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Brucella myocarditis in a 3-month-old: probable transplacental transmission

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Abstract A 3-month-old girl presented with myocarditis owing to brucellosis. Her mother had been diagnosed with brucellosis at 28 weeks gestation but did not receive treatment until after delivery. The infant had intrauterine retardation and had gained little weight since birth. It is considered likely that the brucellosis was transmitted transplacentally.

Introduction

Brucellosis is a bacterial zoonotic infectious disease whose transmission to humans is well known. Although in industrialised countries the prevalence of brucellosis has decreased substantially with effective control in animals and milk products and the pasteurisation of milk, it remains endemic in many regions of the world and is a serious health problem.[1–3]

Transmission to humans occurs through direct contact with infected animals as well as by consumption of infected milk and unpasteurised dairy products. Even though human-to-human and mother-to-infant transmission is rare, neonatal infection via transplacental/perinatal transmission and postnatal infection through breast milk have been reported.[4,5]

Brucellosis is a multi-system disease that may present with a broad spectrum of clinical manifestations. However, cardiac complications are unusual and are mainly in the form of endocarditis. In the absence of endocarditis, myocarditis is a very uncommon complication; most cases reported are in the elderly.[6] We describe a case of myocarditis caused by transplacental transmission of brucellosis.

Case Report

A 3-month-old girl was referred from a local government hospital to the paediatric cardiology department of Inonu University Turgut Ozal Medical Centre with fever, respiratory distress for 5 days and cardiomegaly seen on chest radiograph. She was born in hospital to a 26-year-old gravida 6, para 6 mother by spontaneous vaginal delivery at 38 weeks gestation. She had intrauterine growth retardation. Her birthweight was 2300 g (<3rd percentile) (birth length not known) and she had gained little weight since birth.

Her parents were livestock farmers and the animals had been found to have *Brucella*. The mother had been diagnosed with brucellosis at 28 weeks gestation. Her serum

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agglutination test (SAT) had a titre of 1 : 1280 and *Brucella* was detected in blood cultures. However, the mother had refused therapy for brucellosis during pregnancy. She was given treatment for brucellosis in the 1st 24 hours after delivery. At the same time, a brother and sister of the patient were diagnosed with *Brucella* infection and received treatment.

On examination, the infant looked ill, weight was 3.8 kg (<3rd percentile), length was 53 cm (<3rd percentile) and head circumference 37 cm (<3rd percentile). Temperature was 38°C, heart rate 166 beats/min and respiratory rate 52/min. The apex beat was displaced laterally and on auscultation there was a gallop rhythm without any murmur. The liver was 4 cm and the spleen 5 cm below the costal margins. There were subcostal retractions and crackles were heard bilaterally. The central nervous system was normal.

Investigations

Haemoglobin was 9.4 g/dl, white blood count 12.2×10^9/L, erythrocyte sedimentation rate 30 mm/hr, C-reactive protein 28.3 (0–3.2) mg/L, creatine phosphokinase (CPK) 42 U/L (29–168), CPK-MB (cardiac iso-enzyme of creatine phosphokinase) 44 U/L (0–24) and cardiac troponin I 0.33 ng/mL (0.0–0.02). The Rose-Bengal test was positive and Coombs test titre was 1 : 160. The infant's serum agglutination test (SAT) titre was 1 : 160 and *Brucella* was detected in two blood cultures. Serology for rubella, cytomegalovirus, herpes simplex virus, Epstein–Barr virus and adenovirus were negative and results of metabolic screening were normal. She was fed on breast milk only and no *Brucella* were isolated in the breast milk. Telecardiography showed cardiomegaly and an electrocardiogram showed sinus tachycardia and ST segment depression. Transthoracic echocardiography demonstrated dilatation of the left atrium and left ventricle, trivial mitral valve incompetence and a marked decrease in systolic function of the left ventricle [ejection fraction (EF) 26%, shortening fraction (FS) 12%]. No valvular vegetations were detected and there was no pericardial effusion.

Brucella infection was diagnosed on the basis of the high agglutination titre and the positive blood cultures. The patient was diagnosed as myocarditis secondary to *Brucella* infection and treatment was commenced with parenteral gentamicin (7.5 mg/kg/day in two divided doses), oral trimethoprim (10 mg/kg/day), sulphamethoxazole (50 mg/kg/day) and rifampicin (20 mg/kg/day) in addition to treatment for congestive heart failure (digoxin, angiotensin converting enzyme inhibitor and diuretics).

There was clinical improvement during follow-up: 1 week after initiation of treatment she became afebrile and signs of congestive heart failure resolved; the liver regressed from 4 cm to 2 cm and the spleen from 5 cm to 3 cm. Her SAT titre decreased to 1/80 and repeat blood culture was negative. The patient remained relapse-free during 4-month follow-up. However, cardiac function showed only partial improvement (EF 46%, FS 24%).

Discussion

Brucellosis remains a major health problem in many parts of the world such as the Balkans, the Middle East, Latin America and Mediterranean countries, including Turkey.[2,6] Owing to its intracellular localisation, *Brucella* infection is marked by a prolonged clinical course and relapses. The incidence of *Brucella* in school-age children has increased in endemic areas and it has been reported that children account for 20–25% of cases in endemic areas such as Turkey.[2] It is rare in children under 1 year of age and so transplacental/perinatal infection or transmission through breast milk should be suspected in this age group.[4,5,7]

The major route of transmission of *Brucella* is direct contact with infected animals or intake of infected animal products. Human-to-human transmission and mother-to-infant

transmission in particular is very rare. Neonatal infection acquired transplacentally or during delivery and transmission via breast milk is reported.[4,5,8,9] The course of the disease in pregnant women and neonates and the mode of transmission to the fetus are not well understood. It is believed that transmission to the fetus is via the placenta.[1,5,8] Another route of transmission is through contact with maternal blood, urine or faeces during delivery.[1,8,10] Two infants, one aged 2 months and the other 7 months, in whom transmission through breast milk was suspected were born to mothers who did not have evidence of *Brucella* infection during pregnancy.[4] Similarly, the mother of a 3-month-old infant considered to have been infected through breast milk had postpartum *Brucella* infection.[9] In this case, brucellosis was established in the mother at 28 weeks gestation but she only received therapy after delivery. In view of the intrauterine growth retardation and because the infant gained little weight after delivery, we consider it likely that transmission of brucellosis infection was transplacental rather than perinatal.

Clinical manifestations of neonatal brucellosis vary widely and can involve different organs. Among the most common signs are low birthweight, fever, irritability, retarded development, jaundice, hepatomegaly and splenomegaly which may be difficult to differentiate from other bacterial infections and sepsis.[1,7]

Endocarditis and myocarditis are rare but serious complications of brucellosis. In adults, the incidence of cardiac complications, mainly endocarditis, has been reported to be 0–2%.[6] Cardiac complications secondary to brucellosis have been reported in children too. But data concerning the incidence and details of cardiac complications are limited.[11,12] In a series of five cases, myocarditis owing to *Brucella* infection was reported in three children and endocarditis associated with brucellosis in two; remission followed treatment for *Brucella* treatment in all cases and follow-up echocardiography was evaluated normal.[13]

In this case, however, although *Brucella* infection was eradicated, cardiac function was not restored completely. It is speculated that intrauterine infection, young age, and having serious clinical signs at presentation might have influenced the clinical course of the disease.

Echocardiograpy is useful for detecting valvular vegetations and may facilitate confirmation of the diagnosis of endocarditis.[13] We observed dilatation of the left atrium and left ventricle, trivial mitral valve incompetence and marked decrease in systolic function of the left ventricle, but vegetations and important valve damage that are typical in endocarditis were not detected. These findings strongly support pure myocarditis in our patient.

We consider this to be the first reported case of *Brucella* myocarditis caused by transplacental transmission. In endemic areas, brucellosis should be considered as a cause of myocarditis.

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