Annals of Diagnostic Paediatric Pathology 1997;1(4):267-270

Annals of Diagnostic Paediatric Pathology

Case Report

Congenital toxoplasmosis in a neonate of HIV infected mother*

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Key words: congenital infection, toxoplasmosis, HIV, pregnancy, neonate

ABSTRACT Few references of maternal AIDS associated to *Toxoplasma gondii* infection exist in the literature, in spite of the fact that the newborns of HIV infected mothers are prone to infection. We present a case of a 23-year-old HIV positive mother with accentuated weight loss and Kaposi's sarcoma during pregnancy, having probably been infected by toxoplasma in the last trimester. The newborn showed early and progressive respiratory distress, disseminated petechiae and hepatosplenomegaly. He died eight days after birth. Morphological feto-placentary examination revealed acute, severe and disseminated toxoplasma lesions; noteworthy was the abundance of trophozoites and cysts of the protozoon. HIV p24 antigen was detected in the placentary tissues by immunohistochemistry.

INTRODUCTION

The newborns of Human Immunodeficiency Virus (HIV) infected mothers are possibly more prone to *Toxoplasma gondii* (*T. gondii*) infection, due to maternal immunossupression, giving rise to parasitic recurrences of a previous infection. Remington et al [1] accentuated that the ocurrence of HIV infection and AIDS in pregnant women who also are chronically infected with *T. gondii* has emerged as a significant threat for infection of the fetus with either or both the agents. They complemented that the incidence of congenital transmission of *T.gondii* from those mothers appears to be significantly higher than in non HIV infected women.

Notwithstanding, the literature is lacking in cases of neonatal AIDS and congenital toxoplasmosis. Transmission of *T. gondii* from a chronically infected mother can occur in the setting of an HIV infection but this is not a common phenomenon [2]. We present an observation of neonatal death of a baby infected by *T. gondii*, whose HIV infected mother died six months after delivery.

CASE REPORT

We report the case of a male infant from a 37 week gestation, AGA, weighing 2770g,

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*In part granted by Conselho Nacional de Desenvolvimento Científico e Tecnológico (CNPq), Brazil

Received: July 22, 1997, Accepted: November 5, 1997

Annals of Diagnostic Paediatric Pathology

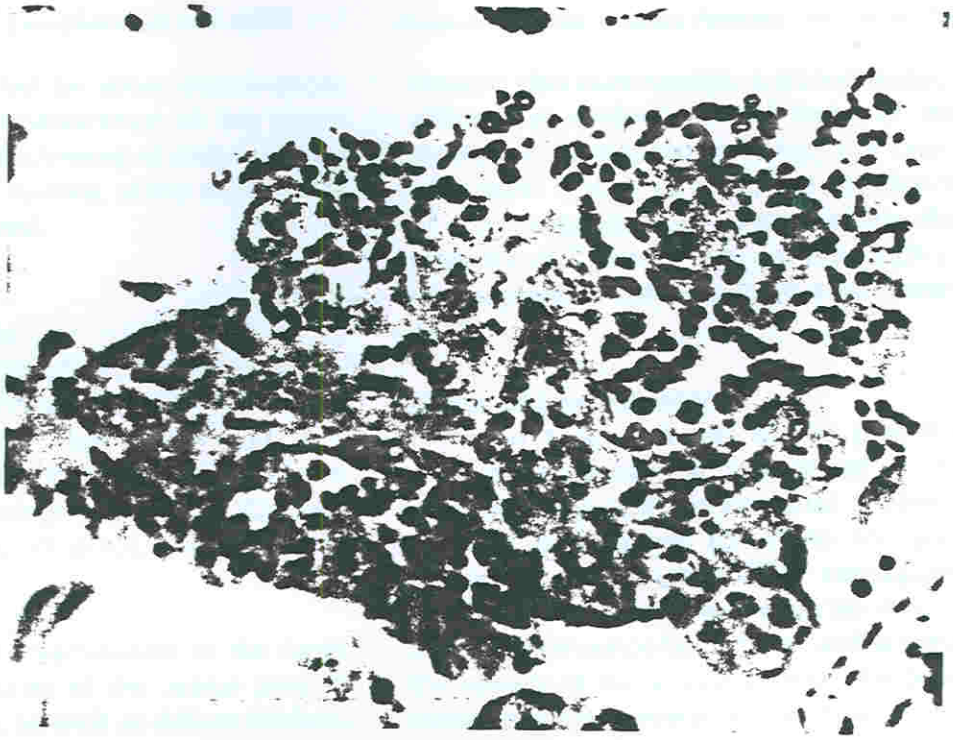

Figure 1. Placenta - Chorionic villus exhibiting extensive necrosis involving trophoblast, capillaries and stroma. Arrow points to group of trophozoites of *T. gondii*. Abundant perivillous cellular exsudate. (HE X 450).

with an APGAR score of 5/3/7 (at 1, 5 and 10 minutes). The amniotic fluid was heavily meconium-stained. Two hours after birth the infant presented mild respiratory distress, petechiae and hepatosplenomegaly.

Laboratory studies showed a white blood cell count of 7100 mm^3 (20% neutrophils, 46% band forms, 31% lymphocytes, 3% monocytes). The hematocrit was 56%. Serologic tests did not indicate infection by rubella virus, herpes simplex virus, cytomegalovirus or syphilis. CFS protein was 40 mg/100ml; glucose was 42 mg/100ml and there were 1200 cells/mm3 (lymphocytes). At 24 hours of life the patient's condition worsened and 30% of oxygen in a hood was deemed necessary. The next day he presented a digestive hemorrhage, fever and his pulmonary condition became increasingly serious. He died 8 days after birth.

His mother was 23-year-old, with a 10 month history of cough, fever, weakness, hair loss; she referred piodermitis and a 10Kg weight loss during pregnancy. Two months before term she had a chest X-ray which showed a discrete diffuse interstitial infiltration; an inconclusive diagnosis of tuberculosis was made, but due to her history, specific medication was started. One month later, she had an HIV positive test. After birth a skin biopsy detected Kaposi's sarcoma; serology later revealed toxoplasma infection. She left the hospital one month after birth and died 5 months later.

PATHOLOGICAL DATA

Gross Examination

At autopsy the neonate weighed 2.520 g, measured 47 cm and had 33 cm of cephalic circumference; no malformations were observed. The child presented mild edema, anaemia, widespread lymphadenopathy and hepatosplenomegaly; the liver was at 4cm and the spleen at 2cm bellow the costal margin. The hepatic parenchyma was dark green and the vascular marks were pronounced. Both lungs were bulky and dark red, weighing 79.3g. No other data were noteworthy.

The placenta was large weighing 805 g and measuring 20 x15cm. Hematogenic placen-

titis was suspected by gross examination, represented by opalescence of the membranes allied to thickening of umbilical vessels. Meconium staining of the membranes and cord was noted.

Histological Data

Sections of formalin-fixed tissues were embedded in paraffin and filled in the department. The routine histological stain was hematoxylin-eosin; for the histological revision special stainings were made (Giemsa, Periodic Acid Schiff (PAS), thrichromic of Gomori).

The microscopic examination of the lungs showed dysmaturity of the acinar area of the parenchyma, as well as diffuse thickening and mononuclear infiltration of the interalveolar septa. Noteworthy was the presence of areas of septal fibrosis and fetalization, chronic bronchiolitis and vasculitis. In the lumen of the alveolar ducts, a few cysts of *T. gondii* were seen. At the nervous tissue there was evidence of meningitis, characterized by leptomeningeal inflammatory cell infiltrates; toxoplasma cysts and glial nodules were abundant. No foci of necrosis and mineralization were observed. The microscopic features were suggestive of the initial phase of the infection.

The heart exhibited the common histologic aspects peculiar to this protozoosis, expressed by chronic endo, myo and pericarditis. Aside the inflammatory lymphocytic infiltration, foci of fibrilar necrosis and the presence of microorganisms were observed. In the liver, foci of coagulation necrosis were common, allied to severe hepatocytic and caniliculi cholestasis and dense mononuclear infiltrate. The paucity of lymphocytes of the spleen, lymphnodes and thymus was pronounced. The thymus was represented by irregularly shaped lobules, exhibiting reticulo-epithelial net, and few enlarged Hassal's bodies. In the kidneys, an interstitial and focal infiltration of round cells mainly perivascular was present; toxoplasma cysts in the gromeruli were observed. In the placenta, aside the dysmaturity of the villous structures and chronic chorioamnionitis, a granulomatous villitis was evident; several forms of the protozoon, as trophozoites (Fig 1) or cysts, were seen. Noteworthy was the abundance of microorganisms in organs where the presence of *T.gondii* is not common such as the esophagus, testicles, kidneys and bone-marrow.

Immunohistochemistry

Formalin fixed samples of the placenta were deparaffinized and processed to immunolabeling with monoclonal antibody anti-HIV p24 antigen (Du Pont) and goat serum anti-IgG mouse FITC conjugated (Sigma). This antigen was detected mainly at the cytotrophoblastic layer and around the vessels of the villous stroma. The positivity revealed severe compromise of the placenta in both maternal and fetal parts. Immunohistochemistry was not done to *T.gondii* in the same tissues.

DISCUSSION

Congenital transmission of *T. gondii* from pregnant women infected with this protozoon and HIV has only recently been recognized as an unique problem [2, 3, 4]. Even in 1988, Belman et al [5] noted that toxoplasmosis, congenital or acquired, was uncommon trait in vertically transmitted HIV infection. Presentely available data reveal that these mothers have chronic toxoplasmosis and do not have demostrable IgM toxoplasma antibodies [1]. Desmonts et al [6], who reported five cases of reactivated toxoplasmosis with vertical transmission during pregnancy, made it plain that this could only account for a few cases of mothers with immunossupressive disorders.

In the case here reported, we have no data which permit us to consider toxoplasma infection as a primary event or as a reactivation due to HIV induced immunossupression. It is worth point out the abundance of microorganisms as trophozoites and cysts of *T. gondii*, wich be due to higher virulence of the protozoon or to decreased maternal and feto-placentary immunity.

In the study of these tissues, immunohisto-

chemistry was employed to verify the presence of the viral antigen and thus the dual infection. The detection of HIV antigens in feto-placentary tissues from infected mothers has been reported [7, 8].

Nevertheless, based on these data, it has been difficult to know if HIV infections do really reach the fetus transplacentally. The simple detection of HIV antigens in placentas of seropositive mothers may reflect the efficiency of the placentary barrier, as well as the viral replication in these sites.

ACKNOWLEDGEMENTS

We are indebted to Cláudia Serpa Osório de Castro for the criticism and revision of the English text and Elizabeth Ferreira da Silva and Daniel Schwartz Pegado for secretarial help.

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