the time of initial admission, the patient complained of abdominal distension and severe weight loss over the past 6-months. During examinations, a splenomegaly (204 × 85 mm) was found. In hemogram: Hb – 151 g/L, WBC – 43 × 10^9/L, PLT – 779 × 10^9/L were. Histological examination of the bone marrow revealed that the bone cavities were filled with fibrotic stroma, no fat cells were detected. Hematopoietic cells were diffusely scattered, the cellular composition consisted of granulocytic and megakaryocytic orders. A reduction in the erythroid order was noted. The number of megakaryocytes was increased, acute polymorphism was noted, atypical forms were abundant. Megakaryocytes formed dense and sparse clumps (up to 6-cells) and layers, their paratrabecular localization was noted. Areas of coarse-fiber collagen fibrosis were noted. During molecular genetic examination, the allelic load of the JAK2V617F mutation was 92.694%. The patient was treated with Hydrea (HY)from June 2019 to September 2019. Since the hemogram did not show positive dynamics, Interferon (IFN) 3 million units was administered intramuscularly 3 times a week from September 2019. After this administration, a relative decrease in spleen size was noted. Starting from March 2020, the patient's condition deteriorated again. In hemogram: Hb – 161 g/L, WBC – 34 × 10^9/L, PLT – 343 × 10^9/L were. The spleen size was 200 × 84 mm on Ultrasound Scan (USS). The patient was prescribed HY 1000 mg p/day along with IFN. Positive dynamics were achieved as a result of treatment with HY+IFN. Hemogram: Hb – 120 g/L, WBC – 4 × 10^9/L, PLT – 476 × 10^9/L; spleen in palpation was +4 cm. Treatment with HY+IFN was continued until April 2021. From April 2021, treatment was continued with HY alone. In May 2024, the patient's condition worsened. Morphological examination of the bone marrow showed 16% blasts, histological examination showed 20% blasts, blasts were of myeloid type. Transformation of the disease to the BP was recorded. The patient was prescribed 2 courses of low-dose Cytosar. Since no positive dynamics were noted and blasts in the bone marrow increased to 78.6%, treatment with Azacitidine (AZA) + Venetoclax (VEN) was initiated in July, and after the 2nd course, clinical-hematological remission was recorded (blasts on myelogram were 0.8%). Although the patient's hemogram and bone marrow results showed Morphological Leukaemia-Free State (MLFS), Ruxolitinib (RUX) 15 mg was added to the treatment with AZA+VEN as a result of the recent sharp increase in spleen size (197 × 78 mm) and abdominal discomfort. As a result of the treatment, the patient's spleen size decreased, abdominal discomfort disappeared. During the examination of the patient, a complete blood count and histological examination of the bone marrow were performed. To confirm myelofibrosis, reticulin stroma examination was performed using the Gomori method, and first- and second-degree fibrosis (M1–MF2) was detected (scale 0–3). In assessment with the Dynamic International Prognostic Scoring System (DIPSS)-2 points-intermediate-1 risk group was formed. The patient's complaints were assessed with Myeloproliferative Neoplasm Symptom Assessment Form Total Symptom Score (MPN-SAF-TSS). Molecular-genetic examination of the JAK2V617F gene was performed using the real-time PCR method of peripheral blood. Spleen size was assessed with USS. AZA was prescribed subcutaneously at a dose of 100 mg for 7 days per course. 6 courses have been conducted so far. VEN was increased according to the scheme and prescribed at a dose of 400 mg; depending on cytopenia's, the dose was reduced by 200 mg, and the number of days of administration varied from 28 to 14 days. RUX was prescribed at a dose of 15 mg daily. **Results:** After transformation of PMF to BP, the patient did not achieve remission despite 2 courses of low dose cytosar treatment. After treatment with AZA+VEN, the patient achieved MLFS. After some time, due to the growth of the spleen, RUX was added to the AZA+VEN treatment protocol, and the spleen's size decreased. **Conclusion:** The use of the AZA+VEN protocol was effective in BP-MF. The subsequent addition of RUX to the treatment further increased the effectiveness of the treatment and led to an improvement in the patient's general condition and a decrease in complaints.

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Adult Hematology Abstract Categories

Multiple myeloma

OP 12_Case report

PROGNOSTIC IMPLICATIONS OF HIGH-RISK GENETIC MUTATIONS IN MULTIPLE MYELOMA PATIENTS UNDERGOING SECOND AUTOLOGOUS STEM CELL TRANSPLANT

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Objective: High-risk genetic mutations significantly influence prognosis in multiple myeloma. Although autologous Hematopoietic Stem Cell Transplantation (auto-HSCT) is a cornerstone of multiple myeloma treatment, the prognostic impact of genetic abnormalities in patients undergoing a second auto-HSCT warrants further investigation. This study aims to evaluate the prognostic significance of high-risk genetic mutations in multiple myeloma patients undergoing a second auto-HSCT. **Methodology:** This retrospective analysis evaluated 26 multiple myeloma patients who underwent a second auto-HSCT between May 5, 2017, and December 10, 2024. Detailed analysis was conducted on 19 patients with available pre-transplant Fluorescence In Situ Hybridization (FISH) data. Among these, 9 patients underwent tandem transplantation, and 10 underwent a non-tandem second auto-HSCT. Prognostic analyses focused on genetic abnormalities detected by FISH. **Results:** The analyzed cohort included 10 males (52.6%) and 9 females (47.4%), with a mean age of 56.79-years (SD = 10.39, range 34–69). Median follow-up post-second transplantation was 31-months (IQR 18–54). Median intervals between two transplantations were 16-months (IQR 4–72.5) overall and 62-months (IQR 31–93) excluding tandem cases. High-risk genetic mutations were detected in 11 of 19 analyzed patients (57.9%): deletion 17p and amplification 1q (each 26.3%), t(4;14) (15.8%), deletion 1p (10.5%), and t(14;16) (5.3%). Patients with high-risk mutations

had a higher mortality rate (54.5% vs. 25%), although not statistically significant overall (p = 0.198). Amplification 1q was significantly associated with increased mortality (80% vs. 28.6%, p = 0.048). Kaplan-Meier analysis revealed significantly shorter overall survival for patients with ≥ 2 high-risk mutations (16.10 vs. 59.43 months, p = 0.017), amplification 1q (32.50 vs. 60.57 months, p = 0.048), and deletion 17p (18.50 vs. 59.12 months, p = 0.030). Platelet engraftment was significantly delayed in patients with at least one high-risk mutation (12.64 vs. 10.88 days, p = 0.033). Neutrophil engraftment and hospital stay durations were not significantly different. Pre-transplant hemoglobin, platelet, and neutrophil counts showed no correlation with survival, engraftment times, or hospital stay duration. Survival outcomes were similar between tandem and non-tandem transplantation groups; however, within the tandem subgroup, genetic mutations were associated with higher mortality (66.7% vs. 0%, p = 0.058). **Conclusion:** High-risk genetic mutations, particularly amplification 1q and deletion 17p, significantly predict poorer survival outcomes following second autologous Hematopoietic Stem Cell Transplantation (auto-HSCT) in multiple myeloma patients. Patients harboring these mutations exhibit higher mortality rates and delayed platelet engraftment, underscoring the clinical importance of pre-transplant genetic profiling. Early identification of these genetic abnormalities through FISH analysis could enable more precise risk stratification, guide personalized therapeutic approaches, and potentially improve clinical management and patient outcomes in multiple myeloma.

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OP 13_Case report

TWO CASES OF PRIMARY AMYLOIDOSIS PRESENTING WITH VERTEBRAL AMYLOIDOMA

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Objective: Amyloidosis is a rare disease in which symptoms occur due to protein misfolding and the accumulation of amyloid fibrils in organs and tissues. 20 types of amyloid proteins have been identified. AL (Amyloid Light chain), AA (Amyloid Associated), Aβ (Amyloid beta) constitute the major types. AL type contains free immunoglobulin light chain amino terminals formed by plasma cells. AA amyloidosis occurs due to the accumulation of Serum Amyloid A (SAA) in tissues due to severe and long-term infection or inflammation. AL type amyloidosis accompanies approximately 10% of multiple myeloma. The frequency of AL amyloidosis is between 3 and 12 per million. It is more common in men and occurs at an average age of 63. AL amyloidosis can affect various tissues. The involvement of AL amyloidosis is in the heart, kidney, liver, nervous system and very rarely in the vertebral area. **Case 1:** A 56-year-old male patient was admitted to the hospital with complaints of low back pain, leg pain, numbness, and inability to walk. After examinations, a compression fracture was detected that caused height loss at L5, and he underwent surgery by a neurosurgeon. After the operation, fixators were placed at L3, L4, and S1. AL amyloidosis was detected as a result of tissue biopsy taken after surgery. Serum free kappa and lambda levels were studied. Lambda light chain was found to be 41.4 mg/dL (8.3–27 mg/dL) high. Other examination results were urea: 53 mg/dL (17–43 mg/dL), creatinine: 0.95 mg/dL (0.67–1.17 mg/dL), calcium 8.8 mg/dL (8.8–10.6 mg/dL), INR = 1.6, PT = 18.1 sec (10–14 sec), APTT 35.6 sec (21–29 sec), Hbg: 9g/dL (12.9–14.2 g/dL), MCV = 78fL (81–96 fL), platelet: 220 10^3/UL (155–366 10^3/U/L), complete urine test was normal, there was no proteinuria. Bone marrow biopsy showed 5%–7% plasma cells. NTpro BNP was 153 pg/mL (< 100 pg/mL), Echocardiography (ECHO) showed the left atrium wider than normal, other heart chambers were of normal width and their wall thicknesses increased. It was evaluated as hypertrophic cardiomyopathy. Ejection Fraction (EF) was measured as 60% with preserved systolic functions. The patient was started on VCD (cyclophosphamide, bortezomib, dexamethasone) and daratumumab treatment with the diagnosis of primary amyloidosis. The patient developed a hematoma in the surgical area, factor levels were studied, factor 10 level was found to be 10% low. Upon numbness, Electromyomography (EMG) was performed on bilateral lower extremities. EMG results showed low bilateral tibial nerve amplitudes. The patient's complaints of loss of strength in the legs and inability to walk began to improve after treatment. **Case 2:** A 60-year-old female patient with diabetes mellitus and coronary artery disease presented with complaints of back pain and difficulty walking. As a result of the vertebral MRI examination performed for the complaints, a lytic mass lesion of 27 × 13 mm in size, extending posteriorly, largely filling the T8 vertebral corpus was observed. The patient was taken to surgery and the mass was removed. In the pathology examination of the mass, CD38, CD138, Lambda positive and Congo red positive staining were observed. In the evaluation of serum free kappa and lambda, free kappa was 111 mg/dL, free lambda was 26 mg/dL, serum free kappa/lambda: 4.17. In the bone marrow examination, the plasma cell ratio was evaluated as 8%. Other laboratory results were as follows: urea: 32 mg/dL (17–43 mg/dL), creatinine: 0.7 mg/dL (0.67–1.17 mg/dL), calcium: 8.9 mg/dL (8.8–10.6 mg/dL), INR: 1.06, PT: 12.2 sec (10–14 sec), APTT: 20.5 sec (21–29 sec), Hbg: 11.4 g/dL (12.9–14.2 g/dL), MCV: 87 fl, platelet: 272 10^3/UL (155–366 10^3/U/L), complete urinalysis showed 3+ proteinuria. Spot urine protein creatinine ratio: 4.5 gr/day was detected. NT pro BNP: 219 pg/mL (< 100 pg/mL) increased, hs Troponin I: 13.3 ng/L. Cardiac examination ECHO showed normal heart chambers, normal left ventricular functions, EF%60. ECG examination was normal. VCD chemotherapy was started in the patient's treatment. The patient's complaints have decreased, and his treatment is continuing. **Results:** AL type primary amyloidosis presenting with vertebral involvement is a rare condition. Amyloidoma is usually a single mass and contains only amyloid in its structure. It can occur in many areas of the body and is hard and fixed. This pattern of involvement is very aggressive and can cause destruction and fractures in the bone. It is most commonly seen in the thoracic and then cervical vertebrae. Lumbar