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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PP 69

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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PP 70

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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PP 71

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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PP 72

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

ongoing chemotherapy. Features similar to immune dysregulation in HLH also occur during pediatric acute leukemias. This immune dysregulation results unexpected cytopenias, fever, and splenomegaly in children with acute leukemia. We aim to analysis the pediatric acute leukemia pateints who had unexpected, prolonged cytopenias, and did not full-fill the HLH-2004 criteria set and received pulse methylprednisolone therapy up to three days **Methodology:** Data was analyzed retrospectively. The diagnosis of HLH was defined according to the HLH-2004 criteria set but two criterias (NK cell activity and sCD25 level) of HLH diagnosis were not studied due to lack of necessary equipment. Treatment response was defined as increasing neutrophil count above 500/mm3 in patients within the first seven days. **Results:** 12 patients received steroid for unexpected, prolonged cytopenias. Five or six of six criteria was not found. Four criteria in four, three criteria in five and two criteria in three patients was determined. All patients had cytopenia at least two of three lineages in peripheral blood, one of which was neutropenia. Hemophagocytosis in bone marrow sample was detected in eight patients. Ten patients (87%) recovered within the first seven days. Seven of nine thrombocytopenic patients recovered. **Conclusion:** In this report, the efficiency of short-term steroid treatment was demostrated in patients with unusual cytopenias who did not full-fill HLH criteria.

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PP 73

EVALUATION OF VACCINATION RESPONSE IN CHILDREN AFTER TREATMENT FOR ACUTE LEUKEMIA

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Objective: Our study aims to evaluate the patients' immunity regarding childhood vaccination after leukemia treatment and determine the vaccines that require additional doses. **Methodology:** Sixty-six patients who were followed up with the diagnosis of ALL and AML between 2013 and 2016 were included in our study. The patient's gender, age at diagnosis, leukemia type, leukemia risk groups, vaccination status before chemotherapy (CT) and serologies of hepatitis A, hepatitis B, varicella, measles, rubella, mumps at the end of CT were recorded. **Results:** At the end of the treatment, loss of protective antibody response against hepatitis A (47.4%), hepatitis B (68.2%), varicella (64.2%), measles (45.5%), rubella (43.9%), and mumps (50%) vaccines were shown. Loss of protective antibodies against hepatitis A (66.7%), hepatitis B (100%), varicella (100%), measles (100%), rubella (91.7%), and mumps (91.7%) in high-risk ALL patients was higher than patients in standard-intermediate risk ALL. **Conclusion:** Loss of humoral immunity against hepatitis A, hepatitis B, varicella, MMR was shown in patients with leukemia at the end of the treatment. Due to the significant decrease in hepatitis B and MMR protective antibodies in the high-risk group, we recommend patients with leukemia who have completed chemotherapy to be vaccinated with hepatitis B vaccine three months and MMR vaccine six months after the treatment.

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PP 74

PONATINIB EXPERIENCE IN A PEDIATRIC CHRONIC MYELOID LEUKEMIA PATIENT

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Objective: Chronic myeloid leukemia (CML) is rarely seen in children. The development of myelofibrosis in CML is not uncommon and is associated with a poor prognosis. In cases unresponsive to treatment, tyrosine kinase mutation should be checked for drug resistance, second generation tyrosine kinase inhibitor (TKI) drugs (dasatinib/nilotinib) should be switched to and a suitable donor for bone marrow transplantation should be sought. Third-choice TKI can be used in children who are unresponsive to treatment and do not have a suitable donor. **Materials and Methods:** Experience of third-choice TKI(ponatinib) in a child with CML diagnosis due to unresponsiveness to treatment. **Results:** A 5.5-year-old female patient with no known disease was referred to us because of hepatosplenomegaly (liver 5 cm, spleen 10 cm). There was no laboratory disorder except for anemia (hgb 8.9 g/dL) and high LDH (1104 U/L). WBC was $11.1 \times 10^3/\mu$L neu $6.92 \times 103/\ \mu$L plt 304000/μL. Peripheral smear showed leukoerythroblastosis. Bone marrow biopsy result was evaluated as compatible with myelofibrosis and an increase in blast rate from 8% to 18% in the bone marrow. The patient was diagnosed CML accelerated phase with cytogenetic (46,XX,t(9:22)(q34;q11))and translocation (t(9:22)- p210,BCR/ABL positive) results and. Imatinib treatment was started at 400 mg/m^2.The copy number of BCR-ABL p210 checked before treatment was 72% IS. However, the patient developed febrile neutropenia, and imatinib dose reduction (< 200 mg/m^2) and interruption were required in the follow-up.Under imatinib treatment, BCR-ABL copy number was 16%IS at 1 month, 11%IS at 3 months, and 95%IS at 5 months. Due to the increase in the BCR-ABL copy number, nilotinib was switched to as a second-choice TKI(230 g/m2/dose, in 2 doses).No mutation could be detected in the c-ABL gene, which was examined for tyrosine kinase resistance. HLA groups were sent from the family and compatible donors were not found. Due to severe neutropenia in the follow-up, nilotinib could be continued at 50% dose. Under nilotinib treatment, the BCR-ABL copy number was 13% IS at 1 month, 10% IS at 2 months, and 31% IS at 3 months. The patient was started on ponatinib (18 mg/m^2/day)