Polymyositis due to *Toxoplasma gondii* in an immunocompetent patient

Polimiosite por Toxoplasma gondii *em paciente imunocompetente*

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ABSTRACT

Toxoplasmosis is a worldwide zoonosis caused by the protozoan *Toxoplasma gondii*, which is mostly acquired by food contaminated with sporulated oocysts present in the feces of cats. The majority of patients evolve without symptoms, if immunocompetent. Its diagnosis is mainly clinical, confirmed by serological study. We report a rare occurrence of toxoplasmosis polymyositis in an immunocompetent patient, emphasing confounding factors, differential diagnoses, complementary tests and therapeutic management, according to the clinical picture.

Keywords: Toxoplasmosis; Polymyositis; Immunocompetent; Case reports

RESUMO

A toxoplasmose é uma zoonose cosmopolita causada pelo protozoário *Toxoplasma gondii*, adquirida por alimentos contaminados com oocistos esporulados, presentes nas fezes de felinos. A maioria dos pacientes evolui de forma assintomática, principalmente os imunocompetentes. Possui diagnóstico principalmente clínico, confirmado pela sorologia. Descrevemos uma rara apresentação de polimiosite por toxoplasmose em paciente imunocompetente, enfatizando os fatores confundidores, diagnósticos diferenciais, exames complementares e conduta terapêutica, de acordo com o quadro clínico.

Descritores: Toxoplasmose; Polimiosite; Imunocompetentes; Relatos de casos

INTRODUCTION

Toxoplasmosis, a zoonosis caused by the protozoan *Toxoplasma gondii*, is a disease of worldwide distribution, more prevalent in underdeveloped countries.(1) Depending on the population group and geographical area, up to 75% of adults have serologic evidence of infection.(2) Cats are infected and definitive hosts; other warm-blooded animals, including humans are intermediate hosts. In humans, the most common sources of infection are consumption of raw or undercooked meat/water contaminated with oocysts. The transmission mother to child during pregnancy is also noteworthy.(2,3) The disease can present itself in different clinical forms, being asymptomatic in most cases, especially in immunocompetent hosts.(1) About 10% of the cases in this group appear as a self-limiting, non-specific outbreaks which rarely needs treatment. Cervical and occipital lymphadenopathies associated with fever, are the most common manifestations.(3) Polymyositis, as well as myocarditis, encephalitis, pneumonitis and hepatitis are described as manifestations acquired after acute infection or after reactivation of latent infection, more frequently observed in immunodeficient individuals.(1,3)

CASE REPORT

NFPM, female, 24 years old, white, single, complaining of upper and lower limbs (LL) pain, associated with proximal muscle weakness and limited abduction movement of the arms. At this time, the patient had no fever, but reported recent history of febrile infection with the presence of painful cervical lymphadenopathy at the beginning. There were no gastrointestinal and genitourinary tract complaints. The patient denied comorbidities, allergies, previous surgeries or blood transfusions and chronic use of medications. She also reported complete vaccination. Physical examination: arterial blood pressure was 120x80mmHg and heart rate was 80 beats/minute; malar erythema and without itch, edema 1+/4+ and 2+/4 in LL; difficulty in walking and remain standing, decreased muscle strength in proximal upper limbs (grade 3) and preserved deep tendon reflexes. Laboratorial findings during hospitalization relevant were: hemoglobin 13.5g/dL; 6.820/mm^3 leukocytes, with differential within normal limits; 194.000/mm^3 platelets; ESR 55mm/h; AST 717U/L; ALT 18U/L; CPK 16,230U/L; aldolase 73.3U/L; LDH 852U/L; FAN 1/640; negative anti-Sm; negative for anti-DNA; anti-SSA (anti-RO) negative; anti-SSB

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Table 1. Laboratory tests performed during follow-up of the patient

	February 7th 2013	March 5th, 2013	March 8th, 2013	April 3rd, 2013	May 6th 2013
FAN	1/640	1/160	1/320	1/320	*
IgG-	Negative	Positive	Positive	Negative	*
IgM-	Negative	Positive	Positive	Negative	*
IgG-	904,9	1200	1230	1111	972,8
IgM-	16,99	13,37	8,55	5,56	4,91

* Measurements not performed.

(anti-LA) negative; negative anti-Jo1; negative for anti-RNP; anti-toxoplasmosis IgM, IgG and IgA reagents; avidity test anti-toxoplasmosis IgG: 0.229U/mL; antidengue sorology IgM and IgG reagents at first, later negatives (Table 1); ophthalmologic examination revealed no abnormalities; computed tomography within the normal ranges; electromyography: myopathic pattern without primary neuronal involvement.

Treatment for toxoplasmosis was started including: clindamycin 600mg intravenous (IV) four times daily, pyrimethamine 25mg orally (PO), one tablet three times daily and prednisone 40mg orally. This regimen lasted 10 days and resultedin progressive clinical improvement and decreased serum titers of IgM anti-toxoplasmosis. Finally, the patient was asymptomatic and was discharged.

DISCUSSION

The investigation of this case included the pathologies associated with polymiositis as autoimmune pathologies, like systemic lupus erythematosus, dermatomyositis, polymyositis, mixed connective tissue disease; infectious diseases as HIV, cytomegalovirus, infectious mononucleosis, dengue fever, toxoplasmosis; and neurological syndromes. Clinical, laboratory and serological findings, in conjunction with electromyographic study, allowed a diagnosis of polymyositis by toxoplasmosis. The anti-dengue serology was a false-positive finding during the clinical course. A muscle biopsy was not necessary, because in most cases the parasite can not be found in the muscle, though it is the most affected tissue.[4-6] The presence of IgG seroconversion with low avidity, significantly elevated titers of IgM, IgA or IgE antibodies for *Toxoplasmosis* in serial samples, in exam and satisfactory clinical response to specific antiprotozoal treatment combined with corticosteroid therapy are sufficient for the diagnosis documentation.[2,4] Based on reports of previous cases, it is evident that the keystone for efficient clinical response is the early initiation of specific antiprotozoal treatment.[4,7,8] Polymyositis is a syndrome secondary to the defect in cellular immunity, which is more commonly associated with systemic autoimmune diseases. However, it can be due to various causes, occurring alone or in association with infections, malignancies or disorders of connective tissue.[2,9] There are problems in establishing the diagnosis of polymyositis toxoplasmosis, which are as follows: the prevalence of Toxoplasma antibodies in the general population; presence of antinuclear antibodies and rheumatoid factors which may cause false positive results; insufficient formation of antitoxoplasmose antibodies to detect active infection in immunodeficient patients.[3,8] The number of adults with serologic reativity for toxoplasmosis is elevated in Brazil, among the highest in the world. Social and environmental peculiarities influence the risk factors and the impact on the seroprevalence.[3] It is noteworthy that the vast majority of those infected are asymptomatic or have mild symptoms, mainly immunocompetent individuals. The implication of myopathic genesis of *Toxoplasma* is rare and poorly understood, however it is suggested that the pattern of cellular immune response, is an important factor in for polymyositis.[5] Is suggested that all patients with polymyositis should have serologic testing for toxoplasmosis as part of their initial evaluation,[1,10] not only for a trial of early antiprotozoal therapy, in the case of positive results, but also to elucidate the relationship between toxoplasmosis and polymyositis.

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