present in 1 each whereas 1 patient had ASXL1, TET2 EZH2, RUNX1 and STAG2 mutations. The median R-IPSS and IPSS scores were 5 (IQR: 4.2-6) and 1.5 (IQR: 0.75-2) respectively. Thirty-one (73.8%) patients received azacytidine and 11 (26.2%) decitabine. ORR at cycle 1, cycle 2 and EOT was 12 (28.6%), 21 (51.2%), 18 (42.9%) with CR rates of 2 (4.8%), 11 (26.2%) and 11 (26.2%) respectively. Febrile neutropenia was observed in 23 (54.8%) and cycles were interrupted due to cytopenia's in 23 (54.8%) patients. Seven (17.1%) patients received allogenic HSCT and 2 (4.9%) received haploidentical HSCT. Five (12.2%) patients received venetoclax maintenance. Eight (21.1%) patients had disease relapse. The OS of MDS cohort was 59.5% with median 907 survival days (95% CI 386-1424) and The DFS was 44.4% with median survival 528 days (95% CI 336-719). Conclusion: Venetoclax in combination with HMA represents an effective therapeutic strategy for AML and MDS in the real-world setting, even in resource limited settings.

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OP 04_Case report

THE ROAD NOT-TAKEN: EXPLORING NON-TRANSPLANT OPTIONS IN DE NOVO PHILADELPHIA - POSITIVE ACUTE MYELOID LEUKEMIA

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Background: Philadelphia chromosome-positive acute myeloid leukemia (Ph+AML) is a rare and aggressive subtype of AML, characterized by the BCR::ABL1 fusion gene. It has historically been considered a high-risk leukemia, with allogeneic Hematopoietic Cell Transplant (HCT) recommended in the first remission. However, the emergence of targeted therapies, particularly potent Tyrosine Kinase Inhibitors (TKIs), has led to reconsideration of this approach. Drawing from advances in Philadelphia-positive acute lymphoblastic leukemia (Ph+ALL), where HCT omission has been explored successfully, this study examines whether a similar strategy can be applied in select Ph+AML cases. Objective: To evaluate the feasibility of a non-HCT approach in Ph+AML by analyzing a case where transplant was omitted, and the patient achieved sustained remission. Methods: We present a case of a 30year-old male diagnosed with de novo Ph+AML, identified through cytogenetics and molecular testing. The patient received induction chemotherapy with cytarabine and anthracycline (3+7) along with a TKI. Due to complications, his initial TKI was switched to ponatinib, and consolidation therapy consisted of azacytidine, venetoclax, and ponatinib. Disease monitoring was performed using quantitative Polymerase Chain Reaction (qPCR) for BCR::ABL1 and Next-Generation Sequencing (NGS). Results: The patient achieved a complete Molecular Response (MR 4.5) after the first cycle of consolidation therapy. Over 12-cycles of treatment, he maintained MRD negativity without emerging mutations. At 30-months post-diagnosis, he remains in sustained remission without undergoing HCT. Key factors that may have contributed to his favorable outcome include the absence of additional cytogenetic abnormalities, early achievement of deep molecular remission, and the use of a third generation TKI with activity against T315I mutations. Conclusion: This case suggests that a transplant-free approach may be a viable option for select patients with Ph+AML. While HCT remains the standard of care, the use of targeted therapies such as ponatinib in combination with venetoclax and azacytidine may provide an alternative pathway to long-term remission. Further studies are needed to validate patient selection criteria and assess long-term efficacy and safety of this approach.

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Adult Hematology Abstract Categories

Cellular therapy

OP 05_Case report

CORRELATION OF CD10+ B-LYMPHOCYTES AND PLASMA CELLS WITH OUTCOME IN BREAST CANCER

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Objective: Bone Marrow (BM) is poorly understood from the point of view of the prognostic role of hematopoietic cells and subpopulations of lymphocytes in patients with Breast Cancer (BC). In recent years, more attention has been paid to the study of the innate immune system, which includes B-lymphocytes. They produce IgM antibodies, which play an important role in the induction of apoptosis. Methodology: Study was carried out in 107 BC patients' stage I-II. Adjuvant chemotherapy - 65.4% of patients, radiation therapy - 49.8%, hormone therapy - 84% of patients. Her2/neu"-" 80%, Her2/ neu"+"-18%, TNBC-12%. The duration of the follow-up period after surgery was 8-years. Multiparameter flow cytometry was used, FACSCANTO II. Studies of BM lymphocyte subpopulations were carried out in the gate of CD45++ cells: CD19, CD20, CD5, CD38, CD10, CD45, HLA-DR, CD27. Radical resection -38.3% of patients, mastectomy - 59.7%. Results: B1-cells was higher in B-Her2"+", stage IIA, with 2 affected lymph nodes. B1-cells correlated with plasma cells. The total percentage of B-cells in BM was significantly associated with the prognosis of BC. B1 cells were associated with progressionfree and disease-free survival. Disease progression was observed at low levels of B1 cells. In cases more than 10% Blymphocytes in the BM of BC patients' Overall Survival (OS)

rates were more favorable (p = 0.01). Especially for BC with a high Ki-67. Disease progression was observed in 1/3 of BC patients with low levels of B1 cells. CD38 expression on B-cells was a prognostically favorable factor: the role is realized during 5–10-years of follow-up after surgery. Level CD38+ B-cells more than 10% correlated with high OS (p = 0.02). The presence of CD10+CD19+ B-lineage precursors was associated with a more favorable prognosis (OS, the threshold level 12%, p = 0.04). The prognostic role of the CD10 antigen was realized when patients were observed for more than 5-years. **Conclusion**: Total relative number of (more than 10%) of BM CD19+ cells were significantly related to OS in BC. B-cell precursors and CD38+ B-cells were associated with favorable prognosis. Prognostic role of B-lineage precursors and CD38- positive cells was in the periods of 5–10 years after surgery.

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OP 06_Case report

NEURON SPECIFIC ENOLASE POSITIVE OVARIAN INTERMEDIATE GRADE SERTOLI LEYDIG CELL TUMOR: A RARE CASE REPORT

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Objective: Ovarian Sertoli-Leydig Cell Tumors (SLCTs) are rare sex cord-stromal tumors, accounting for less than 0.5% of all primary ovarian tumors. They are most commonly diagnosed in adolescents and young women and can present with a variety of symptoms, including abdominal pain, a palpable mass, and, in some cases, signs of hormonal imbalance. While tumor markers such as Cancer Antigen-125 (CA-125) may be elevated, Neuron-Specific Enolase (NSE) is not typically associated with these tumors. Results: A 14-year-old girl presented with diffuse abdominal pain and sudden abdominal distension. Laboratory tests revealed elevated CA-125 (65.3 U/mL, normal: 0 -35 U/mL) and NSE (13.7 μ g/L, normal: 0-12.4 μ g/L). Imaging studies, including transabdominal pelvic ultrasound and abdominal Computed Tomography (CT), demonstrated a large, 23 cm right ovarian mass with predominantly solid but focal cystic components, raising suspicion for malignancy. Given the tumor's size and characteristics, a fertilitysparing right salpingo-oophorectomy and paravertebral retroperitoneal lymph node biopsies were performed. Histopathological examination confirmed an intermediate-grade Sertoli-Leydig cell tumor with frequent mitotic figures but no necrosis. Conclusion: This case is notable as the first reported instance of an SLCT associated with elevated serum NSE levels. While NSE is primarily considered a marker for neuroendocrine tumors and small cell lung carcinoma, its elevation in this case raises questions about its potential role in SLCTs. This finding suggests a need for further research to determine whether NSE could serve as a useful biomarker in the diagnosis or monitoring of SLCTs. Given the rarity of these tumors and the limited data on

their tumor marker profiles, additional studies are needed to explore the clinical significance of NSE elevation in SLCTs.

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OP 07_Case report

POLYRADICULOPATHY FOLLOWING CAR T-CELL THERAPY FOR LYMPHOID MALIGNANCIES, DIAGNOSTIC CHALLENGES: A REPORT OF 3 CASES

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Objective: Neurotoxicity is a serious but incompletely understood complication of CAR T-cell therapy. here we highlight presentation and diagnostic challenges of non-CNS CAR Tcell therapy complication. Case series: Case 1: A 43-year-old female with relapsed DLBCL stage IVB without CNS involvement underwent Axi-Cel CAR T-cell therapy, developing grade I CRS responded to tocilizumab. On day +15, developed diplopia and 6th nerve palsy MRI suggestive CNS lymphomatous infiltration, managed with dexamethasone and intrathecal MTX with a PET scan on day 30 showing CMR. Subsequently, she had limbs weakness with EMG/NCS confirming polyradiculopathy CSF showing high protein but negative for infection, malignant cells and autoimmune. Treatment included methylprednisolone, plasmapheresis, and IVIG, leading to slight improvement. Day 60 PET-CT revealed cervical and lumbar nerve roots neuritis, and by day 90, biopsy-confirmed disease relapse. She had partial response to glofitamab, unfortunately, she passed away four months post-CAR T-cell therapy. Case 2: A 38-year-old male with primary refractory stage III DLBCL received Axi-Cel after lymphodepletion with Flu/Cy. On day +1, grade I CRS developed and was treated with tocilizumab and dexamethasone. By day +4, he exhibited ICANS grade IV with confusion and seizures, managed with methylprednisolone, anakinra, and lorazepam. CSF showed high protein but no infection or malignant cells. MRI suggested viral encephalitis, and acyclovir was started. Extubated on day +8, but showed lower limb weakness (0/5 power) and urinary retention. Spinal MRI showed intramedullary T2 changes. IVIG was given on day +11, and physical therapy started. By day +16, he could stand; MRI normalized by day +29. PET-CT on day +32 showed a very good partial response. The patient was able to walk and discharged on day +39 with ongoing recovery. Case 3: A 36-year-old male with CML transformed to B-cell ALL with CNS involvement refractory to triple IT chemotherapy received IT thiotepa, complicating with lower limb weakness and sphincter loss. NCS confirmed L5 radiculopathy; MRI and CSF were negative for blasts or CNS disease. Post-Brexu-cel infusion (day +5 to +7), he developed CRS grade I, treated with