Congenital Tuberculosis

A Report of a Probable Case

F. B. LE ROUX, J. SCHWERSENSKI, M. J. GREEFF

SUMMARY

Extensive miliary pulmonary tuberculosis in a 17-day-old infant born to a mother with pulmonary tuberculosis is described. It was assumed that the patient probably had congenital tuberculosis. We report our findings to draw attention to a rare disease where a high index of suspicion, timely diagnosis and adequate management are essential if treatment is to be successful.

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Reported cases of congenital tuberculosis are very rare and most of them occurred before the era of chemotherapy.[1,2] Criteria adopted to ascertain the congenital origin of the disease differ considerably according to various authors.[1] In 1935 Beitzke[2] listed conditions which, in his opinion, were necessary for such a diagnosis. These criteria have been accepted by most authors[1,3,4] and are as follows:

(i) the tuberculous nature of the lesions in the infant must be proved;

(ii) a primary complex affecting the fetal liver is proof of the congenital nature of the infection since the organisms must have been carried in the blood of the umbilical vein;

(iii) in the absence of a primary complex affecting the liver, the infection is congenital only (*a*) if the tuberculous lesions are found in the fetus *in utero*, at birth or within a few days of birth; and (*b*) if the child lives longer than a few days, and an extrauterine infection can be excluded with certainty, the child having been separated immediately from the mother and kept in an environment free of tubercle bacilli.

It has been noted that when the infection is acquired at or soon after birth, the onset of the illness is usually delayed until after the first 4 weeks of life.[5,6]

We wish to report a case of an infant who presented at the age of 17 days with extensive pulmonary tuberculosis which was most probably the result of a congenital infection.

Department of Anatomical Pathology, University of Stellenbosch and Tygerberg Hospital, Parowvallei, CP

F. B. LE ROUX, M.B. CH.B., M.MED. (PATH.), *Senior Consultant*
M. J. GREEFF, M.B. CH.B., *Senior Medical Officer*

Department of Paediatrics, University of Stellenbosch and Tygerberg Hospital, Parowvallei, CP

J. SCHWERSENSKI, B.SC., M.B. CH.B., D.C.H., *Registrar*

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CASE REPORT

A 17-day-old baby girl was admitted to hospital with a history of difficult feeding since birth. Twenty-four hours before admission, the infant had become very short of breath and cyanotic. On examination the child was found to be severely distressed and dyspnoeic. Radiography showed a bilateral, diffuse, fine nodularity throughout both lung fields. Fluid from the gastric aspirate contained numerous acid- and alcohol-fast bacilli that were morphologically identical with *Mycobacterium tuberculosis*.

The diagnosis of severe, fulminant bronchopneumonia was made and congenital tuberculosis was regarded as a possibility. Despite intensive therapy, the infant died 24 hours after admission and before antituberculous treatment could be instituted.

On interrogation the mother admitted having had a persistent cough and nocturnal sweating during pregnancy. Subsequent chest radiographs showed a miliary tuberculosis affecting both lungs. Repeated direct examination of sputum and sputum cultures performed after delivery were negative for tubercle bacilli. The mother's response to antituberculous therapy has been good.

POSTMORTEM FINDINGS

The infant's mass was 2 110 g and her length 46 cm. Relevant findings included pericardial and bilateral pleural serous effusions. The right lung weighed 70 g and the left 68 g. Miliary tubercles, 1 - 2 mm in diameter, were diffuse and evenly spread throughout both lungs (Fig. 1). No primary focus could be demonstrated. Although the mediastinal lymph nodes were enlarged, there were no macroscopical signs of necrosis.

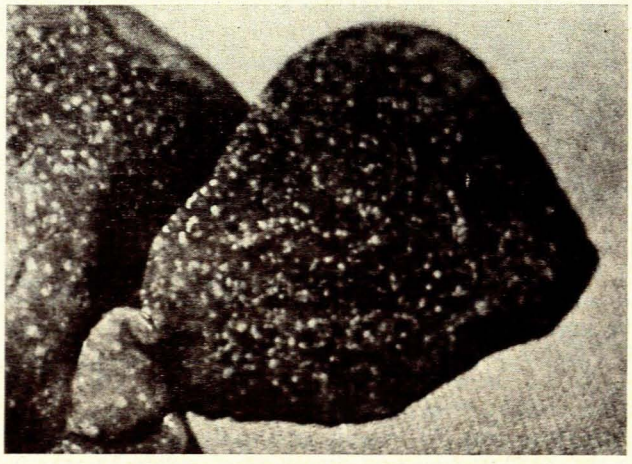

Fig. 1. Right lung showing widespread miliary tubercles.

The liver had a mass of 100 g and was congested, but no macroscopical foci of necrosis were seen. The intestinal tract appeared normal and the portal and mesenteric lymph nodes were not enlarged. The thymus had a mass of 4 g and the spleen (10 g) appeared congested. All the other organs, including the brain and meninges, appeared normal.

Microscopically both lungs had diffuse miliary necrotic foci consisting of cytoplasmic and nuclear debris with a few lymphocytes and neutrophil polymorphs, but no epithelioid cells, histiocytes or giant cells. The hilar lymph nodes showed widespread foci of necrosis and the thymus showed a moderate depletion of cortical lymphocytes. The liver contained a solitary focus of necrosis (Fig. 2). In the spleen there were a number of reactive lymph follicles with germinal centres. In all the necrotic foci in the lungs, lymph nodes and liver, numerous acid- and alcohol-fast bacteria with the morphological features of tubercle bacilli could be demonstrated.

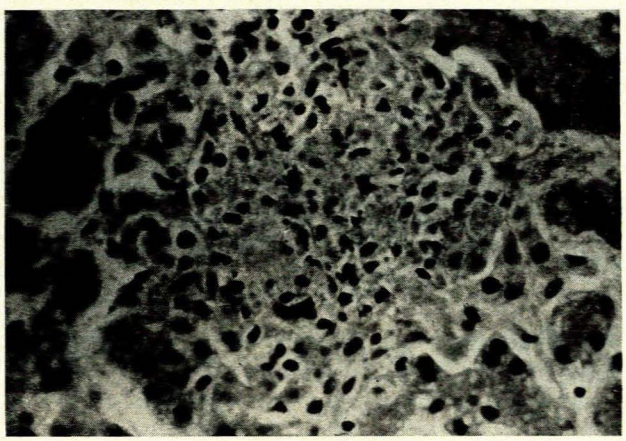

Fig. 2. The solitary tuberculous focus in the liver. Note relative lack of inflammatory cells (H and E × 400).

DISCUSSION

Congenital tuberculosis is very rare, even among infants born to mothers with active, untreated tuberculosis. Ratner *et al.*[7] could not find one instance of congenital tuberculosis among 260 infants born to mothers with tuberculosis. In a pregnant patient suffering from tuberculosis, there may be haematogenous spread to the placenta where a tubercle may form.[8] The fetal vessels supplied by this part of the placenta often undergo thrombosis, thereby preventing the spread of infection to the fetus. Placental tuberculosis need therefore not necessarily affect the fetus.

A fetus may, however, be infected by haematogenous spread or by infected amniotic fluid. Haematogenous spread occurs from the placenta via the umbilical vein. Under these circumstances one of three situations may arise: (*a*) a primary complex involving the liver and regional lymph glands may develop; (*b*) some of the organisms may pass through the ductus venosus and cause a primary complex affecting the lungs, in addition to a primary complex affecting the liver; (*c*) only organisms which reach the lungs may cause a primary lesion with no involvement of the liver.

Amniotic fluid may become contaminated when a tuberculous focus ruptures into the amniotic cavity, and this may cause large numbers of tubercle bacilli to be aspirated by the fetus *in utero* or during birth. In this case large numbers of tuberculous foci, all at the same stage of development, are scattered throughout both lung fields and the mediastinal lymph glands undergo caseation. There is no well-defined single primary lesion.[4] Alternatively, the fetus may ingest infected amniotic fluid *in utero*[9] and thus develop a primary complex affecting the intestinal tract and mesenteric lymph glands.

Morphologically the individual tuberculous foci show a lack of immunological response to the infection. A small number of lymphocytes and neutrophil polymorphs can usually be seen, but no epithelioid or giant cells.[1,4] This lack of response is also reflected in the negative tuberculin test often seen in congenital tuberculosis,[1,4] and in the miliary nature of the disease. Should a tuberculin test give a positive result in the first month of life, however, it must be regarded as strong evidence of congenital tuberculosis.[10]

Infants with congenital tuberculosis may be stillborn[1] or present with clinically evident disease soon after birth.[3] The majority, however, like our patient, appear to be well at birth and remain so until about the end of the 2nd week[1,4] or the 3rd week.[1,9] They then usually present with feeding and respiratory difficulties late in the neonatal period. The rapid progress of the disease after birth seems to be related to better oxygenation of the tissues which favours growth of the bacteria. For the same reason pulmonary lesions may show rapid extension, while the hepatic lesions may remain quiescent.[13] For diagnostic purposes, tubercle bacilli have been demonstrated in material from gastric aspirate, middle ear, lymph nodes, bone marrow and in an endotracheal tube.[4,9]

The prognosis remains poor and the infants often die soon after clinical signs of the disease appear. However, timely diagnosis and specific treatment with isoniazid and streptomycin may be life-saving.[4,11] It is therefore essential to bear this diagnosis in mind when predisposing conditions are present.

The majority of mothers whose infants are affected by congenital tuberculosis suffer from active tuberculosis during pregnancy. If effective antituberculous therapy is not given, these mothers frequently die within days of delivery.[1,3,9] It is important to note that women with apparently inactive tuberculous lesions, and even those with no detectable tuberculous disease, may give birth to infants suffering from congenital tuberculosis.[1,3-5] When the mother is known to be suffering from tuberculosis, the infant must be given antituberculous treatment.[4]

Dormer *et al.*[14] showed that prophylactic isoniazid protection of infants in a tuberculosis hospital was effective. In our patient, the early onset and diffuse, evenly spread tubercles throughout both lungs are typical features of congenital tuberculosis. The miliary pattern would suggest haematogenous spread to the lungs.

The miliary pattern seen in the mother's radiographs and the negative sputum investigations would further support haematogenous dissemination. The solitary focus in the liver without regional lymphadenopathy was probably due to haematogenous spread, but was probably not part of a primary complex. In addition, there was no evidence of intestinal infection which could have spread to the liver.

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Coronary Heart Disease Diagnosed as Cardiomyopathy in Blacks

A Report of Four Cases

M. N. LOWENTHAL

SUMMARY

Four Black South Africans presented with cardiac failure which was diagnosed as being due to cardiomyopathy. Necropsy examination revealed that all 4 patients had had severe coronary atheroma and myocardial infarction during life.

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During 1976 myocardial infarction or very severe coronary atherosclerosis was found in 14 subjects at post-mortem examination at Baragwanath Hospital.[1] In 6 of these patients, coronary heart disease was unsuspected during life. Two of these patients had been admitted in a condition of shock and died shortly thereafter, without proper clinical assessment. Four patients, however, who had been diagnosed as having cardiomyopathy, had been observed for varying periods of time and are described here.

Department of Medicine, Baragwanath Hospital and University of the Witwatersrand, Johannesburg
M. N. LOWENTHAL, M.B. B.CH., M.R.C.P.

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CASE REPORTS

Case 1

An 80-year-old abstemious woman was admitted because of a persistent cough, breathlessness and chest pain. The nature of her chest pains was not elucidated. The signs were those of severe biventricular failure, soft ejection systolic murmur and ventricular gallop. Her blood pressure was 130/70 mmHg. Chest X-ray films showed marked multichamber cardiac enlargement. The clinical diagnosis was cardiomyopathy.

Three days after admission the patient suddenly died. At necropsy examination there was evidence of an old myocardial infarction throughout the posterior portion of the left ventricle. All the coronary arteries showed evidence of severely calcified atheroma.

Case 2

A 78-year-old woman was admitted in October 1975 because of a persistent cough, breathlessness and swelling