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lower risk of death. After adjustment for covariates, the hazard ratio was 0.919 (95% CI: 0.882-0.957; P = 0.0001).

In a real-world population of 1,915 kidney transplant recipients in US, our findings demonstrate that a within-patient increase in serum bicarbonate was associated with a lower risk of all-cause mortality. Each 1-mEq/L increase in serum bicarbonate over time was associated with an 8% decrease in mortality risk. Interventions to increase serum bicarbonate in kidney transplant recipients with metabolic acidosis should be tested.

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LOW SERUM BICARBONATE IS ASSOCIATED WITH GRAFT FAILURE IN KIDNEY TRANSPLANT RECIPIENTS:
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Chronic allograft nephropathy is a major cause of graft loss. Since metabolic acidosis is a risk factor for CKD progression, we examined its role in predicting long-term graft loss in kidney transplant recipients in a large US community-based cohort.

We analyzed data from Optum® EHR+Integrated dataset of US patients (2007-2019) with kidney transplant preceded by ≥1 year of data and no graft loss in the first year post-transplant. The primary outcome was graft failure (i.e., earliest occurrence of return to chronic dialysis, re-transplantation, kidney transplant failure diagnosis or death). The outcome period was -1 year post-transplant to end of the data period.

The primary predictor was the time-dependent change in serum bicarbonate from baseline at each outcome period observation per-unit outcome. Other covariates included demographics, comorbidities, medications, donor type, BMI, smoking status, baseline serum bicarbonate and eGFR. Adjusted Cox PH and Fine and Gray competing risk models were constructed to assess the risk of graft failure over the outcome period (≤10 years; median, 2.6 years) with death as a competing risk.

1,722 patients met inclusion criteria. Donor type was deceased (53%)/living (25%)/unknown (22%), and 27% had a prior transplant. Baseline eGFR and serum bicarbonate, defined as means during second 6 months post-transplant, were 63.4 ml/min/1.73m² and 24.6 mEq/L. 674 (39%) patients had graft failure during the outcome period. An increase in within-patient serum bicarbonate over time was associated with lower risk of graft failure. After adjustment, the hazard ratio per 1-mEq/L increase in serum bicarbonate was 0.899 (95% CI: 0.874-0.924; P < 0.0001), with similar hazard ratio for the competing risk model.

In 1,722 US kidney transplant recipients with a functioning graft at 1 year, a within-patient increase in serum bicarbonate was associated with a lower risk of long-term graft failure. Each 1 mEq/L increase in serum bicarbonate over time was associated with a 10% reduction in graft failure risk. The role of metabolic acidosis as a modifiable risk factor for chronic allograft nephropathy deserves further examination.

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ACYCLOVIR INDUCED ENCEPHALOPATHY IN A PATIENT ON PERITONEAL DIALYSIS:
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Acyclovir is an antiviral agent used for treatment of diverse viral pathologies. The regular pharmacokinetics for acyclovir is altered with kidney dysfunction. At present, our knowledge regarding treatment of acyclovir neurotoxicity in patients undergoing peritoneal dialysis (PD) is limited, as only few case reports have been published. We describe a case of acyclovir induced encephalopathy in a PD patient that was successfully treated with hemodialysis (HD).

34F with a history of systemic erythematosus lupus (on prednisone, cellcept and plaquenil) on PD had subclinical hypothyroidism, anemia and hypertension was admitted for herpetic lesions of her lips and eyelid. She was initiated on IV acyclovir at a dose of 500mg daily. 24 hours later, she developed acute confusion which progressed to myoclonus, lethargy and coma. Labs on admission revealed hb 10, Na 137, K 4.6, Cl 92, CO2 22, BUN 64, Cr 20.85. CT head was negative. EEG did not reveal any epileptiform activity. Lumbar puncture and brain MRI did not show any evidence of viral encephalitis. Acyclovir neurotoxicity was considered as the etiology for her encephalopathy. Acyclovir was held and her CCPD prescription was increased but she continued to remain encephalopathic. Decision was made to initiated hemodialysis. After the first HD session, her mension slightly improved. She received a total of 3 HD sessions, after which her mension completely returned to baseline. Given that she improved after discontinuation of acyclovir and with HD confirmed her diagnosis of acyclovir induced neurotoxicity, PD was resumed after recovery.

Dose adjustment for acyclovir is recommended in patients with ESKD. Despite acyclovir dose being adjusted for these patients, it can still cause neurotoxicity. This complication seems to be more common in those on PD likely due to the slower removal of the medication with PD. Clinicians need to be aware of this potential adverse event, as this diagnosis needs prompt recognition and treatment. Clearance of acyclovir with PD is not completely understood and in fact PD was not adequate to help with clearance in our patient despite increasing her prescription.

The modality of choice for clearance of acyclovir in toxicity is HD

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ACUTE KIDNEY INJURY IN COVID-19 PEDIATRIC PATIENTS: ANALYSIS OF THE VIRTUAL PEDIATRIC SYSTEMS DATA:
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Severe acute respiratory syndrome coronavirus 2 (SARS-CoV-2) is responsible for the 2019 novel coronavirus disease pandemic (COVID-19). Despite vast research about the adult population, there has been little data collected on acute kidney injury (AKI) epidemiology, associated risk factors, treatments, and mortality in pediatric COVID-19 patients admitted to the ICU. AKI is a severe complication of COVID-19 among children and adolescents. Therefore, understanding all aspects of the disease is crucial to further developing treatment and preventative care strategies to reduce morbidity and mortality.

This study aims to assess AKI incidence among COVID-19 pediatric patients in the pediatric intensive care unit (ICU) within North America using the Virtual Pediatric Systems (VPS) database. Additionally, this study assesses AKI associated risk factors, treatments such as kidney replacement therapy (KRT) and associated mortality rates among COVID-19 pediatric patients in North America using VPS data.

This is a retrospective study of COVID-19 pediatric patients (age ≤ 24 years) in the pediatric ICU within North America using the VPS COVID-19 database between January 1, 2020 and June 30, 2020.

Currently, data regarding 1240 pediatric COVID-19 patients has been analyzed. 172 of these patients had renal/urinary system involvement. Of the 172 patients with renal involvement, there were 19 confirmed deaths. This means that 45% of all confirmed pediatric COVID-19 deaths were associated with renal involvement. 36 patients received KRT and there are 2 confirmed deaths in this group. Additionally, 264 (24.67%) patients were diagnosed with Multisystem Inflammatory Syndrome in Children (MIS-C).

Although COVID-19 in the pediatric population tends to present more favorably, renal involvement among the pediatric COVID-19 patient population may be considered a negative prognostic factor with respect to patient outcomes.

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COVID-19 AND KIDNEY TRANSPLANT RECIPIENTS: IMMUNOSUPPRESSION MANAGEMENT AND OUTCOMES:
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The COVID-19 pandemic raises important questions about immunosuppression management and outcomes in kidney transplant recipients.
Kidney transplant recipients with positive SARS-CoV2 PCR seen in outpatient clinics or hospitalized at University and Parkland Hospitals from 3/1-10/1/20 were followed for 90 days. Univariate and multivariate backward selection logistic regression was used to identify risk factors for a composite event of AKI, ICU admission, or death. Non-parametric methods compared biomarkers based on changes in immunosuppressive drugs.

Of 59 patients, mean age (SD) was 51 (14) years, 35 (59%) were male, 13 (22%) black and 36 (61%) Hispanic. 29 (50%) had a baseline eGFR <60 mL/min/1.73 m², 52 (88%) had hypertension and 33 (56%) diabetes. 55 (93%) were on calcineurin inhibitors (CNI) and 49 (83%) on an antimitabolite at baseline. 6 (10%) were treated for acute rejection in the 12 months prior. Initial ferritin level was higher in those who had CNI dose decreased or discontinued vs. those with CNI unchanged, median (IQR) 1271 (839-1932) vs 283 (124-569) ng/mL, p=0.0002. Patients who stopped CNI showed significantly higher peak hsCRP values than those maintained on the same dose; median (IQR) 344 (145-374) vs 41 (22-116) mg/L, P=0.03.

There were 31 composite events, 43 hospitalizations, 13 ICU admissions, and 12 deaths. Of 52 patients with creatinine values, 29 (56%) had AKI, of which 10 (35%) required RRT. 13 (46%) had recovery of AKI at 90 days, defined as serum creatinine within 10% of baseline. Factors associated with the composite are shown (table). An eGFR<60 and peak hsCRP remained in the multivariable model associated with the composite, with area under the curve =0.83.

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**ECULIZUMAB THERAPY FOR GEMCITABINE-INDUCED ATYPICAL HEMOLYTIC UREMIC SYNDROME:**

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Several medications have been implicated as the trigger for atypical hemolytic uremic syndrome (aHUS), including some antibiotics and chemotherapeutic agents. Gemcitabine is seeing expanded chemotherapeutic indications in the field of medicine. We present here a case of aHUS in patient following gemcitabine therapy for advanced ovarian cancer.

A 64-year-old woman with non-metastatic high grade serous ovarian cancer on her seventh line chemotherapy regimen was evaluated for nephrotic syndrome and acute kidney injury (AKI). Her most recent chemotherapy regimen had been gemcitabine monotherapy (dose reduced to 600 mg/m²), with the cumulative dose of gemcitabine roughly 44,000 mg/m² over the last 2 years.

On initial nephrology evaluation, she had anasarca and uncontrolled blood pressure, the serum albumin 2.5 g/dL and the urine protein to creatinine ratio (UPCR) of 15.6 mg/mg. She was started on loop diuretics with improvement of the edema. The gemcitabine therapy was not held as the serum creatinine (sCr) was stable at 1.1 mg/dL.

Over the next few months, the patients sCr started to rise progressively in addition to worsening anemia and thrombocytopenia (56 tho/µL). A hemolysis panel showed elevated lactate dehydrogenase (LDH) of 513 U/L and low haptoglobin to < 30 mg/dL. A renal biopsy was deferred given their thrombocytopenia and risk for bleeding complications and gemcitabine therapy was discontinued.

Despite 6 weeks of gemcitabine discontinuation the AKI and the hemolysis were persistent (sCr up to 3.94 mg/dL). The patient was then started on eculizumab 900 mg weekly infusion. Four weeks into the therapy the patient started to improve and eculizumab was stopped after five infusions. Seven weeks following the initiation of eculizumab, the LDH (174 U/L) and haptoglobin (207 mg/dL) normalized, the sCr improved to 1.55 mg/dL and the nephrotic syndrome resolved with 0.5 mg/dL proteinuria per 24 hour urine collection.

Medication-induced aHUS is traditionally treated with cessation of the drug with or without plasmapheresis. However, given the growing understanding of the underlying mechanism of aHUS involving the alternative complement system, eculizumab, a monoclonal antibody that inhibits the activation of alternative complement cascade, has been receiving increasing use in patients with gemcitabine-induced aHUS progressing after holding gemcitabine.