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outcome was inpatient mortality. The secondary outcomes were length of hospital stay and hospitalization costs. STATA software was used for analysis. Multivariate logistic and linear regression analysis were used accordingly to adjust for confounders.

There were over 71 million discharges in the combined 2016 and 2017 NIS database. Of the 108,670 hospitalizations for UGIB, 10% had coexisting CKD stages III-V, while 5.3% had coexisting ESRD. Those hospitalized for UGIB with concomitant ESRD had statistically significant higher odds of inpatient mortality (AOR 1.85, 95% CI [1.14-3.01], P = 0.023) compared to those with CKD stages III-V. There was also a statistically significant difference in adjusted mean total hospital charge with ESRD hospitalizations spending $10,076 more than hospitalizations for CKD stages III-V (95% CI [3256-16896], P = 0.004) despite there being no significant difference in adjusted length of hospital stay.

Hospitalizations for UGIB with concomitant ESRD had higher odds of inpatient mortality and higher mean total hospital charges compared to those with UGIB and coexisting CKD stages III-V. Prospectively, studies should focus on developing strategies to improve outcomes in patients with UGIB and concomitant ESRD.

331 DUAL PATHOLOGIC MECHANISM OF ACUTE KIDNEY INJURY IN A PATIENT FOLLOWING PERCUTANEOUS RENAL BIOPSY:
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Percutaneous renal biopsy (PRB) is the current standard of care to diagnose various diseases affecting native and transplanted kidneys. Here we present a patient who developed acute kidney injury following PRB, and postulate two different pathologic mechanisms for the cause of acute kidney injury with the help of Nuclear Medicine(NM) renal morphology with flow and function scan.

A PRB on the left side was performed on a 36 year old male patient for evaluation of chronic kidney disease with nephrotic range proteinuria and low C3. His bleeding parameters were normal and pre-biopsy hemoglobin was 9.7 grams/dl. He developed bleeding during the biopsy with subsequent hypotension and was shifted to Intensive care unit. Imaging demonstrated bleeding from a branch of left renal artery. His hemoglobin dropped to 4.9 grams/dl and he required 3 units of blood transfusion and had a catheter embolization of the left renal artery to stem the bleeding. He developed frank hematuria followed by oliguria and his creatinine increased to 4.8 grams/dl from his baseline of 1.7 grams/dl. He subsequently improved.

A NM renal morphology scan was performed a month after the PRB and showed a differential uptake of 41% in left kidney and 59% in right kidney which clearly demonstrates the biopsied kidney to be more affected than the other.

We postulate that the acute on chronic kidney injury in the above patient is due to two different mechanisms. One is due to acute tubular necrosis (secondary to hypovolemic shock) which has affected both kidneys equally. The second is tubular injury caused by blood/renal products which selectively affected the biopsied kidney as demonstrated by NM scan. Blood can cause tubular damage by forming obstructing casts, direct oxidative damage and vasoconstriction causing medullary ischemia. This case in unique in that the bleeding associated damage involved only a single kidney in contrast to other entities which involves both kidneys.

Our case illustrates how the NM renal morphology scan can be used to assess the functionality of individual kidneys in cases of kidney injury, which in turn might throw light on the pathogenesis of kidney injury.

332 A CASE OF APPARENT MINERALOCORTICOID EXCESS DUE TO A HOMOZYGOUS RARE VARIANT IN HYDROXYSTEROID 11-BETA DEHYDROGENASE 2 GENE:
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Apparent mineralocorticoid access (AME) is a rare autosomal recessive genetic disorder caused by a deficiency in 11β-hydroxysteroid dehydrogenase type 2 (HSD11B2), which is encoded by the HSD11B2 gene. Patients present with hypertension, renal wasting, hypokalemia, metabolic alkalosis, with low plasma renin and aldosterone. We describe a case with features of AME who underwent genetic testing which revealed a homozygous rare variant in the HSD11B2 gene.

Patient is an 18-year-old Saudi Arabian male who presented for definitive diagnosis of DR with longstanding hypertension and electrolyte abnormalities. Patient had hypertensive hypokalemia since 3 months of age. He also had a niece and nephew with elevated blood pressure and hypokalemia from a young age. His anti-hypertension regimen included lisinopril, hydralazine and amiloride. He was also on 20mg of a day of potassium tablets.

Metabolic panel revealed no abnormal findings. Aldosterone and an uptight were both suppressed at 3.0 ng/dL (3.1 – 35.4 ng/dL) and 0.5 ug/L/hr (0.8-8ug/L/hr) respectively. Blood cortisol and adrenocorticotropic hormone (ACTH) levels were within normal limits.

Genetic testing was sent for both Liddle’s syndrome and AME. No genetic mutations were identified for Liddle’s syndrome. Sequencing of the HSD11B2 gene disclosed a homozygous variant c.266G>A (p.Gly89Asp), which is rare and has not been observed in large population cohorts. In silico analysis, which includes protein predictors and evolutionary conservation, supports a deleterious effect. This variant was previously reported in a few patients with conflicting interpretation of pathogenicity. The phenotype of our patient indicates this variant is likely pathogenic.

The HSD11B2 enzyme converts cortisol to inactive cortisone which does not have an affinity to activate mineralocorticoid receptor (MR). Defects in this enzyme allow circulating cortisol to activate MR, which mimics clinical features of a hyperaldosteronism. Roughly 40 causative mutations in the HSD11B2 gene have been identified to date as a cause of AME. We believe that the Gly89Asp variant is responsible for the patient’s clinical features of AME, which adds to our knowledge about the genetic causes for AME.

333 EFFECT OF COVID-19 VACCINATION DRIVE ON COVID-19 OUTCOMES IN URBAN DIALYSIS CENTERS:
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Vaccination is a critical strategy to prevent COVID-19. We describe the effects of a vaccine drive implemented in Emory Dialysis centers on COVID-19 vaccine uptake, infection rates and outcomes.

Emory Dialysis, serving an urban population, conducted a COVID-19 vaccination drive (i.e. vaccine education and onsite vaccination administration at its 4 dialysis centers (~750 patients) from March–April 2021. Monthly COVID-infection and vaccination rates were tracked from March 2020–September 2021. We assessed the effect of the drive on the COVID-19 vaccine uptake, infection rates and outcomes including hospitalizations and 30-day mortality. Patients were included if they were diagnosed with COVID-19, 14 days after the vaccination drive (to reflect fully vaccinated status). Patients were stratified by vaccination status and descriptive statistics were performed.

From March 2020–April 2021, monthly COVID-19 infection rates were 0.41–4.97% and vaccination rates were 0–68%. From May–September 2021 (post-vaccination drive), the monthly COVID-19 infection rates ranged from 0–2.50% and vaccination rates were 67.4–76.1%. In the post-vaccination period, 34 patients were diagnosed with COVID-19; 26 were fully vaccinated and 8 were unvaccinated. Among the 34 patients, the median age was 57 years (interquartile range [IQR] 47–73), 26% were female and 79.4% were Black. Compared to unvaccinated group, the vaccinated group was older (62 years [IQR 50-73] vs. 50 years [IQR 41-60], p=0.06), and had a higher prevalence of cardiovascular disease (46.2% vs. 25.0%, p=0.02), otherwise, patient characteristics were similar between the groups. Twelve patients (41.2%) in the vaccinated group vs. 6 patients (75.0%) in the unvaccinated group were hospitalized for COVID-infection (p=0.26). Three patients (11.5%) in the vaccinated group vs. 2 patients (25%) in the unvaccinated group (p=0.35) died within 30-days of COVID-19 diagnosis.

Providing vaccinations at dialysis centers may improve COVID-19 vaccine uptake and outcomes. Studies evaluating the long-term effects of vaccination programs in dialysis centers are needed.