Claire Buchan[3], Tayler Buchan[3], Helen Trenz[2], Titte R Srinivas[2], Pietro Canetta[4]. [1]Baylor Scott and White Medical Center; [2]Novartis Pharmaceuticals Corporation; [3]Asclepius Analytics; [4]Columbia University

IgAN and C3G are chronic glomerulopathies in which complement dysregulation drives pathology. Iptacopan, a proximal complement inhibitor that specifically binds factor B to inhibit alternative pathway activity, received accelerated approval from the US Food and Drug Administration for IgAN, and a Phase 3 study in C3G (NCT04817618) is completed. Atrasentan is a potent and highly selective endothelin A receptor antagonist with an ongoing Phase 3 IgAN study (NCT04573478). Existing IgAN and C3G RWD sources have limitations like poor geographic and insurance coverage, low availability and linkage of lab and treatment data, and data lag. Here, we describe APPRISE, intended to be the largest, most comprehensive RWD source on US pts prescribed iptacopan and/or atrasentan.

APPRISE will be a US-wide, living data platform including retrospective, longitudinal, deidentified pt-level data from adults with IgAN or C3G prescribed iptacopan and/or atrasentan (on approval). Patients will be recruited through iptacopan/atrasentan distribution networks and provide written consent to be enrolled. APPRISE will capture pt demographic and clinical characteristics, lab values, medical history, treatment patterns, and clinical outcomes. Recruitment of pts with IgAN began after iptacopan approval in 2024 and will expand with future approvals. The study period will extend from treatment initiation for the first pt to end of follow-up for the last pt.

Analyses, including of clinical characteristics and treatment patterns, adherence, and effectiveness, will be conducted continuously and refined as sample sizes become sufficient.

APPRISE will enable rapid collection of varied RWD from pts prescribed iptacopan and/or atrasentan to overcome limitations of existing RWD sources, characterize real-world disease progression and treatment patterns, and support future studies to provide valuable insights into IgAN and C3G outcomes.

G-473. DESIGN OF A DISEASE-MANAGEMENT APP TO MEASURE HOME-REPORTED OUTCOMES (HROS) IN PATIENTS (PTS) WITH IGA NEPHROPATHY (IGAN) AND C3 GLOMERULOPATHY (C3G):

Pietro Canetta[1], Hiba Anwar[2], Luke Stephens[2], Rahul Khairnar[3], Connie Zhang[2], Samantha McStocker[2], Amanda Healey[2], Jordan Owashi[2], Helen Trenz[3], Anne Alfano[4], Aran Rice[5], Briana Ndife[3], Mohanram Narayanan[6]. [1]Columbia University; [2]Folia Health; [3]Novartis Pharmaceuticals Corporation; [4]N/A; [5]N/A; [6]Baylor Scott and White Medical Center

IgAN and C3G are rare, chronic glomerulopathies; there is a lack of published evidence on the humanistic burden of these diseases. Methods that capture the full spectrum of disease burden are needed as pts with the same diagnosis may experience different symptoms or prioritize different aspects of their condition. The Folia Health (FH) app allows pts to track HROs (structured, quantitative observations) that are most relevant to their own disease experience. Here we describe how pt and clinician input was used in the design of 2 condition-specific (IgAN and C3G) platforms within the FH app, for use in future researchstudies.

Semi-structured interviews were conducted with clinicians and with pts with IgAN or C3G. Interviews were designed to ensure pt and clinician insights shaped key elements of the app design, such as the identification of relevant and/or lesser-known symptoms, treatments, and health-related quality of life (HRQoL) impacts.

Interviews were conducted with 2 clinicians, 3 pts with IgAN, and 3 pts with C3G. Interviews informed a curated list of disease-specific symptoms, treatments, flare events, and HRQoL impacts, all tailored to optimize the FH app for health tracking in these conditions. Symptoms and treatments suggested for tracking will be presented, including symptoms across behavioral, neurological, dermatological, pain, and urinary health domains. These will enable researchers to comprehensively assess treatment impacts beyond traditional pt-reported outcomes.

The condition-specific platforms within the FH app, informed by pt and clinician insights, address the unique needs of pts with IgAN and C3G by enabling the tracking of tailored HROs. In future studies, participants will use the app to capture aspects of their disease that matter most to them, supporting both effective disease management and meaningful research participation.

G-474. THE NEPHRIN LINK TO THE MAXIMUM EFFECT OF MINIMAL CHANGE DISEASE:

Joseph Ogunsulire[1], Amber Bullock[1], Somtochukwu Godwin-Offor[1], Divya Akella[1], Omonigho Sonia Nwoke[1], Leighton James[1]. [1]Wellstar, Medical College of Georgia

Nephrotic syndrome (NS) is a constellation of findings which include nephrotic range proteinuria, low albumin, with hyperlipidemia and edema. NS is usually due to podocyte damage, causing the extensive protein leak. It can also lead to acute kidney injury and frequently requires a kidney biopsy for definite diagnosis of underlying cause.

This is a 72-year-old male with a past medical history significant for hypertension (HTN), hyperlipidemia and type 2 diabetes mellitus (DM2) who was sent from primary cares providers office for worsening weight gain and shortness of breath with edema. The Patient reported a 50-pound weight gain in the last 4 months, associated with significant joint swelling. He denied rash or ulcer(s).

Result of Initial laboratory showed creatinine of 2.6 mg/dL from baseline of 0.9 mg/dL consistent with acute kidney injury (AKI), Albumin was 2.3 g/dL and urinalysis and urine microscopy showed moderate blood but no RBCs, greater than 600 mg/dL protein with spot urine protein to creatinine ratio of 7.8 g/g. Serology was negative. He ultimately had a kidney biopsy which showed minimal change disease, as well as acute tubular injury and diabetic glomerulopathy. Immunofluorescene showed staining for IgG nephrin autoantibodies.

Podocytes foot process interdigitate to form the slit diaphragm which are made up of several podocyte-specific proteins including podocin and nephrin. There is increasing evidence and cases showing a link between nephrin antibody and minimal change disease, as well as, FSGS. The primary driving factor(s) for this patient's presentation is/are unclear especially given significant overlapping causes of proteinuric kidney disease in this patient including HTN and DM2.

This case is another example of the link between nephrin antibody. podocytopathies and proteinuric kidney disease. It also highlights the possibility of a multifactorial cause of kidney disease, which requires different treatment strategies.

G-475. RELENTLESS RELAPSE: A CASE OF RECURRENT IDIOPATHIC MEMBRANOUS NEPHROPATHY (MN) IN A KIDNEY TRANSPLANT RECIPIENT:

Ji-Min Park[1], Mai Nguyen[1]. [1]Brooke Army Medical Center

MN is a common glomerular disease, which may be associated with significant morbidity and mortality. The course of the disease can be variable, with some patients achieving remission with conservative therapy while 50% of untreated MN eventually lead to end stage kidney disease (ESKD). We present a challenging patient with history of idiopathic MN, who received a kidney transplant and has had multiple relapses of MN despite guideline-directed medical therapy.

Patient is a 59 year-old Caucasian man who presented with nephrotic syndrome in 2000 and was diagnosed with idiopathic MN by kidney biopsy. He did not respond to multiple immunosuppressive agents. He subsequently underwent living-unrelated donor kidney transplantation (LUKT) in 2008 and was maintained on Tacrolimus/Mycophenolate mofetil for immunosuppression. In 2016, he was found to have nephrotic range proteinuria (12g in 24 hr urine) on routine labs.

Serologic workup was negative for anti-PLA2R Ab, anti-THSD7A Ab, hepatitis B/C and HIV. Colonoscopy was normal. Transplant biopsy demonstrated stage II MN so he was treated with Rituximab with resolution of proteinuria with urine protein:creatinine ratio (UPCR) of 0.12. Unfortunately, he experienced increasing proteinuria and elected "empiric" treatment with Rituximab in 2019, which again resulted in complete remission (UPCR 0.17). In 2023, proteinuria returned (UPCR 1.09), and repeat transplant biopsy showed recurrence of MN with evidence of obesity-related changes, transplant glomerulopathy without evidence of rejection, and mild interstitial fibrosis and tubular atrophy (IFTA). Despite achieving clinical remission following each course of Rituximab on a background of Tacrolimus/Mycophenolate/Telmisartan, this patient has exhibited a relapsing course.

It is important to recognize that MN can recur in kidney allografts, and further research is necessary to identify risk factors for recurrence