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have an active sediment, hence arguing against GN. However, our patient did have nephrotic range proteinuria at presentation with only minimal albuminuria. This likely represented low molecular weight proteinuria resulting from tubulointerstitial damage in the setting of systemic inflammation related to HGA. Along with his AKI, our patient’s proteinuria also improved with antibiotic therapy, supporting HGA as the driver of both abnormalities. Renal involvement due to HGA is rare. Yet, with the prevalence of tick-borne illnesses rising in the US, recognizing HGA as a cause of AKI is critical for prompt diagnosis and treatment.

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IS IT IDIOPATHIC NODULAR GLOMERULOSCLEROSIS OR DIABETIC GLOMERULOSCLEROSIS?:

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We present a case with the histologic features of diabetic glomerulopathy (DG) without the history of diabetes mellitus. A 67-year-old lady with history of alcohol abuse, active smoking, chronic anemia (myelodysplastic syndrome (MDS)) with multiple blood transfusions, and siderotic liver disease was admitted with worsening edema. She had BMI 24, positive anasarca, hemoglobin 8.5 g/dl, serum creatinine 1.51 mg/dl, glucose 81 mg/dl with HBA1c 4.7%, serum albumin <2 g/dl with urine protein/creatinine ratio 185 mg/gN, ANA positive 1:1280, positive anti CCP antibody, negative hepatitis B/C, low C3 and no monoclonal protein detected on SPEP/UPEP. Kidney biopsy revealed mesangial sclerosis, expansion with thickened basement membrane (BM) and nodular hyalinization of arterioles on light microscopy, and linear staining of IgG, IgA, light chains but no complements on IF microscopy, and extensive thickening of BM (average 1000 nm), effaced foot process but no electron dense deposits on EM. She had positive siderosis and negative Congo red staining on liver biopsy, and negative amyloid on myocardial scan. In the past, she had episodes of hyperglycemia during acute illnesses such as sepsis, otherwise her fasting blood glucose remained mostly under 100 mg/dl and HBA1c between 4.3 to 5.1%.

The histologic findings of our case are more compatible with diffuse glomerulosclerosis (GS) type of DG than nodular glomerulosclerosis (NG) type which is less often seen in DG. Diabetic GS-like, especially nodular GS-like lesion is also seen in membranoproliferative glomerulonephritis, in amyloidosis and light chain deposition disease. When NG occurs in absence of these conditions, it is called idiopathic nodular glomerulosclerosis (ING), and its pathogenesis remains unclear.

Our patient meets the criteria of ING. However, since her histologic findings are strikingly similar to DG, known episodes of hyperglycemia during acute illnesses and her low HBA1c possibly affected by persistent anemia due to MDS, it is possible to believe that she may have increased or reset sensitivity to the non-diabetic range of glucose, which results in exaggerated glomerulo-vascular response to produce diabetic GS, in the setting of her multiple adverse medical conditions.

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AZOLE INDUCED PSEUDO HYPERALDOSTERONISM WITH THERAPEUTIC LEVELS OF POSACONAZOLE:

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Posaconazole is increasingly used for prophylaxis and treatment of fungal infections owing to its broad spectrum and general tolerability. Rarely, posaconazole is associated with pseudo hyperaldosteronism (PHA), usually in the setting of high Posaconazole levels. We present a case of posaconazole induced PHA manifesting as a syndrome of apparent mineralocorticoid excess (AME) in a patient with therapeutic Posaconazole levels.

A 58 yo M with ESRD s/p DDKT 2016 on tacrolimus and prednisone, HTN, and recent diagnosis of Cladophialophora bantiana brain abscess presented to the ED with fatigue, weakness and incontinence. The brain abscess was diagnosed 3 months prior to admission and had been treated with voriconazole. Imaging showed progression of disease and he was switched from voriconazole to posaconazole. The goal posaconazole level of 2 mcg/ml was limited due to rising transaminases. The level peaked at 2.4 but needed dose reduction to a level of 1.3-1.6.

On hospital day 5 he developed watery diarrhea and was diagnosed C diff infection leading to hypokalemia <3.0mmol/L, which persisted for weeks despite resolution of diarrhea, requiring >80meq/day of potassium supplementation. Serum HCO3 21-23 mmol/L. He developed volume overload and HTN with BP >190/80 despite resumption of home antihypertensive. The constellation of hypokalemia, hypertension and volume overload was consistent with AME. Urinary Fractional excretion of potassium was 30%. Serum 11-Deoxy cortisol level was 23 (ref <10). The diagnosis of PHA was made and spironolactone 50 mg daily was started leading to resolution of hypokalemia and hypertension. His course was complicated by multiple bacterial infections. Unfortunately, he passed 5 months after diagnosis.

PHA is thought to be due to posaconazole off target effect of inhibiting 11-beta-hydroxylase, diminishing the conversion of cortisol to cortisone locally at the principal cell. Elevated local cortisol, and its increased affinity for the mineralocorticoid receptor, leads a syndrome of AME. This case highlights the need to remain vigilant for the development of azole-induced pseudo hyperaldosteronism even in patients with therapeutic levels of posaconazole.

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RACIAL DIFFERENCES AND OUTCOMES IN ESRD PATIENTS PRIMARILY HOSPITALIZED WITH EMPIEYA:

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Empyema is a rare but important cause of hospitalization among ESRD patients where pus collects in the pleural space, which can occur when pyogenic bacteria invade the pleural cavity, by direct inoculation or from adjacent pneumonia or any other source. Many of these patients receive long-term hemodialysis. However, we lack studies on the racial differences and outcomes in-hospital mortality and length of stay (LOS) in these patient population.

This is a retrospective cohort study involving index hospitalizations in ESRD patients between 2016 and 2019, using data from National Inpatient Sample (NIS) in patients 18 years or older who were primarily hospitalized for Empyema.

From 2016 to 2019 our final study sample consisted of 3,815 index hospitalizations for Empyema with ESRD with the mean age of 64.0 years old (SD= ±14.4, p<0.001). Among them 62.25% were males and 37.75% were females (p<0.001), 60.08% were White and 39.92% were Blacks (p<0.001), 92.27% had Charlson comorbidity index of three or more (p<0.001) and 78.63% were on Medicare (p<0.001). Weighted bivariate group (Whites vs Blacks) analyses suggested that Blacks were younger (mean age 61.3 vs 66.5, p<0.001), lower annual above $46,000 (44.4% vs 72.42%, p<0.001), and had similar CCI of three or more (93.21% vs 90.33%; p=0.183). Compared to Whites, in-hospital mortality rate (5.26% vs 3.11%, p=0.197) and LOS (12.5 vs 11.3 days, p = 0.240) were similar in Blacks. Multivariate regression showed that compared to whites, blacks did not have statistically significant higher or lower likelihood of in-hospital mortality (aOR 0.71; 95% CI: 0.26, 1.94; p = 0.502) or any difference in length of stay (β= -0.09; linearized SE =0.82; 95% CI: -0.07, 0.25; p=0.279).

The incidence of index Empyema admissions in ESRD patients is increasing with 23.7% in 2016, 20.5% in 2017, 27.1% in 2018, and 28.7% in 2019, p value=0.004.

Among ESRD patients with index hospitalizations for Empyema, there are no racial differences in the outcomes of in-hospital mortality and LOS.