THA1

Nephrotic Syndrome with unexpected findings!

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THURSDAY - Moderated Poster Session, HALL Q, March 12, 2026, 10:00 - 11:00

Introduction

Nephrotic syndrome in adults is most commonly caused by membranous nephropathy (MN). Cellular crescents in MN are extraordinarily rare, with prevalence estimates of only 0.26-0.39%. Most crescentic cases are associated with ANCA-associated vasculitis or anti-GBM disease. We report an unusual case of PLA2R-positive MN with cellular crescents without ANCA or anti-GBM positivity, complicated by paraproteinaemia without monoclonal gammopathy of renal significance (MGRS).

Case Description

A 50-year-old male prisoner presented in July 2025 with two weeks of diarrhoea and vomiting, uncontrolled hypertension, acute kidney injury (creatinine 245µmol/L from baseline 127µmol/L), and nephrotic syndrome (ACR >4g/g, albumin 26g/L). He had hypertension since 2014 and used ibuprofen (6 tablets weekly) from December 2024-July 2025 for abdominal pain. No significant family history was present and no autoimmune features were found. Following stabilisation, he was discharged but re-presented one month later with deteriorating renal function (creatinine 336µmol/L) and profound nephrotic syndrome (albumin 18g/L). Urine dipstick revealed blood and protein. Immunology showed elevated PLA2R antibodies (142 RU/mL), elevated kappa-lambda ratio (2.56) with lambda light chains on urine immunofixation, but no serum monoclonal bands. ANCA, ANA, anti-GBM antibodies were negative; complement levels normal; viral screening and tumour markers negative.

Results

Renal biopsy revealed 23 glomeruli with 4 globally sclerosed. Light microscopy showed membranous glomerulonephritis with cellular crescents in 3 glomeruli (13%), mesangial matrix expansion, and moderate chronic tubulointerstitial damage. Immunoperoxidase demonstrated diffuse granular IgG staining with strong PLA2R positivity, alongside focal IgM and C1q staining. Electron microscopy revealed global subepithelial electron-dense deposits with focal subendothelial and mesangial deposits. The widespread deposit distribution and IgM/C1q co-staining suggested secondary MN; however, strong PLA2R positivity typically indicates primary disease. No light chain deposition was identified despite elevated paraproteins, excluding MGRS.

Discussion

This represents an extraordinarily rare presentation within the already rare entity of crescentic MN. Most crescentic MN cases (78%) are ANCA-positive or anti-GBM disease-associated, with only 31% showing PLA2R positivity. The combination of strong PLA2R positivity with histological features suggesting secondary MN creates unprecedented diagnostic uncertainty. Very few case reports describe this paradoxical presentation with additional paraproteinaemia complexity. A recent series found 69% of crescentic MN cases were PLA2R antigen negative, emphasising how unusual PLA2R-positive crescentic disease

is. Pathogenesis remains unclear, though cryptic epitope release from podocyte damage has been proposed.

Management

Given diagnostic uncertainty and crescents indicating active inflammation, the patient was commenced on high-dose corticosteroids preceded by pulse therapy following multidisciplinary discussion. Due to the complex presentation combining primary and secondary MN features with crescents, the case was referred to the Eastern Network for Kidney Inflammatory Disease (ENKID) for specialist guidance on induction immunosuppression (rituximab versus modified Ponticelli regimen). Follow-up PLA2R levels and treatment response monitoring are pending.

Conclusion

This case highlights diagnostic challenges when primary and secondary MN features coexist with cellular crescents without typical crescentic disease markers. Paraproteinaemia without MGRS adds complexity. Such cases require specialist multidisciplinary input for optimal therapy selection and underscore evolving understanding of membranous nephropathy pathogenesis.

THA2

An unusual presentation of acute kidney injury secondary to Influenza A-induced rhabdomyolysis

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THURSDAY - Moderated Poster Session, HALL Q, March 12, 2026, 10:00 - 11:00

Introduction:
Influenza A is a common respiratory tract infection, which can have a wide range of extra-pulmonary manifestations, including acute myositis and uncommonly, rhabdomyolysis. For those who develop rhabdomyolysis, the clinical course typically begins with respiratory symptoms followed by musculoskeletal symptoms within a week. We present the case of an individual who presented in summer with severe symptomatic rhabdomyolysis without respiratory symptoms, who subsequently developed multiple significant sequelae of the infection.

Case Presentation:
A 28-year-old, with no significant past medical history, attended their local Emergency Department with a three-day history of progressive lower limb weakness and pain and dark-coloured urine, having recently attended a traditional music festival. There was no history of trauma, excessive exertion, or recreational drug use. Physical examination revealed lower limb weakness only. Initial investigations demonstrated apparent hepatic injury (AST 3170 IU/L, ALT 340 IU/L), raised inflammatory markers (WCC 19.2 x 109/L, CRP 100 mg/L), rhabdomyolysis (Creatinine Kinase (CK) >22,000 IU/L), and normal renal function (Creatinine 67 µmol/L). Toxicology screen was negative. Initial management included broad spectrum antibiotics and intravenous fluids (IVF). Despite initial interventions, CK rose, and serial dilutions showed an estimated level of 284,050 IU/L. Serum creatinine rose over the following 4 days to 487 µmol/L and the patient became oliguric. CT KUB revealed oedematous kidneys without evidence of obstruction. Given the rapidly progressive renal dysfunction and rhabdomyolysis of unclear aetiology, the patient was transferred to our tertiary centre for further management.
Myositis and glomerulonephritis screens were negative. Viral screen revealed Influenza A positivity. MRI of the femurs showed diffuse bilateral symmetrical oedema of the thigh and pelvic muscles. Oseltamivir was initiated but subsequently withdrawn as symptom duration exceeded 7 days. IVF and urinary alkalinization were continued and when volume expanded, diuretics were initiated.
By day 9, creatinine had plateaued at approximately 620 µmol/L and CK had fallen to 2967 IU/L. At this point, the patient started to experience headaches. This rapidly progressed to blurred vision and subsequently a generalized tonic-clonic seizure, with a concomitant jump in systolic blood pressure to >180mmHg. CT brain, angiogram and venograms were all normal. The patient transferred to the intensive care unit for management of a hypertensive emergency, requiring labetalol infusion, amlodipine, doxazosin, clonidine and furosemide for blood pressure control. MRI brain revealed subtle features suggestive of Posterior

Reversible Encephalopathy Syndrome. T wave inversions in leads III and aVF associated with a troponin rise were deemed consistent with a type two myocardial infarction secondary to a hypertensive emergency.
On day 14, Cr started to fall allowing antihypertensive therapy to be weaned. On day 22 the patient was discharged home with creatinine 109 µmol/L.

Conclusion:
This case illustrates a rare but serious complication of influenza infection and highlights the importance of screening for influenza virus in the setting of non-traumatic rhabdomyolysis, even in those without typical symptoms and outside of the usual flu season.

THA3

Tirzepatide-associated interstitial nephritis

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THURSDAY - Moderated Poster Session, HALL Q, March 12, 2026, 10:00 - 11:00

Introduction

Tirzepatide aims to improve glucose control through enhancing insulin secretion and reducing appetite. It is licensed in the United Kingdom for Type 2 diabetes mellitus and weight management. Additionally, glucagon-like peptide-1 (GLP-1) agonists have cardioprotective effect and slow the decline of kidney function in type 2 diabetics with chronic kidney disease (CKD). (1)

Diagnosis of tubulointerstitial nephritis (TIN) is often delayed due to non-specific symptoms including fatigue, arthralgia, low-grade fever and rash, or is found incidentally on blood tests (2).

This case entails a patient who developed TIN following private use of Tirzepatide (Mounjaro).

Case presentation

A 35-year-old female presented to her GP with worsening fatigue over several weeks, and blood tests revealed significant renal impairment with creatinine 546µmol/L. Last known renal baseline showed creatinine 70 µmol/L in 2017.

In September 2024 she had commenced tirzepatide on private prescription. She took no other regular medications and nothing regularly over the counter. She had no recent viral illnesses or infections. Medical history included obesity, never smoker and minimal alcohol. She was clinically euvolemic, afebrile and had no skin rash.

Urinalysis showed 1+ blood, 1+ protein, uPCR 42mg/mmol. Eosinophils normal.

Tirzepetide was stopped, and renal biopsy yielded findings consistent with interstitial nephritis. Following histopathological diagnosis, we commenced prednisolone 40mg daily on 8/7/25. Over the next 2 weeks creatinine significantly improved from 590µmol/L to 181 µmol/L. Her clinical course underscores the importance of considering potential TIN in patients on GLP-1 agonists presenting with non-specific constitutional symptoms.

Discussion

Renal impairment is not currently listed as a potential side effect for Tirzepatide in the BNF. “Tubulointerstitial nephritis” is listed as a “rare or very rare” side effect for other medications such as PPIs.

There are a handful of published case reports of TIN following GLP-1 therapy, and rapidly increasing use of these medications means we need to be aware of this potentially serious adverse effect. NICE licencing currently restricts GLP-1 agonist use to patients with BMI>35 and obesity-related health conditions, so more patients are turning to using these mediations privately (3). Healthcare data and analytics provider IQVIA reports approximately 1.4 million people in the UK access these drugs privately, compared to 200,000 through the NHS. (4)

NICE does not specify renal function blood tests are needed as baseline or for monitoring on tirzepetide, but that they could be considered depending on “current comorbidities and to identify unknown comorbidities” (5) NICE does not recommend routine renal function

testing in obesity without associated risk factors, but does recommend it if there is presence of certain obesity-associated health conditions including hypertension and diabetes.

Conclusion

As use of GLP-1 agonists increases both through the NHS and privately, we need to be very aware of the potential of these medications to cause TIN as seen in an increasing number of case reports (6,7,8).

Symptoms of TIN can be insidious and subtle, with diagnosis often delayed, and we need high clinical suspicion for this diagnosis (9) in patients on GLP-1 agonists who present with constitutional non-specific symptoms.

THA4

Systemic lupus erythematosus with nephrotic syndrome in a patient with autosomal polycystic kidney disease: a rare diagnostic and therapeutic challenge

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THURSDAY - Moderated Poster Session, HALL Q, March 12, 2026, 10:00 - 11:00

Introduction

Autosomal dominant polycystic kidney disease (ADPKD) is the most common inherited kidney disorder, while lupus nephritis (LN) represents a severe manifestation of systemic lupus erythematosus (SLE). Tuberculosis (TB) remains a highly prevalent infection worldwide and may present with systemic features such as weight loss, fever and lymphadenopathy. Their coexistence is exceptionally rare with only a handful of cases reported. Diagnosis of LN in ADPKD is particularly challenging, as renal biopsy is often precluded by extensive cystic involvement. We present the case of a young woman with known ADPKD who developed nephrotic syndrome and was subsequently diagnosed with SLE and latent tuberculosis (TB).

Case description

A 29-year-old Nigerian woman with prior diagnosis of ADPKD presented with four weeks of fatigue, cough, body aches, bilateral flank pain, and 18 kg unintentional weigh loss. She reported frothy, dark urine. On admission, she was febrile with nephrotic-range proteinuria [urine protein-creatinine ratio (uPCR) 457 mg/mmol], hypoalbuminaemia (18g/L), but notably no oedema. Renal function was preserved (creatinine 76 µmol/L). Examination revealed lymphadenopathy, and CT confirmed mildly prominent lymph nodes and extensive bilateral renal cysts (largest 7 cm). Lymph node biopsy demonstrated reactive lymphadenitis. Laboratory evaluation also revealed cytopenias.
Autoimmune testing initially showed low complements (C3 0.54g/L, C4 0.13 g/L) but negative CTD screen, anti-GBM and ANCA (MPO/PR3). Approximately 12 days later, repeat testing in the outpatient clinic demonstrated seroconversion with strong positive ANA, high-titre anti-dsDNA (130 IU/ml, ref < 9.9), anti-Ro/SSA (3.0 AI), anti-ribosomal P (6.9 AI), and chromatin antibodies (> 8.0 AI). Antiphospholipid antibodies remained negative. QuantiFERON-Gold was positive. Virology screen for HIV, hepatitis B and C was negative.

Results

She was discharged after treatment for presumed pyelonephritis and extensive investigations. Shortly thereafter, she re-presented with her first generalised tonic-clonic seizure (CT head unremarkable) and new onset oedema/anasarca. In the context of nephrotic syndrome, systemic features and now strongly positive autoantibodies, a working diagnosis of lupus nephritis was made. Renal biopsy was contraindicated due to cystic anatomy.
She was commenced on high-dose prednisolone, hydroxychloroquine, and Euro-lupus intravenous cyclophosphamide regimen alongside latent tuberculosis therapy under respiratory team supervision. Despite early treatment interruptions due to intercurrent cyst

infection, oedema improved, and proteinuria declined. She continues under multidisciplinary follow-up by nephrology and respiratory teams.

Discussion and conclusion

The coexistence of ADPKD and LN is extremely rare. This case illustrates the diagnostic dilemma of attributing nephrotic-range proteinuria in ADPKD patients, where cystic disease may obscure or confound additional glomerular pathology. It highlights the challenge of establishing LN without histology when renal biopsy is unsafe. In this context, the combination of systemic features, nephrotic syndrome, and evolving autoimmune profile supported the diagnosis. Therapeutically, her case underscores the complexities of immunosuppression in the setting of latent tuberculosis. Subsequent management challenges have involved discerning lupus flares from infection.

This report adds to the sparse literature on ADPKD with concurrent LN, reinforces the need to consider autoimmune causes of proteinuria in ADPKD, and demonstrates the importance of multidisciplinary management in complex overlapping renal disease.

THA5

Tacrolimus toxicity precipitated by Paxlovid in a renal transplant recipient: a case report of drug–drug interaction and system-level failures

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THURSDAY - Moderated Poster Session, HALL Q, March 12, 2026, 10:00 - 11:00

Introduction
A renal transplant recipient collapsed at home after only three doses of Paxlovid, later found with tacrolimus levels >43 ng/mL. Nirmatrelvir/ritonavir (Paxlovid) is a potent CYP3A4 inhibitor; co-administration with tacrolimus can cause life-threatening toxicity. Despite repeated safety alerts, such interactions remain a reproducible hazard. This case is novel in demonstrating extreme tacrolimus elevation after only three doses, prescribed at hospital discharge despite existing warnings, exposing a preventable system-level failure.

Case Presentation
A 59-year-old woman with end-stage renal disease from IgA nephropathy underwent deceased-donor kidney transplantation in 2021. She remained stable on tacrolimus, mycophenolate, and prednisolone, with eGFR 46 mL/min/1.73 m² in July 2025. In June 2025, after testing COVID-positive, she was discharged on Paxlovid without adjustment of immunosuppression or transplant team consultation. After three doses she developed dizziness, blurred vision, and collapsed at home, re-presenting to hospital.

Results
Tacrolimus exceeded 43 ng/mL within 72 hours of Paxlovid initiation (trough 43.6 ng/mL; therapeutic 5–8). Admission labs showed glucose 22 mmol/L, CRP 43 mg/L, creatinine 202 µmol/L (eGFR 26). Chest radiograph revealed mild infiltrates. Blood cultures grew Sphingobacterium spiritivorum, regarded as of uncertain pathogenicity. ECG was normal. Paxlovid and tacrolimus were withheld, and IV fluids commenced. Tacrolimus levels declined rapidly without extracorporeal clearance: 26.5 ng/mL (day 2), 11.2 (day 3), 7.1 (day 5). Neurological symptoms and hyperglycaemia resolved in parallel. Renal function returned to baseline (creatinine 149 µmol/L, eGFR 41) within one week. Tacrolimus was reintroduced at reduced dose (1 mg BD) with stable follow-up. No rejection or opportunistic infection occurred. Causality was supported by temporality, mechanistic plausibility, exclusion of alternatives, and positive dechallenge. WHO-UMC classification was “probable” adverse drug reaction.

Discussion
Paxlovid can precipitate profound tacrolimus toxicity within days, even when prescribed at hospital discharge. The rapid fall from >43 ng/mL to therapeutic range illustrates that withdrawal and supportive care may suffice where levels are extreme but stable. The concurrent bacteraemia highlighted diagnostic complexity in immunosuppressed hosts but did not account for the presentation. Most importantly, this case illustrates a preventable prescribing error despite existing safety alerts, exposing gaps in discharge safeguards, cross-

specialty communication, and patient education. It serves as an early warning signal that current UK prescribing safeguards remain insufficient to protect transplant recipients.

Implication
Transplant recipients remain at risk of preventable, life-threatening toxicity unless electronic prescribing systems enforce robust drug–drug interaction safeguards. Prescribers must confirm tacrolimus therapy before initiating Paxlovid, with pharmacists acting as final gatekeepers.

Next step
National pharmacovigilance and mandatory electronic prescribing hard-stops with transplant-specific interaction flags should be embedded across UK discharge systems. Centre-level audit of Paxlovid prescribing in immunosuppressed patients will be essential to track outcomes and eliminate recurrence.

THA6

A diagnostic dilemma: thrombotic microangiopathy in post-renal transplantation

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THURSDAY - Moderated Poster Session, HALL Q, March 12, 2026, 10:00 - 11:00

Introduction

Thrombotic microangiopathy(TMA) after kidney transplantation represents a formidable diagnostic and therapeutic challenge. Multiple, often overlapping etiologies—such as recurrence of hemolytic uremic syndrome (HUS), infection-related complement activation, and drug-induced injury from calcineurin inhibitors (CNIs)—can present with similar clinicopathological features. Distinguishing between these mechanisms is crucial, as therapeutic strategies may differ. Yet, in clinical practice, these boundaries are blurred, and treatment decisions are frequently made before all relevant investigations are completed. We present the case of a young renal transplant recipient with prior pneumococcal HUS who developed post-transplant TMA, illustrating the complexity of teasing apart recurrent disease from CNI-induced injury.

Case details

A female patient had a history of HUS in 2008 at the age of 2. This was attributed to Pneumococcal infection; genetic screen for atypical HUS was negative. She developed end-stage kidney disease at the age of 19, commencing peritoneal dialysis in March 2024 prior to undergoing a live donor transplant from her mother in July 2024. She developed graft pyelonephritis in October 2024 but, despite antibiotic treatment, her renal function progressively deteriorated, and she required a number of graft biopsies:

-November'24 biopsy (creatinine—189 umol/L): mild acute tubular injury, focal interstitial fibrosis, no rejection or BK nephropathy—changes were felt to be consistent with recent pyelonephritis

-December'24 biopsy (creatinine—237 umol/L) : acute TMA; acute and chronic active T-cell mediated rejection (BANFF 1B), but no antibody-mediated rejection.

-January'25 biopsy(creatinine—213 umol/L): persistent feature of acute TMA, mild hyaline changes within arterioles raising suspicion of CNI toxicity. Although tacrolimus trough levels fluctuated, they were seldom significantly above target range.

Given the previous history of HUS, recurrence was suspected, and she received three doses of eculizumab between December'24 and February'25. However, as graft dysfunction persisted (creatinine-159 umol/L)and based on findings of 3rd biopsy, tacrolimus was switched to sirolimus. Following this, her renal function improved and subsequently stabilized (creatinine-86 umol/L June'25).

Discussion

This case illustrates the diagnostic challenge when TMA occurs in the post-transplant setting, and it is often not possible to determine a cause based on histological examination alone. CNI-associated endothelial toxicity is well documented in the literature, with

tacrolimus implicated in both de novo and recurrent TMA, even in the absence of drug levels above the target range. However, HUS recurrence – though less likely with Pneumococcal-associated cases and in those without a genotype predisposing to atypical HUS – remained a possibility, as complement dysregulation may be triggered by immunological/ infectious precipitants or even with severe rejection and hypertension. Eculizumab, a terminal complement pathway inhibitor, has proven benefit in recurrent/ complement-mediated TMA, while CNI withdrawal is the cornerstone for drug-induced TMA. In this patient, the fact that her graft function remained unchanged following treatment for rejection and eculizumab administration, but subsequently improved following withdrawal of tacrolimus favours tacrolimus as the most likely cause.

Conclusion

Our case underscores the inherent diagnostic and therapeutic uncertainty in transplant-associated TMA. When clinical and histological findings suggest overlapping etiologies, management often requires vigilant monitoring, timely biopsy, and willingness to adapt therapy that are essential for preserving graft function.

THA7

Probable digoxin-induced thrombocytopenia in an elderly haemodialysis patient: diagnostic rescue following drug withdrawal

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THURSDAY - Moderated Poster Session, HALL Q, March 12, 2026, 10:00 - 11:00

Introduction

Drug-induced thrombocytopenia (DITP) is an uncommon but clinically serious adverse reaction, typically immune mediated and abrupt in onset. Digoxin is rarely recognised as a culprit, and no guideline recommends routine haematological monitoring. Patients with dialysis-dependent chronic kidney disease (DD-CKD) are particularly vulnerable to drug accumulation and atypical toxicity. We report the first detailed case of probable digoxin-induced thrombocytopenia in a haemodialysis patient, where rapid recognition prevented unnecessary interventions and averted harm.

Case Presentation

An 84-year-old male with DD-CKD, on thrice-weekly haemodialysis since October 2024, was admitted on 26 August 2025 with bradycardia (heart rate 47 bpm). He had permanent atrial fibrillation managed with long-term digoxin. Platelets, which had been 277 $\times 10^9$/L in March 2025, fell to 54 $\times 10^9$/L on 21 August and 30 $\times 10^9$/L by 23 August. Haematology suspected immune thrombocytopenic purpura, prompting anticoagulation interruption and extensive investigations, including autoimmune, haemolysis, thyroid, vitamin B12/folate, and virology screening alongside abdominal imaging. On admission, digoxin was 2.4 ng/mL (toxic). Digoxin and bisoprolol were discontinued, with ongoing haemodialysis and cardiac monitoring. Sepsis, heparin-induced thrombocytopenia, and dialyser reactions were excluded.

Results

After drug cessation, platelet counts rose steadily in parallel with falling digoxin levels. On 26 August, platelets were 66–70 $\times 10^9$/L; by 28 August they had increased to 88 $\times 10^9$/L, and by 29 August to 126 $\times 10^9$/L, with digoxin falling to 1.2 ng/mL. On 31 August, platelets reached 168 $\times 10^9$/L with digoxin 0.8 ng/mL. Between 01 and 04 September, platelets normalised, ranging from 200 to 268 $\times 10^9$/L, and stability was maintained at 213–268 $\times 10^9$/L through mid-September. C-reactive protein remained modest (35 mg/L), making infection unlikely. Recovery occurred during ongoing haemodialysis and importantly without steroids, immunoglobulin, transfusions, or dialyser change. Haemoglobin and white cells remained stable. The Naranjo score was 5, consistent with a probable adverse drug reaction.

Discussion

This case illustrates the rare association between digoxin toxicity and thrombocytopenia, extending the evidence base to the haemodialysis population. The strong temporal relationship between toxic digoxin levels, abrupt thrombocytopenia, and complete recovery after withdrawal strengthens causality. Crucially, initial misclassification as ITP risked unnecessary immunosuppression and delayed anticoagulation. Recognition of digoxin

toxicity provided a diagnostic rescue, sparing the patient harm and guiding safe resumption of therapy. This also strengthens understanding of the pathophysiology, which until now was based only on temporal associations rather than full laboratory-supported recovery.

Implication
In DD-CKD patients with abrupt thrombocytopenia, clinicians should review medications and consider digoxin toxicity before initiating ITP-directed therapies.

Next step
Systematic pharmacovigilance reporting, targeted platelet monitoring in high-risk digoxin users, and regular digoxin level checks in dialysis populations are warranted to reduce avoidable investigations and inform future nephrology–cardiology guideline updates.

THA8

Hyperoxaluria-Induced Renal Failure: A case of secondary oxalosis following high dose vitamin supplementation

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THURSDAY - Moderated Poster Session, HALL Q, March 12, 2026, 10:00 - 11:00

Hyperoxaluria-Induced Renal Failure: A case of secondary oxalosis following high dose vitamin supplementation

Background: An 80-year-old man, previously fit and well, was referred to the emergency department by his GP with an acute decline in renal function, new-onset anaemia, and hypercalcaemia. He had a six-month history of tiredness, exertional shortness of breath, and weight loss. He reported increased urinary frequency as well as frothy urine. He mentioned that for the past three months, he has been taking over-the-counter Vitamin D 10,000 IU and Vitamin C 2,000 mg daily.

Investigations: Patient presented with acute kidney injury stage 3 with creatinine 412 µmol/L. His last creatinine from 2023 was 97 µmol/L. Further biochemistry revealed severe hypercalcaemia (3.10 mmol/L), hyperphosphatemia (2.06 mmol/L), suppressed PTH (0.8 pmol/L), markedly elevated vitamin D (490.9 nmol/L).He was also found to have normocytic anaemia (Hb 83 g/L). Renal immunology screening and myeloma screen results remained within normal limits.

Further tests with a 24hr urine collection showed significant hyperoxaluria (oxalate 514 µmol/day; oxalate/creatinine ratio 48). OGD and colonoscopy showed duodenal polyp, rectal polyp, and diverticular disease.

Renal biopsy showed extensive tubular injury with calcium oxalate crystal deposition, consistent with oxalosis

Management and outcomes: On admission, hypercalcemia was corrected with intravenous fluids, as per hospital guidelines. Anaemia was corrected with a blood transfusion. Further workup was done to rule out any extra-renal cause of Anaemia

Genetic testing was considered; however, in view of his clinical history and age at presentation, this was not pursued as it was unlikely to alter management or outcome.

On discharge, he was scheduled for follow-up in the low-clearance clinic, referred to specialist renal nurses for dialysis counselling, and to vascular surgery for vascular access formation

Discussion: Oxalate, the ionic form of oxalic acid, is a naturally occurring compound found in both plant and animal sources and is primarily excreted via the kidneys. Oxalosis refers to the systemic deposition of calcium oxalate crystals in tissues outside the kidneys, typically occurring in the context of primary or secondary hyperoxaluria. Impaired renal excretion of oxalate leads to crystal deposition, resulting in oxalate nephropathy and, in some cases, severe systemic complications. The clinical presentation of oxalate nephropathy is heterogeneous, ranging from acute kidney injury (AKI) to chronic kidney disease (CKD), and may occasionally be associated with hypercalcaemia

Primary hyperoxaluria is a rare autosomal disorder causing endogenous oxalate overproduction, while secondary hyperoxaluria results from increased intestinal absorption, often due to fat malabsorption or excessive intake of oxalate or precursors like high-dose

vitamin C. Idiopathic hyperoxaluria is diagnosed when no clear cause is found.[2,3] This case highlights the importance of early recognition of oxalosis and the risks of excessive vitamin supplementation, with overuse of vitamins D and C contributing to hypercalcaemia and kidney injury.

THA9

Alemtuzumab-associated accommodative spasm in a kidney transplant recipient: a rare neuro-ophthalmic complication

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THURSDAY - Moderated Poster Session, HALL Q, March 12, 2026, 10:00 - 11:00

Introduction

Blurred vision after kidney transplantation is often attributed to hyperglycaemia or calcineurin inhibitor toxicity. We describe one of the first reported cases of accommodative spasm following alemtuzumab in a renal transplant recipient, an under-recognised neuro-ophthalmic complication with potential for misdiagnosis and unnecessary intervention. Although alemtuzumab has a well-established safety profile, ocular sequelae are scarcely reported and absent from current prescribing information.

Case/Methods

A 39-year-old woman with NPHP1-associated nephronophthisis and F3 liver fibrosis underwent her second kidney transplant in April 2025. In May 2025, biopsy confirmed Banff IIB acute cellular rejection, treated with alemtuzumab 30 mg subcutaneously and methylprednisolone pulses. Within 72 hours she developed bilateral blurred vision impairing both near and distance tasks. Initial attribution was to post-transplant diabetes, supported by transient hyperglycaemia, and tacrolimus neurotoxicity, with trough levels re-checked, but symptoms persisted despite correction and dose adjustment. Ophthalmic review excluded structural pathology on OCT and slit-lamp examination, while corrective lenses gave minimal benefit, delaying diagnosis.

Results

Symptoms persisted for ~5.5 weeks and caused functional visual disability. On 24 June 2025, cycloplegic refraction demonstrated a hyperopic shift (+2.75D right, +2.50D left) with acuity improving to 0.22 bilaterally, confirming accommodative spasm. Infectious screens including CMV PCR and fundoscopy were negative; differential diagnoses such as CMV retinitis, posterior reversible encephalopathy, and optic neuritis were excluded. No pharmacological treatment was required. By 10 July 2025, vision had fully normalised (acuity −0.04 right, −0.1 left; stereopsis 55”; full extraocular movements), with no recurrence on follow-up. Bradford–Hill assessment (6/10 fulfilled) and WHO-UMC classification both supported a “probable” adverse drug reaction.

Discussion

Alemtuzumab-associated accommodative spasm represents a rare but plausible neuro-ophthalmic complication in transplantation. Early misattribution illustrates anchoring bias toward more common metabolic or calcineurin-related causes. The decisive role of cycloplegic refraction highlights a simple, low-cost diagnostic tool that can prevent unnecessary immunosuppression or drug withdrawal. Mechanistically, cytokine-mediated parasympathetic dysregulation during immune reconstitution may underlie the phenomenon. A single comparable case has been reported in multiple sclerosis, suggesting

that similar events in transplant recipients may be under-recognised or unreported. The absence of accommodative dysfunction in alemtuzumab product labelling or transplant AE registries highlights a pharmacovigilance blind spot. This case signals the need for broader surveillance of neuro-ophthalmic outcomes in immunosuppressed patients.

Implication
Transplant clinicians should consider accommodative spasm in patients with unexplained blurred vision and request cycloplegic refraction before attributing symptoms to metabolic derangements or drug toxicity.

Next step
Systematic incorporation of ophthalmic surveillance into transplant pharmacovigilance, registry-based adverse event reporting, and cross-specialty awareness in both nephrology and ophthalmology guidelines are needed to clarify incidence, mechanisms, and long-term outcomes of this rare complication.

THA10

Lightning strikes thrice: a case report of triple positive ANCA associated vasculitis

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THURSDAY - Moderated Poster Session, HALL Q, March 12, 2026, 10:00 - 11:00

Patients who co-present with seropositivity for both ANCA and anti-GBM antibodies, so called “double positive” patients, are not well characterised and are associated with more severe disease manifestations and poorer outcomes. Here, we describe an unusual case of a patient who presented with a rapidly progressive glomerulonephritis, “triple positive” for MPO, dsDNA, and anti-GBM antibodies.

A 54 year old lady of Chinese ethnicity had moved to the UK in 2021. Usually fit and well, six months previous she developed intermittent flu-like symptoms. This was followed by a six week history of progressive joint stiffness, nasal congestion, shortness of breath, fevers, and frothy urine.

At presentation she had deranged renal function (Cr 190) and haemoproteinuria and was treated for a presumed uncomplicated UTI. She re-presented with a further deterioration in renal function (Cr 275). She had no family history of renal disease or autoimmune disease. She had taken short courses of traditional Chinese medicine herbal remedies, ibuprofen, naproxen, and omeprazole for treatment of her joint stiffness.

Investigations on admission: urinalysis positive for blood (3+) and protein (2+). Urine PCR was raised at 254 mg/mmol and uACR was 149 mg/mmol. Immunology was positive for ANA (titre 1:80, speckled), dsDNA (13), MPO positive (100), anti-GBM (11), and anti-RO and anti-LA positive. ESR was markedly elevated at >100. Serum complements were normal and urine/serum electrophoresis unremarkable. HIV, hepatitis C antibodies, and EBV/CMV PCR were all negative. A haemolysis screen was negative. A renal tract ultrasound was unremarkable and CXR showed no evidence of pulmonary haemorrhage. There was no evidence of chronic infection or malignancy which has often been associated with broader autoantibody profiles.

She was treated with methylprednisolone, followed by a weaning course of prednisolone. Her renal biopsy showed crescentic glomerulonephritis with a vasculitic pattern and accompanying interstitial inflammation and acute tubular injury. Immunohistochemistry

revealed linear staining of the glomerular basement membrane with IgG but in the context of high background staining, so significance was uncertain. Electron microscopy showed diffuse podocyte foot process effacement. She was treated for Anca-Associated Vasculitis (AAV) with rituximab and cyclophosphamide: the prednisolone was later converted to avacopan. Hepatitis B core antibody was positive so she was also started on lamivudine prophylaxis. There was significant improvement in renal function and in autoantibody levels.

Double positive patients with both anti-GBM and ANCA antibodies are a poorly delineated population. In a retrospective cohort study described by McAdoo et al, patients display both the rapidly progressive clinical picture associated with anti-GBM disease, but also the risk of recurrence associated with AAV. Few cases have been described where other autoantibodies are also positive.

Patient's consent: the patient has given verbal documented consent and written consent for publication.

THA11

Is it truly benign? A case report of Cystitis Cystica et Glandularis progressing to irreversible renal failure

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Background

Cystitis cystica et glandularis (CCEG) is a common benign, proliferative lesion seen in the bladder mucosa usually as a result of chronic mucosal irritation or inflammation. It typically runs a benign course but rare instances of obstruction and one case of irreversible renal failure from CCEG have been reported. We report another unusual case of CCEG in a 41 year old male with obstruction and progression to renal failure.

Case Summary

A 41 year old man who presented to the emergency department with obstructive and irritative urinary tract symptoms. There was no history suggestive of urinary tract infection, hematuria, or stones. His only past medical history of note was pituitary tumor which was managed conservatively. Urinalysis was bland while serum creatinine was 106 µmol/l (egfr 76ml/min). CT scan showed bilateral hydro nephrosis and hydroureter, grossly distended urinary bladder and enlarged prostate but no stone was seen.

He was referred to the urologist and then catheterized. He had a cystoscopy which revealed a lesion in the trigone and neck of the bladder extending to the prostate. A transurethral resection of bladder tumor (TURBT) was done and microscopic examination of the resected 5 gram of tissue showed multiple portions of fibromuscular stroma including detrusor muscle, lined by cytologically bland urothelium and multiple areas of cystitis cystica et glandularis. There was no intestinal metaplasia, evidence of dysplasia or carcinoma in situ . A final diagnosis of cystitis cystica et glandularis was made and he was to be followed up with surveillance cystoscopies .

Flexible cystoscopy done annually for the next two years showed minor papillary changes in the lower part of trigone with no tumor recurrence. He however did not have repeat renal function bloods done during this 2 year period. He was planned for a repeat cystoscopy in the third year but was lost to follow up. He re-presented again after two years following a referral after bloods done by GP on account of feeling unwell and recurrence of lower urinary tract symptoms had shown an increase in creatinine to 770 µmol/l from his previously known baseline of 106 µmol/l . CT showed worsening of the previously seen bilateral hydro nephrosis and cystoscopy showed abnormal bladder mucosa at trigone with inability to visualize the ureteral orifice .

Bilateral nephrostomy tubes were inserted but unfortunately, this did not lead to improvement in renal function and he was subsequently commenced on hemodialysis via a tunneled jugular line.

Conclusion

This case highlights a very unusual course of CCEG, an ordinarily benign condition who initially presented with obstruction and stable renal function but then progressed to renal failure .The decline in renal function might have been detected earlier if he continued with his annual surveillance and had his kidney function monitored. This case buttresses the need for renal function monitoring in CCEG patients especially in cases of proven obstruction. This is probably the second case in the literature of CCEG progressing to irreversible renal failure suggesting that CCEG is not entirely benign.