HEMATOLOGY, TRANSFUSION AND CELL THERAPY

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POSTER PRESENTATIONS

Adult Hematology Abstract Categories

Acute Leukemias
PP 01

CYTARABİNE-İNDUCED NEUROTOXİCİTY WELL-RESPONDİNG TO METHYL PREDNİSOLONE

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Objective: Neurotoxicity is a well-recognized complication of high-dose cytosine arabinoside (HIDAC). We describe a patient with AML who suffered cerebellar toxicity following high-dose cytarabine and showed excellent response to methylprednisolone. **Methodology:** A 34-year-old male with acute myeloid leukemia (AML) M1 presented with dysarthria in the inpatient clinic. He had been previously diagnosed with myeloblastic leukemia with maturation type AML, negative for t(8:21), t(9:22), CEBPA and FLT3-TKD mutations by PCR. Cytogenetics were 46, XY. Prior to her presentation, he was treated with induction chemotherapy, which consisted of cytarabine 200 mg/m^2 on days 1–7, idarubicin 12 mg/m^2 on days 1–3. After induction chemotherapy, he had complete morphologic and immunophenotypic remission of her leukemia on bone marrow biopsy, which was followed by one consolidation cycle of high-dose cytarabine (3 gm/m^2 on days 1, 3 and 5). After the first cycle of consolidation therapy, on day six he began to complain of dysarthria, dizziness, gait disorder and balance loss. His cumulative dose of cytarabine at that time was 37.400 g. On physical exam, he had not be able to walk in a straight line, he had dysarthria but he had not dysmetria and dysdiadochokinesia. Gait was ataxic and the rest of neurologic examination was generally normal. MRI and CT of the brain showed no acute pathologic findings. His neurologic symptoms were presumed to be secondary to cytarabine neurotoxicity. He was started on prednisone 80 mg daily over 7 days with rapid resolution of his symptoms within a few days of starting corticosteroids. The steroids were tapered by halving the dose each 3 days over the following 2 weeks. The patient did not receive any additional consolidation treatments with cytarabine, though he remained on maintenance allogeneic hematopoietic cell transplantation (HCT), donor was his brother. He is currently doing well and remains in remission from his disease, without neurologic deficits. **Results:** An excellent response to methylprednisolone in our patient strongly suggests an immune mediated mechanism of neurotoxicity. The patient's improvement in symptoms may have been spontaneous or due to the steroid effect but suggests a possible treatment approach. **Conclusion:** There are no standardized treatments for cytarabine- induced neurotoxic effects, besides discontinuation of the drug. There are only a few cases in the literature.(1) We choice treatment with corticosteroids in this case. The patient presented with neurologic deficits soon after followed by rapid resolution of symptoms after initiation of corticosteroids.This case support the theory of an immune-mediated mechanism and will hopefully serve as a potential treatment for those experiencing neurotoxicity with cytarabine in the future

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Adult Hematology Abstract Categories

Chronic Leukemias
PP 02

REACTIVATION OF HEPATITIS B IN A PATIENT WITH UNTREATED CHRONIC LYMPHOCYTIC LEUKEMIA

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Objective Introduction: The natural course of hepatitis B virus (HBV) infection is determined by the interaction between viral replication and the host's immune response. HBV persists in the body of all infected patients, even those with evidence of serological recovery. Therefore, individuals with a history of HBV infection receiving immunosuppressive therapy are at risk for HBV reactivation. HBV reactivation can result in increased serum aminotransferase levels, fulminant liver failure, and/or death. HBV reactivation has been described in patients receiving chemotherapy for various hematological and solid tumors. We present our patient with a diagnosis of chronic lymphocytic leukemia (CLL) who was spontaneously reactivated during follow-up without treatment. **Case report:** A female patient, who had been followed up with the diagnosis of chronic lymphocytic leukemia for 13 years without treatment, presented in August 2022 with complaints of loss of appetite, weight loss, and jaundice in the eye. It was learned in her history that she had not received chemotherapy or radiotherapy before, had no known disease, and did not use any medication. On physical examination, sclera icteric, multiple lymph nodes with a size of 1 cm in the cervical region and splenomegaly were detected. The patient's Rai stage 2, CLL-IPI score of 3, 17p(-), mutation in the IGHV gene was detected. Detection of leukocyte count 157,000/mm^3, lymphocyte count 149.660/mm^3, hemoglobin 13.5 g/dL, platelet count 117.000/mm^3, alanine aminotransferase (ALT) 2045 U/L, aspartate aminotransferase (AST) 2252 U/L, total bilirubin 10.72 mg/dL, direct bilirubin 9.62 mg/dL, protrombin time 18.7 sec, active partial thromboplastin time 30 sec. While IgG was normal, IgA and M were low. HBsAg positive, anti-HBs negative, anti-HBc IgM negative, anti-HBcIgG positive, HBV-DNA 28,000,000 IU/mL were determined to explain liver dysfunction. One year ago, HBsAg was negative, anti-HBs negative, anti-HBc-IgM negative, while anti-HBc-IgG was positive. The patient was accepted as HBV reactivation and tenofovir disoproxil was started. In the first month of treatment, AST-ALT and bilirubin values returned to normal limits. In the 3rd month of the treatment, HBsAg and HBV-DNA became negative and anti-HBs became positive. The patient was followed up without treatment for CLL. Long-term use of tenofovir disoproxil was planned, despite the possibility of spontaneous HBV reactivation again. **Methodology detected:** Detection of leukocyte count 157,000/mm^3, lymphocyte count 149.660/mm^3, hemoglobin 13.5 g/dL, platelet count 117.000/mm^3, alanine aminotransferase (ALT) 2045 U/L, aspartate aminotransferase (AST) 2252 U/L, total bilirubin 10.72 mg/dL, direct bilirubin 9.62 mg/dL, protrombin time 18.7 sec, active partial thromboplastin time 30 sec. While IgG was normal, IgA and M were low. HBsAg positive, anti-HBs negative, anti-HBc IgM negative, anti-HBcIgG positive, HBV-DNA 28,000,000 IU/mL were determined to explain liver dysfunction. One year ago, HBsAg was negative, anti-HBs negative, anti-HBc-IgM negative, while anti-HBc-IgG was positive. The patient was accepted as HBV reactivation and tenofovir disoproxil was started. In the first month of treatment, AST-ALT and bilirubin values returned to normal limits. In the 3rd month of the treatment, HBsAg and HBV-DNA became negative and anti-HBs became positive. The patient was followed up without treatment for CLL. Long-term use of tenofovir disoproxil was planned, despite the possibility of spontaneous HBV reactivation again. **Results:** determined to explain liver dysfunction. One year ago, HBsAg was negative, anti-HBs negative, anti-HBc-IgM negative, while anti-HBc-IgG was positive. The patient was accepted as HBV reactivation and tenofovir disoproxil was started. In the first month of treatment, AST-ALT and bilirubin values returned to normal limits. In the 3rd month of the treatment, HBsAg and HBV-DNA became negative and anti-HBs became positive. The patient was followed up without treatment for CLL. Long-term use of tenofovir disoproxil was planned, despite the possibility of spontaneous HBV reactivation again **Conclusion:** tenofovir disoproxil was planned, despite the possibility of spontaneous HBV reactivation again. Discussion: HBsAg positive individuals are at greater risk for HBV reactivation compared to HBsAg negative individuals. It has been reported that HBV reactivation rate is up to 70% in HBsAg-positive individuals receiving standard chemotherapy.For those with cured infection (defined as HBsAg-negative, anti-HBc-positive, HBV DNA-negative), reactivation ranged from 0.3% to 9.0%.

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PP 03

ESTABLISHMENT OF EX VIVO DRUG SENSITIVITY SCREENING PLATFORM FOR LEUKAEMIA AND MULTIPLE MYELOMA USING A SOUTH AFRICAN PATIENT COHORT

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Objective: Our objective is to develop a functional precision medicine platform designed to directly identify tailored drug regimens that target individual patient cancer cells and give benefit to the same donors by supporting clinical decision-making. We demonstrate our *ex vivo* drug sensitivity screening platform for precision medicine using Leukaemia and Multiple Myeloma samples from a South African patient