INFANTILE MEGALOBLASTIC ANAEMIA*

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INTRODUCTION

Anaemias of infancy and childhood as a major problem confront the pediatric unit of any hospital in India. Rapid growth, incomplete haemopoietic adjustments and easy susceptibility to infection in this age period are important contributory factors. Infantile megaloblastic anaemia is one of those diseases where infections and nutritional deficiencies play an important role in the pathogenesis.

Faber described for the first time two cases of megaloblastic anaemia in infancy, and later Bachman and others reported isolated cases of this disease. The credit for describing this syndrome as a distinct clinical entity goes to Zuelzer and Ogden, who reported twenty-five cases of megaloblastic anaemia during infancy: the diagnosis was confirmed by the demonstration of megaloblastic bone marrow. Amato from Italy reported twenty-five cases of this syndrome who responded to folie acid therapy.

Later, many workers from the United States, Italy, England, Ireland, Australia, Holland, China and Indonesia reported cases of megaloblastic anaemia.

The rarity of reported cases of this disease from India has prompted the authors to make this communication.

REPORT OF A CASE

H., a 9-month-old male infant was admitted to the Children’s Medical Ward on 9-9-59 with the complaints of vomiting for five months, marked pallor for two months, puffiness of face for one month and dyspnoea for one day. The patient had an attack of gastro-enteritis and tonsillitis two months prior to admission. The weight of the child at birth was 4½ lbs., the milestones being normal. The child was given breast milk as well as goat’s milk. The pregnancy was normal and labour uneventful. Family history did not reveal anything of significance.

On examination the conjunctivae were pale; the face was puffy and pitting oedema was present. The child was dyspnoeic but there was no cyanosis or clubbing of fingers. The temperature was 101°F and the pulse 102 per minute with a good volume and tension. Fine and medium crepitations were present all over the lungs. The liver was enlarged two inches below the costal margin and was firm with a smooth surface. The spleen was enlarged one and a half inches below the costal margin; firm with a smooth surface. There was generalized lymphadenopathy, lymph glands being shotty and firm. The cardio-vascular vascular system on auscultation revealed the presence of a mid-systolic murmur at the mitral area.

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Laboratory investigation.—Haematological findings were as follows: R.B.C., 1.3 million per cmm.; Hb., 30 per cent; reticulocytes, 6 per cent; M.C.V., 84 cu.; M.C.H., 33 yy; M. C. H. C., 34 per cent; total nucleated cells, 71,350 per cmm.; polymorphs, 25 per cent; lymphocytes, 55 per cent; monocytes, 2 per cent; lymphoblasts, 6 per cent; immature cells of granular series, 12 per cent; a few premature red blood cells were also seen in the peripheral smears; fragility, 0.64-0.42; Van den Berg test, negative direct; bleeding time, 1 minute 10 second; coagulation time, 1 minute 30 seconds. The urine revealed no abnormality. The stool was well formed and showed no abnormality. Kahn's test was negative.

Skiagrams of the skull and long bones showed no change.

In view of the clinical findings and haematological changes, a diagnosis of acute lymphatic leukaemia was made. Treatment for acute lymphatic leukaemia was started from 15-9-59. Injections of Achromycin, 25 mg. six-hourly and Prednisone, 5 mg. twice daily were tried.

The general condition of the patient remained the same. To confirm the diagnosis of acute lymphatic leukaemia a bone marrow biopsy was done.

On re-examination (20-9-1959) the peripheral blood smear showed marked macrocytosis of the red cells, poikilocytosis and anisocytosis. A large number of immature cells of the erythroid series was present; the morphology was intermediate between megaloblasts and normoblasts. There was a preponderance of lymphocytes with a few prelymphocytes. A few myelocytes and metamyelocytes were also seen. Bone marrow revealed reactive hyperplasia and active erythropoiesis with proliferation of megaloblasts along with cells intermediate between erythroblasts and megaloblasts. There was also a relative diminution of the number of cells of the granulocyte series, and lymphocytosis. (Fig. 1).

Later gastric analysis was done and it revealed an absence of free hydrochloric acid, but free hydrochloric acid appeared after injection of histamine. There was a rise of temperature following the bone-marrow biopsy. The case was finally diagnosed as a case of megaloblastic anaemia of infancy and was treated accordingly (Fig. 2). Folic acid was administered and the response was excellent. Along with the daily haemoglobin check