soho2023
ELEVENTH ANNUAL MEETING

Case Presentation

1- **57-year-old** man with **bicytopenia** (leukopenia, thrombocytopenia). No personal or family history.

2- **Hypoplastic** bone marrow. Karyotype 46XY, del(20q). No abnormal ASXL1, TP53, RUNX1. **Negative** *NPM1*, *FLT-ITD*, *FLT-TKD*, *IDH1*, *IDH2* mutations.

3- Started **Cyclosporin** and experienced opportunistic infections. He became pancytopenic and **AML** was diagnosed, 2 years later (Peripheral and 30% bone marrow blasts CD 117+, CD34+).

4- After 7 + 3 he had **persistent** disease, tso unrelated HLA-C mismatched **allografted**, after FLAMSA sequential therapy and reduced intensity conditioning (Bu 8 mg/m2; Cy 80 mg/Kg; ATG 60 mg/Kg). Achieving **complete** remission and count recovery.

5- 5 years later, he experienced **extramedullary** (pancreas) AML infiltration. Bone marrow **synchronous** relapse was confirmed (hypocellular, 83% blasts). Same immunophenotype and del (20q).

6- **NGS** revealed MDS-related mutations

Post-Transplant Relapsed Secondary Acute Myeloid Leukemia

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ONKOMEDEOR
ONKOLOGISCHE ZENTREN

Secondary Acute Myeloid Leukemia. From Clonal Evolution to Post-Transplant Relapse

Myelodysplastic syndromes (**MDS**) are clonal hematopoietic **neoplasms** characterized by cytopenias, dysplasia, and specific stem cell **mutations**. The acquisition of additional somatic mutations drives the progression to acute myeloid leukemia (**AML**).

Secondary AML is genomically defined and evolves from a **previous** hematologic diagnosis. It has a **poor** prognosis.

Comprehensive molecular profiling is **standard** of care for hematological disorders at **diagnosis** and **relapse**. Mutational architectural understanding has improved risk stratification and personalized therapeutic approaches for **better** outcomes.