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A Case of IgA Nephropathy in the Setting of Sezary Syndrome and Mogamulizumab
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Introduction: IgA Nephropathy (IgAN) is an autoimmune disease with complex pathogenesis. Sezary syndrome (SS) is a leukemic subtype of cutaneous T cell lymphoma (CTCL). A rare association has previously been reported between IgAN and CTCL. Mogamulizumab (MG) is a monoclonal antibody drug targeting C-C chemokine receptor type 4 (CCR4) and is used in the treatment of CTCL and SS. MG has been associated with drug eruptions and systemic immune-mediated adverse events.

Case Description: A 63 year-old woman with SS was treated with MG. Her skin symptoms improved and circulating Sezary cells cleared. Due to a cutaneous drug eruption, the frequency of MG administration was reduced to monthly after cycle 7. Labs prior to cycle 19 demonstrated serum creatinine (Cr) 1.77 mg/dL from a prior baseline ~0.9-1.0 mg/dL. She received intravenous fluids but Cr worsened to 3.97 mg/dL. Uramylis (UA) revealed more than 20 red blood cells (RBCs) per high powered field (HPF). 24 hour urine protein to creatinine ratio (UPCR) was 2.03 g/g. Serologies and complements were normal except double stranded DNA which was 12 IU/mL (normal <4 IU/mL). Kidney biopsy demonstrated mesangial immune complex deposition with IgA, IgG, and C3 predominance consistent with IgAN (Oxford M1091TTIC0). Prednisone was initiated at 1 mg/kg/day and tapered over 6 months. MG was stopped. After 6 months Cr had improved to 1.10 mg/dL. UA showed 3-5 RBCs per HPF with UPCR of 0.122 g/g. Her SS remained well controlled without systemic therapy.

Discussion: This case reinforces the association between IgAN and CTCL which has been described in prior case series. In patients with CTCL, altered T cell populations and a dysregulated immune response may contribute to the pathogenesis of IgAN. Complicating this case is the use of MG which can deplete normal CCR4-expressing regulatory T cells by inducing antibody-dependent cellular toxicity. MG is associated with cutaneous granulomatous drug eruptions in which alterations in T cell populations have been implicated. Systemic autoimmune complications outside of the kidneys have also been reported. The possibility that MG could play a role in IgAN should be considered.

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Filgrastim-Induced Crescentic Glomerulonephritis
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Introduction: Proliferative glomerulonephritis with monoclonal immunoglobulin deposits (PGNMID) is caused by deposition of monoclonal immunoglobulins in the glomeruli. It is one of renal disorders included in the spectrum of monoclonal gammopathy of renal significance (MGRS). IgG3 with kappa light chain is the most common type. Autologous stem cell transplantation (SCT) provides a durable remission and better renal outcomes. Granulocyte colony stimulating factor (GCSF) is a recombinant glycoprotein used for mobilization of bone marrow in SCT. GCSF has been implicated as a cause of crescentic transformation of an acute glomerulonephritis in one prior case with a monoclonal deposits in a kidney transplant patient. In this case, we report the clinical and pathologic findings of GCSF induced exacerbation and crescentic transformation of pre-existing PGNMID with successful treatment and SCT.

Case Description: A 48-year-old male with recent diagnosis of MGRS presenting as MPGN and monoclonal IgG Kappa with C3 deposits on biopsy and treated with Velcade, cyclophosphamide and dexamethasone with a plan for SCT. Patient was admitted after acute increase in creatinine from 2.87 mg/dl to 6.69 mg/dl with hematuria and proteinuria after receiving GCSF during stem cell mobilization. Timing of acute renal injury correlated with increase in WBC after GCSF injections with a peak of 69 K/ul. Repeat kidney biopsy was significant for crescentic membranoproliferative (62% crescents) glomerulonephritis with monoclonal IgG Kappa deposits. Patient received 5 sessions of plasmapheresis, one dose of renally adjusted IV Cytoksan, and pulse steroids followed with a taper. After a month he undergo an Autologous SCT (creatinine at baseline 1.6mg/dl). His kidney function continued to improve and after 16 months post SCT his creatinine is at 1.4mg/dl.

Discussion: GCSF enhances neutrophils activation in large counts and induces its endothelial activation. In the presence of pre-existing renal pathology, MGRS and MPGN with IgG kappa and C3 deposits in this case, the localized immunoglobulin and complement deposits in the glomeruli can attract activated neutrophils leading to its infiltration and degranulation in the glomerular microenvironment, and resulting in rupture of glomeruli basement membrane and formation of crescent. Therefore, GCSF induced kidney injury should be suspected due to its potential risk for exacerbating pre-existing glomerulonephritis.

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Creatinine-Cystatin C Ratio and Mortality in Cancer Patients: A Retrospective Cohort Study
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Background: Muscle wasting is prevalent in cancer patients, and early recognition of this phenomenon is important for risk stratification. Recent studies have suggested that the creatinine-cystatin C ratio may correlate with muscle mass in several patient populations. The association between creatinine-cystatin C ratio and survival was assessed in cancer patients.

Methods: A total of 3,060 patients who were evaluated for serum creatinine and cystatin C levels at the time of cancer diagnosis were included. The primary outcome was 6-month mortality. The 1-year mortality, and length of intensive care unit (ICU) and hospital stay were also evaluated.

Results: The mean age was 61.6±13.5 years, and 1,409 patients (46.0%) were female. The median creatinine and cystatin C levels were 0.9 (interquartile range [IQR], 0.6-1.3) mg/dL and 1.0 (IQR, 0.8-1.5) mg/L, respectively, with a creatinine-cystatin C ratio range of 0.12-12.54. In the multivariate Cox analysis, an increase in the creatinine-cystatin C ratio was associated with a significant decrease in the 6-month mortality (per 1 creatinine-cystatin C ratio, hazard ratio [HR] 0.35; 95% confidence interval [CI], 0.28-0.44). When stratified into quartiles, the risk of 6-month mortality was significantly lower in the highest quartile (HR 0.30; 95% CI, 0.24-0.37) than in the lowest quartile. Analysis of 1-year mortality outcomes revealed similar findings. The highest quartile was also associated with shorter length of ICU and hospital stay (both P<0.001). These associations were independent of confounding factors.

Conclusions: The creatinine-cystatin C ratio at the time of cancer diagnosis significantly associates with survival and hospitalization in cancer patients.

PO1888
Comparison of Kidney Volume-Based Methodology to Glomerular Filtration Rate Estimating Equations to Predict Measured Glomerular Filtration Rate in Cancer Patients
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Background: Total kidney volume (TKV) has been associated with both measured glomerular filtration rate (eGFR) and with equations recommended to estimate GFR (eGFR) in clinical practice. However, there is scarce data comparing measurement of TKV to eGFR equations as predictors of eGFR, particularly in the oncology setting.

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