retrieved and reviewed. The 2 cases diagnosed by our nephrology service on biopsy with PGN MID were included in the data analysis. Case 1 was of a 74-year-old male with chronic back pain status post spinal fusion surgery complicated by surgical site infection, treated with vancomycin and cefepime, who presented to nephrology consult service for elevated Cr from baseline and oliguria. Case 2 was of an immigrant 60-year-old female with history of hypertension, back pain taking NSAIDs who came with lower limb swelling and nephrotic range proteinuria who developed ESRD needing RRT. The diagnosis was confirmed with renal biopsy showing PGN MID.

PGN MID can have different presentations apart from the expected typical nephritic or nephrotic clinical picture. Our data review for 26 case series, including our 2 patients, showed that patients can present with AKI, CKD, proteinuria, and/or hematuria. Only a subset of these had underlying clonal cell proliferative disorder treated with immunotherapy and chemotherapy. The outcomes in these cases were variable. Our case 1 exhibited signs of renal recovery while our case 2 progressed to ESRD needing HD. Hence, it is important to recognize PGN MID as a differential in all patients with unclear underlying cause of AKI or CKD.

Renal biopsy is crucial in diagnosing PGN MID given its complexity, rarity, and clinical importance.

G-445. PAUCI-IMMUNE FOCAL CRESCENTIC AND NECROTIZING GLOMERULONEPHRITIS SUPERIMPOSED ON DIABETIC NEPHROSCLEROSIS: A RARE CASE REPORT:

Sohail Anwar[1], Ankur Singla[1], Jonathan Silverman [1]. [1]Northwest Health-Porter

Pauci-immune crescentic and necrotizing glomerulonephritis (PICNG) superimposed on diabetic nephrosclerosis is a rare but severe condition that mostly occurs in older populations. The prognosis is generally poor despite aggressive therapeutic interventions, and most patients ultimately require dialysis.
We present a case of rapidly progressive glomerulonephritis secondary to PICNG superimposed on diabetic nephrosclerosis.

A 57-year-old white female with a past medical history of long-standing diabetes mellitus type-2, hyperlipidemia, and active cigarette smoking, presented to the hospital with bilateral lower extremity swelling and frothy urine for 1 month. The patient had bilateral lower extremity edema, and hypertension on physical examination and had a serum creatinine of 6.64 mg/dL with a creatinine of 1.1 mg/dL 1 year ago. Urinalysis showed proteinuria(300 mg/dL) with numerous RBC and 21-30 WBC/HPF. Urine cultures were negative. Urine ACR was 7032 mg/g and 24-hour urine protein was 13 grams. ANA was positive but complement levels, hepatitis and HIV serologies, ASLO, and anti-GBM were all negative with normal serum immunofixation. ANCA serology yielded an elevated P-ANCA level at >1:640 and negative C-ANCA. CT scan of the abdomen and pelvis showed normal kidneys. The patient underwent a kidney biopsy, which showed pauci-immune focal crescentic, and necrotizing glomerulonephritis, and nodular diabetic glomerulosclerosis. There was moderate interstitial fibrosis and tubular atrophy. Six out of twenty eight glomeruli were globally sclerosed, and severe arterial hyalinosis and severe arteriolar intimal fibrosis were present. The patient received induction therapy with intravenous methylprednisolone 500 mg daily for 3 days followed by a tapering dose of oral prednisone and oral cyclophosphamide. The patient was started on hemodialysis due to volume overload and remains dialysis-dependent.

This case signifies that a high degree of clinical suspicion is needed for patients with a long-standing history of diabetes presenting with rapidly declining renal functions, microscopic hematuria, or worsening proteinuria to diagnose superimposed glomerulonephritis on diabetic nephropathy and the role of timely intervention in improving clinical outcomes.

G-446. MEMBRANOUS-LIKE GLOMERULOPATHY WITH MASKED IGG KAPPA AND C3 DEPOSITS: A NOVEL CLINICAL ENTITY:

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Membranous-like glomerulopathy with masked IgG kappa and C3 deposits is a relatively new and poorly understood condition. It's typically seen in young women, often surfacing with vague autoimmune features. In many cases, like the one presented here, proteinuria is first detected during pregnancy, raising interesting questions about its pathogenesis.

We present a 24-year-old Caucasian woman, recently postpartum, who came to the clinic for follow-up after a complicated pregnancy marked by severe preeclampsia. She had nephrotic-range proteinuria (8 g/24 hours) and hypertension during her hospital stay. Interestingly, her history revealed episodes of hematuria and proteinuria dating back several years. Her family history was notable for autoimmune conditions, including celiac disease and Hashimoto's thyroiditis, as well as polycystic kidney disease.
A kidney biopsy was performed, showing glomerular changes consistent with membranous-like glomerulopathy. Immunofluorescence on paraffin-embedded tissue, using antigen retrieval, revealed masked IgG kappa and C3 deposits.

This case highlights the importance of considering membranous-like glomerulopathy with masked IgG kappa and C3 deposits in young women with proteinuria, especially those with pregnancy complications like preeclampsia. Although the patient lacked overt autoimmune symptoms, her family history of autoimmune diseases suggests a genetic predisposition. The use of antigen retrieval immunofluorescence was crucial in detecting the weakly staining deposits, which are difficult to identify without this technique.

The emerging recognition of membranous-like glomerulopathy with masked IgG kappa and C3 deposits is particularly important in young women presenting with pregnancy-related proteinuria and a family history of autoimmune conditions. The use of advanced diagnostic techniques such as antigen retrieval immunofluorescence was crucial in identifying this novel glomerulopathy. Recognizing this condition is critical for early diagnosis and management, and future research is needed to better understand its pathophysiology, clinical course, and long-term outcomes in affected individuals.

G-447. AFFINITY STUDY: 1Y RESULTS OF ATRASENTAN IN IGAN IN PATIENTS WITH UPCR <1 AND ≥1G/G:

Jonathan Barratt[1], Nam Dai Vo[2], Sangho Lee[3], Sung Gyun Kim[4], Carol Pollock[5], Dwarakanathan Ranganathan[6], Sylvie Le Mouhaer[7], Soudeh Ansari[8], Ruth Haile-Meskale[8], Helen Twiston Davies[9], Yasmin Brahmbhatt[8], Hiddo J. L. Heerspink[10]. [1]University of Leicester; [2]Mountain Kidney and Hypertension Associates; [3]Kyung Hee University; [4]Hallym University Sacred Heart Hospital; [5]Royal North Shore Hospital; [6]Royal Brisbane and Women's Hospital; [7]Novartis Pharma SAS; [8]Novartis Pharmaceuticals Corporation; [9]Novartis Pharmaceuticals; [10]University Medical Center Groningen

Atrasentan, a potent, selective endothelin A receptor antagonist, is being investigated to treat IgAN and other kidney diseases (KD).

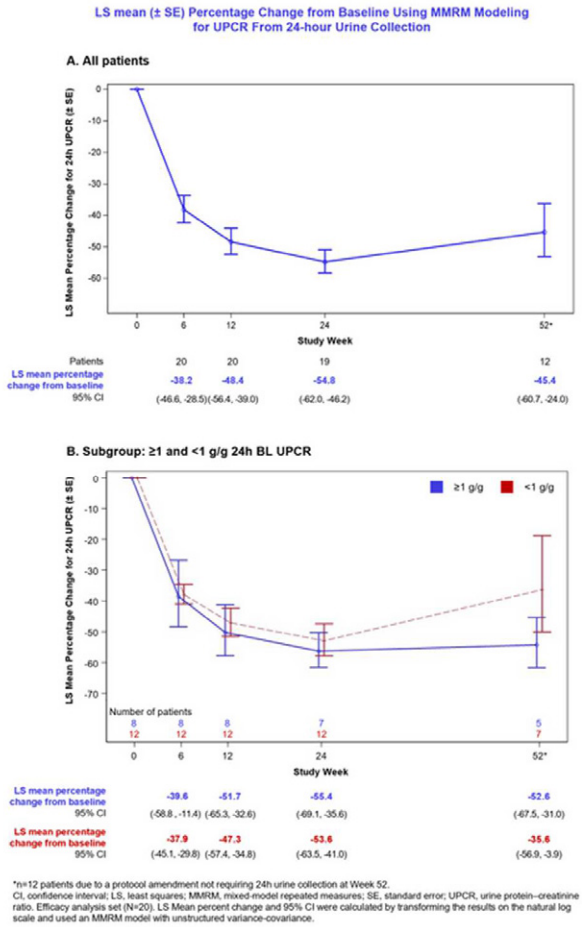
AFFINITY (NCT04573920) is a Phase 2, open-label basket trial of atrasentan in patients (pts) with KD. The IgAN cohort included adults with biopsy-proven IgAN; eGFR ≥30 mL/min/1.73m^2, UPCR ≥0.5 and <1g/g (first morning void at screening), and on maximum tolerated/stable RASi for ≥12 wks. Pts took 0.75mg oral atrasentan daily for 52 wks. The primary endpoint was change in 24h UPCR from baseline (BL) to Wk 12.

In pts with IgAN (N=20; median age 44.5y, 50% women, 45% White, 45% Asian), BL median 24h UPCR was 0.8g/g; 12 pts had BL UPCR <1g/g.

Reduction in UPCR was evident by Wk 6 and sustained through Wk 52 (Fig 1A). Clinically meaningful reductions in UPCR were evident in pts with BL UPCR <1 and ≥1g/g through Wk 52 (Fig 1B). At BL, Wk 12, and Wk 24, 1/20 (5%), 12/20 (60%), and 13/19 (68%) pts had UPCR <0.5g/g, respectively.

One pt discontinued treatment (tx) at Wk 13 due to an AE of headache considered tx-related. There were no tx-related serious AEs or deaths.

Atrasentan was well tolerated and resulted in a stable, clinically meaningful reduction in proteinuria over 1y of tx, comparable between pts with BL UPCR <1 and ≥1g/g.



*n=12 patients due to a protocol amendment not requiring 24h urine collection at Week 52.
CI, confidence interval; LS, least squares; MMRM, mixed-model repeated measures; SE, standard error; UPCR, urine protein–creatinine ratio. Efficacy analysis set (N=20). LS Mean percent change and 95% CI were calculated by transforming the results on the natural log scale and used an MMRM model with unstructured variance-covariance.

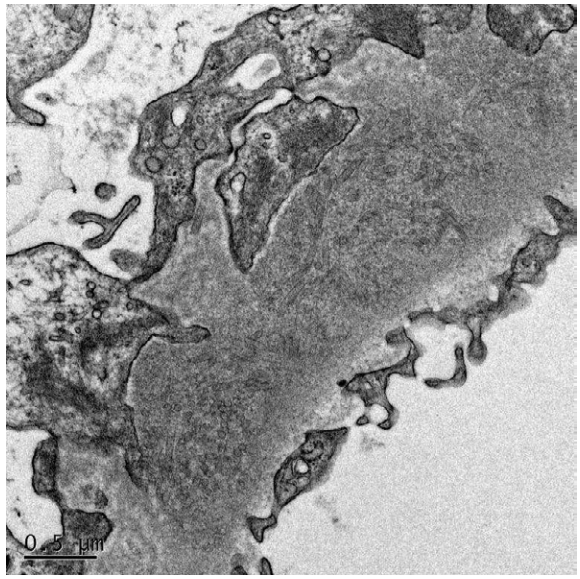
G-448. IMMUNOTACTOID GLOMERULOPATHY AS A PRESENTATION OF UNTREATED CHRONIC LYMPHOCYTIC LEUKEMIA (CLL):

Shreeyukta Bhattarai[1], Elizabeth Cho[1], Kammi Henriksen[2], Basma Merhi[1]. [1]Brown University Health ; [2]University of Chicago Medical Center

Immunotactoid Glomerulopathy (ITG) is rare in glomerulonephritic diseases, accounting for 0.06% of native renal biopsies. Diagnosis is made by renal biopsy with pathognomonic electron-dense immunoglobulin deposits and microtubules. We present a rare case of ITG in an untreated CLL patient.

A 60-year-old Caucasian female with untreated CLL referred for anasarca and nephrotic range proteinuria. Patient was diagnosed 1 year ago with stage 1 CLL after a supraclavicular lymph node and bone marrow biopsy and was treated conservatively. Upon her presentation, blood pressure was 220/98 with facial and 2+ lower extremity pitting edema on exam. Labs showed WBC 18.6, BUN 9 mg/dL, creatinine 0.58 mg/dL, albumin 2.9 g/dL, total protein 5.5 g/dL. Urinalysis had >500 protein with 18 RBC and 24-hour urine protein of 6.6 g/day. Serologies had low C3 43mg/dl (NL: 83-193) and C4 13mg/dL (NL: 15-57), negative ANA, anti-dsDNA, anti-PLA2R, hepatitis B & C, cryoglobulin, and HIV. Urine protein electrophoresis and immunofixation were negative with normal kappa/lambda ratio. Renal ultrasound was unremarkable. Renal biopsy showed membranoproliferative glomerulonephritis with monoclonal IgG kappa light chain deposits and microtubular substructure (Figure) consistent with ITG. She was treated with Obinutuzumab and Venetoclax for her CLL. Ten months later, she had complete resolution of her nephrotic syndrome with negative blood and protein in urinalysis.

Monoclonal ITG is extremely rare but well-known to be associated with hematologic malignancies. It is important to consider it as a differential to expedite diagnosis and treatment. In our patient, the diagnosis of ITG as a renal manifestation of CLL resulted in successful treatment.



G-449. PRIMARY MEMBRANOUS NEPHROPATHY IN A KIDNEY DONOR AFTER COVID-19 INFECTION:

Elizabeth Cho[1], Shreeyukta Bhattarai[1], Basma Merhi[1]. [1]Brown University Health

Kidney dysfunction in COVID-19 is mostly associated with acute tubular injury and collapsing glomerulopathy. Case reports highlight new or recurrent membranous nephropathy (MN) post COVID-19 and those reported were PLA2R negative. We present a case of a kidney donor who developed positive PLA2R MN after COVID-19, 2 years post-donation, with complete remission after treatment.

A 41-year-old female underwent left donor nephrectomy after a normal work-up pre-donation. Post-donation course was unremarkable, with creatinine 0.87 mg/dL, negative blood and protein in urinalysis (UA). Two years later, she had nephrotic range proteinuria after upper respiratory tract infection due to COVID-19. UA showed > 500 protein and 5 RBC with no dysmorphic RBC on urine sediment. Urine protein/creatinine ratio (UPCR) 8.8 g/g, albumin 3.1 g/dL and creatinine 0.8mg/dL. Renal US was normal. She had periorbital and bilateral pitting lower extremity edema; no rash, oral ulcers or joint pain. Serologies with ANA, anti-ds-DNA, C3/C4, hepatitis profile and HIV were negative. Kidney biopsy was deferred due to solitary right kidney and anti-PLA2R IgG titer 1:1280 (Ref interval < 1:10). She was diagnosed with primary MN provoked by COVID-19. Two months later, she had right flank pain with renal infarcts on CT abdomen/pelvis (Figure 1). She was treated with prednisone 1 mg/kg, Rituximab and 6 months of Eliquis. Ten months later, she was in complete remission with UPCR <1 g/g, undetectable anti-PLA2R titers, albumin 3.6 g/dL and creatinine 0.8 mg/dL.

Clinical course and outcomes of MN following COVID-19 are limited and treated with close monitoring and conservative management. Due to high PLA2R titers in our donor patient 2 weeks after COVID-19, we avoided kidney biopsy and treated with immunosuppression. She achieved complete remission of her nephrotic syndrome and maintained a stable kidney function even after renal infarcts.