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. 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 neuro-ophthalmologic 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 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...
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].

**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 re-
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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