"Lysozyme-Induced Nephropathy in Chronic Myelomonocytic Leukemia: A Case Report on Renal Recovery With Pacritinib Therapy"

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Introduction

- Chronic myelomonocytic leukemia (CMML) is a clonal hematopoietic disorder showing both myelodysplastic and myeloproliferative features. While cytopenias and splenomegaly are typical, renal involvement is rare and under-recognized.
- Lysozyme-induced nephropathy (LIN) results from excessive lysozyme production by monocytic cells, leading to tubular injury and progressive renal failure.

Aim

To present a rare case of CMML-associated lysozyme-induced nephropathy (LIN) and highlight the diagnostic challenges, therapeutic considerations, and the clinical benefit of Pacritinib in stabilizing renal function and improving hematologic parameters.

Objectives

1. **Recognize** renal complications, particularly lysozyme-induced nephropathy, as an uncommon but important manifestation of CMML.
2. **Understand** the diagnostic approach, including the role of renal biopsy and molecular profiling.
3. **Evaluate** therapeutic strategies in CMML with renal involvement, with emphasis on the potential role of **Pacritinib**.
4. **Apply** a multidisciplinary management framework (hematology + nephrology) to optimize patient outcomes.

Case Presentation

•65-year-old male with hypertension, cardiomyopathy, orchiectomy, and thrombocytopenia.
•Progressive renal dysfunction: creatinine ↑ from 1.1 → 3.7 mg/dL; proteinuria ↑ to 2.4 g.
•Renal biopsy: tubular deposition of lysosomal material → consistent with lysozyme nephropathy.
•Genetics: ASXL1 and NRAS mutations.
•Treatment course:
- Hydroxyurea (discontinued due to intolerance).
- Pacritinib (JAK2 inhibitor) → stabilized renal function + improved anemia & thrombocytopenia.

Results

Treatment strategy:
Hydroxyurea initially (discontinued due to intolerance).
Transitioned to Pacritinib (JAK2 inhibitor) for cytoreduction and symptom control.
Outcome evaluation:
Stabilization of renal function (creatinine plateau at 2.9–3.7 mg/dL).
Improvement in anemia and thrombocytopenia.
Tolerability of therapy monitored over time.

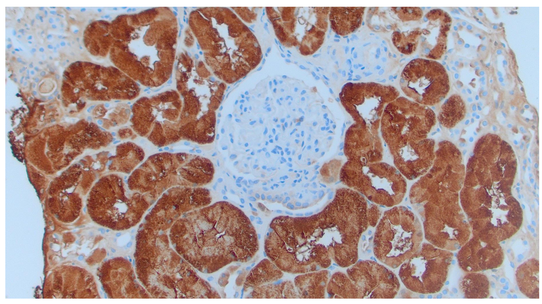


Figure X: Immunohistochemistry showing lysozyme-induced nephropathy in CMML. Strong **diffuse granular cytoplasmic staining (brown)** is observed in the proximal tubular epithelial cells, indicating lysozyme accumulation. The **glomerulus (center) remains** largely unstained, serving as an internal negative control. This staining pattern confirms lysozyme deposition as the cause of tubular injury.

Summary

- Early recognition of renal involvement in CMML is critical.
- Renal biopsy remains the gold standard for diagnosis of LIN.
- Pacritinib may represent a viable therapeutic option in CMML with renal complications.
- Collaborative hematology–nephrology care optimizes patient outcomes.

Discussion

•Renal complications in CMML are under-recognized but carry poor prognosis.
•Studies show up to 22% progress to ESRD within 2 years.
•Prognostic implications: renal dysfunction signals more aggressive disease.
•Therapeutic implications:
- Standard therapies limited by toxicity.
- Pacritinib has shown efficacy in myeloproliferative disorders and here improved both renal and hematologic outcomes.

•Multidisciplinary management is essential for early recognition, biopsy confirmation, and tailored therapy.

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