

## Congenital toxoplasmic myocarditis: case report of an unusual presentation

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**SUMMARY** Extensive calcification of the heart, involving predominantly the right ventricle and interventricular septum, was observed in a 3-h-old baby. The aetiology of the condition was attributed to infection by *Toxoplasma gondii*, based on the presence of several visceral lesions allied to the identification of the microorganism. The case is reported in view of the rarity of the type of the heart lesion.

It is considered that calcification of the myocardium is secondary to vascular, inflammatory or toxic condition. The factors implicated in the pathogenesis of this unusual myocardial lesion are discussed.

### Introduction

According to Eichenwald (1), the clinical features of congenital toxoplasmosis may be so varied that it should be taken into consideration in the differential diagnosis of all obscure neonatal disease. As the maternal infection generally develops in the last trimester of pregnancy (2), the neonatal infection may be clinically inapparent or manifest by several clinical presentations.

The pathological lesions are equally polymorphous, involving several tissues, because of the systemic dissemination which characterizes the first step of the protozoal infection. The myocardium, second only to the brain and the eyes is considered to be commonly affected (3). Although gross examination may furnish some information, the aetiology of the myocarditis is given by the histology of the myocardium, through the presence of inflammatory and degenerative lesions of the muscle fibre caused by the parasite.

We had the opportunity to observe a case of congenital toxoplasmosis in which a rare type of

lesion was detected. Since the autopsy was performed in a private clinic the autopsy findings were incomplete, and some routine data such as weights and measurements were not taken. Notwithstanding, the purpose of this paper refers to the rarity of the type of cardiac lesion.

### Case report

A hydropic white female infant was born after a "normal" pregnancy (36 weeks) and delivery to a primigravida. The infant died 30 min after birth with signs of cardiac failure. There was no information about maternal infection with *T.gondii* during pregnancy but in the puerperium she had a serum dye test titre of 1/16.384, which is compatible with recent primary infection.

Autopsy revealed a white female infant with generalized oedema, pallor and petechial haemorrhages all over the body. A tense abdomen with hepatosplenomegaly was noted. The other noteworthy point was the heart, which was pale, yellow and flabby, with a white friable nodular area near the apex and localized on the wall of the right ventricle (Fig. 1). All the other cardiac structures looked normal except for the ventricular septum, where

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**Figure 1** Heart and great vessels—on the right ventricle wall a whitish nodular area is observed.

some irregular white areas were observed.

When the head was examined an abundant yellow turbid fluid flowed. Over the leptomeninges and covering the cerebral cortex were areas of opacity. Nodular white lesions were scattered on the cortex, the basal nuclei and the brainstem, which were observed to be identical to those on the heart. The placenta was voluminous, hydropic and fragmented. The villous tissue was oedematous, pale and friable. Areas of opacity on the membranes and thickening of the umbilical vessels were identified.

Microscopy demonstrated visceral lesions attributed to *Toxoplasma gondii*. The following abnormalities were noted: haematogenous placentitis, meningoencephalitis with glial nodules, areas of necrosis and mineralization; retinochoroiditis, hepatitis, adenitis, interstitial nephritis, pancreatitis, hypertrophy of the lymphoid tissue, interstitial pneumonitis, myositis and pancarditis. Throughout the brain, brainstem, spinal cord, retina, striated muscle, lung, adrenals, umbilical cord and placenta cystic forms of the protozoa were detected. Histologic examination of the heart disclosed involvement of all structures of the organ, including the pericardium, the myocardium and the endocardium. The inflammatory

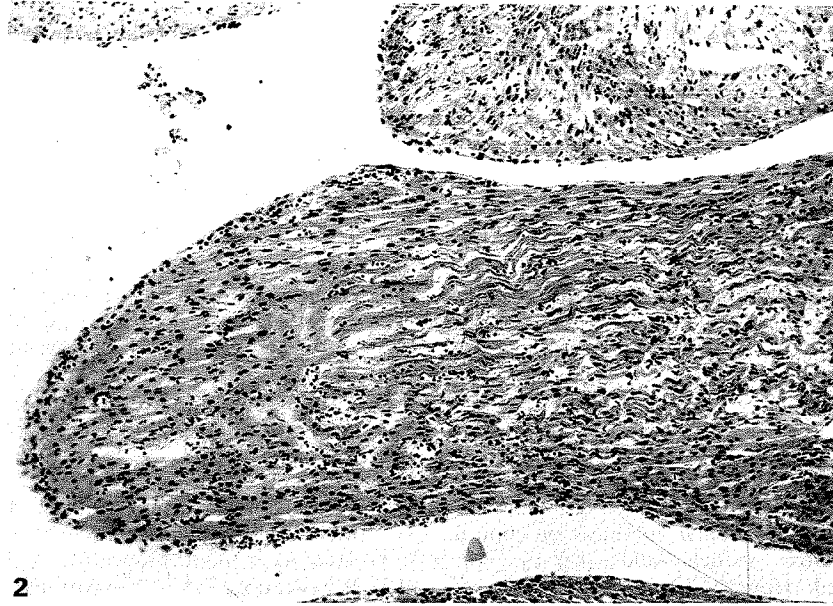
infiltrate was mostly interstitial, patchy in distribution and sited in the perivascular tissues. It consisted of histiocytes, lymphocytes, plasma cells and eosinophils. Myocardial fibres were separated by oedema and inflammation. There was necrosis, fragmentation and hyalinization of individual fibres (Fig. 2). Noteworthy was the presence of extensive areas of necrosis and calcification of the right ventricle and interventricular septum (Fig. 3). The coronary vessels were normal. A few, free and intracellular trophozoites were identified but encysted parasites were not seen.

### Discussion

Torres (4) was the first to describe the presence of *T. gondii* in the myocardium of a newborn. This single observation remained the only example for several years. Now, however, myocarditis is considered to be nearly always present in the instances of generalized toxoplasmosis and often present in the congenital infection (5). Experimentally, it was demonstrated that in acute generalized toxoplasmosis the myocardium is always involved (6, 7). The purpose of this paper is to demonstrate the rarity of this type of cardiac lesion which we have not seen described and which has not been observed in 47 cases of congenital toxoplasmosis on which we have performed autopsies.

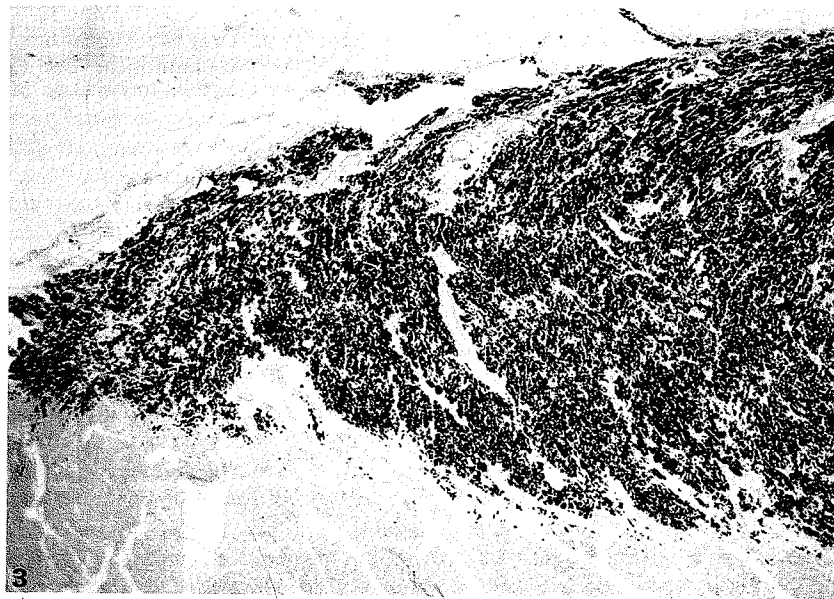
Although pathological calcification is not uncommon in perinatal pathology, calcified changes in the myocardium are rare. It is postulated that calcification of the myocardium is secondary to vascular, inflammatory or toxic conditions (8). In the literature there are some cases of calcification of the heart that resemble the one we describe. Definite infections had been present in all of the cases (9). Vássina *et al.* (10) relate a case of a microcephalic child who presented with myocarditis associated with calcifications of the heart muscle, without a definite aetiology being demonstrated. Probably the myocardial calcification resulted from a severe myocarditis. Ernstene (11) emphasized that mineralization is always preceded by degenerative and necrotic lesions in the muscle fibres and this can occur in any pathological condition capable of destroying the cardiac muscle. The fact that the blood vessels were normal points to a non-vascular cause of the fibre necrosis.

Niedmann *et al.* (12) quoted that the inflammatory reaction in *T. gondii* infection is independent of the virulence of the parasite as well as of the host defences. They attributed the inflammatory phenomenon to immunological factors and to antibodies produced by the protozoa in the myocardium.



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**Figure 2** Focal and diffuse inflammatory infiltration of the myocardium and overlying endocardium. Focal necrosis of the heart fibres (H & E  $\times$  125).

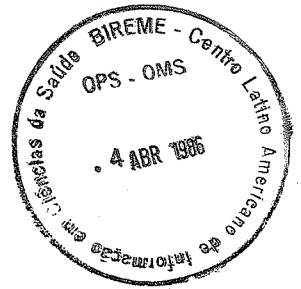


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**Figure 3** Extensive area of cardica mineralization involving pericardium and myocardium (H & E  $\times$  125).

Shevkunova *et al.* (10) considered that necrotic and destructive lesions of the myocardium resulted from the mechanical action of the parasite in its reproduction and to the action of toxins which may be respon-

sible for the vascular lesions. Henry and Beverley (6) verified in mice that, usually, the highly virulent strain of *T. gondii* produced larger areas of myocardial necrosis but that the low virulent strain



produced less severe but more consistent and widespread lesions with necrosis of single fibres or small groups of fibres. In rabbits inoculated with a highly virulent strain they (7) observed that myocarditis was much less severe than that caused by the low virulent strain.

The paucity of polymorphs, the predominance of lymphocytes and the presence of plasma cells in the inflammatory exudate may indicate that a hypersensitivity process is involved, particularly in the necrosis of the muscle fibre. The unusual aspect of the heart lesion in the present observation may be linked to these factors.

#### References

- (1) Eichenwald AF. A study of congenital toxoplasmosis with particular emphasis on clinical manifestations, sequels and therapy. In: Siim JC, ed. Human Toxoplasmosis, Copenhagen: Muksgaard, 1979.
- (2) Desmonts G, Couvreur J. Congenital toxoplasmosis. A prospective study of 378 pregnancies. *N Engl J Med* 1974; 290: 110-6.
- (3) Zuelzer WW. Infantile toxoplasmosis with report of three cases including two in which the patients were identical twins. *Arch Pathol* 1944; 38: 1-19.
- (4) Torres CM. Sobre uma nova entidade mórbida do homem caracterizada por meningoencefalite, miosite e miocardite aguda congénitas, associadas à presença, nos respectivos tecidos, de um novo parasita. *Encephalitozoon Chagasi*. *Brasil Médico* 1928; 42: 1-14.
- (5) Lelong M, Bernard J, Desmonts G, Couvreur J. La toxoplasmose acquise (Étude de 227 observations). *Arch Fr Pédiatr* 1960; 17: 281-331.
- (6) Henry L, Beverley JKA. Experimental toxoplasmic myocarditis and myositis in mice. *Br J Exp Pathol* 1969; 50: 230-8.
- (7) Henry L, Beverley JKA, Archer JF, Johnson SG. Experimental toxoplasmic myocarditis in rabbits. *J Pathol* 1973; 109: 141-51.
- (8) Diamond M. Calcification of the myocardium in a premature infant. *Arch Pathol* 1932; 14: 137-45.
- (9) Scholz T. Calcification of the heart: its roentgenologic demonstration. Review of the literature and theories on myocardial calcification. *Arch Intern Med* 1924; 34: 32-59.
- (10) Schevkunova EA, Novikov FE, Varlamova TA. Lesions of the heart in toxoplasmosis. *Kardiologia* 1976; 16: 132-40.
- (11) Ernstene AC, Hazard JB. Extensive calcification of the myocardium. Report of a case. *Circulation* 1951; 3: 690-3.
- (12) Niedmann G, Del Campo E, Thierman E, Sánchez J, Levy M. Miocarditis de probable etiología toxoplasmica. *Bol Chil Parasitol* 1962; 17: 58-63.