DYNAMICS OF IgG TOXOPLASMA GONDII TITER IN BLOOD OF DOGS DURING THERAPY

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ABSTRACT

The aim was to analyze the dynamics of IgG Toxoplasma gondii titer in dogs for several years after therapy, taking into account the seasonality of the disease. Daraprim has been shown to be the most effective drug for the treatment of acute toxoplasmosis, but it has the disadvantage that it affects only endozoites and does not affect Toxoplasma gondii cysts. IgG titer was recorded by serological method (ELISA test). The study was conducted on dogs of the German Shepherd breed, in which at the initial visit to the clinic were found titers of antibodies against toxoplasmosis. The level of antibodies in the repeated study was lower than in the primary; in the third study - lower than in the primary and secondary. That is, a decrease in the level of antibodies was observed in each subsequent study. This may indicate a decrease in the activity and amount of the pathogen or the tolerance of the immune system to the antigen.

Keywords: IgG, Toxoplasma gondii, Daraprim, Sulfadiazin, toxoplasmosis treatment, dogs

INTRODUCTION

Toxoplasmosis is common in countries with different climatic and geographical conditions, due to the presence of a wide range of hosts - more than 350 species of mammals [1]. The causative agent of toxoplasmosis - Toxoplasma gondii (group of coccidia) is an intracellular obligate parasite that is able to form cysts. The pathogen belongs to the type Apicomplexa, class Sporozoa, party Coccidiida, subfamily Isosporinae, genus Toxoplasma, and species Toxoplasma gondii. Italian researchers L. Rinaldi and A. Scala prove that toxoplasmosis is widespread from Alaska to Australia [2]. According to some authors, the infected organism remains a carrier of the pathogen =all life. Most animals do not show clinical symptoms of toxoplasmosis. Clinical manifestations of toxoplasmosis are related to species and age of the animal, stage and location of the parasite [3]. Mostly in asymptomatic forms of the disease, the ambiguity of clinical manifestations in the absence of specific symptoms causes difficulties in the diagnosis of toxoplasmosis. This may be redness of the eyes, a slight runny nose, short-term diarrhea. The animal (cat) may lose some weight, has short-term loss of appetite. With the transition to a chronic form of toxoplasmosis, the symptoms disappear [3]. In dogs older than 6 years of age, toxoplasmosis manifests itself in the form of intermittent fever, anorexia, depression, indigestion, dermatitis, weight loss and lasts several months. With the defeat of the nervous system there is increased aggression and excitability, convulsions, paralysis, paresis of the hind limbs [4]. Acute toxoplasmosis has been observed in dogs that have not been vaccinated against the immunosuppressive canine distemper virus (CDV) [5,6]. Clinical manifestations of toxoplasmosis are largely associated with the state of the immune system of the macroorganism and can vary from latent to septic forms with fatal outcome. The big problem of this invasion is the lack of specific clinical manifestations in patients with normal immunoresistance. As a result, animals are either not examined for toxoplasmosis, or diagnostic errors often occur, so the attention of doctors on this problem is very important [7]. Currently, for preventive measures and for the treatment of animals from toxoplasmosis, sulfadiazine and pyrimitamine are offered [8]. However, these drugs often cause side effects.

Indications for the diagnosis of animals and humans are a complete protocol of testing for the activity of the pathogen toxoplasmosis, which includes additional determination of IgA class antibodies and DNA toxoplasma in serum by polymerase chain reaction. If the results of these tests

are negative, the patient is excluded from the risk group for the disease and does not require re-examination [10].

AIM

The aim of our study was to analyze the dynamics of the titer of IgG Toxoplasma gondii in dogs for several years after therapy.

MATERIALS AND METHODS

The study involved three dogs of the German Shepherd breed aged 4-6 years, in which at the initial visit to the clinic were found titers of antibodies against toxoplasmosis. Clinical signs: lameness (arthritis and arthrosis with deformation of cartilage tissue) (Fig. 1), skin lesions, itching, nervous system disorders (convulsions during sleep, restless behavior, bouts of aggression).

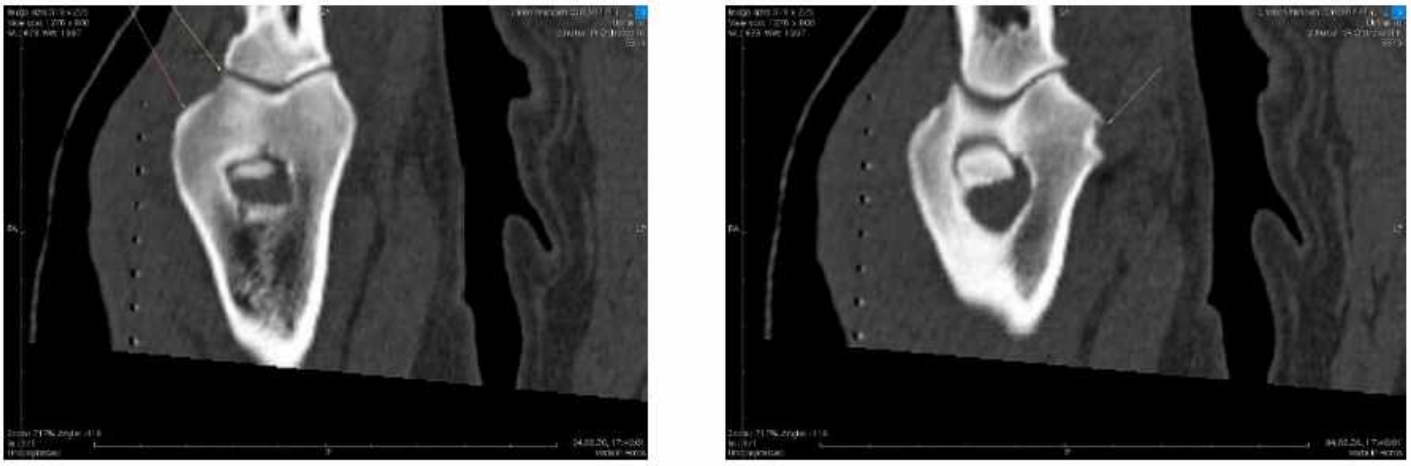

Fig. 1. Computer tomography with a quick test of cyst tissue

Animals were treated with Daraprim 2 mg / kg (active substance Pyrimethamine) once daily for 14 days, Sulfadiazin (150 mg / kg) twice daily for 14 days, Folic acid (5 mg) twice daily for 21 days. All animals that participated in the clinical experiment underwent a repeat course of therapy in connection with the restoration of the clinical picture, approximately one year later. After treatment and obtaining a clinical effect, the animal was observed with the help of monitoring studies of IgG Toxoplasma gondii. IgG titer was recorded by serological method (ELISA test) using a test system from Hema (Ukraine). Blood for the study was taken from the ulnar vein in the morning on an empty stomach.

RESULTS AND DISCUSSION

The most effective drug for the treatment of acute toxoplasmosis is Daraprim, but it affects only endozoites, which are actively multiplying and does not affect on cysts of Toxoplasma gondii. It should be noted that one of the three animals during therapy in the period from 4 to 6 days had signs of depression, refusal to feed, vomiting. This animal was additionally administered intravenous solution of reosorbilact in an amount of 200 ml and saline at a dose of 200 ml two times a day. The probable cause of this complication is an individual reaction to drugs in combination with toxins formed as a result of the death of endozoites Toxoplasma gondii.

Analyzing the graph (Fig. 2) of the results, it is seen that during the study period, IgG titers increased several times.

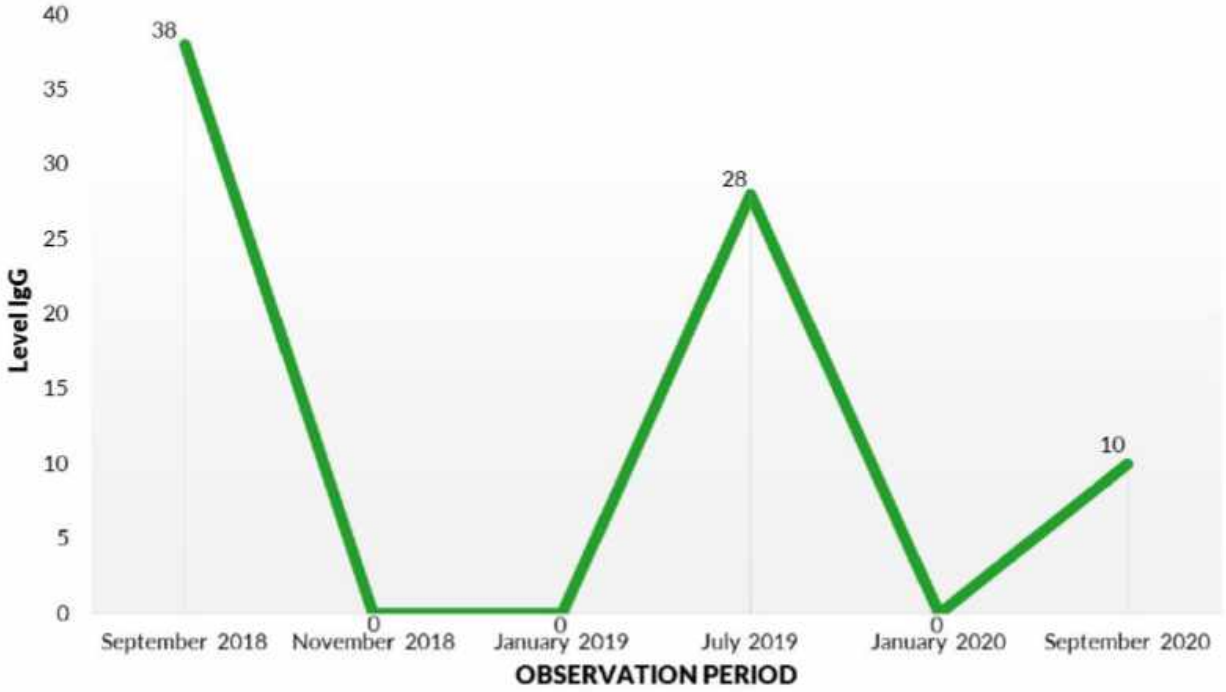

Fig 2. Dynamics of IgG titers during therapy

The increase in IgG titer is corresponded to the exacerbation of clinical signs of the disease. After therapy (November - January) a decrease in IgG titer was observed. Re-examination of serum in dogs for IgG titers of Toxoplasma gondii showed their presence. Therefore, the level of antibodies in the repeated study was lower than in the primary. The IgG titer in the third study was lower than in the primary and secondary. That is, a decrease in the level of antibodies was observed in each subsequent study, which may indicate a decrease in the activity and amount of the pathogen or the tolerance of the immune system to the antigen. It was observed a dependence that exacerbations are registered with the beginning of the autumn period of the year.

Our previous studies show that dogs have the lowest levels of cellular immunity at this time of year compared to other times of the year. At this time, the length of daylight is significantly reduced, weather conditions change: the level of solar radiation decreases, frosts begin. It is obvious that these factors lead to a pronounced immunosuppression, namely to a decrease in the number of lymphocytes on the background of reduced activity of microorganisms in the environment. In various experimental models of Toxoplasma persistence in cellular systems, Daraprim showed the effect of complete purification of cell cultures from Toxoplasma without further recurrence. Daraprim also formed three types of persistence.

In our experimental studies, re-establishment of the activity of the humoral part of the immune system against Toxoplasma gondii may indicate the need to change the treatment protocol.

CONCLUSIONS

In dogs, Toxoplasma gondii (as an intracellular parasite) retains activity and leads to clinical exacerbations, mainly in the autumn. It is likely that the autumn period of activity of the pathogen is associated with seasonal physiological depression of the immune system.

Preservation of individual foci of "dormant" intracellular forms of Toxoplasma gondii leads to reactivation of the symptoms of this disease, which in turn requires a comparative study of additional treatment protocols.

REFERENCES

Annual Epidemiological Report on Communicable Diseases in Europe (2014). https://www.ecdc.europa.eu /sites/portal/iles/media/en/publications/Publications/1011_SUR_Annual_Epidemiological_Report_on_Communicable_Diseases_in_Europe.pdf

Khan, M.U. (2017). Seroprevalence of Toxoplasma gondii in South Asian countries. 36(3),981-996. doi: 10.20506/rst.36.3.2730.

Rinaldi, L.& Scala, A. (2008). Toxoplasmosis in livestock in Italy: an epidemiological update. *Parassitologia*. 50 (1-2),59-61.

Limon, G. & Burrells, A. (2016). Toxoplasma gondii: Level of exposure in pigs and cattle in the UK and a hypothetical model for human exposure. https://www.food.gov.uk/sites/default/files/media/document/fs517004 finalreport.pdf

Ravilov, R.H., Gerasimov, V.V., & Vorobieva, M.N. (2008). Toxoplasmosis of domestic carnivores. Kazan, 98. Dubey, J.P., Beattie, C.P. (1988). Toxoplasmosis of Animals and Man. CRC Press, Boca Raton. 220

Capen, C.C.& Cole, C.R. (1966). Pulmonary lesions in dogs with experimental and naturally occurring toxoplasmosis. Pathol. Vet. 3. 40–63.

Rhyan, J.& Dubey, J.P. (1992). Toxoplasmosis in an adult dog with hepatic necrosis and associated tissue cysts and tachyzoites. Canine Practice 17.129.

Ferguson, D.J. (2008). Toxoplasma gondii: 1908–2008. Clin. Microbiol. Infect.8, 634–640.

Jurankova, J., Opsteegh, M., Neumayerova, H., Frencova, A., Balaz, V. & Koudela B. (2013). Quantification of *Toxoplasma gondii* in tissue samples of experimentally infected goats by magnetic capture and real-time PCR. *Vet. Parasitol*, № 193 (1-3). P. 95-99.