International Journal of Infectious Diseases (2008) 12, 671–674


http://intl.elsevierhealth.com/journals/ijid

## CORRESPONDENCE

### Acute immune-mediated thrombocytopenic purpura related to *Toxoplasma gondii* infection

Acute toxoplasmosis has a benign course in immunocompetent adult patients. However, complications following infection can occur in congenital disease, immunocompromised patients, or with the ocular form.[1] Hematological disorders associated with acute toxoplasmosis are characterized by the mononucleosis-like form of the disease with leukopenia and lymphocytosis, including atypical lymphocytes in some cases. Anemia and thrombocytopenia are extremely rare events.[2]

The association of toxoplasmosis and low platelets has been described in the congenital form of the disease, whereas acute thrombocytopenic purpura has rarely been described.[3] This disorder can be a complication of the primary infection with poor outcome due to the risk of severe bleeding.

We describe a case of acute immune-mediated thrombocytopenic purpura (IMTP) related to *Toxoplasma gondii* infection in an immunocompetent adult patient.

A 49-year-old man presented with a 3-week history of fever, malaise, and myalgia. On physical examination, severe cervical lymphadenopathy and mild hepatomegaly were present. This patient was diagnosed as having a mononucleosis-like syndrome. Serologic examination for recent HIV, Epstein–Barr virus (EBV), rubella, and cytomegalovirus (CMV) infections were negative. Serology for hepatitis B virus, hepatitis A virus, and hepatitis C virus were also negative. Other laboratory examinations showed hemoglobin (Hb) of 12 g/dl, hematocrit (Ht) of 36.4%, white blood cell count of $9.4 \times 10^9$/l, platelets of $395 \times 10^9$/l, aspartate aminotransferase (AST) of 67 U/l, and alanine aminotransferase (ALT) of 58 U/l (normal AST = 37 U/l and ALT = 41 U/l). Antibodies against toxoplasmosis were positive (ELISA IgM = 9.93 and ELISA IgG $>$ 300 IU/ml). An indirect immunofluorescence IgM assay was positive for *T. gondii*. No ophthalmologic examination was performed.

Three weeks later, the patient returned to hospital complaining of petechial lesions on his legs. He denied other hemorrhagic symptoms and recent use of medication. Despite no visual symptoms, an ophthalmologic examination was performed and revealed a right acute chorioretinitis, sparing the macula, not presenting with associated old scars, suggesting acute toxoplasmosis. Laboratory testing showed a platelet count of $5.0 \times 10^9$/l, Hb 12.4 g/dl, and Ht 39.3% (Table 1). Coagulation tests were normal, and anti-nuclear antibodies and rheumatoid factor were negative. A bone marrow aspirate revealed a normal cell count, including normal platelet-producing megakaryocytes. An acute IMTP related to *T. gondii* infection was diagnosed, and prednisone 1 mg/kg/day was prescribed as well as sulfadiazine and pyrimethamine with folinic acid. After five days, the platelet count returned to normal levels ($172 \times 10^9$/l). At the one-month follow-up, the patient had maintained a normal platelet level with tapering of steroids. The treatment duration was 4 weeks and ophthalmologic examination after two months showed improvement of the ocular lesion with a tendency to scar.

The first case of thrombocytopenia associated with toxoplasmosis was reported by Borderon et al. in 1969.[4] Cases of chronic IMTP are the most commonly described, whereas there are only four cases in the medical literature of the acute form of IMTP associated with this parasitic infection.[2,5,6]

The case we have reported had a mononucleosis-like syndrome with ocular lesion and two different serological tests that were highly suggestive of acute toxoplasmosis. Three weeks later, the sudden appearance of petechial lesions and thrombocytopenia suggested the diagnosis of purpura. IMTP associated with *T. gondii* infection was diagnosed after other etiologies had been ruled out, including viral, bacterial, and other parasitic infections. Vaccination can be another cause of IMTP and was ruled out. Autoimmune tests should be performed in these cases; all described above were negative. The bone marrow aspirate also corroborated the diagnosis, showing normal platelet production.

The pathogenesis of IMTP associated with toxoplasmosis is not understood. The mechanism is probably similar to that of other infections associated with thrombocytopenic purpura, such as CMV, EBV, mumps, and rubella.[7] Nevertheless, despite increasing knowledge about IMTP, controversy surrounding treatment persists, and there is no consensus among experts on how to treat this complex disease. The management of IMTP is predicated on the extent of the thrombocytopenia and the symptoms of the disease. Corticosteroids and intravenous immune globulin are standard treatments, although the use of new therapies, such as monoclonal antibodies, has increased. In our case, a short course of corticosteroids for the treatment of the toxoplasmosis was enough to return the platelet count to normal levels.

**Table 1** Laboratory findings of immune-mediated thrombocytopenic purpura related to *Toxoplasma gondii* infection

| Variable | Period of the toxoplasmosis and immune-mediated thrombocytopenic purpura | | | |
|---|---|---|---|---|
| | Mononucleosis-like | Immune purpura | 1 week after | 1-month follow-up |
| Hemoglobin (g/dl) | 12.0 | 12.4 | 11.8 | 13.4 |
| Hematocrit (%) | 36.4 | 39.3 | 38.2 | 43.6 |
| White cell count ($\times 10^9$/l) | 9.46 | 6.74 | 7.74 | 7.77 |
| Neutrophils ($\times 10^9$/l) | 4.90 | 2.40 | 3.90 | 4.30 |
| Lymphocytes ($\times 10^9$/l) | 3.00 | 3.90 | 2.20 | 2.50 |
| Platelets ($\times 10^9$/l) | 395 | 5 | 172 | 299 |
| C-Reactive protein (mg/l) | 29.4 | 2.58 | 1.59 | 0 |

## Acknowledgement

We thank Helena Petta, André Siqueira Machado, and Silvia Goulart for clinical and laboratory assistance.

*Conflict of interest:* No conflict of interest to declare.

## References

1. Bhopale GM. Pathogenesis of toxoplasmosis. *Comp Immunol Microbiol Infect Dis* 2003;**26**:213–22.
2. Deutsch M, Nezi V, Kountouras D, Belegrati M, Kalmantis T, Dourakis SP. Immune-mediated thrombocytopenic purpura associated with *Toxoplasma gondii* infection in an immunocompetent patient. *Hematol J* 2004;**5**:538–9.
3. Hohlfeld P, Forestier F, Kaplan J, Tissot J, Daffos F. Fetal thrombocytopenia: a retrospective survey of 5,194 fetal blood samplings. *Blood* 1994;**84**:1851–6.
4. Borderon JC, Pesnel G, Canet J, Fabre A, Solle R, Lajouanine P. Thrombopenic purpura and acquired toxoplasmosis. *Ann Pediatr (Paris)* 1969;**16**:129–32.
5. Gurkan E, Baslamisli F, Guvenc B, Bozkurt B, Unsal C. Immune thrombocytopenic purpura associated with Brucella and Toxoplasma infections. *Am J Hematol* 2003;**74**:52–4.
6. Michez D, Quintart C, Noel E, Lepage P. Peripheral thrombopenic purpura associated with acquired toxoplasmosis. *Rev Med Brux* 1998;**19**:135–7.
7. Yenicesu I, Yetgin S, Ozyurek E, Aslan D. Virus-associated immune thrombocytopenic purpura in childhood. *Pediatr Hematol Oncol* 2002;**19**:433–7.

Felipe Francisco Tuon
Hermes Ryoiti Higashino
Antonio Carlos Nicodemo*
*Department of Infectious and Parasitic Diseases, Medical School, University of São Paulo, São Paulo, SP, Brazil*

Valdir Sabbaga Amato
*Infectious and Parasitic Diseases Clinic, Hospital das Clínicas, Medical School, University of São Paulo, São Paulo, SP, Brazil*

*Corresponding author.
Current address: Rua Barata Ribeiro, 414, Conjunto 104, CEP 01308-000, São Paulo, SP, Brazil.
Tel.: +55 11 32144251; fax: +55 11 32591318
*E-mail address:* ac_nicodemo@uol.com.br (A.C. Nicodemo)

**Corresponding Editor:** William Cameron, Ottawa, Canada

23 August 2007

doi:10.1016/j.ijid.2007.12.009

## An unusual case of *Corynebacterium striatum* endocarditis and a review of the literature

An 83-year-old man was admitted with a 3-day history of fever and joint pain (mostly knee and shoulder). Over the previous three weeks he had been treated in the community with ciprofloxacin for a presumed recurrent urinary tract infection. He was known to have metastatic prostate cancer and had developed secondary hyperfibrinolysis syndrome with an intracerebral bleed from which he had made a good recovery.

On examination, he was afebrile. His pulse rate was 85 per minute and his blood pressure was 98/60 mmHg. An ejection systolic murmur was heard. His knees and shoulders were tender and hot to the touch. Respiratory and abdominal examinations were normal. Neurological examination revealed increased tone in the muscles.

Polymyalgia rheumatica, polyarthritis, rheumatoid arthritis, and metastatic effects of prostate cancer were considered in the differential diagnosis. Laboratory studies revealed elevated urea and creatinine (12.5 mmol/l and 148 μmol/l, respectively), high alkaline phosphatase (351 U/l), and a markedly elevated C-reactive protein (CRP) (565 mg/l). He had a mild leukocytosis ($11.4 \times 10^9$/l) and a raised erythrocyte sedimentation rate (74 mm/h). Tests for rheumatoid factor and anti-neutrophil cytoplasmic antibody were negative. A chest roentgenogram showed clear lung fields, but extensive bony sclerosis was noted consistent with bony metastases.

Four separate sets of blood cultures taken at different times grew *Corynebacterium striatum*. In the absence of definitive laboratory guidelines for determining the antibiotic susceptibility of diphtheroids, we established the minimum inhibitory concentrations (MIC) for penicillin,