Acute renal failure in a patient with chronic myelomonocytic leukemia

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72 year old male admitted to nephrology with **AKI and edemas** in lower limbs

**Personal record**

- HT

- Prostatic adenocarcinoma (in remission treated with hormonetherapy and radiotherapy.

- Chronic myelomonocytic leukemia, stable and treated with Azacitidin (33 cycles).
In urine
Proteins **50 mg/dl**
Blood: Negative
Leucocytes: Negative
Sediment: no abnormal sediment.

Prot/Cr **585 mg/g**
MAU/Cr **220 mg/g**

Complement in range, ANAs, ANCAs, proteinogram, Serology (VIH VHC and VHB) negative.
Renal ultrasound

Normal size
Normal ecostructure
**Moderate bilateral hidronephrosis**
(no clear obstruction)
No litiasis.
Cortical cysts in right kidney.
Renal ultrasound

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Normal ecostructure
**Moderate bilateral hidronephrosis**
(no clear obstruction)
No litiasis.
Cortical cysts in right kidney.

Abdominal CT scan

Thickening of urothelium
(inflammatory vs tumoral) renal pelvis and both proximal ureters
Bone marrow aspiration:

Immature granulocytic hyperplasia.
Blasts (myeloid appearance) account for **18% of global cellularity**.
Monocytes: <10% (mostly immature monocytes).
**Cytology suggests a myelodisplastic/myeloproliferative disorder type CMML-2 (similar to previous)**
Bone marrow aspiration:

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Nephrology

Bilateral double J catheter

Cr 4 mg/dl
Nephrology

Bilateral double J catheter

Renal biopsy

Steroids (3 bolus 250mg MP)
### Causes of acute kidney injury in leukaemia

| Prerenal | Intravascular depletion  
| NSAIDs, ACEs, ARB,… |
|----------|-------------------------|
| Renal    | Glomerular: GN, vasculitis, TMA… |
|          | Tubulointerstitial: infiltration of renal parenchyma, lysozyme, TIAN,… |
|          | Tubular, light chain deposition disease, tumoral lysis syndrome, ATN by chemotherapy… |
| Postrenal| Obstruction by urethral infiltration, tumoral masses or nephrolithiasis. |

**Lysozyme serum** 36.8 mg/L (normal range: 4 - 13)

**Lysozyme urine** 27.9 mg/L (normal until 2)
• 2 cores: corticomedullary junction
• 9 glomeruli (none sclerosed)
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• 9 glomeruli (none sclerosed)
• Architectural distortion
Lysozyme
Lysozyme
On a Remarkable Bacteriolytic Element found in Tissues and Secretions.

By Alexander Fleming, M.B., F.R.C.S.

(Communicated by Sir Almroth Wright, F.R.S. Received February 13, 1922.)

(From the Laboratory of the Inoculation Department, St. Mary's Hospital.)

[Plate 9.]

In this communication I wish to draw attention to a substance present in the tissues and secretions of the body, which is capable of rapidly dissolving certain bacteria. As this substance has properties akin to those of ferments I have called it a "Lysozyme," and shall refer to it by this name throughout the communication.
SERUM AND URINARY LYSOZYME (MURAMIDASE) IN MONOCYTIC AND MONOMYELOCYTIC LEUKEMIA*

BY ELLIOTT F. OSSERMAN,‡ M.D., AND DOLORES P. LAWLOR

(From the Department of Medicine, Columbia University College of Physicians and Surgeons, and the Francis Delafield Hospital, New York)

PLATES 91 AND 92

(Received for publication 14 July 1966)

By electrophoretic analysis, large quantities of an exceptionally basic, cationic protein (CP) have been found in the urine of ten consecutive cases of monocytic and monomyelocytic leukemia.¹ The protein CP has been isolated and characterized as a relatively low molecular weight constituent with the enzymatic properties of lysozyme (muramidase).² Increased levels of CP-lysozyme have been demonstrated in the serum of monocytic leukemics by immunochromic and enzymatic techniques but not by electrophoresis. Evidence has been obtained that CP-lysozyme is the normal enzyme which is elaborated in excess in the monocytic leukemics. The relatively small molecular size of CP-lysozyme is apparently responsible for its urinary excretion.
Acute Myelogenous Leukemia Treated with Daunomycin Associated with Nephrotic Syndrome

M. Thomson, G. de Arriba, J. Ordi, H. Oliva, L. Hernando
Servicios de Medicina Interna, Nefrología y Anatomía Patológica, Fundación Jiménez Díaz, Madrid, Spain

Myelodysplastic Syndromes With Nephrotic Syndrome

Takayuki Saitoh,¹* Hirokazu Murakami,² Hideki Uchiumi,¹ Kazuaki Moridaira,¹ Tadashi Maehara,¹ Takafumi Matsushima,¹ Norifumi Tsukamoto,¹ Jun'ichi Tamura,¹ Masamitsu Karasawa,³ Takuji Naruse,¹ and Jun Tsuchiya²
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A Forgotten Cause of Kidney Injury in Chronic Myelomonocytic Leukemia

Tejas V. Patel, MD, MPH¹, Helmut G. Rennke, MD², J. Mark Sloan, MD³, Daniel J. DeAngelo, MD, PhD³, and David M. Charytan, MD, MSc¹
**DIAGNOSIS:**

Infiltration of renal parenchyma by leukemic cells with superimposed acute tubular damage by lysozyme.
renal biopsy: Infiltration by CMML + acute tubular damage by lysozyme

3 bolus of MP (250 mg each)
Acute renal failure is an uncommon complication of CMML that can be multifactorial.

- Tumoral lysis
  - Infiltration of renal parenchyma by leukemic cells
- Toxicity by chemotherapy
- Hyperviscosity syndrome
- Thrombotic microangiopathy
  - Acute tubular damage by lysozyme

Lysozyme is the less common pathogenic cause of AKI in patients with CMML (scarcey reported in literature).

In patients with CMML it should be considered in differential diagnosis and lysozyme levels in serum and urine should be measured in patients with CMML and AKI
Thank you!

Cristina Ortiz