Tuberculous Meningitis in Early Infancy

Tuberculous meningitis (TBM) is a common and serious complication of primary tubercular complex in preschool children in our country. Following exposure to tubercle bacilli, it takes about 6-8 weeks for a primary complex to develop(1). Six to twelve months after the primary infection, tuberculous meningitis, secondary to hematogenous spread, may occur. The commonest age group for tuberculous meingitis is 9 months to 3 year(2). BCG vaccination prevents hematogenous dissemination and development of TBM to the extent of 60-80%. Immunity following vaccination takes about 8 to 10 weeks to develop during which interval exposure to tubercle bacilli can cause disseminated infection(3). TBM has been reported to occur in a 4-month-old infant(4). We report a case of TBM which occured in a BCG vaccinated infant at an early age.

A 16-week-old infant was admitted with irregular fever, cough and breathlessness of 21 days duration. He had generalized convulsions for one day prior to admission. He was delivered normally at term with a birth weight of 2.8 kg and received BCG vaccine at 8 weeks of age. He had history of contact with infective tuberculosis in family from his uncle since birth. The mother had no history suggestive of tuberculosis. On examination the patient was toxic and showed poor activity. The infant weighed 4.5 kg, heart rate was 130 per minute and respiratory rate was 50 per minute. Subcostal and intercostal recession were seen. The breath sounds were diminished in the right infraclavicular and mammary region. Coarse crepitations were heard on both

sides. Abdominal examination revealed a firm hepatomogaly of 5 cm and splenomegaly 4 cm below the costal margin. Examination of central nervous system was normal except lethargic behavior.

Laboratory investigations showed a hemoglobin level of 5.5 g/dl, leucocyte count of 14,800/cu mm with 80% polymorphoneuclear cells, 15% lymphocytes, 2% monocytes and 3% eosinophils. The erythrocyte sedimentation rate was 68 mm at the end of first hour. The reticulocyte and platelet counts were 7% and 225000/cu mm, respectively. The peripheral smear showed microcytic hypochromic anemia and 6 nucleated red cells per 100 leucocytes. Lumbar puncture revealed clear CSF with, markedly elevated pressure and cobweb formation. The CSF protein level was 120 mg/ dl, sugar 25 mg/dl and 60 lymphocytes/cu mm. The cobweb on Zeihl Neelsen staining showed acid fast bacilli. X-ray chest showed evidence of upper zone pneumonitis on right side with bilateral miliary mottlings. The Mantoux test was 15 mm. Fetal hemoglobin estimation was 30%. The X-ray chest of the mother was normal Mantoux test was negative. Treatment comprised oxygen, intravenous dextrose, blood transfusion, streptomycin, INH, rifampicin, pyrazinamide, dexamethasone and phenobarbitone. Within a few days, the child showed remarkable improvement. The liver and spleen were still enlarged, though regressed, at the time of discharge on the 21st day. Eight weeks after discharge, the infant was still moderately pale, and the liver and spleen were enlarged 3 cm and 2 cm, respectively. There was no neurological deficit. Fetal hemoglobin was 15%. Hemoglobin electrophoresis did not reveal any other abnormal hemoglobin. A repeat X-ray chest was normal.

The child had progressive primary

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tuberculosis with miliary tuberculosis and TBM. At term, HbF accounts for 70-90% of the total hemoglobin. It then falls rapidly to 25% at 1 month, and 5% at 6 months. The child had elevated fetal hemoglobin level for his age possibly due to thalassemia major. BCG vaccination is protective for hematogenous dissemination. However, the present case developed TBM due to delayed BCG vaccination. The clinical signs of TBM were minimal. BCG vaccination which can modify the clinical profile was responsible for this. It is important to suspect TBM even if the child is BCG vaccinated as typical or a typical manifestations can occur in such children particularly when he is in contact with a case with tuberculosis(1). The case shows that BCG vaccination must be done soon after birth to allow prevention of hematogenous dissemination

in areas with a high prevalence of infective adult tuberculosis.

J.B. Ghosh, Swapan Senapati, B.S. Medical College Hospital, Bankura (W.B.)

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