



Congress Abstract

Rapidly Progressive Acute Tubulointerstitial Nephritis Masquerading as RPGN: The Vital Diagnostic Role of Renal Biopsy in Advanced AKI

Assel Tolezhanova¹, Meruyert Madikenova^{2,3}, Abduzhappar Gaipov^{2,3}

¹Department of Medicine, Outpatient Hemodialysis Center, LLP "FMNQAZAQSTAN", Astana, Kazakhstan

²Department of Medicine, Nazarbayev University School of Medicine, Astana, Kazakhstan

³Clinical Academic Department of Internal Medicine, University Medical Center, Astana, Kazakhstan

Proceedings of Central Asian Congress
of Nephrologists with International
Participation, October 9-10, Aktau,
Kazakhstan

Corresponding author's email:
abduzhappar.gaipov@nu.edu.kz

Copyright: by the author(s).

License: A non-exclusive license by the
publisher. Published by Australasia
Publishing Group LLP (Astana,
Kazakhstan) on behalf Kazakh Kidney
Foundation.

Open Access: This article is an open access
article distributed under the terms and
conditions of the CC-BY Creative
Commons Attribution license
<https://creativecommons.org/licenses/by/4.0>



ABSTRACT

Background & Objective: Acute tubulointerstitial nephritis (ATIN) is a potentially reversible cause of acute kidney injury (AKI). However, when presenting with rapid loss of renal function and fever of unknown origin, ATIN frequently mimics rapidly progressive glomerulonephritis (RPGN). In Kazakhstan, patients presenting with severe renal dysfunction (eGFR <10 mL/min/1.73m²) are frequently denied or delayed from undergoing diagnostic kidney biopsy due to institutional safety concerns, procedural hesitation, and a regional deficit in subspecialized nephropathology services. This abstract demonstrates the critical diagnostic value of renal biopsy in advanced AKI with clinicopathological dissociation and highlights systemic diagnostic barriers in Central Asia.

Case Presentation: A 41-year-old female with no prior history of kidney disease presented in June 2026 with an unexplained persistent fever (37.0–38.0°C). Despite empirical antibacterial therapy ('ex juvantibus'), her fever persisted. Over 8 weeks, serial laboratory testing revealed a dramatic, rapidly progressive decline in kidney function (Table 1): serum creatinine escalated from a baseline of 70 µmol/L (eGFR 96 mL/min/1.73m²) in June 2026 to 130 µmol/L (09.07.2026), 381 µmol/L (31.07.2026), 441 µmol/L (06.08.2026), and peaked at 524 µmol/L (20.08.2026, eGFR 8.6 mL/min/1.73m²). Remarkably, diuresis remained fully preserved, and urinary abnormalities were strikingly mild (proteinuria 0.13–0.60 g/L, absence of active urinary sediment/hematuria). Immunological evaluation showed positive immunoblot reactivity to SS-B, RP11, and Mi-2α; however, classic systemic connective tissue disease criteria were not met, and ANCA, anti-GBM, and C3/C4 levels were unremarkable. Due to severe renal failure (eGFR <10 mL/min), local clinical evaluation in Kazakhstan hesitated to perform a kidney biopsy, categorizing the condition as RPGN vs. ESRD (CKD Stage 5). The patient subsequently traveled to Turkiye (Istanbul) for an urgent ultrasound-guided renal biopsy on August 20, 2026. Light microscopy (22 glomeruli) revealed intact glomeruli without crescents or necrosis, but severe active interstitial inflammation (mononuclear and neutrophilic infiltrate, tubulitis, leukocyte casts) and acute tubular injury, alongside early chronic tubulointerstitial changes. Direct immunofluorescence was negative. Immediate high-dose corticosteroid therapy (IV Methylprednisolone pulse 80 mg followed by oral Prednisolone 32 mg/day with gradual taper) was initiated. Renal function responded dramatically: serum creatinine dropped from 524 µmol/L to 294.1 µmol/L within 7 days (27.08.2026), 218.9 µmol/L (02.09.2026), and reached 148.2 µmol/L (10.09.2026, eGFR 39.0 mL/min/1.73m²), with complete normalization of inflammatory markers (CRP 1.1 mg/L). Hemodialysis was completely avoided.

Conclusions & Policy Implications: Discrepancy between profound renal failure and mild urinary sediment (clinicopathological dissociation) strongly points toward acute tubulointerstitial disease rather than RPGN. Severe reduction in eGFR should not be considered an absolute contraindication to diagnostic kidney biopsy. Without biopsy, this patient would likely have been mislabeled as end-stage renal disease (ESRD) and initiated on lifelong maintenance dialysis. There is an urgent health policy imperative in Kazakhstan to modernize nephrobiology protocols, invest in state-of-the-art biopsy technology, and establish dedicated nephropathology training to prevent irreversible progression of treatable renal diseases.

Keywords: Acute Tubulointerstitial Nephritis (ATIN); Rapidly Progressive AKI; Kidney Biopsy; Low eGFR; Nephropathology; Kazakhstan Healthcare Policy.

Table 1: Serial changes in serum creatinine, eGFR, proteinuria, and key diagnostic/therapeutic milestones from baseline to post-treatment follow-up.

Date	Serum Creatinine (μmol/L)	eGFR (mL/min/1.73m ²)	Proteinuria (g/L)	Clinical Milestone / Event
June 2026	70.0	96.0	Not detected	Preserved baseline kidney function; persistent fever of unknown etiology (37-38°C).
09.07.2026	130.0	45.7	n/a	Onset of rapid kidney function decline; empirical antibiotics prescribed.
13.07.2026	167.0	33.8	n/a	Progression of AKI; initial nephrology consultation.
20.07.2026	206.0	26.3	n/a	Immunoblot positive for SS-B, RP11, Mi-2α. Suspected connective tissue disease / RPGN.
23.07.2026	334.0	14.7	0.148 g/L	Severe AKI progression; urinary sediment remains remarkably benign.
31.07.2026	381.0	12.6	0.033 g/L	Nephrology consult (Astana): AKI of unknown origin vs RPGN; biopsy delayed due to low eGFR.
06.08.2026	441.0	10.6	0.148 g/L	Profound renal deterioration in the absence of nephrotic syndrome or severe hematuria.
17.08.2026	479.6	9.5	0.552 g/d	UMC Consult: eGFR <10. Recommended urgent renal biopsy & immunomodulation.
20.08.2026	524.0	8.6	0.168 g/L	Urgent Biopsy at Univ. Hosp. (Istanbul): Severe active ATIN, acute tubular injury, early chronicity. High-dose IV Methylprednisolone (80mg) started.
27.08.2026	294.1	17.2	0.330 g/L	7 days post-steroid: Marked 44% reduction in serum creatinine. Tapering oral Prednisolone (32mg/d).
10.09.2026	148.2	39.0	0.132 g/L	Dramatic recovery of kidney function (eGFR 39); CRP normal (1.1 mg/L); dialysis completely avoided.