Innumerable Non-Bone-Contiguous Hepatic Plasmacytomas in Relapsed Multiple Myeloma

Kacie Staples, DO; Amy Courtney, DO; Jason Moran, MD
MaineHealth Maine Medical Center, Portland Maine

MaineHealth
Department of Internal Medicine

Introduction

Extramedullary disease (EMD) in multiple myeloma (MM) definitionally occurs in tissues without para-skeletal disease. It portends a worse prognosis with poor response to traditional therapies and occurs in up to 14% of patients with myeloma recurrence after remission. Notably, EMD is frequently associated with mutations in the MAPK pathways and gain of function mutations in chromosome 1q21. This case is unique because EMD to the liver is uncommon, and numerous lesions of disease in the liver is quite rare.

Case

An 83-year-old male with history of MM presented electively to the hospital after new liver lesions were incidentally noted on outpatient ultrasound.

- He was previously treated with a fourteen-month course of daratumumab, lenalidomide, and dexamethasone.
- He had recently reached remission with lambda free light chains stable at 3.09 mg/dL.
- Prior Fluorescence in Situ Hybridization (FISH) analysis showed gain of function mutation at 1q22.
- Computed tomography completed during admission showed “innumerable” liver lesions with accompanying hepatomegaly and a left hemipelvis lytic lesion.
- Lambda free light chains were measured showing a sharp increase to 358.76 mg/dL.
- Biopsy of one of the liver masses resulted with plasma cell proliferation with histochemistry positive for lambda protein, consistent with MM relapse with EMD.
- He subsequently restarted daratumumab, lenalidomide, and dexamethasone.



Top image: computed tomography in soft tissue window, showing numerous hepatic masses. Middle image: computed tomography in liver window, showing a more defined image of many hepatic masses. Bottom image: computed tomography in bone window showing lytic lesion of the left hemipelvis (white arrow).

Discussion

This case highlights a rare presentation of EMD in a patient with a high-risk mutation. First line therapy agents for EMD in MM are the same in patients without EMD, with a common combination being daratumumab, lenalidomide, and prednisone.

These patients often have poor response to treatment, although limited data show that treatment with lenalidomide can completely eradicate EMD in up to 44% of patients.

Given this patient’s age and comorbidities, including poor cardiac function and chronic obstructive pulmonary disease, proteasome inhibitors were deferred. He unfortunately did not tolerate his triple-therapy chemotherapy regimen and continued to clinically decline, subsequently moving to inpatient hospice.

There is growing evidence that chimeric antigen T-cell (CAR-T) therapy with immunomodulators may show benefit in patients with EMD and may represent a promising new therapy to an otherwise challenging to treat malady.

Conclusion

- Extramedullary disease portends worse prognosis in patients with MM.
- The treatment for MM with EMD remains the same as for patients with MM without EMD.
- There exists genetic mutations which can be associated with a higher risk of EMD in MM.

References

1. Tirotta D, Lena C, Torre C, Muratori P. Hematologic focal hepatic lesions in the absence of liver cirrhosis: Consider lymphoma and myeloma, *Clinics and Research in Hepatology and Gastroenterology,* 2026; 50(2): 2210-7401. doi.org/10.1016/j.clinre.2025.102749.
2. Bladé J, Beksac M, Caers J, Jurczyszyn A, et al. Extramedullary disease in multiple myeloma: a systematic literature review. *Blood Cancer J*. 2022;12(3):45. doi: 10.1038/s41408-022-00643-3.