in peripheral smear. On scrotal ultrasonography (USG), the left epididymal head had a mildly heterogeneous appearance and the patient was treated for epididymitis with suspicion of epididymitis. Approximately one week later, the patient presented with fever . The patient's peripheral smear showed 36% blasts, 38% eosinophils, 2% monocytes, 6% lymphocytes, 10% segments and 8% bands. Bone marrow aspiration was performed for the diagnosis of acute leukemia and PreB-ALL was diagnosed. Control testicular USG was evaluated as testicular involvement of leukemia. During follow-up, the patient had nausea, vomiting, dizziness, decreased visual field, nuchal rigidity, and outward gaze limitation. Magnetic resonance (MR) venography revealed thrombosis in the inferior sagittal sinus and anticoagulant therapy was initiated. The patient with central nervous system symptoms was considered to have leukemic involvement and his treatment was adjusted. ALL is a condition that can cause HE. The prognosis is poor in ALL patients presenting with HE. HE may occur before the classical ALL symptoms therefore the diagnosis of ALL should also be considered in patients presenting with HE.

https://doi.org/10.1016/j.htct.2024.11.031

PP 04

EVALUATION OF DRUG EFFECTIVENESS AND CONTROLLED RELEASE PROFILES OF CLAY MINERALS LOADED WITH ANTI-CARCINOGENIC AGENT AS A DRUG DELIVERY SYSTEM ON LEUKEMIA

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Objective: This study aimed to evaluate the slow release and internalization of azacitidine-bentonite combination in THP-1 and K562 cell cultures in acute myeloid leukemia morphology. Methodology: The morphology of bentonite clay was assessed using two Scanning Electron Microscopes. The bentonite-azacytidine combination was assessed in THP-1 and K562 cell cultures via in vitro cell proliferation tests, proliferation with CCK-8, and drug release tests with dialysis membranes. Additionally, apoptosis and internalization were determined using the Annexin V-FITC Kit and fluorescence methods, respectively. Results: Our findings showed that azacytidine achieved complete and controlled release within 8 hours. Bentonite displayed significant antiproliferative effects at concentrations of 10, 25, 50, and 100 μ g/ml in both cell lines. The combination of azacytidine and bentonite exhibited a synergistic effect in inhibiting cell proliferation, with significant decreases in cell viability in the 1 μ M azacytidine + 10 μ g/ml bentonite, 5 μ M azacytidine + 10 μ g/ml bentonite, and 10 μ M azacytidine + 10 μ g/ml bentonite groups compared to the controls. The drug release profile of the

bentonite-azacytidine combination demonstrated slow release, with 50% released in the first two hours and approximately 90% released in the fourth hour, with prolonged release exceeding eight hours, potentially reducing side effects and increasing efficacy in target cells. **Conclusion:** In conclusion, bentonite NPs exhibited significant potential as drug carriers for azacytidine in the treatment of leukemia and offered benefits such as improved solubility, bioavailability, controlled release, protection from harsh environments, and cost-effectiveness.



https://doi.org/10.1016/j.htct.2024.11.032

PP 05

LIVER INVOLVEMENT IN ACUTE MYELOID LEUKEMIA: A CASE REPORT

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Objective: Although rare, extramedullary involvement can be observed in patients with acute myeloid leukemia (AML). These extramedullary involvements are also known as myeloid sarcoma, granulocytic sarcoma, or chloroma. The most common sites of involvement are soft tissues, bone, periosteum, and lymph nodes. Patients with extramedullary involvement may exhibit a more aggressive clinical course. In this case report, we evaluated an AML patient with liver involvement at the time of diagnosis. Case Report: A 66-year-old female patient presented to our hospital with complaints of fatigue, bruising on the skin, and yellowing of the eyes for about a month. Physical examination revealed icterus in the sclera, and widespread ecchymoses on the arms and abdomen. Laboratory findings showed a hemoglobin level of 7.8 g/dL, a leukocyte count of $4.4 \times 10^{9/L}$, a neutrophil count of $1.1 \times 10^{9/L}$, a platelet count of 30 \times 10^9/L, CRP at 29 mg/L, and direct bilirubin at 5.8 mg/dL. Peripheral blood smear revealed notable myeloblasts and auer rods. Bone marrow aspiration smear showed over 20% myeloblasts, supporting the diagnosis of acute myeloid leukemia. Flow cytometry analysis was evaluated as consistent with AML. Abdominal ultrasonography revealed the liver was 19.5 cm and the spleen was 16 cm in size. The patient underwent 7+3 remission induction chemotherapy. After chemotherapy, bilirubin levels returned to normal, and the patient was diagnosed with liver involvement of AML. **Conclusion**: The clinical presentation of extramedullary involvement varies depending on the affected organ and region. A definitive diagnosis is made through biopsy. In patients with AML, as in our case, a biopsy may not always be feasible due to the risk of bleeding. Therefore, in cases where hepatomegaly, abnormalities in liver function tests, and elevated bilirubin levels cannot be explained by other diseases, liver involvement should be considered.

https://doi.org/10.1016/j.htct.2024.11.033

PP 06

IS ALL-TRANS RETINOIC ACID EFFECTIVE IN PULMONARY HAEMORRHAGE IN PATIENTS WITH ACUTE PROMYELOBLASTIC LEUKAEMIA?

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Objective: Acute myeloid leukaemia (AML) develops from myeloid precursor cells in the bone marrow. Acute promyelocytic leukaemia (APL) is an aggressive subtype (5-10%) of AML with the t(15;17) translocation. It is sensitive to all-trans retinoic acid (ATRA). The aim of this article is to emphasise the efficacy of ATRA treatment in pulmonary haemorrhage associated with APL and to contribute to the literature. Case Report: A 14-year-old girl presented with malaise, pallor and bruises since 1 month. On examination, she was pale and had bruises on the trunk and extremities, but no organomegaly. Investigations revealed pancytopenia and atypical mononuclear cells in peripheral blood smear. Bone marrow aspirate showed promyeloblasts (80%) and AML-M3 surface markers were positive in flow cytometry. Cytogenetic analysis revealed t(15;17) translocation. AML-BFM 2012 chemotherapy protocol was initiated. During induction chemotherapy, the patient developed dyspnoea and pulmonary haemorrhage. The child was transferred to intensive care unit and ATRA was added to the chemotherapy at a dose of 25 mg/m2/day. Coagulation tests improved 2 days after ATRA treatment and clinical findings improved 4 days later. On the 9th day of intensive care unit admission, the patient was transferred to inpatient ward and there was no bleeding during follow-up. Discussion: Haemorrhagic complications are frequent in APL patients and are one of the main causes of early death (5-9%) (1,2). Increased plasmin production (60-fold) due to excessive annexin-II receptor expression in promyeloblasts has been shown to cause fibrinolysis. It is thought that patients develop increased hyperfibrinolysis rather than consumption coagulopathy (2). ATRA is highly effective in bleeding control (3). Conclusion: Patients should be monitored with coagulation tests at regular intervals due to the high risk of bleeding. Undesirable haemorrhagic conditions may develop before and during treatment. ATRA can provide effective control in treatment.

https://doi.org/10.1016/j.htct.2024.11.034

Adult Hematology Abstract Categories

Chronic Leukemias PP 07

MULTI-TYROSINE KINASE INHIBITOR-ASSOCIATED APLASTIC ANEMIA AND A BRIEF LITERATURE REVIEW

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Objective: Chronic myeloid leukemia (CML) is a malignancy classified under the group of chronic myeloproliferative neoplasms. It is characterized by uncontrolled leukocytosis, bleeding, thrombosis, recurrent infections, and hepatosplenomegaly. With the introduction of imatinib in 2001, followed by the second- and third-generation tyrosine kinase inhibitors (TKIs), a new era in the treatment of CML began, as overall survival rates have since reached levels comparable to normal life expectancy. In this article, we review the first case of aplastic anemia that developed after bosutinib treatment, along with other cases of aplastic anemia reported in the literature following the use of TKIs. Case Report: A 57-year-old female was referred for leukocytosis identified during evaluation for fatigue, weakness, and early satiety. Initial lab results showed a WBC of 384×10^{9} /L, NEU of 246×10^{9} /L, Hb of 7.2 g/dL, and Plt of $281\times10^9/L.$ Abdominal ultrasound revealed splenomegaly (23 cm), and peripheral blood smear suggested chronic myeloid leukemia (CML), leading to BCR-ABL transcript testing. Hydroxyurea was initiated while awaiting results. Two weeks later, the BCR-ABL transcript level was 49%, and imatinib 400 mg/day was started on December 15, 2022. The February 2023 earthquake disrupted the patient's imatinib use for three months. Upon return in May 2023, labs showed a WBC of 17×10^{9} /L, NEU of 14×10^{9} /L, Hb of 14.1 g/dL, Plt of $424\times10^9\text{/L}\text{,}$ and BCR-ABL remained at 49%. Imatinib was resumed. In August 2023, BCR-ABL decreased to 41%. However, in October 2023, pancytopenia emerged, leading to imatinib discontinuation (WBC: $2.98\times10^{9}/L,$ NEU: $0.5\times10^{9}/L,$ Hb: 4.1 g/dL, Plt: $2\times10^{9}/L).$ ABL mutation analysis showed no resistance mutations (Hemogram values at diagnosis and after treatment are shown in Figure 1). After two weeks without medication and no improvement in pancytopenia, bone marrow biopsy confirmed aplastic anemia (Figure 2A). Following ten weeks of recovery, normocellular marrow was observed (Figure 2B), and dasatinib 50 mg/day was started on February 1, 2024, later increased to 100 mg/day. Due to worsening cytopenias in late February, dasatinib was reduced and eventually discontinued in March. Bosutinib 500 mg/day was initiated in May, with BCR-ABL at 27.9%. As cytopenias progressed, bosutinib was reduced to 300 mg/day. Despite a BCR-ABL decrease to 8.63% in August, cytopenias persisted, and bosutinib was further