

Apatinib-Associated Nephrotic Syndrome with Membranoproliferative Glomerulonephritis and Post-Biopsy Renal Hemorrhage: A Case Report and Literature Review

Weidong Huang#

the Fifth Affiliated Hospital of Wenzhou Medical University

Yiling Zhu#

the Fifth Affiliated Hospital of Wenzhou Medical University

Tingyan Xiang

Liandu District Baiyun Street Community Health Service Center, Lishui, Zhejiang, China

Qiankun Zhang

the Fifth Affiliated Hospital of Wenzhou Medical University

Mingfeng Mao

the Fifth Affiliated Hospital of Wenzhou Medical University

Lingfeng Huang

sch1f@163.com

Suichang County People's Hospital, Suichang County

Case Report

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Abstract

Background: Anti-VEGF therapies, including apatinib, are widely used in oncology and have been associated with renal adverse events such as proteinuria and nephrotic syndrome. However, reports of nephrotic syndrome directly attributed to apatinib remain rare, and evidence to guide clinical management and peri-procedural decision-making is still limited.

Case Presentation: We describe a 60-year-old man with hepatocellular carcinoma treated with apatinib who developed nephrotic-range proteinuria. After a prospective diagnostic workup, apatinib was discontinued. The patient subsequently underwent a renal biopsy three days after cessation of apatinib, which revealed membranoproliferative glomerulonephritis (MPGN). The patient experienced remission of nephrotic syndrome following drug discontinuation. Notably, post-biopsy renal hemorrhage occurred, requiring emergency renal embolization.

Conclusions: Nephrotic syndrome is a rare adverse event of apatinib. Clinicians should intensify renal monitoring during cancer treatment, perform individualized risk–benefit assessments if renal injury occurs, and adjust therapy accordingly. If renal biopsy is required, apatinib should be discontinued and meticulous peri-procedural monitoring implemented. Further research is needed to establish evidence-based guidance and optimized strategies to maximize patient outcomes.

Introduction

Vascular endothelial growth factor (VEGF) inhibitors exert therapeutic effects by suppressing VEGF-driven angiogenesis and are widely used in ophthalmology and oncology. Their primary mechanism involves inhibition of VEGF-A signaling through VEGF receptors (VEGFRs)(1). These agents include monoclonal antibodies, antibody fragments, fusion proteins, and small-molecule tyrosine kinase inhibitors (TKIs). Proteinuria is a recognized class effect of VEGF inhibitors(2, 3). Apatinib, a novel small-molecule VEGFR-2 TKI developed in China and approved in October 2014, competitively inhibits ATP binding to VEGFR-2, thereby blocking downstream signaling and impairing endothelial cell migration and proliferation, resulting in potent anti-angiogenic and antitumor activity(4, 5). Well-documented adverse events include hypertension, proteinuria, hand–foot syndrome, leukopenia, and diarrhea(6, 7). Reports of TKI-associated nephrotic syndrome remain rare. We present a case of an elderly male who developed nephrotic syndrome during apatinib therapy and experienced renal bleeding following renal biopsy. This case aims to (i) inform clinical safety considerations in the use of the TKI apatinib and (ii) discuss the appropriate timing of renal biopsy when nephrotic syndrome occurs during apatinib therapy.

Case Presentation

A 60-year-old man was diagnosed with poorly differentiated, giant hepatocellular carcinoma (HCC) in 2017 and underwent a right hepatectomy at an external center, with unremarkable postoperative follow-up. In July 2020, he presented with abdominal distension. Contrast-enhanced CT revealed multiple intra-abdominal and pelvic metastases. Following disease progression, the patient initiated treatment with camrelizumab monotherapy. However, due to significant treatment-related fatigue, therapy was discontinued after two cycles. The switch transition to the vascular endothelial growth factor receptor (VEGFR)-targeted tyrosine kinase inhibitor (TKI) apatinib in August 2020 was guided by the patient's socioeconomic constraints—limiting access to standard first-line combination regimens—and intolerance to prior immunotherapy. The use of apatinib in this setting aligns with guideline recommendations supporting VEGFR-TKIs in advanced HCC(8).

Starting in August 2020, he received TKI apatinib (500 mg/day), a vascular endothelial growth factor receptor 2 (VEGFR-2) TKI, with dose reduction to 250 mg/day after one year; this therapy has continued. In addition to the aforementioned anti-tumor therapy, the patient did not take other medications.

From September 2020 until the current hospitalization, routine laboratory monitoring consistently showed trace to moderate proteinuria (1 + to 2+), with serum creatinine levels ranging from 78 to 99 $\mu\text{mol/L}$ and an estimated glomerular filtration rate (eGFR) between 65 and 76 mL/min/1.73m^2 . No peripheral edema was documented during this period. Persistent proteinuria did not lead to a formal nephrology consultation or specific intervention. On February 6, 2025, he was admitted with facial and lower-extremity edema lasting approximately one month, accompanied by a 5 kg weight gain. His past medical history included chronic hepatitis B virus (HBV) infection diagnosed 18 years earlier, for which he had been receiving entecavir 0.5 mg daily for the past eight years. During this period, regular monitoring of HBV DNA consistently showed undetectable viral loads, indicating sustained virological suppression. He also had a history of hypertension, managed with amlodipine 5 mg/day and valsartan 80 mg/day, with adequate blood pressure control. He reported no prior use of antiplatelet agents, anticoagulants, or lipid-lowering medications.

Height: 1.60 m; Weight: 67 kg; BMI: 26.2 kg/m^2 . On examination, marked edema of the face and lower extremities was observed; other findings were unremarkable. Laboratory investigations revealed nephrotic-range proteinuria (Grade 3 according to CTCAE v5.0) accompanied by hypoalbuminemia. Renal function remained preserved, and no hematuria was present (Table 1). Nephrotic syndrome was diagnosed based on the triad of nephrotic-range proteinuria (24hUP 4.99 g/day), hypoalbuminemia (28.8 g/L), and peripheral edema, along with hyperlipidemia (TC 7.63 mmol/L). Investigations including HBV DNA, tumor markers, antinuclear antibodies, ANCA, anti-GBM antibodies, serum immunofixation, echocardiography, abdominal ultrasound, and chest CT did not identify alternative etiologies. Given the temporal association between initiation of TKI apatinib and proteinuria, and the absence of other explanatory causes, TKI-related nephrotoxicity leading to nephrotic syndrome was considered, although glomerulonephritis due to other etiologies could not be entirely excluded.

Table 1
Laboratory findings on admission

The laboratory parameters	Results(Reference Range)
WBC	4.0×10 ⁹ /L(4.0–10)
HB	124 g/L(120–160)
Neut%	50.5%(40.0–75.0)
ALC	1.6×10 ⁹ /L(1.1–3.2)
Plate	126×10 ⁹ /L(125–350)
Alb	28.8 g/L(40–55)
Cr	99 μmol/L(57–111)
BUN	7.3 mmol/L(2.9–7.8)
TC	7.63 mmol/L(<5.20)
LDL-C	4.82 mmol/L(<3.12)
PT	10.2s(9.2–13.9)
APTT	30.3s(21.2–34.8)
D-Dimer	1.19mg/L(0-0.55)
UP	3+(-)
RBCE	4–8 /μL(0–3)
UPCR	10.8 mg/g(≤0.2)
24hUP	4.99 g/day(≤0.15)
eGFR	69.8 mL/min(80–120)
ANA	Negative(-)
ANCA	Negative(-)
Anti-GBM antibody	Negative(-)
Abbreviations: WBC=white blood cells; Hb=hemoglobin; Neut%=neutrophil percentage; ALC=absolute lymphocyte count; Plate=platelets; Alb=albumin; Cr=creatinine; BUN=blood urea nitrogen; TC=total cholesterol; LDL-C = low-density lipoprotein cholesterol; PT=Prothrombin Time;s=second;APTT=Activated Partial Thromboplastin Time;UP=urinary protein; RBCE = red blood cells in urine; UPCR=urine protein–creatinine ratio; 24hUP = 24-hour urinary protein excretion; eGFR=estimated Glomerular Filtration Rate;ANA=antinuclear antibody; ANCA=antineutrophil cytoplasmic antibody; Anti-GBM Ab=anti–glomerular basement membrane antibody.	

To determine the histological pattern of kidney injury and guide subsequent clinical management, the TKI was temporarily withheld three days before a planned renal biopsy. On February 14, 2025, the patient underwent a percutaneous ultrasound-guided biopsy of the right kidney. Pathological examination of the renal biopsy specimen revealed findings consistent with membranoproliferative glomerulonephritis (MPGN) (Fig. 1). No evidence of extrarenal involvement or identifiable secondary etiologies was found.

Immediately following the biopsy, the patient developed right flank pain. Post-procedural ultrasound revealed a right renal hematoma with signs of progressive expansion (Fig. 2), accompanied by a decline in hemoglobin from 124 g/L pre-procedure to 97 g/L post-biopsy, consistent with active hemorrhage. Emergency renal angiography with selective embolization was performed on the same day (Fig. 3), achieving successful hemostasis.

The nephrotic syndrome was attributed to TKI, and tumor specialists were consulted regarding treatment decisions. The patient's HCC remained radiographically stable, and discontinuation of TKI was being considered. Once renal status stabilized, re-evaluation of oncologic therapy would be conducted; accordingly, TKI was discontinued.

During follow-up, regular outpatient visits were maintained. Two months after stopping TKI, on April 22, 2025, clinic assessment showed resolution of generalized edema, proteinuria decreased to 1+, serum albumin rose to 38.6 g/L, and the urine protein-to-creatinine ratio (UPCR) normalized to approximately 1.0, indicating remission of nephrotic syndrome.

Discussion

This case describes a 60-year-old man who developed nephrotic syndrome during therapy with TKI, a VEGF receptor tyrosine kinase inhibitor. To exclude secondary etiologies, a renal biopsy was performed after a 3-day washout of the drug; however, post-biopsy renal bleeding necessitated embolization. The "histological pattern" of kidney injury showed MPGN, and the nephrotic syndrome improved after discontinuation of TKI. Key take-home messages are: (1) recognize TKI-associated nephrotic syndrome as a potential adverse event; (2) initially discontinue the drug and monitor the renal response before pursuing invasive diagnostics to exclude secondary etiologies; and (3) renal biopsy should be considered only after a washout period and clinical stabilization, given the bleeding risks in this context.

Membranoproliferative glomerulonephritis (MPGN) is a histologic pattern of injury rather than a specific disease entity, requiring systematic exclusion of secondary etiologies. In our patient, an extensive workup excluded chronic hepatitis B (undetectable HBV DNA on entecavir), autoimmune disorders (negative ANA, ANCA, and anti-GBM antibodies), and monoclonal gammopathy (negative serum immunofixation). Cryoglobulinemia was not tested due to assay unavailability at our institution. Complement levels (C3 and C4) were normal. Immunofluorescence showed granular capillary loop deposition of IgG and C3, with weaker staining for IgM and IgA, consistent with immune complex–mediated MPGN. The absence of dominant C3 deposition without immunoglobulins argues against a primary complement-driven process such as C3 glomerulopathy. Although the mechanism by which apatinib induces MPGN remains unclear, VEGF signaling is critical for glomerular endothelial and podocyte integrity; its disruption may alter the local immune microenvironment and predispose to immune complex deposition. This case broadens the spectrum of anti-VEGF–associated glomerular diseases, identifying MPGN as a rare but biopsy-proven pattern of TKI-related renal injury. Distinguishing immune complex–mediated MPGN from complement-mediated glomerulopathies has therapeutic and prognostic relevance. Notably, the favorable renal response after drug withdrawal supports the reversibility of apatinib-induced injury upon cessation of the offending agent.

TKI apatinib, a novel anti-VEGF receptor tyrosine kinase inhibitor, can cause proteinuria through multiple mechanisms. One possible mechanism is increased glomerular capillary wall permeability. Podocytes and glomerular endothelial cells express VEGF receptors, and disruption of signaling between these cell types can compromise the glomerular filtration barrier, leading to proteinuria(1, 9). Another mechanism is VEGF inhibition–mediated downregulation of endothelial nitric oxide synthase (eNOS) and reduced nitric oxide (NO) production. NO is a key vasodilator, and VEGF signaling promotes endothelial NO synthesis; impairment of this pathway may injure endothelial cells and alter glomerular hemodynamics, contributing to proteinuria(1, 10). Some reports also implicate VEGF-inhibitor–associated hypertension in the pathogenesis of proteinuric states(11). Although proteinuria has been described with TKI apatinib(12, 13), nephrotic syndrome remains a rare manifestation. By contrast, nephrotic syndrome has been documented with other anti-VEGF agents, including bevacizumab(14), dasatinib(15), and sunitinib(16). The present case of TKI apatinib-associated nephrotic syndrome with remission after drug cessation adds clinically relevant evidence to inform safer use of this agent.

A growing body of evidence indicates that anti-VEGF therapies can cause a spectrum of renal injuries; the relevant cases are detailed in Table 2. Reported pathologies include thrombotic microangiopathy (TMA), podocytopathy, membranoproliferative glomerulonephritis (MPGN), collapsing glomerulopathy, cryoglobulinemic GN, and immune complex–mediated GN, with TMA described most frequently(11, 17, 18). Historically, many TMA cases have involved agents targeting VEGF-A or VEGF ligands (e.g., bevacizumab), whereas TKI agents that inhibit VEGF receptors (VEGFRs) have fewer reported instances of TMA. In our case of TKI apatinib, a VEGFR-2–targeted TKI, the renal histology did not reveal TMA features.

Table 2
Clinicopathological features of renal injury associated with VEGFR-TKIs

Author(s)	Age / Sex	Primary Diagnosis for TKI Therapy	Agent(s)	Time to Onset	Clinical Presentation	Proteinuria (at onset)	SCr (Onset / Baseline)	Renal Biopsy Finding	Management
Ruebner et al. (2014)-Case 1 [14]	3 y / F	CML, Ph+	Dasatinib	~ 2 years	NS, edema	UPCR 17 mg/mg	99 (78)	FSGS-like (IgM+C1q)	Withhold
Ruebner et al. (2014)-Case 2 [14]	15 y / F	Ph + ALL	Imatinib	NR	NS, edema, CMV colitis	UPCR 31 mg/mg	27 (NR)	Not performed	Withhold
Ruebner et al. (2014)-Case 3 [14]	1 y / F	Pre-B ALL	Quizartinib + Temozolomide	~ 6 months	NS, hypertension	4+ (quantitative NR)	18 (NR)	Not performed	Prednisone (ineffective)
Ruebner et al. (2014)-Case 4 [14]	15 y / M	Glioma + RCC	Sunitinib	4 months	NS + TMA (HTN, AKI, MAHA)	UPCR 15 mg/mg	150 (62)	Not performed (clinical TMA)	Withhold
ElShaer et al, (2021) [15]	46/F	Ph + CML	Dasatinib	~ 5 years	NS, edema, dyspnea	UPCR 5.33 mg/mg; 24hUP 5.9 g/d	70 (NR)	Endotheliosis + podocytopathy (IF-)	Withhold switch to nilotinib
Takahashi et al, (2012) [16]	72/M	GIST (imatinib-resistant)	Sunitinib	~ 6 months	NS + AKI + HTN	10.1 g/d	171 (79)	TMA-like endotheliosis + FSGS (IF-)	Withhold challenge, reduced
Izzedine et al (TMA group), (2014) [18]	60 y (mean) / 71% F	Mixed (RCC, breast, ovarian, lung)	Bevacizumab (61), VEGF-Trap (5), TKIs (7)	3 mo (median)	Proteinuria (100%), HTN (83.5%), hematuria	2.58 g/d (mean)	95 (NR)	TMA (intraglomerular)	Withhold anti-hypertensives
Izzedine et al (MCN/cFSGS group), (2014) [18]	59 y (mean) / 81% M	Mixed (RCC, ovarian, breast, lung)	TKIs (20), Bevacizumab (1)	2 mo (median)	Proteinuria (100%), HTN (48.1%)	3.15 g/d (mean)	111 (NR)	MCN/cFSGS (podocytopathy; KI-67 ⁺)	Withhold anti-hypertensives

Abbreviations:

y = years; M = male; F = female; CML = chronic myelogenous leukemia; Ph + = Philadelphia chromosome positive; ALL = acute lymphoblastic leukemia; RCC = renal cell carcinoma; NS = nephrotic syndrome; TMA = thrombotic microangiopathy; AKI = acute kidney injury; MAHA = microangiopathic hemolytic anemia; HTN = hypertension; CMV = cytomegalovirus; UPCR = urine protein-to-creatinine ratio; SCr = serum creatinine; Cr = creatinine; FSGS = focal segmental glomerulosclerosis; IF- = immunofluorescence negative; GIST = gastrointestinal stromal tumor; IHC = immunohistochemistry; CKD = chronic kidney disease; TKI = tyrosine kinase inhibitor; MCN = mesangiocapillary glomerulonephritis; CG = collapsing glomerulopathy; BP = blood pressure; RAS = renin-angiotensin system; d = day(s).

Notes:

All SCr values are expressed in $\mu\text{mol/L}$. Values in parentheses indicate baseline SCr before TKI initiation; values outside parentheses indicate SCr at the time of renal biopsy.

For the Izzedine et al. (2014) rows, values represent group means or medians from a case series of 73 patients with TMA and 21 patients with MCN/cFSGS, data are not available.

For the Takahashi et al. (2012) case, sunitinib was administered on a 4-weeks-on, 2-weeks-off schedule with dose reduction from 50 to 25 mg/day. For the ElShaer et al. (2021) case, dasatinib was maintained at 50 mg/day after prior dose reduction.

In Ruebner Case 2, concurrent CMV infection may have confounded the clinical presentation. In Ruebner Case 3, the lack of remission was confounded by pancytopenia and concomitant temsirolimus use.

Cases without a renal biopsy are indicated as "Not performed" in the Biopsy Finding column.

TKI apatinib's anti-angiogenic mechanism raises bleeding risk, with reported sites including epistaxis, hemoptysis, hematemesis, melena, tumor hemorrhage, and central nervous system bleeding(19–21). Across studies of TKIs, patients receiving these agents experience bleeding events of varying severity, with overall incidence rates around 9.1% for all grades and 1.3% for high-grade events. Targeted TKIs as a class are associated with elevated bleeding risk; the pooled relative risk is approximately 1.67 (95% CI, 1.19–2.33)(22). A retrospective study of tumor patients undergoing renal biopsy reported in reference (23) showed that renal biopsy is relatively safe in this population and provides important information for clarifying renal disease etiology and guiding subsequent therapy.

There is currently no universal standard for when to discontinue anti-VEGF therapy before invasive procedures; washout periods vary by agent. For bevacizumab, the American Society of Clinical Oncology recommends a 6-week pause prior to surgery to reduce operative bleeding and wound-healing complications(24). Ellis et al. suggested an 8-week interval before selective liver resection to minimize intraoperative bleeding risk(25). Arevalo et al. recommend stopping bevacizumab 4 weeks before vitrectomy without increasing intraoperative bleeding or retinal detachment risk(26). For intravitreal anti-VEGF agents such as ranibizumab and aflibercept, systemic concentrations fall below detectable levels within roughly 7 days after administration, supporting a practice of withholding therapy for about 1 week prior to ocular surgery to reduce bleeding risk(27).

Bleeding events attributed to TKI apatinib are uncommon. To our knowledge, this report describes the first case in which TKI apatinib-associated nephrotic syndrome was followed by post-biopsy postbiopsy renal hemorrhage. According to study(28), the half-life of TKI apatinib is 9 hours. Based on this half-life, the drug is expected to be completely cleared from the patient's system in at least 2.5 days. Therefore, a renal biopsy was performed 3 days after drug discontinuation. Despite the relatively well-defined half-life of TKI apatinib, the patient developed post-biopsy bleeding after a 3-day washout period. This observation underscores the need for an extended washout period before invasive renal procedures in patients receiving TKI apatinib and highlights the importance of heightened perioperative surveillance. Based on the case described in this report, we suggest that patients receiving TKI apatinib who are scheduled for invasive procedures discontinue the drug for at least three days preoperatively to reduce the risk of postoperative bleeding. However, as a single-case report, this study has inherent limitations and cannot establish a precise or generalizable discontinuation interval. Larger studies are needed to determine a more accurate preoperative cessation period.

Most cancer patients can derive long-term benefits from anti-VEGF-based therapies, but failure to monitor and address proteinuria can lead to renal injury. Clinically, nephrotoxic proteinuria typically emerges within one to four weeks after initiation of therapy(29). Currently, there is no universally accepted standard treatment for anti-VEGF-induced renal injury. Previous reports indicate that discontinuation of anti-VEGF therapy may lead to rapid complete or partial remission of proteinuria, whereas some patients progress to chronic kidney disease(30, 31). In the present patient, drug withdrawal was followed by resolution of edema, reduction in proteinuria, normalization of serum albumin, and stable renal function, consistent with partial remission. Reports of death directly attributable to anti-VEGF-induced renal injury are rare. However, cessation of anti-VEGF therapy may be associated with tumor progression or recurrence in some cases(32), highlighting the need to balance renal safety with ongoing oncologic control. In this patient, longitudinal oncologic follow-up after discontinuation of TKI apatinib showed tumor stability with no signs of recurrence at the time of reporting.

Several limitations should be acknowledged. This is a single case report with inherent limitations in generalizability. Testing for cryoglobulinemia was not performed due to assay unavailability at our institution. The short washout period (3 days) before biopsy and the limited follow-up duration (2 months) preclude definitive conclusions regarding optimal biopsy timing and long-term renal outcomes. The underlying mechanisms of apatinib-induced MPGN remain speculative in the absence of serial biomarker or complement profiling data. Larger studies are needed to confirm these observations.

Conclusions

Nephrotic syndrome is a rare but important adverse event of apatinib therapy. Clinicians should enhance renal monitoring during anti-cancer treatment and perform individualized risk-benefit assessments when renal injury occurs, adjusting and optimizing the therapeutic strategy as needed. If renal biopsy is considered in patients receiving TKI apatinib, the drug should be discontinued with careful peri-procedural monitoring. Additional research is needed to establish evidence-based guidance and more precise management strategies that maximize patient benefit while minimizing nephrotoxicity.

Abbreviations

24hUP, 24-hour urinary protein excretion

AKI, acute kidney injury

ALC, absolute lymphocyte count

ALL, acute lymphoblastic leukemia

ANA, antinuclear antibody

ANCA, antineutrophil cytoplasmic antibody

Anti-GBM Ab, anti-glomerular basement membrane antibody

APTT, activated partial thromboplastin time

BP, blood pressure

BUN, blood urea nitrogen

CG, collapsing glomerulopathy

CKD, chronic kidney disease

CML, chronic myelogenous leukemia

CMV, cytomegalovirus

eGFR, estimated glomerular filtration rate

FSGS, focal segmental glomerulosclerosis

GIST, gastrointestinal stromal tumor

H&E, hematoxylin-eosin

HTN, hypertension

IF-, immunofluorescence negative

IHC, immunohistochemistry

LDL-C, low-density lipoprotein cholesterol

MAHA, microangiopathic hemolytic anemia

MCN, minimal change nephropathy

MPGN, membranoproliferative glomerulonephritis

Neut%, neutrophil percentage

NR, not reported

NS, nephrotic syndrome

PAS, periodic acid-Schiff

PASM, periodic acid-Schiff with silver

Ph+, Philadelphia chromosome positive

PT, prothrombin time

RAS, renin-angiotensin system

RBCE, red blood cells in urine

RCC, renal cell carcinoma

SCr, serum creatinine

SOB, shortness of breath

TC, total cholesterol

TKI, tyrosine kinase inhibitor

TMA, thrombotic microangiopathy

UP, urinary protein

UPCR, urine protein–creatinine ratio

VEGFR-TKIs, vascular endothelial growth factor receptor tyrosine kinase inhibitors

WBC, white blood cells

Declarations

Ethics approval and consent to participate

The patient provided written informed consent for the publication of this case report. According to institutional guidelines, ethical review was not required for this case report.

Consent for publication

Written informed consent was obtained from the patient for the publication of this case report and any accompanying images. A copy of the written consent is available for review by the Editor-in-Chief of this journal upon request.

Availability of data and materials

All data generated or analysed during this study are included in this published article. Further inquiries can be directed to the corresponding author.

Competing Interests

The authors declare that the research was conducted in the absence of any commercial or financial relationships that could be construed as a potential conflict of interest.

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Authors' contributions

Conceptualization and writing - original draft: Weidong Huang

Data curation: Yiling Zhu

Methodology: Tingyan Xiang

Supervision: Qiankun zhang, Mingfeng Mao

Writing-review & editing: Lingfeng Huang

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Figures

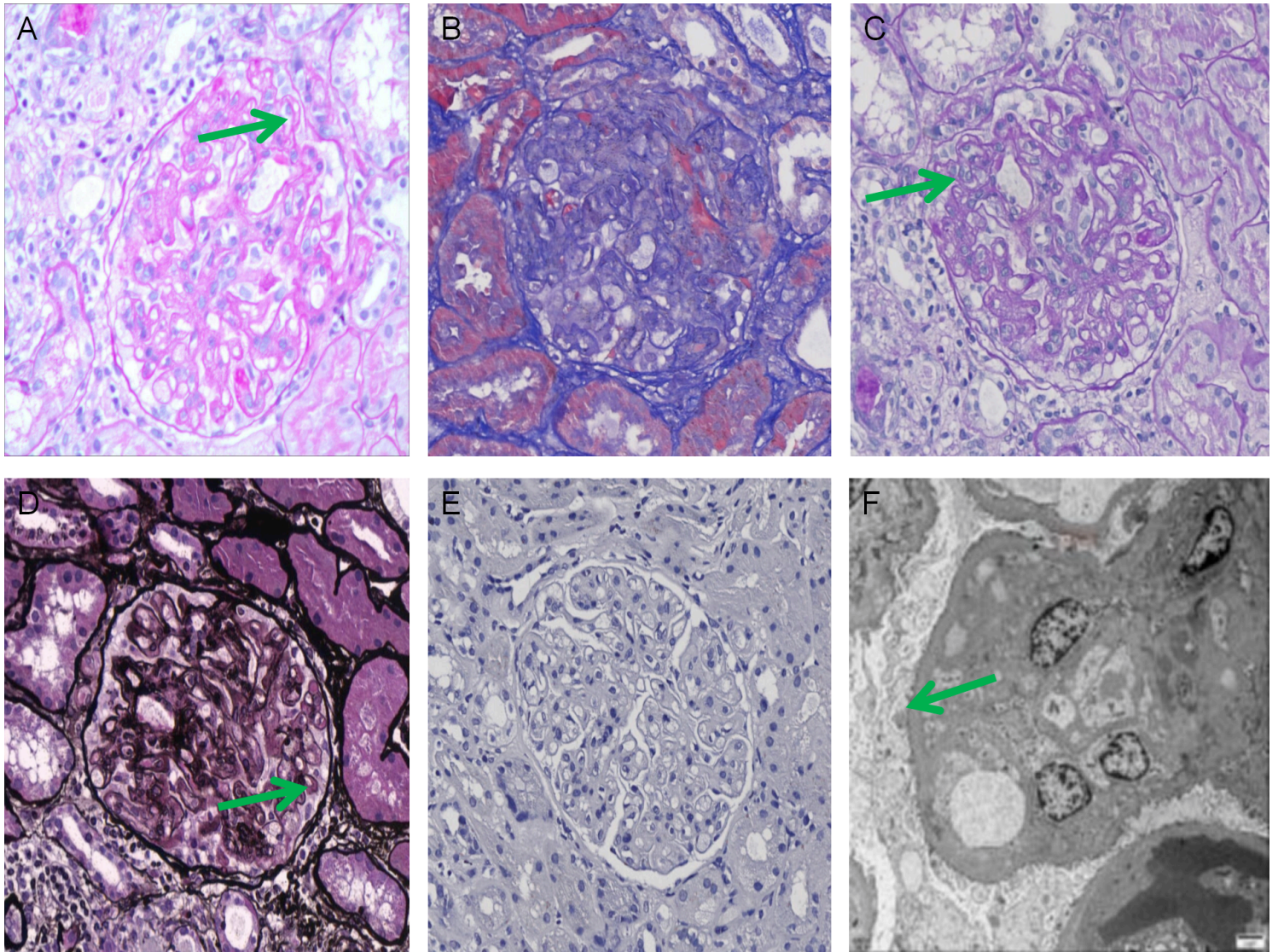


Figure 1

Histological findings of kidney injury. A. Hematoxylin–eosin (H&E) at $\times 400$: diffuse glomerular basement membrane thickening (green arrow) with mesangial expansion and double contours. B. Masson's trichrome at $\times 400$. C. Periodic acid–Schiff (PAS) at $\times 400$: mesangial cell and mesangial matrix proliferation (green arrow). D. PAS with silver (PASM) at $\times 400$: diffuse basement membrane thickening (green arrow) with mesangial expansion and double contours. E. Congo red stain at $\times 400$: negative. F. Electron microscopy at $\times 2500$: segmental thickening of the glomerular basement membrane in the capillary loops; diffuse foot process effacement.

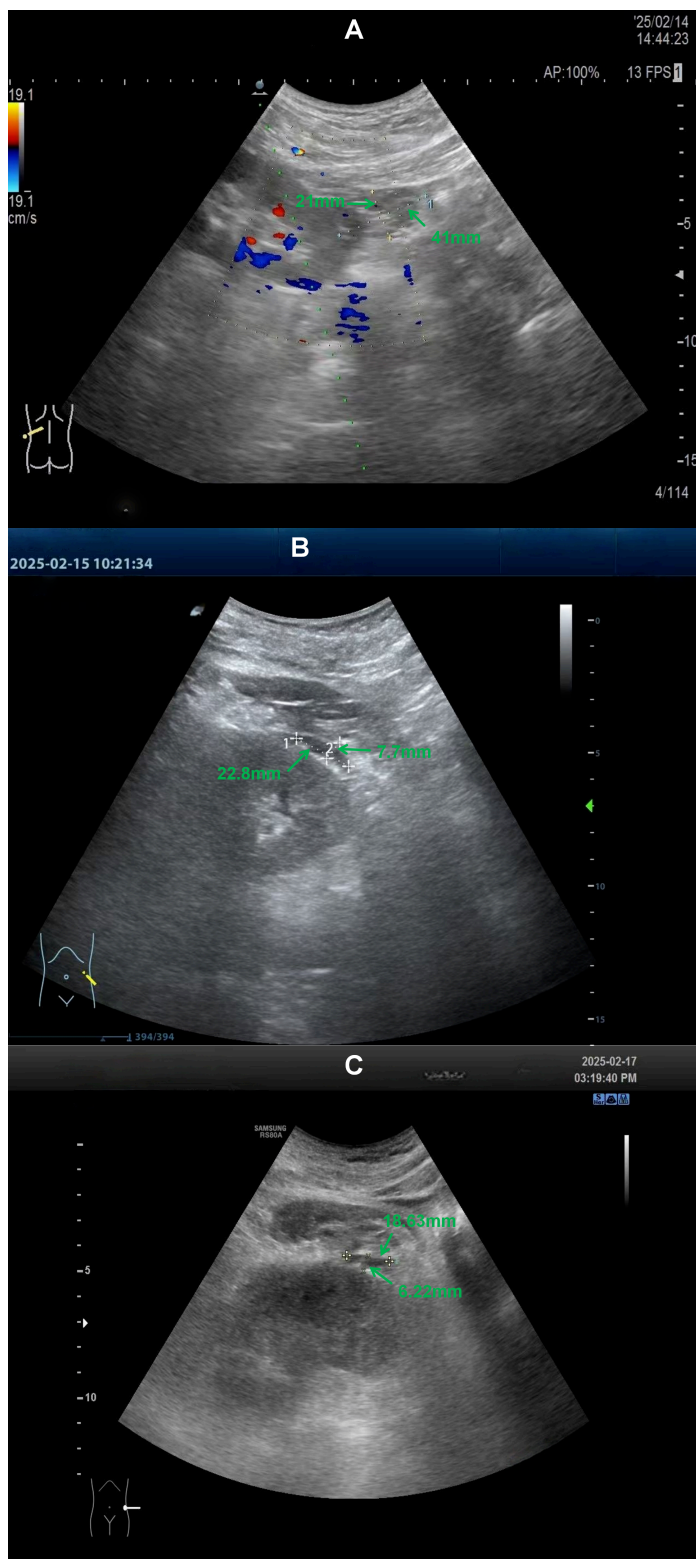


Figure 2

Ultrasound images showing post-biopsy evolution of a perinephric hematoma.

Green arrows indicate the extent of the hematoma.

A. Immediate post-biopsy view at 5 minutes.

B. Follow-up ultrasound on postoperative day 1.

C. Follow-up ultrasound on postoperative day 3.



Figure 3

Pre- and post-embolization angiographic images of the renal artery.

A. Pre-embolization renal arteriography: site of renal bleeding indicated by the green arrow. B. Post-embolization angiography: no contrast extravasation and effective hemostasis; the embolized bleeding site is indicated by the green arrow.

Supplementary Files

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