18 months later, the time when this article was written.

Discussion

Although T. gondii infections are common in domestic dogs worldwide, most infections are subclinical [17]. Clinical toxoplasmosis is considered rare, and the suggestion has been made that viral infections, such as CDV may predispose to those infections [1,6,8,18]. Clinical toxoplasmosis in dogs has a wide range of presentations, and when specific signs involving the neurologic system are present, a comprehensive differential diagnosis becomes a key element [1]. Neurologic deficits suggesting damages to the nervous system are one of the symptoms observed in T. gondii infections in dogs. The type and severity of clinical illness depend on the degree and localization of tissue injury, in which cell necrosis is caused by the intracellular growth of the parasite. In one report on dogs that had died presumably from neurologic CDV infection, the presence of focal hemorrhagic and malacia in brain was described related to the detection of T. gondii cysts [12]. In a report from Brazil, almost 40% of the dogs infected with CDV were seropositive for T. gondii in IFA titers [13]. In another report, toxoplasmosis was diagnosed in 11 of 50 dogs that had neurologic signs based on IFA antibodies against T. gondii [17]. Although the IFA test is considered parasite-specific, the magnitude of titer is not associated with the presence or severity of the clinical signs. According to Dubey [2] a tentative antemortem diagnosis of clinical toxoplasmosis in dogs can be based on: (1) a combination of serology and clinical parameters such as high IgM titers or a fourfold or greater-increasing or decreasing-titer of IgG (after treatment or recovery) indicating recent or active infection, (2) the exclusion of other

causes of the clinical syndrome, and (3) a beneficial clinical response to an anti-Toxoplasma drug.

Learning points/Take home messages

In view of the similarity of the clinical signs, serological tests should be performed to differentially diagnose infection by N. caninum and T. gondii in all cases of dogs with neuromuscular signs, even if CDV virus has been identified because of the likely possibility of virus-parasite coinfection.

Consideration should be given to determining the serologic status against T. gondii before using drugs that are potent inhibitors of cell-mediated immunity, such as corticosteroids.

Confirming the presence of T. gondii in a dog from Córdoba, Argentina is relevant, because of the potential clinical relationship of this host to human beings through functioning as a canine sentinel of T. gondii proximity to humans.

Declarations

Conflict of interest statement: The authors do not have any financial or personal relationships with other individuals or organizations that could inappropriately influence the case report.

Ethics statement: This case was approved for presentation by the committee of the Veterinary Hospital of the Catholic University of Córdoba, which is responsible for the patient.

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