pattern, biatrial enlargement, moderate tricuspid regurgitation, and mild pulmonary hypertension. The constellation of findings was inconsistent with hypertensive heart disease, raising suspicion for infiltrative cardiomyopathy. Given the typical echocardiographic appearance, the patient was referred to hematology for amyloidosis evaluation. Laboratory assessment revealed elevated inflammatory markers (CRP: 59-74 mg/L) but notably, serum protein electrophoresis showed no distinct M-band. However, serum immunofixation was positive only for lambda light chains with negative IgA, IgG, and IgM, suggesting an oligosecretory plasma cell disorder. Bone marrow biopsy revealed 40% plasma cell infiltration with immunophenotype showing CD38(+), CD56(+), and CD19 (-) with lambda light chain restriction. Critically, Congo red staining was positive, confirming amyloid deposition and establishing the diagnosis of AL amyloidosis. Cytogenetic analysis by FISH was negative for high-risk abnormalities including p53 deletion, RB1 deletion, t(11;14), and t(4;14). Additional imaging revealed multisystem involvement: chest CT showed ground-glass opacities in lower lobes with reactive mediastinal lymphadenopathy, while abdominal ultrasound demonstrated grade 1 hepatosteatosis, minimal splenomegaly, and mild ascites, consistent with systemic amyloid deposition. The patient's medical history was notable for appendiceal mucinous neoplasm in 2022, raising questions about potential relationships between these conditions. Clinical presentation included progressive heart failure symptoms with peripheral edema, confirming cardiac involvement as the primary manifestation. **Discussion:** This case illustrates several important clinical aspects of AL amyloidosis. The presentation in a 43-year-old patient is relatively uncommon, as AL amyloidosis typically affects older adults with median age around 65 years. The cardiac-predominant presentation with characteristic echocardiographic findings enabled early recognition and appropriate referral. The oligosecretory nature of the underlying plasma cell dyscrasia initially complicated diagnosis, as conventional serum protein studies were unrevealing. This emphasizes the importance of comprehensive light chain analysis in suspected cases, as oligosecretory variants can account for up to 15% of cases. The "sparkling" myocardium appearance on echocardiography, while not pathognomonic, represents a classic finding in cardiac amyloidosis resulting from increased acoustic reflectance of amyloid-infiltrated myocardium. Combined with restrictive physiology and biatrial enlargement, these findings strongly suggest amyloid cardiomyopathy. The multisystem involvement demonstrated by imaging studies indicates advanced disease requiring prompt treatment initiation. Cardiac amyloidosis carries poor prognosis without treatment, with median survival often less than one year in symptomatic patients.

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PP 34

High-Risk IgA-κ Myeloma with Sacral Mass in a 31-Year-Old: Deep Response to Daratumumab −Lenalidomide−Dexamethasone plus Local RT without ASCT

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Introduction: Multiple myeloma (MM) in young adults is uncommon, and high-risk cytogenetics complicate standard pathways. We report a 31-year-old woman with IgA-κ MM, large sacral involvement, and adverse genetics, achieving a deep remission with daratumumab−lenalidomide−dexamethasone (DRd) plus focal radiotherapy (RT), electing to defer autologous transplant. **Methods:** Single-patient case review from prospectively maintained records. Data included presenting features, MRI/PET-CT, serum/urine monoclonal studies, bone-marrow histology/flow, and plasma-cell FISH. Treatment, response, and tolerability were documented. **Results:** A previously healthy 31-year-old presented with severe nocturnal lumbosacral pain and right-sciatic radiation. MRI revealed a left-lateral sacral mass (77 × 56 mm) with contrast enhancement; PET-CT demonstrated focal hypermetabolic lytic lesions in sacrum, L1, pubis, and scapula (SUVmax 5.4−5.9), with no visceral/extramedullary organ disease. Serum studies showed an IgA-κ M-component with elevated free light-chain ratio; β2-microglobulin was 4.2 mg/L (ISS stage II). Bone-marrow biopsy displayed intertrabecular plasma-cell infiltration; immunophenotype CD38+, CD56+, κ-restricted, CD19−; reticulin 0−1/4; amyloid negative. Plasma-cell FISH identified t(14;20)(IGH−MAFB) in ~35% of cells, indicating high-risk disease. She commenced DRd and received concurrent local RT to the sacrum (fractionated) for rapid pain control. Treatment was well tolerated, without renal or calcium derangements. Clinically, pain resolved; biochemically, the M-component cleared; radiologically, bone foci regressed with disappearance of pathologic uptake on interval imaging. Bone-marrow reassessment showed marked reduction of clonal plasma cells, consistent with deep response. Given age, recovery, and patient preference, autologous transplant was performed; she continued maintenance (daratumumab ± lenalidomide) with sustained remission on follow-up. **Discussion:** This case underscores four practice points. (1) Aggressive osseous disease at young age can herald high-risk biology; early, integrated MRI/PET staging captures true burden and guides focal RT for symptom control while systemic therapy acts on disseminated marrow disease. (2) Immunophenotype and marrow context (CD38 +/CD56+, κ-restriction; low reticulin) affirmed clonal

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.

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PP 35

FAMILIAL MULTIPLE MYELOMA: SIBLING CASES WITH DISTINCT CLINICAL MANIFESTATIONS

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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.

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PP 36

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

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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.

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