

therapy ($p = 0.978$, $p = 0.902$ respectively). Following low-dose maintenance therapy, the median PFS2 was 26.167-months (95% CI 5.571–46.762) in the lenalidomide free therapy group and 25.433-months (95% CI 0.08–50.787) in the lenalidomide-based second line therapy group ($p = 0.581$). Although median OS2 could not be calculated, at 60-months, the survival rate was 74.5% in patients receiving lenalidomide-based treatment, while it was 62.3% in patients receiving treatment without lenalidomide ($p = 0.637$). **Conclusion:** This study introduces the concept of low dose versus MTD lenalidomide maintenance. MTD does not confer a survival benefit and is associated with increased toxicity. Our findings support low-dose maintenance as a preferable approach, although lenalidomide refractoriness remains a concern.

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PP 26_Case report

ASSESSMENT OF INTERPHASE FLUORESCENCE IN SITU HYBRIDIZATION (FISH) TEST IN A PATIENT WITH MULTIPLE MYELOMA: EXPERIENCE OF OUR MEDICAL GENETICS DEPARTMENT

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Objective: Multiple Myeloma (MM) is an orphan disorder of end stage plasma cells with acquired genetic abnormalities of clinical importance not captured by conventional cytogenetic analysis because of the low proliferation of malignant plasma cells. Thus, interphase Fluorescence In Situ Hybridization (FISH), performed on sorted plasma cells detected abnormalities independently of a proliferative and infiltrative index. The purpose of this study was to explore, for the first time in our Medical Genetics department the molecular genetics features in a Tunisian patient with multiple myeloma. A 35-year-old Tunisian man, followed-up for MM since two years and received VTD chemotherapy protocol (bortezomib, thalidomide et dexaméthasone). Actually, as part of evaluation of his disease, and in the presence of infectious syndrome, the MM's relapse is suspected. Magnetic cell separation of PCs was performed using the Whole Blood CD138 MicroBeads, Whole Blood Column Kit, and the QuadroMACS Separation Unit (Miltenyi Biotec) according to the manufacturer's protocol. Slides were pretreated according to the manufacture's protocol. The FISH probes used in this study included IGH/FGFR3(4p16/ 14q32; DC.DF)/vysis, TP53/CEP 17(17p11.1-q11.1/ 17p13.1) FISH probe, Vysis. **Results:** Revealed the presence of three signals of IGH in 75% of nuclei and one signal of TP53 in 96% of nuclei. These results demonstrated the deletion of the short arm of chromosome 17 (del(17p)) and the absence of t(4;14). However, the presence of three signals of IGH indicated either the IGH amplification or the IGH rearrangement

involving other partner chromosomes. These results were consistent with patient's relapse. The t(4;14) and del (17p) are high-risk markers associated with adverse prognosis. Patients with these genomic aberrations should be treated with targeted therapy. The detection of the 1q21 'gain' could be considered in further studies because it is the most frequent structural abnormality, observed in 35%–40% of the patients with MM which is an independent poor prognostic factor.

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PP 27_Case report

TREATMENT MANAGEMENT IN MULTIPLE MYELOMA WITH RENAL DISORDER

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Introduction: Multiple Myeloma (MM) is a plasma cell neoplasm characterized by the accumulation of monoclonal plasma cells in the bone marrow, leading to osteolytic lesions, anemia, infections, hypercalcemia, and kidney impairment. This review focuses on managing kidney disease in MM, particularly light chain cast nephropathy. **Case:** A 66-year-old male with progressive fatigue, dizziness, weight loss, and recurrent pneumonia was referred for anemia evaluation on 28.11.2024. Laboratory results showed:

- Hemoglobin: 5.4 g/dL, Hematocrit: 18.3%, Platelets: $56 \times 10^3/\mu\text{L}$
- Creatinine: 1.39 mg/dL, Calcium: 9.7 mg/dL, Total Protein: 9 g/L, Albumin: 2.9 g/L
- IgA: 4295 mg/dL, IgG: 141 mg/dL, IgM: < 5 mg/dL

Peripheral smear showed rouleaux formation, and protein electrophoresis revealed a gamma peak. Immunofixation detected IgA-lambda bands, with bone marrow biopsy confirming 70% plasma cell infiltration. The patient was started on VCD chemotherapy (bortezomib, cyclophosphamide, dexamethasone). Neutropenia worsened, requiring G-CSF support. Renal function improved, and zoledronic acid was given for widespread lytic lesions. Due to Febrile Neutropenia (FEN), treatment was switched to VRD (bortezomib, lenalidomide, dexamethasone). After four cycles, symptoms improved, and cytopenia's resolved. Although serum immunofixation remained positive, the patient achieved a Very Good Partial Response (VGPR). A follow-up bone marrow biopsy is planned after four more cycles, with Autologous Stem Cell Transplant (ASCT) scheduled if the response continues. **Conclusion:** Managing MM with renal impairment requires balancing efficacy and toxicity. A four-drug regimen (Dara-CyBorD or Isa-CyBorD) is preferred in fit patients with severe AKI, while a three-drug regimen (Dara-Vd) is recommended for frail patients. If daratumumab or isatuximab is unavailable, CyBorD is an alternative. Bortezomib, daratumumab, and isatuximab can be safely used in kidney dysfunction without dose adjustments. Lenalidomide is avoided in AKI unless refractory. In this case, VCD was chosen initially, and after renal improvement, VRD

was used despite FEN episodes. The patient achieved VGPR and is now planned for ASCT.

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PP 28_ Case report

THE SUCCESS LIES ON CLINICAL SUSPECT: THE SYNCHRONOUS CANCERS PRESENTING AS PULMONARY AND VERTEBRAL MASS LESIONS

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Objective: It is a well-known epidemiological research issue that cancer patients are at high risk for developing multiple primary cancers. The risk increase is more likely among cancer survivors and elderly people. We present a case of synchronous cancers with pulmonary cancer and extramedullary plasmacytoma. A 68-year-old male patient was evaluated for back pain, walking difficulty, and urinary incontinence. MRI showed a vertebral mass lesion on T10–11, with a pre-diagnosis of metastatic bone disorder. A PET-CT scan was performed to find out the primary cancer. This time, two mass lesions were striking: one on the right infrahilar region of the lungs and the other as a large lesion on the vertebrae, as seen on MRI, which seemed to be two separate malignant lesions. Two biopsies were decided. The patient's clinical picture deteriorated, and an urgent surgery for decompression and a diagnostic lung biopsy by bronchoscopy were performed. Histology of the vertebral lesion revealed kappa monotypic cell infiltration consistent with plasmacytoma, and histology of the lung revealed non-small cell lung carcinoma. He had a monoclonal gammopathy as IgG kappa with a level of 1.24 g/dL. Further investigation covered bone marrow, which confirmed the diagnosis of solitary plasmacytoma and primary lung carcinoma. Treatment was designed as radiotherapy for plasmacytoma and referral to the oncology unit with a recommendation for three monthly follow-ups for pursuing active myeloma development. **Results:** Multiple cancers comprise two or more primary cancers occurring in an individual originating in a primary site or tissue and are neither an extension nor a recurrence or metastasis. According to the timing of the cancers' diagnosis, the development of different cancers may be differentiated as synchronous or metachronous. The risk for the development of multiple primary cancers may be multifactorial as inherited predisposition to cancer; the lifestyle, cancerogen exposure related with environmental factors; previous cancer and increased survival and surveillance of cancer patients. We highlighted the need for comprehensive epidemiological data collection in cancer patients by publishing this case.

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PP 29_ Case report

ACUTE LYMPHOBLASTIC LEUKEMIA DIAGNOSED FOUR YEARS AFTER HSCT IN A BETA THALASSEMIA PATIENT: A CLINICAL CASE

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Introduction: Beta thalassemia is an inherited blood disorder caused by defective synthesis of the beta chains of hemoglobin. This results in the production of ineffective red blood cells, leading to anemia and a severe reduction in the ability to transport oxygen to organs and tissues. In some cases, patients with beta thalassemia, due to prolonged treatment processes and other factors, may develop malignant hematologic disorders. This case presentation describes a patient diagnosed with beta thalassemia major who developed Acute Lymphoblastic Leukemia (ALL) four years after undergoing Hematopoietic Stem Cell Transplantation (HSCT). **Materials and methods:** A patient diagnosed with beta thalassemia major, registered at the Thalassemia Center (TC), underwent allogeneic HSCT in 2020 and was later diagnosed with T-cell Acute Lymphocytic Leukemia (T-ALL) four years post-transplant. **Results:** A 19-year-old male patient was diagnosed with beta thalassemia major at the age of one year. He has been under regular follow-up at the Thalassemia Center since the age of six. At seven years old, he was officially diagnosed with "Beta Thalassemia Major" (HbA2 – 3.9%; HbF – 57.1%) and has since been on a transfusion regimen with chelation therapy. On February 23, 2020, he underwent an allogeneic bone marrow transplantation from his HLA 10/10 matched sibling using the BU/Flu/CY/ATG/TT myeloablative conditioning regimen. Post-transplant chimerism analysis showed 93% donor cells. The patient was regularly monitored at the TC-HSCT outpatient clinic. **Medical history:** The patient was born from his mother's third pregnancy and third delivery.

- Two siblings from previous pregnancies did not survive.
- He was born at term with a birth weight of 3500g.
- He had incomplete routine vaccinations.
- He had a history of measles and chickenpox infections.
- The family denies a history of tuberculosis or venereal skin diseases.
- One healthy sibling lives at home.
- Parents are not consanguineous.
- The father was diagnosed with Hodgkin lymphoma two months ago and started treatment.

On November 5, 2024, the patient presented with extensive bruising and petechiae over his entire body. His general condition was severe, and laboratory findings were:

- Leukocytes (L): $284.32 \times 10^3/\mu\text{L}$
- Hemoglobin (Hb): 121 g/L
- Platelets (Tr): $30 \times 10^9/\text{L}$
- Blast cells: 80%