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identified variants affect the C-terminal half of the cubulin protein, which is associated with benign chronic proteinuria in the absence of IGS. The early use of genetic screening in the diagnostic workup of proteinuria in the pediatric setting can minimize the need for further invasive testing even in cases without suspicious family history. Future studies of these patients are necessary to inform long-term outcomes and management of proteinuria.

PERICARDIAL EFFUSION: A POTENTIAL LATE COMPLICATION OF COVID-19 INFECTION: Olusola Sofegen1, Ann Kathleen Gamilla-Crudo2, Muhammad A. Mujtaba3, Syed A. Hussain3. University of Texas Medical Branch COVID-19 infection involves entry of SARS-CoV-2 virus into cells via interaction between its spike protein and angiotensin converting enzyme resulting in an NF-kB mediated inflammatory response. Cardiac manifestations without pulmonary symptoms is uncommon but has been described in the literature during an acute infection. We report a rare case of a potential late cardiac complication months after an acute COVID-19 infection. A 62-year-old male with hypertension and end stage renal disease on hemodialysis three times a week. Patient presented with fever, anemia and myalgia. He denied chest pain or respiratory symptoms. Patient tested positive for COVID-19 and received only treatment of symptoms. Over the next nine months he reported persistent fatigue and new onset of shortness of breath. He continued dialysis without interruption. His symptoms progressed resulting in hospital admission. All laboratory investigations, including BUN (27mg/dL), were within normal limits. Chest X-ray revealed cardiomegaly. Echocardiogram showed a large pericardial effusion without tamponade. Pericardiocentesis was accomplished with removal of 1700 mL of bloody fluid. Cell count, LDH, protein and glucose was normal. Fungal, viral, aerobic and anaerobic cultures of the pericardial fluid was negative. No malignant cells detected. Serial echocardiograms at 1, 3 and 5 months revealed a persistent small pericardial effusion. Cardiac manifestations of SARS-CoV-2 includes myocarditis, pericarditis and pericardial effusions. In case reports, the presence of the cardiac inflammatory state occurred simultaneously with an acute COVID-19 infection. In our case the COVID-19 infection occurred over nine months earlier yet remains a plausible explanation for his hemorrhagic pericardial effusion due to the absence of other identified causes. Further, COVID-19 molecular PCR testing of pericardial testing remains low yield due to its specific development for nasopharyngeal swab sampling. Cardiac manifestations of SARS-CoV-2 infection typically occur at the time of diagnosis. A late cardiac complication of COVID-19 may include pericardial inflammation with effusion. Further data and testing needs to be developed to confirm the diagnosis and guide therapy.

TRANSPANT GRAFT TMA INJURY POST COVID VACCINATION: Olusola Sofegen1, Ann Kathleen N. Gamilla-Crudo1, Muhammad A. Mujtaba3, Syed A. Hussain3. University of Texas Medical Branch Vaccination against SARS-CoV-2 is essential. Complicating this effort are reports of a suboptimal response to the SARS-CoV-2 spike protein in patients on immunosuppressive medications and possible thrombotic microangiopathy (TMA) in renal transplant patients. A 48-year-old male who received a living unrelated transplant in 2015. Pre-operative creatinine was 10.42 mg/dL and decreased to 2.48 mg/dL within a week. Patient received Basiliximab induction and maintained on tacrolimus and mycophenolate (MMF). One month post-transplant patient was diagnosed with TMA. Tacrolimus was stopped and patient was switched to Sirolimus and continued on MMF. Patient was followed closely by transplant nephrology for the next 5 years with a baseline creatinine of 1.9 mg/dL, protein to creatinine ratio below 0.5 mg/mg and well controlled diabetes. No DSA Class I or II detected on regular testing. Patient was compliant with all prescribed medications. On January 25 2021 patient received Pfizer Vaccine. Second Pfizer vaccine administered on February 18 2021. A week later creatinine was noted to be 3.44 mg/dL. Repeat creatinine of 4.27 mg/dL. Biopsy revealed diffuse lymphocytic interstitial inflammation, perihilar capillaritis, and C4D negative. Findings consistent with chronic TMA. DSA testing revealed Class II DSA:DQs (SI: 5933), Allosure 1.2 %. BK < 500 and CMV undetected. Patient received therapeutic plasma exchange, IV Ig infusion and steroids while on MMF and sirolimus. His creatinine decreased to 2.9 mg/dL on discharge. Over the next 6 months graft function deteriorated. He is now CKD stage 5 and under evaluation for a second transplant.

EVALUATING CLINICAL OUTCOMES IN PATIENTS (PTS) WITH CKD WITH RAPID AND NON-RAPID EGF: DECLINE: A REPORT FROM THE DISCOVER CKD RETROSPECTIVE COHORT: H Heerspink1, G James1, S Nolan1, JJ Carrero2, Matt Arnold2, R Pecotis-Filho3,4, JJ Garcia Sanchez5, CSP Lam6,7, H Chen6, E Kanda8, M Lainscak9, C Pollock10, DC Wheeler11, 1University of Groningen; 2AstraZeneca; 3Karolinska Institute; 4Pontifical Catholic University of Parana; 5Arbor Research Collaborative for Health; 2Department of Cardiology, National Heart Centre; 6Duke-NUS Medical School; 7AstraZeneca; 8Kawasaki Medical School; 9General Hospital Murska Sobota and University of Ljubljana; 10Kolling Institute, Royal North Shore Hospital, University of Sydney; 11University College London. CKD progression is associated with poor clinical outcomes, yet there is limited evidence evaluating clinical outcomes in pts with rapid and non-rapid eGFR decline. Data were extracted from the US TriNetX hospital-EMR and integrated Limited Claims and Electronic Health Record (LCCD) data (IBM Health, Armonk, NY), UK Clinical Practice Research Datalink (CPRD-GOLD) linked to hospital data, and Japan Medical Data Vision (JMDV) databases. Eligible pts were aged ≥18 yrs (≥20 yrs for JMDV), with 2 consecutive eGFR measurements 5-75 mL/min/1.73 m2 recorded 90-730 days apart. Index date was the 2nd eGFR. Linear mixed-effect models were used to calculate 2-patient-level eGFR slopes using the CKD-EPI equation. Pts with an annual eGFR decline ≥4 mL/min/1.73 m2 were considered rapid progressors. Adjusted Cox proportional hazard models were used to compare the risk of all-cause mortality, renal composite, MACE and all-cause hospitalizations after 2-years post index, in pts with rapid vs non-rapid eGFR decline. The study cohort included 1-1.2 M pts. Rapid eGFR decline occurred in 13.8% of pts overall who had a greater comorbidity burden. Risk of adverse clinical outcomes was significantly higher for pts with rapid eGFR decline (except MACE in US LCCD), Table 1. Approximately 1 in 10 pts in the DISCOVER CKD retrospective cohort experienced rapid eGFR decline which was associated with a significantly higher risk of adverse clinical outcomes.

COMPARING CHARACTERISTICS OF PATIENTS (PTS) WITH CKD AND HYPERKALEMIA (HK) WITH AND WITHOUT A DIAGNOSTIC (DX) CODE FOR HK: A REPORT FROM THE DISCOVER CKD RETROSPECTIVE COHORT: C Pollock1, 1G James2, JJ Carrero2, M Kherzian2, S Fishbane3, H Chen2, E Kanda4, K Jäbrink5, CSP Lam6,7, M Lainscak9, P Stenvinkel10, DC Wheeler11, 2R Pecotis-Filho3,4, 5Kolling Institute, University of