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sensitive only to vancomycin. He completed 21 days of vancomycin and resumed PD via his existing catheter.

The pathogenicity of Corynebacterium is often unrecognized as it is considered a skin or nasopharyngeal contaminant. There are only a few reported cases of pathogenic Coryneforms and as such, a paucity of data in PD patients. The delay in speciation with lack of antibiotic susceptibilities prompted us to consider it a contaminant. The microbiology lab checked susceptibilities from infectious disease consult orders and ultimately, vancomycin monotherapy was continued. In a patient with diminishing dialysis options, we were able to successfully treat Coryneform bacterium peritonitis and salvage the PD catheter.

324 FAMILY WITH AUTOSOMAL DOMINANT TUBULOTINTERSTITIAL KIDNEY DISEASE (ADTKD) DIAGNOSTIC JOURNEY:
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Autosomal dominant tubulointerstitial kidney disease (ADTKD) is a group of rare genetic disorders characterized by progressive decline in kidney function and bland urinary sediment with no or mild proteinuria. Hematuria is generally not present. Diagnosis of ADTKD can be challenging due to its nonspecific presentations and late diagnosis is not uncommon. Here we present a complex diagnostic case of ADTKD who presents with long standing history of chronic kidney disease (CKD) and extensive family history of renal failure.

Patient is a 40 year old female with CKD, persistent microscopic hematuria, mild proteinuria and extensive paternal family history of renal failure (Figure 1) of unknown etiology who was referred from an outside hospital system to Cleveland Clinic’s Renal Genetics Clinic. She underwent urinalysis due to her family history and was noted to have hematuria and mild proteinuria at 28 years. She had a kidney biopsy in an outside hospital which showed thin basement membrane nephropathy with normal collagen staining. She previously completed an 18 gene kidney disease panel which was nondiagnostic. The physical exam was unremarkable. Her urinalysis on dipstick showed 2+ hematuria and 2+ proteinuria, eGFR was 79ml/min/1.73m², and uric acid was 4.6mg/dl. Renal ultrasound was unremarkable. Exome sequencing was completed that identified a likely pathogenic variant in UMOD (c.317G>T; p.C106F) that established a genetic diagnosis of ADTKD. Given this diagnosis, patient started allopurinol 100mg daily and targeted variant testing was offered to her at risk family members.

ADTKD should be considered in cases with CKD of unknown etiology, particularly those with strong family history. This case indicates persistent microscopic hematuria with mild proteinuria may be present in some cases of ADTKD and highlights the importance of reevaluation and genetics involvement in diagnostic dilemmas.

326 KDQOL SURVEY SCORES IN DIALYSIS PATIENTS DECREASE FOLLOWING THE FIRST YEAR OF THE COVID-19 PANDEMIC WITH CONCURRENT CHANGES IN PATIENT COMPLIANCE:
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The Kidney Disease Quality of Life (KDQOL) survey is a review of patients’ quality of life (QOL) on hemodialysis. Lower survey scores in depression, burden of disease, and treatment satisfaction are associated with worse compliance to treatment and poorer outcomes.

KDQOL surveys were extracted from and stratified by year, with duplicate entries removed. Annual mean scores for each component of the survey were calculated for each clinic. The KDQOL data represents the mean scores for 2017-2019 compared to the first three quarters of 2021. 2020 was excluded due to sampling challenges and high patient turnover creating potentially inaccurate data. Mean scores were compared by Student’s t-test with Bonferroni adjustment for multiplicity. Phosphorus and PTH levels were used as a surrogate for treatment compliance.

Patients reported lower QOL scores during the COVID-19 pandemic compared to pre-pandemic (baseline). All KDQOL metrics were significantly lower in 2021 compared to the mean of three years prior to the pandemic. A two sample Student’s T test was used to determine the change in mean score for each category: Physical Component Score (t(2)= 14.5, p = 0.009), Mental Component Score (t(2)= 36.7, p = 0.0004), Burden of Kidney Disease (t(2)= 6.1, p = 0.01), Symptoms of Kidney Disease (t(2)= 22.8, p = 0.0009), Effects of Kidney Disease (t(2)= 8.8, p = 0.006).

Phosphorus was significantly higher in 2021 compared to the mean of 2018 and 2019 when calculated via t-test (t(31)= -2.72, p = 0.01).

Parathyroid Hormone quarterly data was evaluated via t-test for 2018 to 2020 vs. the first three quarters of 2021 (t(12)= -7.15, p = 0.01). Using the KDQOL survey to measure patients’ QOL, we found that all measures were significantly lower in 2021 following the pandemic. Using markers of bone metabolism as measures of treatment compliance, phosphorus and PTH levels were also significantly higher in 2021. In ESRD patients who survived the trauma of the pandemic, QOL is perceived to be worse and is likely associated with lower quality of life.
ROLE OF FENOFIBRATE IN LIPOPROTEIN GLomerulopathy: Case Report:

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Lipoprotein glomerulopathy (LPG) is a unique disorder characterized by lipoprotein thrombi in glomeruli, an abnormal plasma lipoprotein profile that resembles type III hyperlipoproteinemia and a marked increase in serum apolipoprotein E (1). Rare mutations in apolipoprotein E gene may contribute to the pathogenesis of the disease. Unfortunately, there is still no effective therapy for this disorder, but some case reports have highlighted the benefits of fenofibrate use.

A 25 ½ Asian male with hypertension and hyperlipidemia was referred to nephrologist for evaluation of nephromegaly. His medications included hydrochlorothiazide, losartan and atorvastatin. His previous labs showed creatinine 1.24 mg/dL, BUN 25 mg/dL, potassium 4.7 mmol/L, albumin 1.8 g/dL, total cholesterol 574 mg/dL, HDL 53 mg/dL, LDL 468 mg/dL, triglycerides 203 mg/dL, AST 17 IU/L, ALT 12 IU/L. Renal duplex revealed enlarged kidneys with increased cortical echogenicity. He complained of leg swelling. No family history of kidney and cholesterol problems.

Urinalysis revealed 2+ protein, 30 RBCs. Urine protein to creatinine ratio was 5 g. Hepatitis B antigen, Hepatitis C antibody, HIV, SPEP, C3, C4, ANA, Anti PLA2R antibody, serum free light chains were negative. Renal biopsy revealed Lipoprotein glomerulopathy, moderate interstitial fibrosis and Oil Red O stain positive for lipid deposits in glomerular capillary loops. Electron microscopy showed glomerular capillary loops are distended and thrombosed with amorphous material containing numerous vacuoles. His proteinuria improved to 3.2 g in two months after treatment with fenofibrate. Apolipoprotein E genetic testing is pending.

LPG is a unique disorder with potentially harmful consequences whose treatment and outcomes are poorly understood. Prompt diagnosis is pertinent to slow and hopefully prevent progression to end stage kidney disease. Although no definitive treatment exists for LPG, our case highlights the importance of early initiation with fenofibrate (2). After 2 months of treatment with fenofibrate, a remarkable decrease in urinary protein excretion was noticed. Long term follow up is needed to further elucidate response to treatment.

Treatment with fenofibrate will benefit in improving proteinuria in LPG.

METFORMIN ASSOCIATED LACTIC ACIDOSIS:

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Metformin is a first line medication for anti-glycemic control because of its low cost, effectiveness, weight neutrality, and unique mechanism of action. The most common side effect of this biguanide is gastrointestinal distress. However, the relatively rare complication of metformin associated lactic acidosis (MALA) is the most serious adverse effect of this drug, because of its substantial morbidity and mortality. Metformin is cleared from the body by tubular secretion, which may be impaired during kidney injury.

A 61-year-old man presented to hospital for confusion and abdominal pain after heavy alcohol consumption for several days. He had severe oligoanuric AKI, profound volume depletion, an anion gap metabolic acidosis, type II lactic acidosis, and elevated metformin level.

Patient was found to have elevated serum creatinine to 14.2 (baseline at 1), potassium 6.4, bicarb 6, anion gap of 35, PH 7.01 and lactic acid 16.3. His volatile toxic alcohol analysis was negative and CT scan did not show evidence of bowel ischemia. He received aggressive volume resuscitation and isotonic bicarbonate infusion and boluses and was admitted to MICU. Hemodialysis was immediately initiated for a duration of 3 hours with standard blood flow and dialysate flow rates, and he was transitioned to CVVH after this initial high efficiency HD. Within 24 hours, he was treated with a second full HD session followed by CVVH again. By the end of day 2 admission, he started to have increased urine output and CRRT was discontinued. Patient was normotensive during the hospitalization, his lactic acid was slowly downtrend to 2.7 after 48 hours of treatment. His metformin level found elevated to 10 mcg/ml (therapeutic range is 1-2 mcg/mL). Patient was discharged from hospital after 7 days with corrected electrolytes and fully recovered renal function (creatinine 1.2).

We present a relatively rare but serious complication of a widely used medication, metformin, – MALA, which occurred in an individual with normal baseline kidney function in the setting of volume depletion and oligoanuric AKI. Nephrologist should consider MALA in unexplained high level of lactic acidosis in patients using metformin. Early initiation of hemodialysis can correct the lactic acidosis, aid with metformin clearance, and improve the clinical outcome in patients presenting with MALA in the setting of AKI.