plasmacytosis consistent with MM rather than solitary plasmacytoma or IgG4-related processes. (3) Cytogenetic risk—notably t(14;20)—supports intensified monoclonal-antibody –based induction (DRd) and vigilant surveillance, as this lesion associates with inferior outcomes on IMiD/PI-only backbones. (4) In select young patients achieving deep remission, deferring ASCT after robust daratumumab-based induction and consolidative RT can be reasonable when aligned with patient values and close monitoring—especially if toxicity, fertility considerations, or personal preference weigh heavily. **Conclusion:** Young-onset, high-risk IgA-κ MM with a large sacral mass achieved a durable, deep remission on DRd plus focal RT, permitting ASCT deferral with maintenance therapy and sustained disease control. Pairing comprehensive imaging with cytogenetic risk and early antibody-based induction may optimize outcomes in comparable high-risk, bone-predominant presentations.

https://doi.org/10.1016/j.htct.2025.106168

PP 35

FAMILIAL MULTIPLE MYELOMA: SIBLING CASES WITH DISTINCT CLINICAL MANIFESTATIONS

Ali Turunç *, Birol Güvenç

Çukurova Üniversitesi, Türkiye

Introduction: Multiple myeloma (MM) is a malignant plasma cell disorder that typically occurs sporadically. Familial clustering is rare, with only a limited number of cases reported worldwide. Such familial presentations suggest a possible hereditary predisposition or shared environmental risk factors contributing to disease development [1,2]. Here, we present two siblings with distinct plasma cell neoplasms: one with recurrent extramedullary plasmacytoma and the other with multiple myeloma. **Case Presentation:** The first case was a 69-year-old woman who underwent surgery in 2017 for a proximal femoral mass, diagnosed as plasmacytoma. In 2024, she presented with a cervical swelling; excisional biopsy of a right level-5 lymph node again revealed plasmacytoma. Bone marrow biopsies performed at that time did not show features of multiple myeloma. Her brother, one year older, was diagnosed with multiple myeloma in June 2025. PET-CT revealed lytic lesions in the axial skeleton, and systemic therapy was initiated. **Discussion:** Familial occurrence of plasma cell neoplasms is exceedingly uncommon. Reported cases often involve either multiple relatives with MM or, less frequently, different manifestations of plasma cell disorders within the same family [3,4]. The present siblings illustrate divergent clinical phenotypes: persistent extramedullary plasmacytoma without myeloma progression in the sister, versus classical MM with lytic bone disease in the brother. This highlights the potential role of shared genetic background with variable penetrance and expression. Genetic susceptibility loci, immune dysregulation, and epigenetic mechanisms have all been proposed as contributors to familial myeloma [5]. Recognizing such familial patterns may have implications for surveillance strategies in high-risk relatives. **Conclusion:** We report a rare familial clustering of plasma cell neoplasms in siblings, underlining the importance of considering hereditary predisposition in plasma cell disorders. Further genetic and epidemiological studies are warranted to elucidate the underlying mechanisms.

https://doi.org/10.1016/j.htct.2025.106169

PP 36

ACQUIRED PYRUVATE KINASE DEFICIENCY FOLLOWED BY MYELODYSPLASTIC SYNDROME: A CASE REPORT

Salih Sertaç Durusoy *, Sinem Çubukçu, Gönül Irmak

Ali Osman Sönmez Onkoloji Hastanesi, Türkiye

Introduction: Pyruvate kinase (PK) deficiency is an autosomal recessive red blood cell (RBC) enzymopathy leading to chronic hemolysis. It is the second most common RBC enzymopathy and the most frequent cause of chronic hemolytic anemia due to an enzyme defect. PK enzymes consist of various isoforms encoded by PKLR and PKM genes, which catalyze the conversion of phosphoenolpyruvate (PEP) to pyruvate and ATP in the final step of glycolysis. Clinically significant PK deficiency is associated with PKLR mutations. Acquired PK deficiency is extremely rare, and its molecular basis remains unclear. Some cases have been associated with AML. Here we present a rare case of acquired PK deficiency followed by myelodysplastic syndrome (MDS). **Case Presentation:** A 70-year-old male presented with fatigue, weakness, and jaundice. Laboratory findings were as follows: WBC: 7.0×10^9/L, Hemoglobin: 7.9 g/dL, MCV: 101 fL, Platelets: 601×10^9/L, Total bilirubin: 1.6 mg/dL (indirect: 1.0 mg/dL), LDH: 280 U/L. Other biochemical parameters were within normal limits. Hemoglobin electrophoresis was normal. Direct and indirect Coombs tests were negative. Haptoglobin was 14 mg/dL (low). Erythrocyte PK activity was reduced at 3.16 U/g Hb (reference: 4.4–5.9). G6PD activity and osmotic fragility were normal. The patient had no prior anemia history. Genetic analysis for PKLR mutations was negative, supporting an acquired form. During follow-up, bilirubin increased to 8.6 mg/dL, LDH rose to 800 U/L, and hemoglobin decreased to 6.0 g/dL. The patient was taking gliclazide for diabetes mellitus, which was discontinued due to suspicion of hemolysis induction. Bilirubin subsequently decreased. Bone marrow biopsy showed dysplastic erythroid changes without blast increase, consistent with MDS. The patient initially required two RBC transfusions weekly, but after gliclazide withdrawal, the requirement decreased to one unit every two weeks. Genetic testing for MDS is ongoing. **Discussion & Conclusion:** Acquired PK deficiency is extremely rare. In this case, a 70-year-old patient developed PK deficiency followed by a diagnosis of MDS. While congenital hemolytic anemias usually present in younger patients, clinicians should be aware that acquired cases may appear later in life. Careful evaluation of medications and bone marrow disorders is essential in elderly patients with unexplained hemolysis.

https://doi.org/10.1016/j.htct.2025.106170