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

Berrin Balik Aydin [1]

[1] *Gazi Yasargil Education And Training Hospital*

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

Sinan Demircioğlu [1], Atakan Tekinalp [1], Ali Demir [2]

[1] *Necmettin Erbakan University Meram Faculty of Medicine, Department of Hematology, Konya, Turkey*
[2] *Necmettin Erbakan University Meram Faculty of Medicine, Department of Gastroenterology, Konya, Turkey*