PP 02

Acute cerebellar syndrome after high dose cytosine arabinoside treatment: case report

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Objective: Cytosine Arabinoside (cytarabine, ARA-C) discovered in the early 1950s from crptathetia crpta which is a type of sponge. It is a chemotherapeutic agent that is an analogue of primidine from the group of antimetabolites and it is used in AML (acute myeloid leukemia), ALL (acute lymphoblastic leukemia), CML (chronic myeloid leukemia), relapse or refractory hodgkin lymphoma, non-hodgkin lymphoma, primary central nervous system lymphoma treatment. The most common side effect is cytopenias due to the bone marrow suppression. In addition; side effects may occur such as nausea, vomiting, diarrhea, abdominal pain, hepatic dysfunction, neurological side effects. Beside many neurological side effects such as peripheral neuropaty, convulsion, cerebral dysfunction can be seen in systemic and intrathecal treatments, classical cytarabine neurotoxicity is acute cerebellar syndrome caused by high-dose systemic therapy.

Case report: A 61 year-old man who had lung cancer history and not suitable for allogeneic stem cell transplantation; was planned HyperCVAD A/B chemotherapy protocol with the diagnosis of B-ALL. Dysarthria and impaired coordination of hand and foot movements occurred on the sixth days of second cycle of HyperCVAD-B chemotherapy. In neurologic examination, dysarthric speech, measured and sequential motion tests for cerebral examination was failed and no motor deficits. No mass or vascular pathology were detected in imaging examinations that could explain the patient's complaint. As a result, the patient was evaluated acute cerebellar syndrome caused by high dose cytosine arabinoside side effect by neurology department. From the eleventh day of treatment patient's complaints was regressed and come back to normal at fifteenth days of treatment.

Conclusion: Acute cerebellar syndrome begins 3–8 days after the start of drug administration. Cerebellar symptoms such as dysarthria, dysdiadokokinesia, dysmetry and ataxia greatly improved after stopping the drug however these symptoms may not full recover in approximately 1/3 of patients. Therefore there is not any treatment for neurological side effects, it should be kept in mind in chemoterapy treatment planning. While planning treatment, dose adjustment considering side effects as performance and age of patient. Nearly monitored is most important for early diagnosed of neurological symptoms.

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PP 03

An unusual case report: myeloid sarcoma presented with appendicitis

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Objective: Myeloid sarcomas (MS) also called as granulocytic sarcomas, myeloblastoma or chloromas are the representatives of extramedullary infiltrates of immatur myeloid cells. According to 2016 revısıon to the World Health Organization classification of myeloid neoplasms and acute leukemia; it is classified a subgroup of Acute myeloid leukemia (AML) and related neoplasms. MS has more aggressive clinical course than AML. MS may present simultaneously with or precede bone marrow disease or may be seen in relapse. Isolated myeloid sarcoma involvement is most frequently detected in the bone, periosteum, soft tissues and lymph nodes. Herein we reported an unusual case of a 44 year old man who presented to appendicitis and diagnosed with MS with appendicitis pathology report.

Case report: A 44 year-old man who had no history of smoking, alcohol or any chronic disease, was admitted to the hospital with sudden abdominal pain, nause and vomiting. The patient, who was diagnosed with appendicitis and was underwent appendectomy. The patient's appendectomy result was reported as MS. In the blood test examination; white blood cells (WBC): $43.2 \times 10^3/\mu L$; neutrophil: $36.3 \times 10^3/\mu L$; monocyte: $2 \times 10^3/\mu L$, hemoglobin:11.5 g/dL, and plateletes:$100 \times 10^3/\mu L$ were detected. Peripheral smear was applied to the patient due to leukocytosis. Blastic cell infiltration was detected in peripheral smear and the patient underwent bone marrow biopsy. More than 20% monoblasts were observed in bone marrow aspiration. Flowcytometric examination was performed and immunfenotypic finding MS of the patient were interpreted of AML M5. Remission induction chemotherapy (daunorubucin 60 mg/m^2 110 mg/daily throughout 3 days and ARA-C 100 mg/m^2 180 mg/daily, throughout 7 days) was planned.

Conclusion: Hematological malignancies could involve extramedullary soft tissue in relatively rare cases. MSs are rare extramedullary tumors, most commonly occur in patient with acute or chronic myeloid leukemia (1–3). De novo MS may represent the first sign of systemic disease. Untreated MS usually progress to AML in about 1 year. The clinical presentation of MS depends on location, size of mass. In the current case a sudden right lower abdominal pain, nause and vomiting due to blastic infiltraion and obstruction of appendix were initial symptoms. Total excision of the mass is to gold standard for diagnosis. In the current case, appendix patology results showed that monoblastic and localize inflitration of cell form of monocytoid with large nucleolus, prominent nucleoli, wide cytoplasm, Ki 67 proliferative activity >. There is no consensus of MS treatment. Recommended treatment regimen for isolated MS or MS with AML is conventional AML protocols. In conclusion, MS is a subgroup of AML present with myeloblastic

infiltration of soft tissue. MS may present at any time of disease process and any localization. It should be kept in mind that hematological malignancies may be seen all over the body and may be present atypically because early diagnosis and treatment are very important cause of MS's aggressive clinical course.

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PP 04

Postdural puncture superior sagittal sinus thrombosis during remission induction therapy for acute lymphoblastic leukemia

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Objective: Superior sagittal sinus thrombosis (SSST) during the course of acute lymphoblastic leukemia (ALL) may arise during or even after treatment. Majority of the cases are either directly attributed to ALL or considered as a consequence of using chemotherapy agents including prednisone, vincristine, cytarabine and especially L-Asparaginase. Post-lumbar puncture intracranial hypotension is a rarely encountered cause of SSST in ALL.

Case report: A 27-year-old man was admitted with fatigue and following bone marrow aspiration and biopsy, he was diagnosed as having B-ALL. He received HyperCVAD regimen as remission induction therapy, which included doxorubicine, vincristine and cyclophosphamide, dexamethasone and intrathecal administration of methotrexate. Cranial computerized tomography (CT) prior to intrathecal methotrexate was normal. Cerebrospinal fluid analysis was acellular and showed no ALL infiltration. He complained of mild postural headache intrathecal treatment. Eight days after intrathecal administration of methotrexate on day 13 of HyperCVAD, he complained of newly developing non-postural headache and vomiting.

Methodology: At the time of symptoms, complete blood count showed the following: WBC: 5480/uL, Hgb: 12.3 g/dL and PLT: 148,000/uL. Coagulation profile studies showed prothrombin time of 13 s, partial thromboplastin time (PTT) of 30.9 s, and normal concentrations of fibrinogen. Neurologic examination including evaluation of his mental status, sensory, motor, and reflex functions of his extremities. Coronal plane contrast enhanced T1-weighted MRİ demonstrated a nonenhancing superior sagittal sinus with the empty delta sign, compatible with a diagnosis of SSVT. Enoxaparin 2×1 mg/kg was initiated. Platelet transfusions were given to keep platelet count over 50,000/uL during the course of anticoagulant therapy.

Results: SSST in the context of ALL has been ascribed to lymphoblastic infiltration of the superior sagittal sinus wall or to the chemotherapeutic agents used. L-Asparaginase decreases plasma antithrombin, plasminogen, and fibrinogen concentrations while prednisone may increase the levels of factor VIII. These hemostatic changes may predispose to thrombosis, especially in the setting of the turbulent flow in the superior sagittal sinus. Our patient harbored none of the aforementioned risk factors except for the use of corticosteroids. Any cause of intracranial hypotension, which induces a downward shift and traction of the brain, may disrupt the veins/sinus and hence may lead to venous dilatation and thrombosis.

Conclusion: Our patient most probably developed intracranial hypotension due to lumbar puncture, which resulted in SSST. The possibility of a dural venous thrombosis should be suspected in patients with ALL who had treatment with L-asparaginase and prednisone. However, SSST thrombosis should also be an important consideration in patients with dural puncture who report a changing pattern of their headache (postural headache becoming nonpostural in character) and severe nausea and vomiting.

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PP 05

Retrospective analysis of all patients single center experience

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Objective: The aim of study was to evaluate the demographic, clinical, laboratory, genetic and pathological features of the patients followed with the diagnosis of adult ALL in our center and to evaluate their contribution to the prognosis, treatment responses and overall survival rates of the patients and to contribute to the literature.

Methodology: A total of 116 patients diagnosed with ALL in our center between 2006–2018 were included in the study. Patients under 18 years of age and patients with active solid organ malignancies were not included in our study. The data of the patients were obtained by scanning the hospital computer automation system and patient files.

Results: Sixty-two of our patients are male and 54 are female. The mean age of the patients was 43.1 years. Twenty patients were T-ALL and 96 patients were B-ALL. In a quarter of patients, the Philedelphia chromosome was positive. 22 of our patients had standard risk and 94 had high risk class. Total survival rate was 52.6%. The mean total survival time was 41,4 months. 83.6% of the patients were in remission with induction therapy. Forty patients underwent allogeneic stem cell transplantation. There was no statistically significant difference between B-ALL, T-ALL and Ph+ ALL patients in terms of remission induction and survival. Tyrosine kinase inhibitors improved the prognosis of Ph+ ALL patients. In patients who received TKI treatment, the decrease in PCR values at the 3rd month was found to be a good prognostic factor. PCR monitoring is important in predicting prognosis in patients receiving