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 thirdchoice 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 $304000/\mu L$. 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/m². 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/m²) 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 secondchoice 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 ($18 \text{ mg/m}^2/\text{day}$)