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.

https://doi.org/10.1016/j.htct.2020.09.065

PP 04

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

A. Ozturkmen[1], E. Gulturk[1], o. Yildiz[2], F. Hindilerden[1,*]

[1] *University of Health Sciences Bakırkoy Dr. Sadi Konuk Training and Research Hospital, Division of Hematology, İstanbul, Turkey*
[2] *University of Health Sciences Bakırkoy Dr. Sadi Konuk Training and Research Hospital, Department of Radiology, İstanbul, Turkey*

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.

https://doi.org/10.1016/j.htct.2020.09.066

PP 05

Retrospective analysis of all patients single center experience

E. Kelkitli[1,*], E. Arslan[2], M. Turgut[1]

[1] *19 Mayıs University Department of Hematology, Samsun, Turkey*
[2] *19 Mayıs University Department of Internal Medicine, Samsun, Turkey*

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

TKI. The presence of severe fibrosis in the bone marrow (Grade 2–4) was found to be poor prognostic.

Conclusion: In our study, although the overall survival rate is consistent with the literature, it is evident that it is still insufficient. Therefore, more study and innovation are needed in the treatment of adult ALL.

https://doi.org/10.1016/j.htct.2020.09.067

PP 06

Case report: acute lymphoblastic leukemia with bone involvement

F. Yavaşoglu[1,*], C. Özdemir[2]

[1] *Afyonkarahisar Health Sciences University Hospital, Hematology Department, Afyonkarahisar, Turkey*
[2] *Afyonkarahisar Health Sciences University Hospital, Pathology Department, Afyonkarahisar, Turkey*

Objective: ALL is the most common type of acute leukemia in children, after AML in adults. At the time of diagnosis, there may be weakness due to anemia, signs of bleeding due to thrombocytopenia, signs of infection related to neutropenia. There may be bone pain due to expansion of the medullary cavity by the leukemic process. However, low back pain due to vertebral body collapse is one of the rare symptoms at the time of diagnosis. We are reporting an adult male patient with acute lymphoblastic leukemia who presented with paraparesis and multiple osteolytic lesions in lomber and thoracal vertebra.

Case report: A 63-year-old male patient had a complaint of back pain for 4 months, spreading to the left leg, accompanied by numbness and loss of strength. The patient without incontinence and painful walking was operated by the neurosurgery department. The patient with pancytopenia was consulted to us. In physical examination peripheral LAP was not detected and spleen size was determined as 16.5 cm by ultrasound. In the laboratory examination was remarkable for Hb: 9 g/dL, MCV: 79 fL, plt: $13*10^3$/uL, sedim 76 mm/h LDH: 1092 u/L. Other biochemical tests are normal. The L2 corpus pathological fracture biopsy result was determined as CD45+, Cd19+, Cd10+, TDT+, PAX 5+, c myc 30%+, Kİ 67% 50+, and was compatible with B lymphoblastic lymphoma infiltration. In bone marrow biopsy, 98% cellularıty, 99% blastic infiltration was detected. Blasts were CD34+, CD19+, PAX 5+, 80% CD10+, 80% TDT+, 50% CD22+, 30% CD20+, CD123+, respectively. Cytogenetics and fluorescence in situ hybridization (FISH) panel for ALL were normal; Philadelphia chromosome was not present. HyperCVAD chemotherapy was started for the patient who was diagnosed with B-ALL+ bone involvement. Intrathecal chemotherapies were given. After Hyper CVAD 2B chemotherapy, the patient was clapped due to sepsis.

Conclusion: Skeletal lesions can occur in a variety of malignant hematological conditions. In diseases such as multiple myeloma and waldenstrom macroglobulinemia, bone involvement is a common finding in diagnosis. Acute lymphoblastic leukemia and lymphomas can rarely present with osteolytic lesions and neurological involvement. ALL is a chemosensitive tumor, so chemotherapy is the main treatment option. In patients with bone involvement, radiotherapy and surgical resection are the other treatment options that can be applied.

https://doi.org/10.1016/j.htct.2020.09.068

CHRONIC LEUKEMIAS

PP 07

Chronic lymphocytic leukemia presenting as pulmonary involvement in an elderly patient: a case report

O. Ekinci[1,*], A. Dogan[2], M. Aslan[1], I. Aras[3], C. Demir[2]

[1] *Department of Hematology, Faculty of Medicine, Fırat University, Elazığ, Turkey*
[2] *Department of Hematology, Faculty of Medicine, Yüzüncü Yıl University, Van, Turkey*
[3] *Department of Pathology, Faculty of Medicine, Yüzüncü Yıl University, Van, Turkey*

Objective: A significant part of chronic lymphocytic leukemia (CLL) cases receive a diagnosis during the examination of routinely detected lymphocytosis or the investigation of the causes of lymphadenopathy or hepatosplenomegaly. Apart from these, CLL cases may rarely manifest as pulmonary involvement, which can include broncho-pulmonary infiltration, pleural effusion, or an endobronchial lesion. In the literature, cases presenting with CLL-associated broncho-pulmonary infiltration are extremely rare. Here, we present an elderly case with CLL presenting as pulmonary involvement.

Case report: An 82-year-old male patient presented to our hospital with progressive dyspnea, non-productive cough, and weight loss, which had persisted for one month. Chest X-ray radiography revealed opacity in the lower zone of the right lung. Contrast computed tomography (CT) of the chest visualized a soft-tissue density measuring approximately 74 mm × 75 mm in maximal axial dimensions in the inferior segment of the right middle lobe with surrounding ground-glass density and some air bronchogram localized near the medial hilum. Laboratory test results were as follows: hemoglobin level, 13.4 g/dL; total leukocyte count, 174×10^9/L; lymphocyte count, 148×10^9/L; platelet count, 192×10^9/L. Peripheral blood smear showed diffuse mature small lymphocytes and smudge cells. Peripheral blood flow cytometry revealed strong positivity for the CD5, CD20, CD19, and CD23 markers, consistent with CLL. A bronchoscopy was performed for diagnostic purposes and a transbronchial biopsy was taken from the lung parenchyma, and bronchoalveolar lavage (BAL) was performed. BAL cytology and microbiological tests were not diagnostic. On immunohistochemical examination of the parenchymal biopsy, neoplastic cells showed a CD20(+), CD5(+), CD23(+), CK(−), CK7(−), CK20(−), CD56(−), synaptophysin(−), chromogranin-A(−), CD3(−), TTF-1(−), Napsin A(−), and P63(−) staining pattern. The Ki67 proliferation index was 10%. The pathology clinic reported the result to be consistent with a chronic lymphocytic leukemia/small lymphoma infiltration. Cervical and abdominopelvic CT results of the patient were also considered and the CLL stage was determined as RAI 2