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A 65-year-old female presented to a nephrology clinic for evaluation of worsening renal dysfunction. The patient is well known to have systemic sarcoidosis under complete remission on low dose prednisone and likely membranous nephropathy (no previous biopsy) with mildly elevated phospholipase A2 receptor (PLA2R) antibodies. Her membranous nephropathy was in partial remission on angiotensin receptor blockade, with urine to protein creatinine ratio (UPCR) of 1.5 g/dL. Five months after receiving the single dose SARS-CoV-2 vaccine (Johnson & Johnson®), she started having a flare up of her systemic sarcoidosis with worsening joint, skin and respiratory symptoms. Blood chemistry revealed worsening renal dysfunction with elevated creatinine up to 1.7 mg/dL from her baseline of 1.0 mg/dL. UPCR was also elevated at 3.4 g/dL. Urine sediment revealed no red blood cells or casts, only several calcium oxalate dihydrate crystals. A kidney biopsy was performed and showed a combination of membranous nephropathy (PLA2R positive) along with granulomatous interstitial nephritis with well-formed epithelioid granulomas characteristic of sarcoidosis. She was started on high dose prednisone and her renal function improved to 1.2 mg/dL. UPCR decreased to 1.8 g/dL and serum PLA2R antibodies became undetectable. She is still being monitored.

After many years of renal sarcoidosis and membranous nephropathy remission, the relapse of renal disease after receiving the SARS-CoV-2 vaccine (Johnson & Johnson®) suggests the

HYPEROAGULOPATHY AND ASCITES INDUCED BY PRIMARY MEMBRANOUS NEPHROPATHY IN A PATIENT WITH SARCOIDOSIS:
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Although rare, sarcoidosis can cause membranous nephritis (MN). [1] Herein, we present a case of a patient with sarcoidosis who developed MN with elevated PLA2R Ab and subsequently improved with RAS inhibition and Rituxan. A 49-year-old male with sarcoidosis that was treated a year prior for inferior vena cava (IVC) thrombus was evaluated for worsening ascites and lower extremity edema.

Labs on admission revealed a normal serum calcium as well as renal function with serum albumin of 0.7 mg/dL. A paracentesis was performed which was consistent with transudative ascites. Urine dipstick had 3+ proteinuria and urine to creatinine (UPC) ratio of 17.5 g/day. Urine was devoid of RBCs and WBCs. Autoimmune workup including ANA, anti-smooth muscle Ab, c- and p- ANCA, IgG4, and IgA was negative; infectious workup was also negative. Doppler ultrasound showed patent IVC. His serum phospholipase A2 receptor (PLA2R) antibody was significantly elevated at 101 RU/mL.

Because patient had a history of sarcoidosis, a renal biopsy was performed showing primary membranous nephropathy with glomeruli positive reaction to PLA2R and IgG4 Abs. Two weeks after his first Rituxan infusion, his symptoms improved and his proteinuria decreased to 5.5 g/day.

Sarcoidosis commonly causes tubular interstitial nephritis. Rarely, primary membranous glomerular nephritis can coexist with sarcoidosis. In secondary MN, PLA2R abs is infrequently seen but with sarcoidosis, it may incite PLA2R antibody activation causing primary MN. [1-2]

RELAPSE OF MEMBRANOUS NEPHROPATHY AND RENAL SARCOIDOSIS FOLLOWING EXPOSURE TO SARS-COV-2 VACCINE (JOHNSON & JOHNSON®):
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The role of infectious agents derived antigens including severe acute respiratory syndrome coronavirus 2 (SARS-CoV-2) has been recognized as a trigger for development of autoimmune mediated disorders following natural infection or immunization. However, there is a scarcity of reports of occurrence of autoimmune associated kidney disorders or flare ups following exposure to a SARS-CoV-2 vaccine. A well-designed clinical trials are needed to answer these questions.

nephrotic syndrome patients due to a poor quality of evidence. Well-designed clinical trials are needed to answer these questions.
association between receiving the vaccine and the recurrence of renal sarcoidosis and membranous nephropathy.

ESKD PATIENT EXPERIENCES DURING THE COVID-19 PANDEMIC: A SURVEY STUDY;
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End stage kidney disease (ESKD) patients are vulnerable to medical and psychosocial difficulties. We aimed to identify specific concerns for ESKD patients during the early months of the COVID-19 pandemic. Surveys were administered to adult ESKD patients receiving dialysis in three units run by a large dialysis organization affiliated with an academic nephrology practice. Multiple choice and open-ended questions were used to assess patients’ perceptions of access to care and essentials of daily living, and effects of changes in dialysis schedules or prescriptions. Screening questions were used to assess patient anxiety and depression.

172 ESKD patients on dialysis were surveyed. Participants on home dialysis modalities (peritoneal dialysis (PD) or home hemodialysis (HHHD)) more commonly reported feeling “very connected” to their dialysis care teams compared to patients on in-center hemodialysis (ICHD) (PD: 74.1%; HHHD: 66.7%; ICHD: 62.3%). Patients who identified as White more commonly reported feeling “very connected” compared to patients who identified as non-White (White: 74.4%; Black/African American: 60.5%; Hispanic: 69.6%). Patients with histories of anxiety or depression more commonly reported feeling less cared for during the pandemic. 16.9% of participants reported new transportation issues, 6.4% reported difficulty obtaining medications, and 9.3% reported difficulty getting groceries. A minority of patients met screening criteria for depression or anxiety, though patients with self-reported histories of anxiety or depression had higher screening scores. Five themes emerged as influencing patient experiences: 1) the positive influence of relationships with dialysis staff; 2) the value of interactions with family or other caretakers; 3) difficulties with access to care; 4) changes in physical and mental health; and 5) awareness of, and response to, the COVID-19 pandemic.

Our study identifies sub-populations of ESKD patients who may be more vulnerable during the COVID-19 pandemic: those with histories of anxiety or depression, non-White patients, and patients on ICHD. Use of home dialysis modalities may be associated with better patient perceptions of care.

END-STAGE KIDNEY FAILURE IN AN AMISH FAMILY DUE TO A HETEROZYGOUS PATHOGENIC VARIANT IN THE INF2 GENE ENCODING INVERTED FORMIN 2:
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Approximately 10% of adults with chronic kidney disease have a monogenic cause. Focal segmental glomerulosclerosis (FSGS) of unknown cause is more likely to have a genetic etiology in the setting of treatment resistant, progressive kidney disease in children or adults under 50 years. Identifying genetic FSGS is often followed by medical management changes, and accurate familial recurrence counseling can be given. Genetic FSGS can be caused by more than 50 genes, including INF2. We report an adult Amish male with a pathogenic variant in INF2, and a significant family history of end-stage kidney disease (ESKD) with recurrent kidney transplant.

A 30 year old male self-referred to Renal Genetics Clinic at the Cleveland Clinic for evaluation due to his family history of ESKD. One uncle had kidney transplanted due to FSGS. Physical exam showed blood pressure 130/80mmHg. Labs showed mild proteinuria with protein/creatinine ratio 0.4 and estimated glomerular filtration rate 99 mL/min/1.73m2. With a history of family history and mild proteinuria, a 72 gene next-generation sequencing FSGS panel was performed which identified a heterozygous pathogenic variant (c.653 G>A, R218Q) in the INF2 gene. With this genetic diagnosis, patient was initiated on Lisinopril and thorough genetic counseling was offered including targeted genetic testing for his at-risk family members.

Pathogenic variants in INF2 cause progressive kidney disease. A minority of cases also present with neuropathy (Charcot-Marie-Tooth disease). Recurrent transplant is not common for monogenic causes of FSGS, however multiple family members of this patient have needed multiple kidney transplants, which deserves further investigation. The prevalence of monogenic kidney disease in the Amish population is not well studied, and to our knowledge this is the first report of INF2-related kidney disease in an Amish family.

PAYMENT POLICY SUPPORT FOR INTEGRATING HEALTHY EATING AND HEALTHY MOVING WITH TRADITIONAL MEDICAL CARE FOR CHRONIC KIDNEY DISEASE CARE:
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Chronic kidney disease (CKD) is due to diabetes and/or hypertension in almost two-thirds of cases in the U.S. Overall outcomes for diabetes2 and hypertension3 improve with integration of healthy eating and increased physical exercise (healthy moving) with conventional medical care. Integrating healthy eating with conventional medical care for patients with CKD due to diabetes and/or hypertension might reduce adverse outcomes. Individuals of low socioeconomic status (SES) are at disproportionately high risk for CKD4 and Medicaid is the US Federal government program designed to support health needs of those of low SES.

We examined if there were offerings within Medicaid that might support integration of healthy eating and healthy moving with traditional medical care in the treatment of individuals with diabetes and/or hypertension.

Medicaid is increasingly exercising regulatory options to address health needs of the population it serves that go beyond provision of conventional medical care through non-medical drivers of health, including healthy eating and healthy moving.5 Many state Medicaid programs are addressing these needs through managed care programs and other authorities.6 Some states use flexibility with Medicaid to support provision of home delivered meals5 and fund statewide infrastructure to support healthy eating.7 Medicaid also provides grants to support incentives for its participants to join programs for healthy moving.8 Medicaid provides opportunities to states to support integration of healthy eating and healthy moving with conventional medical care in the treatment of individuals with diabetes and/or hypertension associated with CKD. This integration holds promise to reduce increasing adverse outcomes in all patients with CKD and should be explored as a mechanism to reduce adverse outcomes especially in low SES populations whose health needs are supported by Medicaid.

SEVERE HYponATREMIa AND PREECLAMPSIA:
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Hyponatremia in pregnancy, defined as serum sodium (Na) <135mEq/L, is a rare but important marker of severe preeclampsia.

We present a 35 year old G1P0, with migraines and pregnancy-associated hypothyroidism at 33 weeks gestation of twin pregnancy (vanishing twin at 8 weeks), who was admitted for intrauterine growth restriction and preeclampsia. One week prior to admission she was diagnosed with gestational hypertension with blood pressures ranging in the low 130’s. Upon admission, blood pressure was 142/86 and physical exam notable for +1 pitting edema bilaterally. Laboratory studies were significant for a serum sodium (Na) of 119 with a serum osmolality of 246, creatinine was 0.51mg/dl, uric acid was elevated at 7.6 and TSH was normal. Urine protein/creatinine was 0.3, urine osmolality