

CONGENITAL TOXOPLASMOSIS IN IRAN

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ABSTRACT

This is a report of the first proved case of congenital toxoplasmosis in Iran in an infant who died with hypothermia and lethargia 29 days after birth. Both parents showed serological evidence of toxoplasmosis and the finding in the mother indicated recent infection. The baby, during her short life, showed prominent neurological disorders and hepatosplenomegaly. Toxoplasms were demonstrated in the ventricular fluid and brain tissue. The parasite was also isolated by mice inoculation.

INTRODUCTION

Toxoplasmosis is one of the infections most commonly seen in man and animals throughout the world. Congenital toxoplasmosis, which is acquired by the foetus during an asymptomatic or mild primary infection of the mother with parasitaemia, is probably the most serious form of infection (WHO, 1969).

According to views of most investigators, congenital toxoplasmosis or abortion due to *Toxoplasma* infection results only from primary infection during pregnancy and any woman who has experienced an acute *Toxoplasma* infection with antibodies present before becoming pregnant will not have a congenitally infected offspring or abortion due to toxoplasmosis (Desmonts et al. 1965, WHO, 1969, Kimball et al. 1971, Frenkel 1971).

Although congenital toxoplasmosis has not been reported from

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Iran, *Toxoplasma gondii* has been isolated from human lymph node, stray cats and guinea pigs in this country (Ghorbani and Samii 1973, Anwar et al. 1973, Ghorbani and Hafizi, 1974).

This paper reports the first human case of congenital toxoplasmosis in Iran, diagnosed by demonstration of the parasite in ventricular fluid and isolation of the causal organism after mice inoculation.

CASE REPORT

Maternal history: Sh. Y. 24 years old, primipara, B Rh⁻, had normal birth under medical care. At 4th month of pregnancy she had herpes simplex around her mouth and a mild cervical lymphadenitis in the 5th month of pregnancy.

Baby girl S.Y., a product of 39 weeks gestation was born on Dec. 11th, 1976. She was 2,550 gm, 47 cm, with head circumference of 36 cm, AB Rh⁺, and negative Coombs' test. Her liver and spleen were not enlarged. The baby developed generalized seizure 3 days after birth. On Dec. 17th, head circumference was 36.5 cm and right eye seemed to be smaller than left with enophthalmia. On Dec. 20th, head circumference was 38 cm and liver and spleen were enlarged. On Dec. 22nd, head circumference was 38.5 cm, the skull X-ray showed remarkable increase in general size with prominent fontanells but did not show calcification. Examination of the eyes showed enophthalmos, hypotropia, convergent squint and microphthalmos of the right eye. Left fundus was seen but right fundus was not seen due to low set globe, small pupil and cloudy media. On Dec. 27th head circumference was 40 cm, the liver and spleen were 5 and 6 cm below right and left costal margin respectively. Fresh urine for cytomeglovirus infection was negative, reducing substance, ferric chloride tests and V.D.R.L. were negative. Haemagglutination inhibition test for rubella was negative, serum IgM was 350 mg %. Ventriculus tap was done: WBC=640 (PMN 60%, Lymph. 20 %), RBC=1820, protein 3500 mg %. In the ventricular fluid *Toxoplasma* tachyzoites were observed. Indirect fluorescent antibody test (IFA) for toxoplasmosis were performed on the sera from the mother and child which revealed an antibody titre of 1:6400 on the serum from the mother and 1:400 on the serum from the child.

On Jan. 4th, 1977, the child had right irregular pupil, post synechae iritis and cataract. Left eye showed chorioretinitis.

The child was under treatment with antibiotic and I.V. fluids before the diagnosis of toxoplasmosis was made, but after the diagnosis she was treated with pyrimethamine and sulfadiazine. She expired on Jan. 8th 1977 with hypothermia and lethargia.

weeks to months after birth.

In the described case most of the characteristic symptoms of congenital toxoplasmosis were observed shortly after birth. We tried to find out the source of infection of the mother by reviewing her history. As mentioned, she had herpes simplex around her mouth and mild cervical lymph-adenitis during pregnancy. Presumably, adenopathy during second trimester of pregnancy has been due to toxoplasmosis. Furthermore, high antibody titre to *Toxoplasma* in the mother confirms her recent acute infection, which produced serious hazard to the foetus. IFA done on her husband's serum showed an antibody titre of 1:1600. The family did not keep a cat or any other pet in their house, but underdone kebab (steak) was usually consumed by the family. Therefore, the source of infection seems to be consumption of underdone meat.

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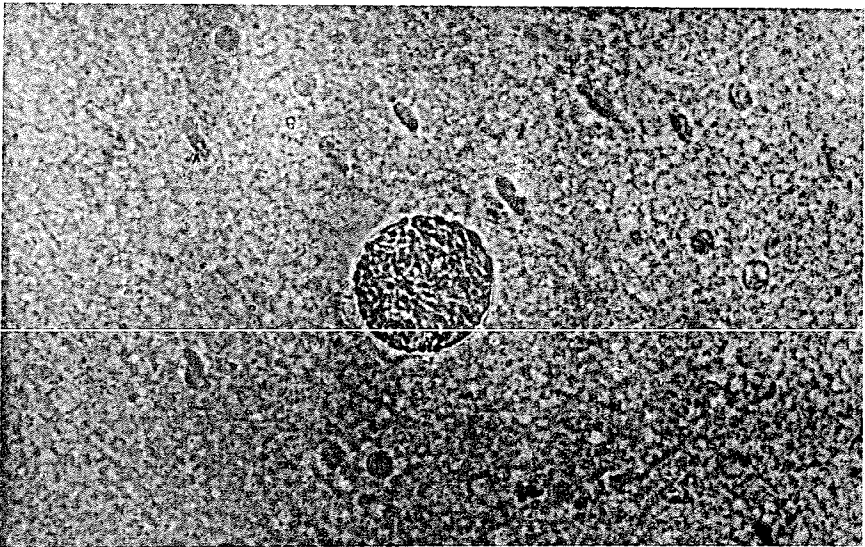


Fig. 1. *Toxoplasma gondii* in cystic stage. Unstained wet film of the infant's brain tissue (X500).