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In FIDELITY, a prespecified pooled analysis of the FIDELIO-DKD and FIGARO-DKD phase III trials, finerenone reduced the risk of cardiorenal outcomes vs placebo in patients with T2D and CKD. Hispanic patients are more likely than non-Hispanic patients to develop end-stage kidney disease. This analysis explored outcomes in Hispanic patients. FIDELITY included patients with T2D and either CKD (UACR ≥ 30–<300 mg/g and eGFR 225–590 mL/min/1.73 m², or UACR 2300–5000 and eGFR 225 mL/min/1.73 m²) treated with optimized RAAS blockade (randomized to finerenone or placebo). Efficacy outcomes included a CV (death, nonfatal MI, nonfatal stroke, or hospitalization for heart failure) and kidney composite (kidney failure, sustained 25% eGFR decline, or renal death) endpoints.

Of 13025 patients in FIDELITY, 2099 (16.1%) self-identified as Hispanic; 50.7% received finerenone. Median follow-up was 3 years. In Hispanic patients, incidence rates were lower for the CV composite with finerenone (3.7/100 patient-years [PY] vs placebo (4.65/100PY) and kidney composite outcomes (2.59/100PY vs 2.65/100PY). No treatment differences were observed between Hispanic and non-Hispanic patients in both the CV (HR 0.80; 95%CI 0.62–1.04 and HR 0.87; 95%CI 0.79–0.97, respectively, pinteraction=0.59) and kidney composite outcomes (HR 0.94; 95%CI 0.67–1.33 and HR 0.75; 95%CI 0.64–0.87, respectively, pinteraction=0.22). Finerenone reduced UACR by 32% at month 4 in Hispanic patients (LS-mean treatment ratio: 0.68; 95%CI 0.63–0.73).

The efficacy and safety of finerenone were similar in Hispanic and non-Hispanic patients with CKD and T2D.

COVID 19 ASSOCIATED NEPHROTIC SYNDROME:
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Since the outbreak of SARS-COV-2 (COVID-19) in late 2019, there is a wide variety of renal pathology that is most associated with collapsing focal segmental glomerulosclerosis and acute tubular injury. This case presents a patient with new-onset focal segmental glomerulosclerosis - not otherwise specified (FSGS-NOS), in a previously healthy adult after a COVID-19 infection.

A 30-year-old Caucasian male with no significant past medical history presented with increasing severe generalized edema for 6 months following COVID-19 infection. The patient was admitted to the hospital with a significant acute kidney injury and hypalbuminemia. Subsequent kidney biopsy revealed focal segmental glomerulosclerosis (FSGS variant) with widespread foot process effacement that strongly supported a primary podocytopathy. Serological work-up was unrevealing: C3/C4, HIV, Hepatitis B and C, UPEP, SREP, and light chains were negative. COVID-19 anti-nucleocapsid and anti-spike antibodies were positive. The patient was started on high-dose prednisone, RAAS blockade, statin therapy, and diuretics. Outpatient follow-up has revealed significant symptomatic improvement of edema with a return to baseline creatinine with initial interventions.

COVID-19 infections have been most closely associated with collapsing focal segmental glomerulosclerosis, termed COVAN (corona virus-associated nephropathy), and acute tubular injury. Although the incidence of a non-collapsing FSGS secondary to COVID-19 infection is much lower than COVAN, FSGS-NOS increases the risk of permanent damage to kidney parenchyma if untreated. At this time, management and outcomes of non-collapsing FSGS associated with COVID-19 are not thoroughly studied. Therefore, we present this case to make physicians aware of the risk of interplay between non-collapsing FSGS and COVID-19, as early recognition and treatment can promote recovery.

RIBOCICLIB-INDUCED PSEUDO-ACTIVE KIDNEY INJURY:
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Cyclin-dependent kinase (CDK) 4/6 inhibitors, such as ribociclib, have been used to treat hormone receptor (HR)-positive, human epidermal growth factor receptor 2 (HER2)-negative advanced or metastatic breast cancer in combination with an aromatase inhibitor (AI).

A 69-year-old woman was diagnosed with HR-positive, HER2-negative metastatic breast cancer to the sternal manubrium after she had presented with a left breast mass. She was started on letrozole 2.5 mg orally daily (AI) and ribociclib 600 mg orally daily. After 3 treatment cycles, a repeat PET-CT scan revealed partial response with improvement in all her lesions. On laboratory work-up, however, she was found to have persistently elevated creatinine, prompting nephrology referral. Before treatment, her baseline creatinine was 1.13 mg/dL (eGFR of 50 mL/min/1.73m²). Both her creatinine and cystatin C were 1.36 mg/dL (eGFR of 39 mL/min/1.73m²) while her cystatin C was 1.24 mg/dL (eGFR of 53 mL/min/1.73m²). Since initiating treatment, her creatinine had increased and ranged between 1.25 mg/dL and 1.45 mg/dL. Her urinalysis was bland with no active sediment. She also did not have proteinuria. It was suspected that at baseline, she had mild chronic kidney disease secondary to microvascular disease related to aging and hypertension. Further evaluation was performed.

On follow-up, repeat creatinine was 1.39 mg/dL while repeat cystatin C was 1.37 mg/dL. Iohamate clearance revealed a measured GFR of 56 mL/min/1.73m² compared to a creatinine-based eGFR of 46 mL/min/1.73m². Random urine retinol-binding protein to creatinine ratio was also checked, and it was normal. Based on these findings, her elevated creatinine was concluded to be related to ribociclib’s interference with tubuline creatinine secretion. Active tubular creatinine secretion is mediated by multiple solute carrier (SLC) transporters in the kidney, including organic cation transporter 2 (OCT2) and multidrug and toxin extrusion protein (MATE) 1 and MATE 2-K. Ribociclib inhibits OCT2 and MATE 1, thereby interfering with creatinine secretion.