

Collapsing Focal Segmental Glomerulosclerosis Progression to ESRD Following Combination Therapy with Pembrolizumab and Enfortumab Vedotin: A Case Report

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Case Report

Keywords: Enfortumab vedotin, Pembrolizumab, Collapsing focal segmental glomerulosclerosis, Antibody-drug conjugate, End-stage renal disease

Posted Date: October 11th, 2026

DOI: <https://doi.org/10.21203/rs.3.rs-10936687/v1>

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Additional Declarations: No competing interests reported.

Abstract

Introduction:

Pembrolizumab (PB), a PD-1 immune checkpoint inhibitor, and enfortumab vedotin (EV), a Nectin-4 targeting antibody-drug conjugate delivering the microtubule-disrupting payload MMAE, represent the current standard of care for advanced urothelial carcinoma. While PB-associated glomerulopathy is a recognized rare complication, rapid one-month progression to end-stage renal disease (ESRD) following the EV-PB combination has not been previously described.

Case Presentation:

A 76-year-old male with high-grade urothelial carcinoma and baseline Stage 4 CKD (eGFR 25 mL/min/1.73 m², creatinine 2.5 mg/dL) secondary to prior cisplatin exposure was initiated on PB 200 mg/IV and EV 1 mg/kg IV on a 21-day cycle. Within one month, he developed AKI presenting with a creatinine of 3.1 mg/dL, eGFR of 20 mL/min/1.73 m², and nephrotic-range proteinuria of 13.1 g/24 hours. Renal biopsy confirmed cFSGS with 53% global glomerulosclerosis and 70% cortical fibrosis. Both agents were discontinued, and rituximab was initiated; however, renal function still deteriorated. Six months later he presented with labs of: creatinine 8.4 mg/dL, eGFR 6 mL/min/1.73 m²; requiring emergent hemodialysis. Renal function did not recover, and the patient progressed to permanent ESRD.

Discussion/Conclusion:

This case highlights three key teaching points. First, pre-existing CKD from prior nephrotoxic chemotherapy amplifies the risk of irreversible cFSGS progression following ADC-immunotherapy, underscoring the need for baseline nephrology evaluation in this population. Second, we propose a novel synergistic mechanism whereby PB-induced podocyte dedifferentiation and cell-cycle re-entry renders those podocytes selectively vulnerable to MMAE-mediated cytotoxicity which can potentially explain the unusually rapid deterioration observed. Third, once advanced interstitial fibrosis is established, immunosuppressive salvage is unlikely to alter the trajectory, and its toxicity must be weighed against a negligible probability of benefit. Notably, PB-induced cFSGS typically remits within 2–3 months of drug discontinuation; however, the literature on dual therapy with EV remains limited.

Introduction

Immune checkpoint inhibitors (ICIs) have reshaped the treatment of advanced solid tumors. Tumors are marked as foreign by the cell-surface neoantigens their unregulated proliferation generates, but that same rapid turnover lets them adapt to the selective pressure that the immune system subjects onto them [1, 2].

The best characterized of this adaptation is the programmed cell death protein 1 (PD-1)/programmed death-ligand 1 (PD-L1) axis: engagement of PD-1, an inhibitory receptor on activated T lymphocytes, by PD-L1 dampens cytotoxic T-cell activity and protects healthy tissue from immune-mediated injury [2, 3]. Because cells expressing PD-L1 preferentially survive T-cell-mediated lysis, selective pressure enriches it across the tumor until the tumor is read as self [1].

In advanced urothelial carcinoma, pembrolizumab is now given first-line with enfortumab vedotin, an antibody-drug conjugate (ADC) directed against Nectin-4, a cell adhesion molecule overexpressed on urothelial carcinoma. Once internalized, it releases monomethyl auristatin E (MMAE), a microtubule inhibitor that prevents tubulin polymerization and drives cell-cycle arrest and apoptosis [4, 5, 6]. The two agents are complementary: enfortumab vedotin induces immunogenic tumor cell death and releases tumor antigen, while pembrolizumab lifts the PD-1 brake on the T cells that respond [7, 8].

Podocyte loss of this kind produces a podocytopathy, most often focal segmental glomerulosclerosis (FSGS). Its collapsing variant (cFSGS) carries one of the worst prognoses in glomerular disease, progressing to end-stage renal disease (ESRD) in 71% of patients compared with 20% for other FSGS variants [9]. Reported ICI-associated glomerular disease has nonetheless largely remitted after withdrawal, in patients with preserved renal function on checkpoint blockade alone [10]. Whether that holds in established chronic kidney disease (CKD) with concurrent ADC therapy is unknown: renal safety data for this combination in advanced CKD are limited to one retrospective cohort reporting comparable survival without graded renal toxicity.

We describe a patient with biopsy-proven cFSGS that progressed to dialysis-dependent ESRD after a single month of enfortumab vedotin plus pembrolizumab, with a prior history of stage 3 chronic kidney disease (CKD) caused by cisplatin and gemcitabine. Two features separate this case from the published literature: the speed of onset and the complete absence of recovery after drug withdrawal.

Case Presentation

A 76-year-old white male with a history of high-grade papillary urothelial carcinoma and malignant neoplasm of the urinary bladder, originally diagnosed in February 2020, presented to the nephrology clinic in October 2024 for evaluation of suspected cancer recurrence. The patient had undergone urethrectomy one year prior for a cancerous growth, but a follow-up PET/CT and MRI urogram revealed an enlarging, enhancing lesion at the base of the penis with nodular enhancing focus that had developed rapidly over a two-month interval, raising suspicion for recurrence. Comorbidities included Type 2 Diabetes Mellitus, Hypertension, Hepatic Cirrhosis (related to a history of heavy alcohol use during his thirties and forties, with a 50 pack-year smoking history), and Hypothyroidism.

Because the patient was not a surgical candidate, combination systemic therapy was initiated with pembrolizumab 200 mg IV every 21 days and enfortumab vedotin 1 mg/kg IV every 21-day cycle. The patient received his first dose of pembrolizumab and enfortumab on day 1 of prescription and another dose of enfortumab on day 8. Baseline laboratory values prior to the first infusion included: WBC 3.9 x

$10^3/\mu\text{L}$, RBC $2.91 \times 10^6/\mu\text{L}$, Hgb 10.3 g/dL, Hct 30%, platelets $211 \times 10^3/\mu\text{L}$, BUN 54 mg/dL, creatinine 2.5 mg/dL, eGFR 25 mL/min/ 1.73m^2 , and a 24-hour urine protein of 2.1 g/24 consistent with Stage 3 CKD at baseline. Pertinent vitals included a BP of 171/89 mmHg and HR of 62 bpm (Table 1, Fig. 2). On physical examination, the patient had an ECOG performance status of 2, pain score of 0, and was alert and oriented to person, place, and time. Upon physical examination, there were no notable signs of edema, no lymphadenopathy, and the cardiac, pulmonary, and abdominal exams were insignificant.

Within one month of initiating combination therapy, the patient experienced a precipitous decline in renal function and was diagnosed with acute kidney injury (AKI) superimposed on his baseline CKD. Repeat labs at that time revealed: BUN 68 mg/dL, creatinine 3.1 mg/dL, eGFR 20 mL/min/ 1.73m^2 , WBC $4.4 \times 10^3/\mu\text{L}$, RBC $2.85 \times 10^6/\mu\text{L}$, Hgb 10 g/dL, Hct 29.8%, platelets $152 \times 10^3/\mu\text{L}$, and a 24-hour urine protein of 13.1 g/24h consistent with nephrotic-range proteinuria (Table 1, Fig. 2). All physical exam findings remained unchanged from baseline except 1 + edema was noted bilaterally. Both pembrolizumab and enfortumab vedotin were discontinued, and a high-dose prednisone taper was initiated (100 mg daily for one week, followed by 80 mg, 60 mg, 40 mg, and 20 mg over subsequent weeks) while awaiting biopsy. A renal ultrasound performed two weeks later demonstrated increased renal echogenicity with parenchymal volume loss compatible with chronic kidney disease, no hydronephrosis, and bilateral renal cysts. A subsequent renal biopsy revealed glomerular injury with collapsing features in 20% of glomeruli, 53% diffuse global glomerulosclerosis, extensive interstitial fibrosis and tubular atrophy involving 70% of the cortex, moderate arteriosclerosis, and severe arteriolar hyalinosis. These findings led to the diagnosis of cFSGS superimposed on chronic cisplatin-induced damage (Fig. 1).

3 months following the biopsy, the patient was initiated on rituximab at 1000 mg every 2 weeks in an attempt to arrest the immune-mediated glomerular injury and nephritis. Despite treatment, his renal function continued to deteriorate over the subsequent months without meaningful recovery. Repeat labs demonstrated: BUN 75 mg/dL, creatinine 3.9 mg/dL, eGFR 15 mL/min/ 1.73m^2 consistent with progression to Stage 4–5 CKD, albumin 3.5 g/dL, alkaline phosphatase 127 U/L, AST 12 U/L, ALT 23 U/L, WBC $5.4 \times 10^3/\mu\text{L}$, RBC $2.68 \times 10^6/\mu\text{L}$, Hct 24.1%, and platelets $164 \times 10^3/\mu\text{L}$ (Table 1, Fig. 2). A significant clinical dilemma arose regarding escalation to another prescription of high-dose corticosteroids (prednisone taper starting at 80 mg and incrementally decreasing by 20 mg every week), as the patient's comorbid Type 2 Diabetes and alcohol-related hepatic cirrhosis made the risk of glycemic instability and severe infection prohibitive relative to the low probability of reversing such extensive fibrosis. Due to this risk and the lack of clinical recovery during prior administration, prednisone was not readministered, and rituximab was discontinued.

Approximately six months after the initial diagnosis of cFSGS, the patient presented to a follow-up appointment in a clinically decompensated state, reporting profound fatigue, generalized weakness, and "pink-colored" urine. He was emergently referred to the emergency department, where he was admitted with HR 130 BPM, BP 91/62 mmHg, SpO_2 100%, and RR 19 respirations/min. On exam he appeared ill

with pale skin, although he was not diaphoretic. It was noted that he was not experiencing acute respiratory distress and presented with normal breath sounds with no chest pain. A small amount of blood was noted in his urostomy bag; he denied hemoptysis. Bilateral 2 + lower extremity edema was noted (was noted on physical assessments from nephrologist and oncologist check-ups starting from two months prior to ED visit). Admission laboratory values revealed: RBC $2.06 \times 10^6/\mu\text{L}$, Hgb $6.5 \times 10^3 \text{ g/dL}$, Hct 19.3%, BUN 220 mg/dL, creatinine 8.4 mg/dL, eGFR 6 mL/min/ 1.73m^2 , sodium 134 mEq/L, chloride 86 mEq/L, potassium 4.4 mEq/L, and calcium 7.8 mg/dL (Table 1, Fig. 2). Urinalysis demonstrated significant nephrotic-range proteinuria and tests were negative for GI bleeding and hypovolemia.

Table 1

Laboratory findings mapping the patient’s gradual renal decline as time progressed after his first administration of the combination therapy. The key timestamps used were the values taken prior to administration (baseline), the first AKI diagnosis (one month after starting the therapy), once the biopsy gave the cFSGS diagnosis (6 months later), and finally the ED admission (1 year later).

Lab Test	Baseline (pre-infusion)	AKI diagnosis (~ 1 month later)	Post-biopsy/rituximab (~ 6 months later)	ED Admission (~ 1 year later)
RBC	$2.91 \times 10^6/\mu\text{L}$	$2.85 \times 10^6/\mu\text{L}$	$2.68 \times 10^6/\mu\text{L}$	$2.06 \times 10^6/\mu\text{L}$
Hct	30.0%	29.8%	24.1%	19.3%
Creatinine	2.5 mg/dL	3.1 mg/dL	3.9 mg/dL	8.4 mg/dL
eGFR	25 mL/min/ 1.73m^2	20 mL/min/ 1.73m^2	15 mL/min/ 1.73m^2	6 mL/min/ 1.73m^2
WBC	$3.9 \times 10^3/\mu\text{L}$	$4.4 \times 10^3/\mu\text{L}$	$5.4 \times 10^3/\mu\text{L}$	N/A
Platelets	$211 \times 10^3/\mu\text{L}$	$152 \times 10^3/\mu\text{L}$	$164 \times 10^3/\mu\text{L}$	N/A
Hgb	10.3 g/dL	10 g/dL	N/A	6.5 g/dL
BUN	54 mg/dL	68 mg/dL	75 mg/dL	220 mg/dL

The patient was also found to have acute esophageal necrosis of unclear etiology. He had no hemoptysis, but upper endoscopy revealed diffuse, severe mucosal changes characterized by sloughing and congestion involving the entire esophagus. Hemodialysis was initiated emergently via a temporary catheter. Although the acute uremic symptoms of esophageal necrosis and anemia resolved with dialysis, renal function did not recover. The patient progressed to permanent ESRD and remains dependent on chronic hemodialysis. Repeat MRI urogram demonstrated no active cancer-related lesions, and all cancer-directed therapy has been discontinued. The patient is scheduled for a repeat MRI urogram in approximately four months (from the submission date of this paper) for restaging.

Discussion

We report a 76-year-old man with a history of stage 3 CKD from prior cisplatin and gemcitabine exposure who developed biopsy-proven cFSGS within one month of initiating enfortumab vedotin and pembrolizumab for recurrent urothelial carcinoma. In contrast to previously published cases, discontinuation of both agents did not produce remission: renal function declined progressively despite corticosteroids and rituximab, and the patient progressed to ESRD requiring permanent hemodialysis. We describe this course, propose a sequential mechanism by which checkpoint-induced podocyte dedifferentiation may render podocytes susceptible to MMAE, and consider the implications for renal surveillance in patients with reduced renal reserve who are candidates for ADC-immunotherapy combinations.

To our knowledge, this rapid deterioration of renal function following combination pembrolizumab and enfortumab vedotin therapy has not been previously described in the literature. Reported cases of just pembrolizumab-induced FSGS described a slower onset over the course of a one-year treatment cycle with full recovery within approximately two months of drug discontinuation [11]. However, a similar rapid-onset of drug-induced cFSGS has been reported with two other agents: ado-trastuzumab emtansine (T-DM1) and zoledronate. The T-DM1 case is mechanistically informative, as T-DM1 is also an antibody-drug conjugate that delivers a microtubule-disrupting payload closely analogous to MMAE in enfortumab vedotin. The patient in that case-study developed nephrotic-range proteinuria and biopsy-proven cFSGS within weeks of initiating therapy. Although those authors could not adequately conclude that the cFSGS was directly attributed to the usage of T-DM1, it is important to note that the patient did enter remission back to baseline kidney function three weeks after drug discontinuation [12]. The zoledronate case demonstrated that even a single dose of a triggering agent could produce cFSGS. Zoledronate is a bisphosphonate whose mechanism of inducing podocyte dedifferentiation and hyperplasia, hallmarks of cFSGS, are still unknown [13]. Together these reports suggest that microtubule-disrupting conjugates and immune-modulating agents can accelerate podocyte injury, particularly where renal reserve is already reduced (Table 2).

Table 2

Summary comparing other case studies where patients experienced some form of similar outcome, signs/symptoms, or presentations as the patient depicted in this paper by the authors. The similarities were highlighted either the targeted effect of the drugs due to other studies highlighting the same drug, drugs with similar mechanisms of action, or entirely different targets. Additionally, the onset of cFSGS and the remission pattern were also major points of comparison used to demonstrate similarities between the different cases.

Case/Agent	Targeted effect of drug	Onset of cFSGS	Outcome after discontinuation	Notable features
Pembrolizumab + enfortumab vedotin (this case)	- Pembrolizumab: anti PD-1 antibody - Enfortumab Vedotin: Antibody-drug conjugate microtubule disrupting payload	Rapid: within 1 month of starting combination therapy	No recovery; progressive decline to ESRD-range CKD	Not previously described in prior literature per authors
Pembrolizumab alone (prior report) [11]	anti-PD-1 antibody	Slower: over course of 1 year of treatment	Fully recovery within about 2 months of discontinuation	N/A
Ado-trastuzumab emtansine (T-DM1) [12]	Microtubule disrupting payload which binds to Her2 receptors	Rapid: within weeks of initiating therapy	Remission to baseline kidney function 3 weeks after discontinuation	Antibody-drug conjugate delivering microtubule-disrupting payload analogous to MMAE (enfortumab's payload mechanism). Causal link between the drug and patient outcome was not definitively established
Zoledronate [13]	Bisphosphonate: kills osteoclasts and prevent bone degradation	Rapid: after a single dose	Not specified	Mechanism of podocyte injury unknown; patient had prior renal dysfunction (mild proteinuria from a prior medication)

This patient's renal architecture was compromised before the first infusion. Generally, cisplatin has been linked to acute tubular necrosis and apoptosis in proximal tubules through activation of inflammatory cytokines [14]. Because of its low molecular weight, unbound cisplatin is freely filtered by the glomerulus and accumulates within proximal tubular cells at approximately five times the serum concentration [15]. In a cohort of 198 patients receiving cisplatin-based regimens, 8.1% discontinued treatment specifically for renal toxicity [16]. Reduced renal function also narrows what can be given next. Among patients with

urothelial carcinoma, an objective response to first-line gemcitabine-carboplatin was achieved in 12.5% of those with a GFR below 30 mL/min compared with 56.7% of those with a GFR of 30 to 60 mL/min, and the median number of tolerated cycles fell from 6 to 2 [17].

Evidence on this combination in advanced CKD rests on a single retrospective cohort. Among 462 patients starting first-line enfortumab vedotin plus pembrolizumab, the 65 (14.1%) with a creatinine clearance below 30 mL/min had overall survival (adjusted hazard ratio, 0.95; 95% CI, 0.61–1.49) and progression-free survival (adjusted hazard ratio, 0.70; 95% CI, 0.47–1.06) comparable to those with better renal function [18]. That reassurance has limits directly relevant to this case. The severe-dysfunction group was small, renal function was defined by a single creatinine clearance threshold rather than by CKD stage or degree of proteinuria, and the analysis reported survival rather than graded treatment-emergent toxicity. Whether a patient with an eGFR of 25 mL/min/1.73 m² and established structural injury shares that risk profile is unresolved.

We propose a sequential mechanism for the accelerated injury observed in this patient presentation. MMAE preferentially kills actively dividing cells while sparing mitotically quiescent ones, so mature podocytes should be relatively insensitive to it. Pembrolizumab changes that condition. Blocking PD-1 withdraws the inhibitory signal that protects podocytes from T-cell-mediated attack, and the injured podocyte responds by dedifferentiating, downregulating synaptopodin and WT-1, and re-entering the cell cycle [19]. A cell forced back into cycle is precisely the target MMAE is designed to kill. In this model pembrolizumab does not simply add to the toxicity of enfortumab vedotin but creates the substrate for it, converting a payload that should be renally inert into one capable of eliminating podocytes that cannot be replaced.

The observed failure to salvage renal function with rituximab further underscores the irreversible nature of cFSGS when it occurs in a previously damaged kidney. Rituximab targets CD20-positive B-cells and is often employed to dampen the humoral immune response in glomerular diseases [20]. However, its efficacy is limited in cases driven primarily by T-cell-mediated cytotoxicity or where global glomerulosclerosis is already widespread. In this patient, biopsy confirmed that 53% of glomeruli were already globally sclerosed, suggesting that structural damage had surpassed the point of functional recovery. The decision to forgo high-dose corticosteroid therapy reflected a similar consideration of futility versus toxicity. Although remission rates with corticosteroids in idiopathic FSGS range from 31–74% depending on histologic variant and renal function, the response rate in cFSGS specifically is markedly lower with one prospective study reporting complete remission in only 13.6% and partial remission in 4.5% with the remainder demonstrating steroid resistance, progression to ESRD, or death [21, 22]. KDIGO 2021 restricts immunosuppression to primary FSGS and advises against it in secondary FSGS and FSGS of undetermined cause; a collapsing lesion should prompt a search for a secondary or APOL1-related cause [23]. Given this patient's biopsy demonstrating 70% tubular atrophy and 53% global glomerulosclerosis, combined with comorbid Type 2 Diabetes and hepatic cirrhosis, the risk of glycemic instability and infection was deemed to outweigh the negligible probability of reversing such extensive fibrosis with corticosteroid administration [24].

The downstream consequences of this patient's renal failure manifested as the systemic ischemic complications that ultimately precipitated his emergent admission. Acute esophageal necrosis is a rare condition with mucosal ischemia serving as the primary driver [25]. In the setting of ESRD, hemodynamic compromise and low-flow states reduce mucosal perfusion, rendering the esophagus vulnerable to ischemic injury—a risk further compounded in this patient by Type 2 Diabetes, Hypertension, and Hepatic Cirrhosis [25, 26]. Similarly, his severe anemia was likely a dual consequence of uremic bone marrow suppression and the loss of erythropoietin-producing cells within the 70% of the renal cortex that had become fibrotic [27, 28]. Once renal reserve is exhausted by prior injury, the aggressive nature of cFSGS can rapidly overwhelm the remaining nephrons, leading to permanent reliance on hemodialysis. Among patients who progress from FSGS to ESRD, in-center hemodialysis is the most common initial renal replacement therapy, utilized in approximately 72.1% of cases [29].

Several limitations qualify these conclusions. Causality cannot be established from a single case: both agents were started and withdrawn together, rechallenge was neither performed nor appropriate, and MMAE concentrations were not measured. Furthermore, the proposed pembrolizumab–MMAE interaction is a hypothesis generated by this case and requires experimental confirmation. Collapsing lesions involved only 20% of glomeruli, against a background of 53% global glomerulosclerosis, 70% interstitial fibrosis and tubular atrophy, and severe arteriolar hyalinosis. Diabetic and hypertensive nephrosclerosis, together with prior platinum exposure, therefore account for much of this chronic damage. As a result, the combination's contribution could have plausibly been simply an acute insult to a kidney with no reserve to absorb one.

Conclusion

In this patient, combination pembrolizumab and enfortumab vedotin was followed within one month by biopsy-proven cFSGS and progression to permanent ESRD. Three points follow. First, although an immune checkpoint inhibitor–associated cFSGS is rare, patients with pre-existing CKD or prior nephrotoxic chemotherapy may face a substantially higher risk of accelerated, irreversible progression than the published literature in healthier populations suggests. Second, the proposed synergy between pembrolizumab-induced podocyte dedifferentiation and MMAE-mediated cytotoxicity offers a mechanistic explanation for why this combination may be particularly hazardous to a compromised kidney, and warrants investigation as ADC–immunotherapy regimens are more widely adopted. Third, once cFSGS develops alongside extensive interstitial fibrosis, salvage with corticosteroids or rituximab is unlikely to alter the outcome, and their toxicity must be weighed against a negligible probability of benefit.

For patients with recurrent urothelial carcinoma in whom surgery is not feasible, baseline renal function and prior nephrotoxic exposure should therefore be treated as critical variables in treatment selection, with a low threshold for nephrology referral before an ADC–immunotherapy combination is started. Where such therapy is unavoidable, surveillance of creatinine, eGFR, urinalysis, and the urine protein-to-creatinine ratio may detect glomerular injury early enough to intervene before irreversible nephron loss.

Prospective renal safety data for the enfortumab vedotin–pembrolizumab combination in patients with advanced CKD are needed.

Declarations

Ethics Approval and Consent to Participate:

Our institution does not require ethical approval for reporting individual cases.

Consent to Participate:

Written information consent was obtained from the patient to participate in this case report.

Consent for Publication:

Written information consent was obtained from the patient for their anonymized information to be published in this article.

Competing Interests:

The authors declare that they have no known competing financial interests or personal relationships that could have appeared to influence the work reported in this paper.

Funding:

This research received no specific grant from any funding agency in the public, commercial, or not-for-profit sectors. Article processing charges were covered by the Division of Nephrology, University of California, Irvine, which had no role in the design, conduct, analysis, or reporting of the study.

Author Contribution

(Corresponding Author) George Hitti – Primary/Corresponding author who wrote the main text of the manuscript along with synthesizing, interpreting, and cataloging the patient data that was noted in the patient’s chart throughout the course of his clinical presentation. Author also prepared Figure 2 along with tables 1 and 2. Caroline Gee – Helped edit and review the manuscript to ensure that all the pertinent information relevant to the case was reported. Johnathn Zuckerman – Helped produce, interpret, and analyze the renal biopsy images that are present in figure 1. Ramy Hanna - Helped edit and review the manuscript to ensure that all the pertinent information relevant to the case was reported.

Acknowledgements:

Not applicable

Data Availability

Data sharing is not applicable to this manuscript as no datasets were generated or analyzed during the current study since only a single patient case was looked at. The information regarding this patient was stored on EPIC and is not available to anyone outside of the patient's care team.

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Figures

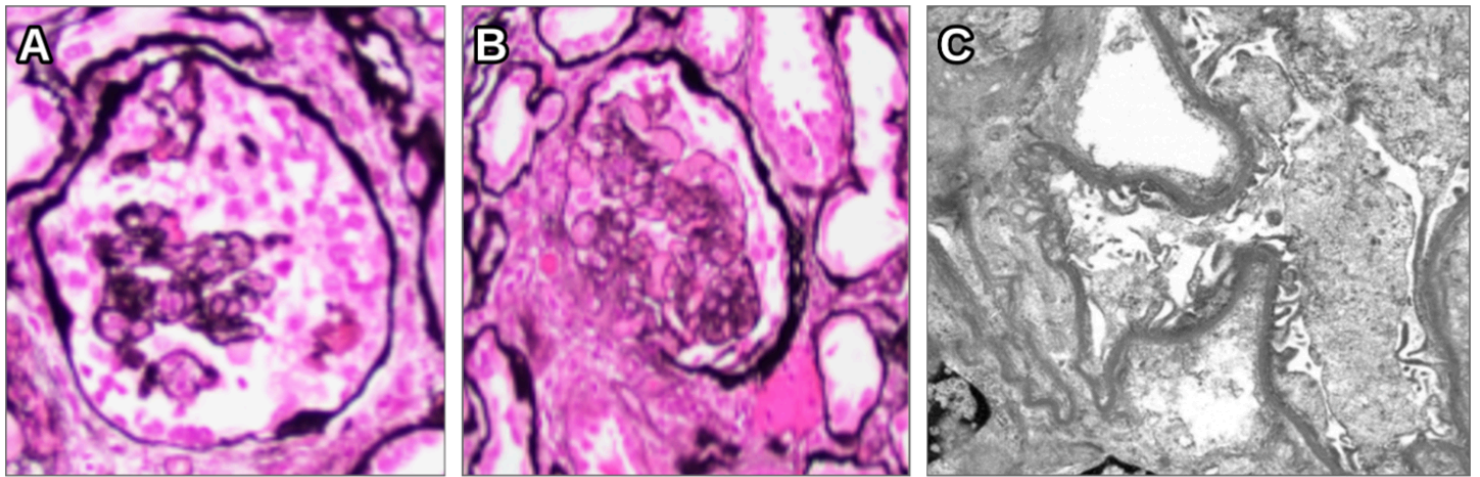


Figure 1

Kidney biopsy demonstrates glomeruli with collapsing glomerulopathy. **(A)** Active appearing collapsing lesion represented by epithelial cell hyperplasia forming a pseudocrescent and underlying implosion of capillary loops (Jones silver stain, original magnification 400x). **(B)** Chronic appearing collapsing lesions represented by sclerotic collapsed glomerular capillary loops with less exuberant epithelial cell hyperplasia (Jones silver stain, original magnification 400x). **(C)** Electron micrograph demonstrating segmental podocyte foot process effacement (EM tissue from re-processed paraffin block; original magnification 4300x).

Longitudinal Renal Function and Therapeutic Interventions

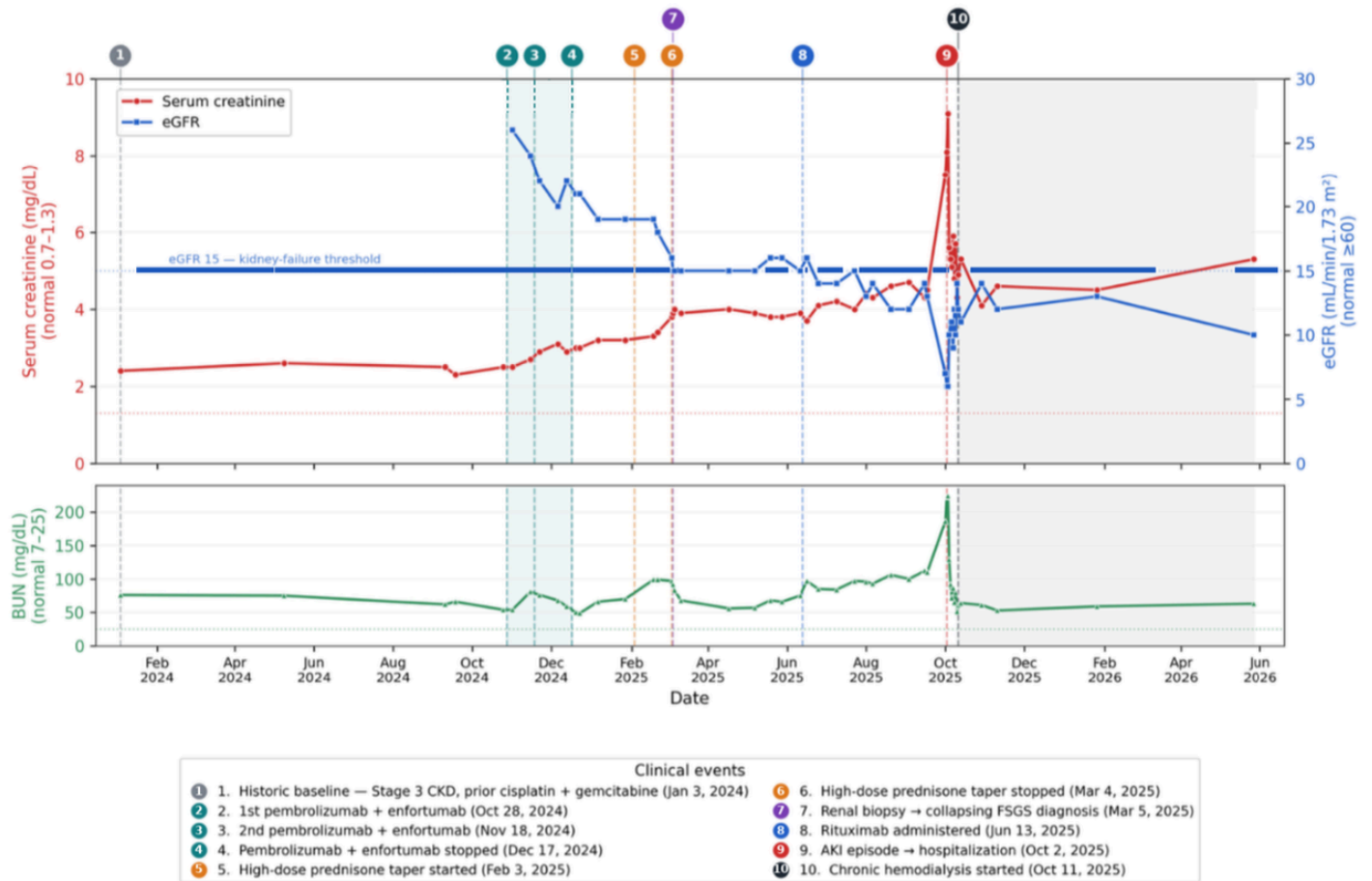


Figure 2

Kidney function markers (eGFR, serum creatinine, and BUN) mapped against the timing of treatment milestones and adverse events over the patient's clinical course. The key clinical events that were used as timestamps were the (1-3) the combination therapy administration, (5-6) prednisone taper, (7) Renal Biopsy, (8) Rituximab administration, (9-10) AKI which progressed to hemodialysis.