Journal of Population Therapeutics & Clinical Pharmacology

RESEARCH ARTICLE
DOI: 10.53555/e3a56931

A RARE CASE PRESENTATION OF MULTIPLE MYELOMA AND HEPATIC ENCEPHALOPATHY IN A PATIENT WITH DECOMPENSATED CHRONIC LIVER DISEASE: A CASE REPORT

Dr Rishabh Ahlawat[1], Dr Deepak Sharma[2], Dr Vinay Mathur[3], Dr Saket Kanodia[4*], Dr Rudra Dutt Kaushik[5], Dr Deepak Singla[6]

[1](MBBS , junior resident , Department of General medicine), Sharda University, rishabhahlawat@gmail.com
[2] (MBBS , MD , DM(NEPHROLOGY), FIMSA) Professor And Head , Department Of General Medicine , Sharda University
[3] (MBBS , junior resident , Department of General medicine), Sharda University, Vinaymathur146@gmail.com
[4*](MBBS , junior resident , Department of General medicine), Sharda University, kanodiasaket98@gmail.com
[5] (MBBS , junior resident , Department of General medicine), Sharda University, rudra.d.kaushik@gmail.com
[6] (MBBS , junior resident , Department of General medicine), Sharda University, singlad7@gmail.com

***Corresponding Author:** Dr Saket Kanodia
(MBBS ,junior resident , Department of General medicine), Sharda University, kanodiasaket98@gmail.com

Abstract

Background: Multiple myeloma rarely presents concurrently with hepatic encephalopathy, especially in patients with decompensated chronic liver disease (CLD). Overlapping biochemical abnormalities may delay diagnosis.

Case Presentation: A 76-year-old male presented with altered sensorium and hepatic encephalopathy. Laboratory findings showed renal dysfunction, hypercalcemia, anemia, reversed albumin-globulin ratio, and monoclonal protein, raising suspicion for multiple myeloma. Lytic bone lesions and bone marrow biopsy confirmed the diagnosis.[1–3]

Discussion: Hepatic encephalopathy generally reflects advanced CLD; however, co-existing plasma cell dyscrasias complicate the diagnostic picture. The presence of CRAB criteria—hypercalcemia, renal impairment, anemia, bone lesions—necessitates evaluation for myeloma, even in CLD patients.[4–10]

Conclusion: Plasma cell dyscrasias should be considered in CLD patients who present with unexplained metabolic derangements.

Keywords: Multiple myeloma, hepatic encephalopathy, CRAB features, liver disease.

Introduction

Hepatic encephalopathy is a well-recognized complication of decompensated chronic liver disease (CLD). However, when metabolic abnormalities exceed the expected severity of hepatic dysfunction, alternative or coexisting etiologies must be considered[5,9] Multiple myeloma, a plasma cell malignancy characterized by bone marrow infiltration and CRAB criteria (hypercalcemia, renal impairment, anemia, and bone lesions), may coexist in such patients and delay diagnosis.[1–3]. This report highlights such a diagnostic dilemma in a patient presenting with hepatic encephalopathy.

Case Presentation

A 76-year-old male was brought to the Emergency Department with altered mental status and absent response to verbal or painful stimuli. Patient also had abdominal distention from the past 6 months. Family reported absence of stool passage for past two days. No history of alcohol use, fever, seizures, trauma, or prior neuropsychiatric symptoms was noted.

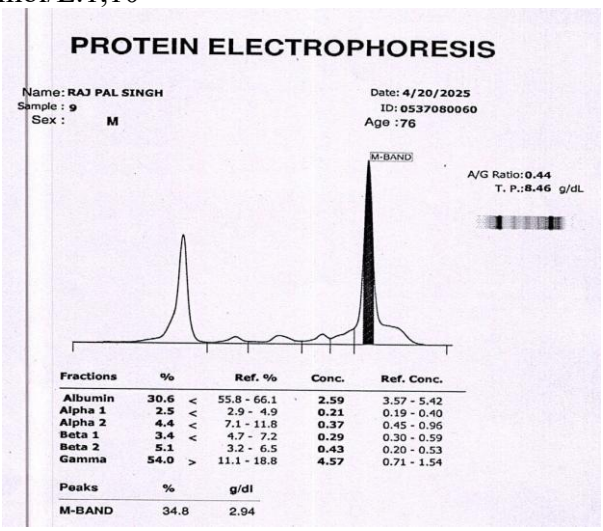
Vital signs: BP 100/60 mmHg, HR 88/min, Temp 98.6°F, SpO_2 95% RA.

General examination: Mild pallor, abdominal distension with ascites, sluggish bowel sounds.

Neurological examination: GCS E2 V1 M3; no focal deficits.

Laboratory Findings

- **Liver function tests:** Total bilirubin 5.4 mg/dL, AST 96 IU/L, ALT 84 IU/L, albumin 2.2 g/dL, globulin 4.1 g/dL (A:G ratio 0.53).
- **Renal function:** Serum creatinine 3.2 mg/dL, BUN 68 mg/dL.
- **Hematology:** Hemoglobin 8.1 g/dL, platelets 140,000/mm³.
- **Metabolic parameters:** Serum calcium 11.2 mg/dL.
- **Serum protein electrophoresis:** M-spike present.
- **Serum immunofixation:** IgM λ.
- **Serum ammonia:** 172 μmol/L.1,10



Imaging and Additional Evaluation

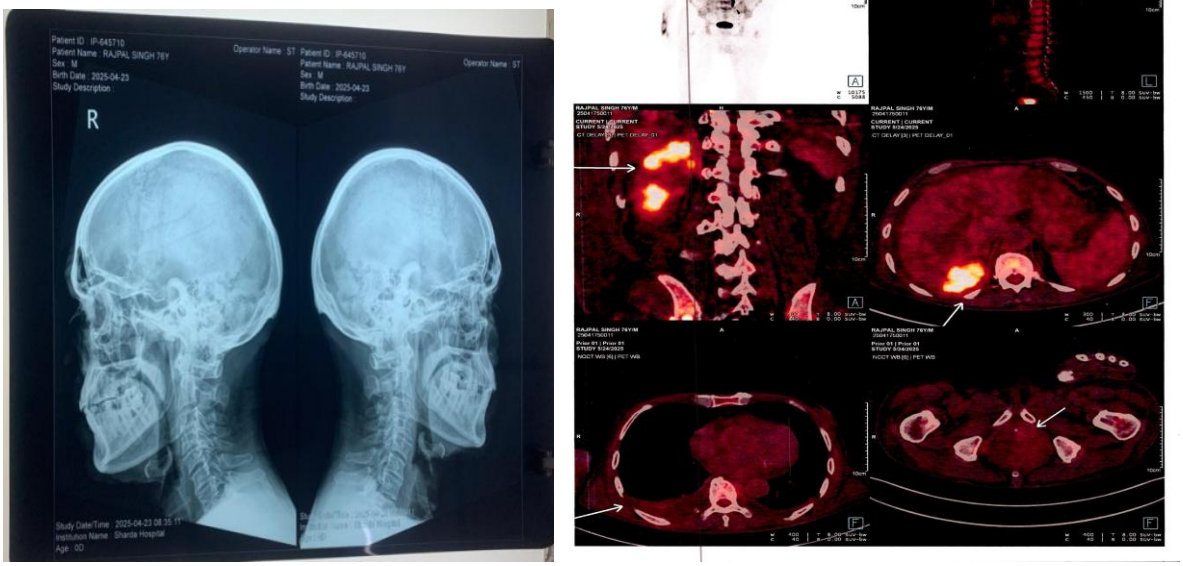
- **Ultrasound abdomen:** Coarse liver echotexture, splenomegaly, ascites.
- **CT brain:** Normal.
- **18F-FDG PET** CT: 1. Hypermetabolic involvement appearance of right kidney, may be due to stasis of radiotracer in the kidney and due to likely patient movement there is possibility that hypermetabolism appears to involve cortex at places. There is no known malignancy in kidney- in view of these findings possible pyelonephritis is not ruled out.

2. Bone marrow shows relatively high metabolic activity- likely involved by mitotic process.

3. No definite study evidence of metabolically active disease elsewhere in the body regions surveyed.

- **Skeletal survey:** Lytic lesions in skull and vertebrae.

• **Bone marrow biopsy:** Hypercellular marrow with >30% plasma cells → confirming multiple myeloma.[2,3,8]



Management

The patient was admitted to the ICU and managed with lactulose, rifaximin, bowel clearance, hydration, electrolyte correction, and empirical antibiotics due to the risk of spontaneous bacterial peritonitis. Hematology consultation guided initiation of myeloma treatment upon stabilization.[7,12]

Discussion

This case demonstrates a diagnostic challenge where CLD and multiple myeloma coexisted. CRAB features—hypercalcemia, renal impairment, anemia, and lytic lesions—guided hematologic evaluation.[1–4,6–10] Myeloma may be overlooked in CLD patients due to symptom overlap.Hypercalcemia and bone pain are particularly important diagnostic clues.[3,6]

In this case, several findings were disproportionate to typical hepatic encephalopathy:

- Hypercalcemia
- Markedly raised creatinine
- Significant anemia
- A reversed A:G ratio beyond what is typical for CLD
- Lytic lesions on skeletal survey

These findings fulfilled **CRAB criteria**, prompting further hematologic evaluation. A timely bone marrow biopsy confirmed multiple myeloma.

This case emphasizes the importance of maintaining diagnostic vigilance in CLD patients, especially when metabolic derangements cannot be fully attributed to hepatic dysfunction.

Conclusion

Multiple myeloma should be considered in patients with hepatic encephalopathy who present with unexplained metabolic abnormalities such as hypercalcemia, renal impairment, severe anemia, or bone pain.[1,3,9] Early diagnosis and multidisciplinary management significantly improve clinical outcomes

References

[1]Rajkumar SV. Multiple myeloma: 2022 update. Am J Hematol. 2022;97(8):1086–107.

[2]Kyle RA, Durie BGM, Rajkumar SV, et al. MGUS and smoldering myeloma. Leukemia.2010;24(6):1121–7.
[3]Kumar SK, Rajkumar V, Kyle RA, et al. Multiple myeloma. Nat Rev Dis Primers. 2017;3:17046.
[4]EASL Clinical Practice Guidelines: Hepatic encephalopathy. J Hepatol. 2022;77(3):827–44.
[5]Palumbo A, Anderson K. Multiple myeloma. N Engl J Med. 2011;364:1046–60.
[6]Rajkumar SV, Dimopoulos MA, Palumbo A, et al. IMWG diagnostic criteria. Lancet Oncol.2014;15:e538–48.
[7]Bird JM, Owen RG, D'Sa S, et al. Guidelines for multiple myeloma. Br J Haematol.2011;154(1):32–75.
[8]Dimopoulos MA, Terpos E, et al. Renal impairment in myeloma. Leukemia. 2010;24:525–34.
[9]Schreiber YS, Schafer DF. Hepatic encephalopathy. World J Gastroenterol. 2015;21:11523–35.
[10]Ludwig H, Sonneveld P, et al. Myeloma complications. Haematologica. 2018;103:1866–77.
[11]Tapper EB, Sharma P. Liver disease in elderly. Clin Geriatr Med. 2020;36:277–88.
[12]Chim CS, Kumar SK, et al. Relapsed myeloma. Leukemia. 2018;32:252–62.