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transient ischemic attack and tachycardia. The patient’s renal failure was attributed to acute kidney injury (AKI) and diabetic nephropathy. Renasight testing was performed that revealed a heterogeneous autosomal dominant TTR mutation c.424G>A (p.Val142Ile) and heterogeneous carrier status for HBB Beta-Hemoglobinopathy. Following genetic testing, the patient was referred back to cardiology for re-evaluation.

This patient had a significant history of edema and cardiovascular disease (CVD) believed to be secondary to nephrotic syndrome. Genetic testing revealed an amyloidogenic pathogenic variant, known to be associated with CVD. Following this diagnosis, the patient opted for a living-related transplant. For the patient, the findings could affect donor selection and may pose risk for increased risk of CVD in his children. Family testing and genetic counseling would be appropriate for monitoring and implementing suitable medications such as ACEi/ARBs to slow the progression of heart and kidney disease.

Mid-level healthcare providers can effectively take advantage of Renasight to improve patient management and care. This case provides an example where correct diagnosis using Renasight can influence treatment decisions, selection of a suitable donor for transplant, and future genetic testing for families.

150 CLINICAL TIMELINE OF DECREASED GFR FROM HYPOTHYROIDISM CAUSED BY AMIODARONE TOXICITY: A REVERSIBLE YET UNDERAPPRECIATED ENTITY:

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While the differential diagnosis of acute kidney injury and chronic kidney disease include states of distant organ dysfunction that can lead to renal dysfunction, e.g. cardiorenal and hepatorenal syndromes, thyroid dysfunction causing renal dysfunction is not typically included in the classic differential despite its well-understood pathophysiology and reversible nature. We present a case of amiodarone-induced hypothyroidism leading a rapid worsening of clearance, reversible with thyroid replacement.

A 69-year-old male with a history of coronary artery disease, hypertension, heart failure with reduced ejection fraction, CKD stage 3, and type 2 diabetes was referred for evaluation of CKD. His history included a new diagnosis of paroxysmal atrial fibrillation for which he was started on amiodarone. His sCr had risen from a baseline of 1.6 mg/dL to 2.9 mg/dL over 5 months. Urinalysis was bland with absent proteinuria. Thyroid stimulating hormone was profoundly elevated to 181.31 mIU/ml; thus amiodarone was discontinued and levothyroxine was started. Once euthyroid, sCr improved to 1.4 mg/dL.

The numerous effects of thyroid hormone on kidney function include increased cardiac output, renal blood flow, renin-angiotensin-aldosterone system activity, filtration pressure, tubular glomerular feedback, sodium potassium ATPase activity, and urinary concentrating ability. Thyroid hormone also decreases peripheral vascular resistance. Through these mechanisms, hypothyroidism reduces GFR which is reversible with thyroid replacement.

In spite of the well understood mechanism as well as numerous epidemiologic reports, few clinical reports of the syndrome of thyroid dysfunction resulting in reversible renal dysfunction have been published. This is an underrecognized important reversible cause of CKD.

151 STRATEGIES TO CONTAIN COVID-19 INFECTION IN A HOSPITAL BASED OUTPATIENT HEMODIALYSIS UNIT IN THE SOUTH BRONX:

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New York City was the epicenter of COVID-19 infections within the United States in the spring of 2020. Our public, hospital-based hemodialysis (HD) unit is located in Bronx County, which had the highest rates of infections and deaths due to COVID-19. We retrospectively investigated the prevalence of COVID-19 in our HD unit and the effectiveness of expanded infection control measures implemented during the surge.

Charts were reviewed for all 61 patients receiving maintenance HD between March 1-July 15, 2020. 4 HD patients and 2 HD healthcare providers (HCP) developed symptoms from COVID-19 infection between March 17-23, followed by another 5 patients and 2 HCP. HD patients underwent SARS-CoV-2 PCR nasal swab, regardless of symptoms, allowing detection of 4 asymptomatic COVID-19 cases. Positive cases were cohorted. Patients were screened for fever and COVID-19 symptoms before each HD, advised to wear face masks and practice hand hygiene. 5 patients were hospitalized with COVID-19 within 14 days of the screening period with no additional cases detected afterwards.

During the surge, patients requiring bedside HD increased exponentially so HD frequency or treatment hours were reduced for some patients and 20 were temporarily transferred to other units. In May, all 32 HCP were tested for COVID-19 antibody with 18.8% (5 with and 1 without symptoms) testing positive. In June, 51 HD patients were tested for antibodies with detection of 6 additional asymptomatic individuals who had been SARS-CoV-2 PCR negative. In total, 26 patients (42.6%) tested positive for COVID-19, of which 42.3% were asymptomatic, and with 1 death.

Early identification and isolation of both symptomatic and asymptomatic patients by universal screening along with stringent infection control measures limited the spread of COVID-19 infection in our unit.

152 OUTCOMES OF HOSPITALIZED PATIENTS WITH COVID-19 AND ACUTE KIDNEY INJURY REQUIRING RENAL REPLACEMENT THERAPY:

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The incidence of acute kidney injury (AKI) in COVID-19 patients has been reported as high as 47%, with mortality ranging from 35-80% in this population. AKI patients requiring renal replacement therapy (RRT) likely have a greater mortality risk. Our aim was to describe outcomes of AKI patients who required RRT among hospitalized COVID-19 patients from a large diverse population in Southern California.

We conducted a retrospective cohort study of COVID-19 patients with AKI requiring RRT defined as conventional hemodialysis, continuous renal replacement therapy, or both, within Kaiser Permanente Southern California in the period of 3/14/2020 through 9/30/2020. We collected information on patient characteristics,
A MYSTERIOUS MULTI-SYSTEM DISEASE AND RENAL BIOPSY REVEALING CONCURRENT IgG4-RELATED DISEASE, C3 GLOMERULONEPHRITIS, AND ANCA-ASSOCIATED GLOMERULONEPHRITIS:

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IgG4-related disease (IgG4 RD), C3 glomerulonephritis (C3 GN), and anti-neutrophil cytoplasmic antibody-body-associated glomerulonephritis (ANCA GN) are rare entities with presumed incidences of 1: 100,000, 1: 1,000,000, and 17: 1,000,000, respectively. We report an extremely rare case of a patient with concurrent IgG4 RD, C3 GN, and ANCA GN.

A 62 y/o Asian M with hypertension and active tobacco use presented with fatigue, erosive polyarthritis, neuropathic pain, numbness of lower extremities, pruritic macular rash on his back, and unintentional weight loss of 10 lbs over a period of 3 months. Laboratory work up showed creatinine 3.18 mg/dl (baseline 0.8 mg/dl); microscopic hematuria; dipstick-positive proteinuria, quantified by a spot urine protein/creatinine ratio of 2.45 g/d of which 1.809 g/d was albumin; ANA titer 1:2560, homogeneous pattern; dsDNA <1:10; ANCA titer 1:2560; cytoplasmic staining (anti-MPO 220 AU/ml, anti-PR3 8 AU/ml, reference range 0-19 AU/ml for both anti-MPO and anti-PR3); ESR 76 mm/h and CRP 17 mg/L; C3 69 mg/dl (reference range 90-180 mg/dl); C4 26 mg/dl (reference range 12.47 mg/dl); rheumatoid factor 26 IU/ml (reference range 0-14 IU/ml); anti-cyclic citrulline peptide 8 U (reference range 0-19 U); normal SPEP/UPEP/immunofixation; negative hepatitis B, C and HIV. Serum IgG4 level was 120 mg/dl (reference range, 1-123 mg/dl).

Chest CT 5 months prior to admission was suggestive of interstitial lung disease. Given the above work up, and the presumed acute onset of kidney injury, supported by a PTH of 42 pg/ml, the patient underwent kidney biopsy, which showed cellular, fibro-cellular, and fibrous crescents in 15 of 24 glomeruli; moderate tubular atrophy with marked interstitial fibrosis; patchy dense interstitial lymphoplasmacytic inflammation with mild tubulitis; increased IgG4: IgG ratio of 0.26 (normal <0.10); subendothelial, subepithelial, intramembranous, and mesangial electron dense deposits; and glomerular staining positive only for C3 by immunofluorescence. The patient was started on steroids and cyclophosphamide and creatinine improved to 1.85 mg/dl. Functional and genetic testing revealed elevated C3c, Factor H levels, deletion of CFHR1-CFHR4 gene and ADAMTS13 gene mutation of unknown significance.

IgG4 RD is a multi-organ fibro-inflammatory condition, with a prevalence of 0.28 to 1.08 per 100,000 of population. Renal involvement typically manifests as interstitial nephritis. C3 GN is another rare disease resulting from abnormal regulation of the complement pathway. The estimated incidence is 1 to 3 cases per million with a point prevalence of 0.6 to 140 cases per million. Although ANCA vasculitis is more commonly seen than both IgG4 RD and C3 GN, it remains rare. We present a case of a patient with biopsy-proven concurrent IgG4 RD, C3 GN, and ANCA vasculitis.

To the best of our knowledge, only one case with concurrent IgG4 RD, C3 GN, and ANCA GN has been published worldwide [1]. A deeper understanding of the pathophysiologic factors that predispose to each of these diseases may help to elucidate whether their linkage is coincidental or the result of a common immunologic disturbance.

154 A MYSTERIOUS CASE OF HYPERCALCEMIA IN NON-UREMIC CALCIPHYLAXIS PATIENTS:

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Hypercalcemia is a common electrolyte disturbance in clinical practices. Hyperparathyroidism and hypercalcemia of malignancy constitute more than 70% of total cases. Immobilization is one of the rare causes of hypercalcemia and is often overlooked. It should be considered in cases with a history of prolonged immobilization and unexplained hypercalcemia.

A 50-year-old obese woman was referred to our center due to history of non-uremic calciopathy on her thighs for 1 year. 14 months prior, she developed acute kidney injury in the setting of acute alcoholic liver failure. At that time, her serum creatinine (Scr) peaked at 3.92, but decreased to 1.3 ranges after discharge. Serum calcium (Ca) was 9.4 mg/dl 1 month after discharge, she developed tender skin lesions on her thigh that eventually became eschar. This was biopsied and diagnosed with calciphylaxis. Her serum Ca still remained in a normal range. She then was commenced on sodium thiosulfate (STS) 12.5 mg thrice weekly for 4 months with partial improvement, thus the dose was reduced to twice weekly. Nonetheless, the lesions worsened leading to resumption of the previous dose. Throughout the course, she had been debilitated and in a wheelchair. Of note, her serum Ca began to increase at 7 months (Ca 12.4 mg/dl) after the initiation of STS. PTH was notably suppressed (6.3 pg/mL). At our center, she still had residual shallow ulcers with some eschars on both thighs. Laboratories showed Scr 1.53 mg/dl, total Ca 12.5 mg/dl, phosphorus 3.5 mg/dl, PTH <6 pg/mL, 25-OH vitamin D 25 16.5 pg/mL, and 1,25-OH vitamin D <8 pg/mL. Concurrently, her bone mineral density (BMD) showed significant reduction in T-score of both hips from 1.3 to -1.4. This prompted us to consider immobilization resulting in rapid bone turnover as a cause of hypercalcemia. Pamidronate was subsequently initiated both diagnostically and therapeutically. Ca quickly normalized in 3 days, followed by an improve in Scr at 0.92 mg/dl.

Rapid bone turnover secondary to immobilization in this patient, reflected by drastic changes in BMD, resulted in hypercalcemia in this case. Furthermore, metabolic acidosis-induced STS may further enhance this process.

Hypercalcemia from immobilization is frequently diagnosed by exclusion. It is more common in pediatric and geriatric populations. Anti-resorptive agents are not only an important treatment option but also used for diagnostic purposes.

155 FANCONI SYNDROME SECONDARY TO NON-CRYSTALLINE LIGHT CHAIN PROXIMAL TUBULOPATHY IN CHRONIC LYMPHOCYTIC LEUKEMIA: A RARE PRESENTATION OF A COMMON DISEASE:

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Light chain proximal tubulopathy (LCP) is a rare form of monoclonal protein related kidney disease. We report the first case of non-crystalline LCP in CLL patients who presented with Fanconi syndrome.

An 81-year-old white male with CKD stage 5 not yet on dialysis baseline Cr 4.5 mg/dl, hypertension, chronic hypokalemia on potassium supplement and CLL. His CKD was thought to be due to hypertensive nephrosclerosis. He was diagnosed with CLL 5 years ago which did not require any treatment until 2 years ago when the CLL began to progress. He was treated with rituximab and acalabrutinib with various side effects leading to discontinuation. Upon further evaluation, Cr was notably elevated at 11.2 mg/dl. However, he was not uremic and had good urine output. Laboratories showed BUN 64 mg/dl. Na 144 mEq/L. K 3.3 mEq/L, bicarbonate 16 mEq/L, uric acid 5.4 mg/dl, and WBC 68.7x10^9 cells/L. Monodonal study showed IgG kappa with M spike of 0.5 g/dl. Urinalysis showed non-dysmorphic microhematuria.