rescue therapy to prevent toxicity. Besides myelosuppression and mucositis, other side effects of methotrexate are hepatotoxicity, erythema, desquamation, allergic reactions and neurotoxicity. Methotrexate is also associated with radiation recall and false photosensitivity. A 10-year-old girl with pre-B ALL underwent hematopoietic stem cell transplantation two times due to marrow and central nervous system (CNS) relapse. On the follow-up, 3 months later she had a bone marrow relapse. After remission obtained with high dose chemotherapy, maintenance treatment was given due to relapse/refractory disease. One year later she had isolated CNS relapse again and treated with intrathecal methotrexate, Ara-C and dexamethasone. The patient was started on relapse/refractory maintenance therapy, and 1 g/m2 methotrexate was given every 4 weeks. Immediately after intravenous methotrexate was given to the patient in the 13th week of her treatment, she complained of burning, pain and redness in the areas that had previously been desquamated due to sunburn. No additional treatment was given, except alkaline hydration and calcium folinate, when the findings were observed. The patient was started on antihistamine therapy. Methotrexate drug level reached 0.02 umol/L at the 54th hour, the i.v. hydration was stopped. The patient's red and itchy lesions healed within 2 days by benefiting from the antihistamine. She is being followed-up at our outpatient clinic weekly chemotherapy without any sign of relapse. This sunburn-like erythema after methotrexate administration might be associated with impaired mononuclear cell response in sun-exposed tissues. Our case stated that he went to the sea two weeks ago and that the bullae secondary to the sunburn that developed afterwards peeled off after they burst. In conclusion, patients with a history of recent generalized sunburn should have their methotrexate delayed to avoid this complication.

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UNUSUAL METABOLIC COMPLICATIONS OF CHILDHOOD ACUTE LYMPHOBLASTIC LEUKEMIA: HYPERCALCEMIA, HYPERAMONEMIA, LACTIC ASIDOSIS

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Case report: We present three children with precursor B acute lymphoblastic leukemia (ALL). The first one had malignancy associated hypercalcemia at diagnosis. The second one experienced hyperamonemia during induction. Both of them had been treated successfully. The last one had refractory leukemia and died because of lactic acidosis due to extensive infiltration of the liver by tumor cells. The rare but potential fatal metabolic complications of ALL needs high clinical suspicion and prompt treatment.

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CENTRAL HYPOTHROIDISM DUE TO ACUTE LYMPHOBLASTIC LEUKEMIA WITH CENTRAL NERVOUS SYSTEM INFILTRATION

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Case report: We describe a five-year-old girl with high risk B precursor acute lymphoblastic leukemia with central nervous system involvement. Laboratory tests suggested the presence of central hypothyroidism (thyroid-stimulating hormone [TSH]: 0.30 mU/ml, normal range 0.64–6.27 mU/ml; serum free thyroxine [FT4]: 0.70 ng/dl, normal range 0.86–1.4 ng/dl). Magnetic resonance imaging detected heterogeneous contrast enhancement of pituitary gland in addition to cerebral and cerebellar atrophy.

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BONE AS A SITE OF EXTRAMEDULLARY DISEASE IN ACUTE LYMPHOBLASTIC LEUKEMIA

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Case report: We describe 3 children with pre B acute lymphoblastic leukemia (ALL).The first two were evaluated in orthopedic clinics because of limping due to ischium involvement and bone fracture suspicion due to involvement of upper limb bones.As a result of normal hemograms in both cases, leukemia diagnosis delayed.The third patient experienced bone marrow and vertebral column relapse of ALL presenting with nuchal rigidity mimicking meningitis.Bone should be considered as a site of extramedullary disease.

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A CHALLENGE IN PEDIATRIC ACUTE LEUKEMIA TREATMENT: UNEXPECTED, PROLONGED CYTOPENIA. IS IT BE CALLLED 'INCOMPLETE HLH'?

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Objective: The diagnostic criteria set for HLH may look like symptoms of cancer or a severe bacterial infection common occurring when patients are immunosuppressed due to