Since January 2020 Elsevier has created a COVID-19 resource centre with free information in English and Mandarin on the novel coronavirus COVID-19. The COVID-19 resource centre is hosted on Elsevier Connect, the company's public news and information website.

Elsevier hereby grants permission to make all its COVID-19-related research that is available on the COVID-19 resource centre - including this research content - immediately available in PubMed Central and other publicly funded repositories, such as the WHO COVID database with rights for unrestricted research re-use and analyses in any form or by any means with acknowledgement of the original source. These permissions are granted for free by Elsevier for as long as the COVID-19 resource centre remains active.
cause interference in the measurement of sodium, calcium and glucose, among others. It arises due to an error occurring when venous samples are diluted. However, when measuring ionised calcium direct potentiometry is used and calcium can be measured accurately as the sample is not diluted. In pseudohypercalcemia serum calcium will be high but ionised calcium will be in normal range, thus separating it from the much more common true hypercalcemia.

**Conclusion:** In patients with high paraprotein levels, lab assays may be unreliable, and it is important to deploy good clinical judgement and to consider checking ionised calcium.

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**204 TELEMEDICINE FOR PEDIATRIC NEPHROLOGY IN THE AGE OF COVID-19 AND BEYOND: PERSPECTIVES ON FUTURE PRACTICES AND WORKFLOW CHANGES FOR GLOMERULAR DISEASE MANAGEMENT:**

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While the use of telemedicine has increased steadily over the years, it was rapidly implemented during the onset of the COVID-19 crisis. Due to the lack of standardized workflows for telemedicine, the authors of this paper have developed a standardized workflow via the Delphi method to provide a foundation/suggestion for future standardized workflows.

A literature search and article review was completed by the authors for the Delphi Panel to discuss. The panel did not grade the evidence, but instead used them as part of a suggested guideline as there are very limited RCTs.

There are four key components when managing glomerular diseases during the COVID-19 pandemic including immunosuppression, monitoring and diagnosis, supportive care, and in-person visits. In the case of confirmed or suspected infection, it is important to cease all medication to avoid an exacerbation of the infection. It is important to consider an alteration of medication till the end of the pandemic to short reversible agents. This allows for easy cessation of medication in case of infection. Until the end of the pandemic, providers should avoid immunosuppressive therapy for patients who are otherwise stable. For diagnosis and monitoring, any lab tests should be done outside of a hospital. Along with this, biopsies should be done only when absolutely necessary. All visits should be conducted electronically with an initial telederm visit to assess the patient’s symptoms and triage them for further treatment.

In order to deliver the most effective treatments for patients standardized telemedicine workflows are imperative. We believe adaptation of this workflow can provide the foundation towards delivering standardized care to patients across the nation.

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**205 NOVEL REGIMEN FOR REFRACTORY PRIMARY MEMBRANOUS NEPHROPATHY:**

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Primary membranous nephropathy (PMN) is the most common cause of nephrotic syndrome. Although there are multiple regimens for PMN, complete remission is typically not achieved until months or years after full treatment. We present a case of early, complete and sustained remission of refractory PMN using a single course of rituximab with low-dose cyclosporine and prednisone.

A 49-year-old female presented with leg swelling and proteinuria of 31 g/day. She was diagnosed with PMN by kidney biopsy in August 2016 and was started on daily prednisone. In April 2017, due to lack of complete response, she was started on cyclosporine (goal trough level 100-200 mg/L) resulting in partial remission (defined as proteinuria 0.3-3.0 g/dl). After 15 months, the patient relapsed with proteinuria to 4.9 g/d. Cyclosporine and prednisone were tapered off, and she was started on a course of rituximab in July 2018, followed by cyclosporine (trough level 58-85 mg/L) and prednisone. Following this, partial remission occurred within 6 weeks, and complete remission within 10 months. Serum creatinine remained stable at 0.56-0.71 mg/dl throughout. She was tapered off cyclosporine and prednisone from May 2019 to April 2020 with remission sustained for over 2 years.

This case demonstrates the efficacy of a single course of rituximab with low-dose cyclosporine and prednisone for the treatment of refractory PMN. Previous studies using rituximab or cyclosporin took 3 months to achieve partial remission, while our patient did so in 6 weeks. Our patient was able to achieve complete remission without a second course of rituximab and, by using a lower dose of cyclosporine, we minimized nephrotoxicity.

This novel combination regimen of rituximab with low-dose cyclosporine and prednisone offers a potentially effective approach in the management of refractory PMN. Clinical trials are needed to formally compare this regimen to current therapies for PMN.

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**206 STUDYING AKI IN COVID-19 PATIENTS IN THE ICU USING NEPHROCHECK BIOMARKER:**

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COVID-19 patients have a high incidence of acute kidney injury (AKI) and a notable increase in mortality once AKI has developed. The goal of this study is to help identify critically ill COVID-19 patients who are at higher risk of developing severe AKI and renal failure in hopes that earlier identification can lead to improved outcomes.

We performed a retrospective study of COVID-19-positive patients in the intensive care unit (ICU) at a major Southwest United States tertiary hospital from March 20 to November 26, 2020 who had a Nephrocheck® test, an FDA-approved lab test that allows for assessment of AKI risk. Patients who met the criteria were put into risk groups based on their Nephrocheck® value: low-risk (<0.3), intermediate-risk (0.3-2.0), and high-risk (>2.0). Patients in the study were evaluated for outcomes of mortality, ICU length of stay, need for renal replacement therapy, urinary output, and degree of
207 THE IMPORTANCE OF CYP2D6 POLYMORPHISM IN ACUTE KIDNEY INJURY (AKI):

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The CYP450 subfamily CYP2D6 metabolizes approximately 25% of commonly prescribed medications. The concomitant drug use and CYP450 metabolism correlations increases the risk of drug induced rhabdomyolysis and potential nephrotoxicity. We report an unusual case of AKI and rhabdomyolysis following a low risk surgery and correlate the CYP2D6 metabolism to multiple hypothetical etiologies of AKI.

45-year-old African-American male with history of obstructive apnea, intensive work out and use of anabolic steroids was admitted for elective laparoscopic umbilical hernia repair. Postoperatively, he developed an abrupt reduction in kidney function, from a baseline creatinine 1.1mg/dl up to 1.8mg/dl, severe hyperkalemia and respiratory acidosis. Due to disproportional hyperkalemia to the AKI, potassium peaked to 7.5meq/L, rhabdomyolysis was suspected. CK was trended to a peak of 33,050U/L in 36h. Urine toxicology was positive for methamphetamine. Hemodialysis was initiated for AKI with refractory hyperkalemia. Within 48h after surgery his renal failure resolved. Patient was discharged after 9 days.

The genetic polymorphisms of CYP2D6 have a functional impact on the metabolism and clearance of many drugs including methamphetamine and anabolic steroids. African-specific alleles metabolize CYP2D6 at a slower rate predisposing to drug toxicity, increasing the risk for rhabdomyolysis and direct drug-induced kidney injury. It was felt that the interaction between illicit drugs and commonly used medications that share the same metabolic pathway, in a potentially poor metabolizer, may have contributed to his renal failure.

This case provides a novel insight on the correlation of CYP2D6 polymorphism and drug interaction in the pathophysiology of AKI. In potentially poor metabolizers, non-nephrotoxic commonly used medications and illicit drugs may have a synergic effect causing prolonged serum drug level and increased risk for AKI.

208 THE KIDNEY DIET CHALLENGE: AN EXPERIENTIAL EDUCATIONAL EXPERIENCE:

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Nutrition is under-emphasized in medical curricula, and the kidney diet is one of the most challenging diets. We hypothesized that attempting adherence to this diet will improve knowledge and the ability of participants to counsel their patients with chronic kidney disease.

In the first part of this study was a knowledge assessment administered to all nephrology fellows during the 2020 National Board of Medical Examiners Nephrology In-Training Exam. We later opened the assessment to a broader, global audience via social media. Respondents included trainees and practicing nephrologists. Participants self-identified willingness to participate in the second part of the study, the Kidney Diet Challenge (KDC). We provided daily individualized kidney diet plans to our participants and asked them to follow the diet for 5 days and included daily webinars by experts. A daily survey captured self-reported adherence to the diet. Social media maintained active engagement (#kidneydietchallenge via @dietkidney). We measured social media activity by tracking the official hashtag. All participants received a follow-up knowledge assessment.

Among the Nephrology fellows (n=289), the median score was 2 out of 5 (40%) questions correct, and results did not differ by year of training (p=0.310). Of the participants (n=70) who completed the 5-day challenge, the post-test score was significantly higher (p=0.01) and ‘ability to advise patients about kidney diet’ improved (p=0.01). The social media campaign resulted in 974 posts and reached an audience of 406,241 users (Picture 1)

The KDC is an immersive, experiential educational tool to help participants learn how to better counsel their patients about adherence to a complex kidney diet.

209 RECOGNIZING PSEUDOHYPERKALEMIA TO PREVENT UNNECESSARY DIALYSIS:

Safa Osman1, Melinda Talley1, Alisha Parker1, Neville R. DossabhoY1. 1UMMC, Jackson, MS, United States

Pseudohyperkalemia is a reported rise in serum potassium (K) concentration along with a normal effective plasma K concentration. High K can result from cell lysis and release of intracellular K due to concentration along with a normal effective plasma K concentration. High K can result from cell lysis and release of intracellular K due to concentration along with a normal effective plasma K concentration. High K can result from cell lysis and release of intracellular K due to concentration along with a normal effective plasma K concentration. High K can result from cell lysis and release of intracellular K due to concentration along with a normal effective plasma K concentration. High K can result from cell lysis and release of intracellular K due to concentration along with a normal effective plasma K concentration. High K can result from cell lysis and release of intracellular K due to concentration along with a normal effective plasma K concentration. High K can result from cell lysis and release of intracellular K due to concentration along with a normal effective plasma K concentration. High K can result from cell lysis and release of intracellular K due to concentration along with a normal effective plasma K concentration. High K can result from cell lysis and release of intracellular K due to concentration along with a normal effective plasma K concentration. High K can result from cell lysis and release of intracellular K due to concentration along with a normal effective plasma K concentration. High K can result from cell lysis and release of intracellular K due to concentration along with a normal effective plasma K concentration. High K can result from cell lysis and release of intracellular K due to concentration along with a normal effective plasma K concentration. High K can result from cell lysis and release of intracellular K due to concentration along with a normal effective plasma K concentration. High K can result from cell lysis and release of intracellular K due to concentration along with a normal effective plasma K concentration. High K can result from cell lysis and release of intracellular K due to concentration along with a normal effective plasma K concentration. High K can result from cell lysis and release of intracellular K due to concentration along with a normal effective plasma K concentration. High K can result from cell lysis and release of intracellular K due to concentration along with a normal effective plasma K concentration. High K can result from cell lysis and release of intracellular K due to concentration along with a normal effective plasma K concentration. High K can result from cell lysis and release of intracellular K due to concentration along with a normal effective plasma K concentration. High K can result from cell lysis and release of intracellular K due to concentration along with a normal effective plasma K concentration. High K can result from cell lysis and release of intracellular K due to concentration along with a normal effective plasma K concentration. High K can result from cell lysis and release of intracellular K due to concentration along with a normal effective plasma K concentration. High K can result from cell lysis and release of intracellular K due to concentration along with a normal effective plasma K concentration. High K can result from cell lysis and release of intracellular K due to concentration along with a normal effective plasma K concentration. High K can result from cell lysis and release of intracellular K due to concentration along with a normal effective plasma K concentration. High K can result from cell lysis and release of intracellular K due to concentration along with a normal effective plasma K concentration. High K can result from cell lysis and release of intracellular K due to concentration along with a normal effective plasma K concentration. High K can result from cell lysis and release of intracellular K due to concentration along with a normal effective plasma K concentration. High K can result from cell lysis and release of intracellular K due to concentration along with a normal effective plasma K concentration. High K can result from cell lysis and release of intracellular K due to concentration along with a normal effective plasma K concentration. High K can result from cell lysis and release of intracellular K due to concentration along with a normal effective plasma K concentration. High K can result from cell lysis and release of intracellular K due to concentration along with a normal effective plasma K concentration. High K can result from cell lysis and release of intracellular K due to concentration along with a normal effective plasma K concentration. High K can result from cell lysis and release of intracellular K due to concentration along with a normal effective plasma K concentration. High K can result from cell lysis and release of intracellular K due to concentra...