in all cases(4). However, absence of palpable spleen in Kala-Azar had been reported. In a series by Mallick *et al.* of 450 cases, spleen was not palpable in 2 cases (0.4%) but both of them were partially treated(5). In another series of over 600 patients, Prasad made an observation that in children with Kala-Azar, hepatomegaly was more consistent finding(1). Three of his 330 pediatric patients (<1%) did not have palpable spleen but in all of them liver was enlarged. One adult patient did not have hepatosplenomegaly. The present case is unusual for its absence of palpable liver and spleen both inspite of two months of illness. Even on careful questioning history of having receiving treatment for Kala-Azar was denied.

It appears that in this case the splenic enlargement was slower and had the duration of illness been more, spleen would have become clinically palpable. The fact that later inspite of usual treatment, spleen became palpable and ultrasonographic demonstration of mild enlargement further substantiate this hypothesis. This case highlights the importance of ultrasonography in assessment of splenic enlargement and it should be used in clinical conditions in which enlargement of spleen is suspected.

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Hypoplastic Anemia: A Preleukemic State in Acute Lymphocytic Leukemia

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Preleukemic state is well known to precede acute myelogenous leukemia. About 50% of myelodysplastic syndromes are known to progress to acute nonlymphocytic leukemia (ANLL)(1). Unlike ANLL, preleukemia in acute lymphocytic leukemias (ALL) still remains an ill defined and retrospective diagnosis. Rarely hypoplastic

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anemia/aplastic anemia (HA/AA) has been reported to precede overt ALL in children(2-4). This communication reports a case of hypoplastic anemia (HA) with atypical clinical features which evolved to ALL within 7 weeks.

Case Report

A 3-year-old boy, presented with fever and cough for one month in October, 1987. He had received no drugs such as chloramphenicol, phenylbutazone or corticosteroids in the recent past. Physical examination revealed a sick and toxic looking child with severe anemia, bilateral small cervical lymphadenopathy and an abscess on the right leg. There was no evidence of bony tenderness or petechiae. Hepatosplenomegaly of 4 and 1 cm, respectively were present. Other systemic examination was essentially normal. Investigations revealed a hemoglobin of 2.4 g/dl, with a total leucocyte count (TLC) of 1000/μL and platelet count of 1,80,000/μL. The peripheral smear showed a normocytic, normochromic picture without any immature cells. Bone marrow revealed hypocellularity with erythroid prominence, normal megakaryocytes and myeloid elements. The myeloid:erythroid ratio was 1:1. There was prominence of monocytic and reticulum cells. Erythroid component showed moderate dyserythropoiesis with binucleate red cells, nuclear budding and megaloblastosis. Bone biopsy could not be performed due to lack of parental consent. Blood and urine cultures were sterile, pus culture from the abcess grew *Pseudomonas aeruginosa*. Serological tests for typhoid and infectious monoucleosis were negative.

The child was treated with blood transfusion and a combination cephexin and gentamician for a period of two weeks. The fever responded in five days. He was then started on iron, B12 and folic acid. Over the next 18 days his hepatosplenomegaly regressed and the hemoglobin rose to 10 g/dl.

Seven weeks from his initial presentation he was readmitted with fever, purpura and ecchymotic patches all over body of ten days duration. There was severe pallor, generalised lymphadenopathy, a hepatosplenomegaly of 4 and 3 cm, respectively with ecchymoses. Investigations showed a hemoglobin of 5.6 g/dl, TLC of 10,000/μL and a platelet count of 60,000/μL. The peripheral smear revealed neutrophils 4%, lymphocytes 72%, lymphoblasts 20% and monocytes 4%. The marrow at this time was cellular and replaced by lymphoblasts with a L_1 (FAB classification) morphology. The cytochemistry showed PAS positivity and Sudan black and peroxidase stains were negative. Immunophenotyping showed the blasts to be non-T, non-B but the common ALL antigen (CALLA) was positive. The cerebrospinal fluid cytopathology was negative for blast cells.

A diagnosis of ALL, L_1 type, CALLA positive was made. The child was given an induction therapy with vincristine, adriamycin and prednisolone(5), with supportive measures. He went into complete remission in four weeks. The patient was advised consolidation therapy with cyclophosphamide and L-asparaginase along with central nervous system prophylaxis with intra-thecal methotrexate and cranial irradiation. He received only one dose of methotrexate and discontinued therapy due to socio-economic factors.

Seven months from the initial presentation he returned with fever and petechiae for 8 days. He had generalized petechiae, fever and hepatosplenomegaly of 2 and 1 cm, respectively. Hemoglobin was 6.6 g/dl,

TLC 7800/μL, platelet count 78,000/μL and the peripheral smear showed 30% lymphoblasts. The bone marrow was replaced by lymphoblasts. The cerebrospinal fluid cytopathology was positive for blasts. A diagnosis of ALL in relapse with central nervous system involvement was made and the child was reinduced with the same regime as before. This time he was given intrathecal triple therapy (8 injections) with cytosine arabinoside, methotrexate and hydrocortisone followed by cranial radiation of 2000 rads. Subsequent therapy was continued as per the AIIMS protocol(5). He remained in remission for 12 months when he again developed fever, petechiae and pallor with splenomegaly of 3 cm and was diagnosed to be in relapse on bone marrow examination. The parents refused further therapy because of economic factors.

Discussion

Preleukemia is a state characterized by an abnormality of the blood and/or the bone marrow which precedes the manifestation of leukemia. The percentage of blast cells in the peripheral smear or the marrow is inadequate to justify the diagnosis of leukemia(6).

Some authors have observed evolution of HA/AA into ALL and rarely to ANLL in children(2,3,4,7). Breatnach *et al.*(4) observed eight cases in their series of 360 patients with ALL in whom AA preceded the onset of ALL. Development of overt ALL with CALLA positive blasts was observed within 7 weeks. Our observation support the findings of Breatnach *et al.* that children with HA/AA preleukemia develop CALLA positive lymphoblastic leukemia(4).

Hypoplastic or aplastic marrow in these children has been postulated to occur secondary to (*a*) a bacterial or viral infection(8); (*b*) an insult to the marrow initially causing hypoplasia and leading to the development of ALL(3); and (*c*) focal leukemia not detected even on multiple biopsies(3).

Spontaneous remission of the HA preceding leukemia as seen in our case has been rarely reported(2). Melhorn *et al.*(3) had observed a dramatic response to corticosteroid therapy in all their 6 cases who subsequently developed ALL. It has been argued that steroid therapy may induce remission in these children and diagnosis of ALL will be established during a relapse. Androgens have been recommended as they may stimulate the underlying clone of leukemic cells to manifest ALL early(7). In general patients with CALLA positive ALL at diagnosis have better long term survival(9). However, when CALLA positive ALL is preceded by a preleukemic state it has a poor outcome as seen in the few cases reported in literature(4).

The diagnosis of hypoplastic anemia as a preleukemic state is retrospective. The presence of hepatosplenomegaly and/or lymphadenopathy, with cytopenias, dyserythropoiesis in the bone marrow, a dramatic response to steroids and/or spontaneous remission in a child with HA/AA suggests a preleukemic state. These cases need close clinical and hematological follow up to study the evolution of the disease.

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Tuberculides: An Uncommon Manifestation of Tuberculosis

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Tuberculosis is a common disease of childhood in India. However, skin tuberculides though common once are rather rare now(1). We report a child of miliary tuberculosis with skin tuberculides.

Case Report

A 1½-year-old male child was brought to the hospital with the presenting complaints of low grade intermittent fever and cough for 1½ months. In the preceding one month the child had developed raised reddish-purplish spots on the entire body. These came in crops appearing first on the lower trunk and then spread to involve the whole body especially the extensor aspects of the extremities. There was no itching. There was history of decreased oral intake and loss of weight. The child was unimmunised. The father was a known case of tuberculosis taking irregular treatment from a local doctor.

On examination, the general condition of the child was unsatisfactory. He was irritable with a pulse rate of 108/min, a respiratory rate of 40/min and a temperature of 100°F. He was pale and had significant cervical and axillary lymphadenopathy. There was a reddish-purplish papular rash that was symmetrically distributed but more marked on the extensor surfaces (*Fig. 1*). The abdominal examination revealed hepatomegaly of 6 cm and a splenomegaly of 4 cm. The rest of the systemic examination was essentially normal.

Investigations revealed a hemoglobin of 7.5 g/dl, TLC of 10,000/cu mm. DLC $P_{50}L_{46}M_2E_2$, ESR 64 mm and peripheral smear showed microcytic hypochromic anemia. Mantoux test was 15 mm at 72 hours with 5 TU. *X*-ray chest showed soft

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