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Letter to Editor

Oncology Section

Atypical Presentation of Childhood Leukemia

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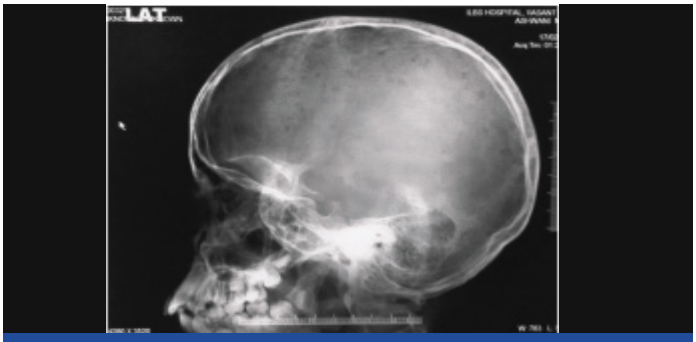
To,

The Editor,

Leukemia is the most common childhood cancer worldwide [1]. Acute lymphoblastic leukemia (ALL) usually manifests with pallor, hepatosplenomegaly, lymphadenopathy, fever, bone pain, and bleeding [2]. Presence of frank osteolytic lesions with hypercalcemia is infrequent [3].We describes here a case of paediatric ALL who presented with multiple lytic lesions, hypercalcemia and absence of blasts in the peripheral blood film. A 10-year-old boy presents with generalised weakness, pain in right hypochondriac region which was non radiating, aggravated by coughing, associated with vomiting which was non bilious and non projectile for last 15 days. On examination, mild pallor was present with no icterus and lymphadenopathy. Liver was palpable 2 cm below the right costal margin. On investigations, complete hemogram showed Hb of 9.2g/dl, WBC count of 8.0×10^9 /L with neutrophils 54%, lymphocyte 36%, monocyte 7%, eosinophil 2%, myelocyte 1% and platelet count 271 $\times10^9$ /L. Peripheral blood film showed mild anisocytosis with microcytes and hypochromia. His serum biochemistry studies were as follows: sodium: 134.8 mmol/L, potassium: 4.2 mmol/L, Crealinium: 0.93mg/dl, Calcium: 14.2 mg/dl, Phosphorous 6.4 mg/dl, uric acid: 7.6 mg/dl, LDH: 654 IU/L, Alkaline phosphatase: 110U/L, Albumin: 3.6g/dl. His ESR was 100 mm/h. Serum vitamin D3 level was 16.3 (15-30ng/ml). His serum parathyroid hormone (PTH) level was 7.2 (13.7-77.2 pg/ml). His abdominal ultrasound was normal except for hepatomegaly. The skeletal survey showed multiple lytic lesions in skull [Table/Fig-1] and ill defined translucent areas in pelvic bone. In view of anemia and multiple lytic lesions, bone marrow examination was done. Bone marrow examination revealed hypercellular marrow with 55% blasts which were positive for CD 10, CD 19, CD 22 and negative for myeloperoxidase confirming to diagnosis of precursor B acute lymphoblastic leukemia. Cytogenetic analysis of bone marrow showed 46XY karyotype. Molecular analysis for t (9; 22), t (12; 21), t (1; 19), and t (4; 11) using PCR method were negative. Patient was started on aggressive hydration, bisphosphonate and frusemide with monitoring of tumour lysis markers. For ALL, he was started with chemotherapy as per BFM 2002 protocol. His serum calcium levels decreased to 11.0 mg/dl after 48 hrs. Currently he is undergoing chemotherapy and his Day +15 marrow was hypocellular with 3% blasts (M1 marrow). Hypercalcemia is a rare finding in paediatric ALL ranging in frequency from 0.6- 4.8% [4]. Hypercalcemia of malignancy can occur due to two mechanisms. First is localized bone destruction by invasive cancer cells and second mechanism involves osteoclastic bone resorption after the release of humoral derived factors from tumour cells. Although hypercalcemia is assumed to be linked with high tumour bulk, a series of 22 patients showed no difference in event free survival in children with hypercalcemia at presentation. It has been reported that t (17;19), which is associated with poor prognosis, is frequently seen in patients with hypercalcemia due to humoral factors [5]. In our patient, hypercalcemia was due to direct bone invasion and cytogenetics was normal. The presence of multiple lytic lesions and hypercalcemia with no blasts in the peripheral blood are uncommon findings in ALL, which has prompted us to report this case.



[Table/Fig-1]: X-ray Skull showing multiple lytic lesions

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