Introduction

• The vast majority of collapsing FSGS have an associated HIV component, coined HIV nephropathy.
• In one report, 1.4 of all kidney biopsies over a 15 year period demonstrated this pathology.
• Both genders are impacted equally, but there appears to be an increased incidence in the African American population.
• Due to its pathogenesis of damaging glomerular podocytes and parietal epithelial cells, the presentation often revolves around nephrotic syndrome symptoms. [1]

We present a 56 year old African American female with a history of asthma, who was admitted with significant edema, nausea, vomiting and abdominal discomfort for the third time in one week. 
Vitals were generally normal, except for a mild temperature of 99F and tachycardia. Her initial CMP showed hypoalbuminemia, and mild transaminitis. Her BUN/Cr was consistent with an AKI, and had an elevated value relative to baseline which was normal. Initial urinalysis demonstrated significant proteinuria without hematuria. Initial CT abdomen and pelvis was inconclusive other than bilateral renal calculi. She was admitted to the hospital with studies ordered for EBV, CMV, HIV, anti-smooth muscle antibody, and anti-ANA to search for a potential auto-immune etiology.

Case Presentation

On admission there was a slight transaminitis. A hepatic work up as well as GI consult was requested. Imaging was unremarkable. Acute viral hepatitis, Tylenol level and coagulation factors were within normal limits.
Serum creatinine was found to be 1.3 at admission, with a baseline of around 1.1 with minimal variability. Urinalysis was significant proteinuria in nephrotic range. Initially, the suspicion was for prerenal azotemia, but the creatinine continued to climb up to 6.8. There was no response to crystalloid challenge or 5% albumin which caused concern for glomerulonephritis. She was began on high dose steroids, and worked up for an auto-immune etiology which came back insignificant, however, was positive for IgM kappa light chain disease which may have also played a role in renal damage. A renal biopsy was completed that demonstrated cFSGS and severe tubular injury. A progressively worsening renal function made her a candidate for hemodialysis and a permacatheter was placed. Renal function began to stabilize in just over 2 weeks and the patient was discharged with outpatient follow up.

Hospital Course

Discussion

• High dose steroid treatment is used in the setting of FSGS, and is also the backbone of malignant lymphocytotic disease such as multiple myeloma [3].
• Although prednisone is used in the setting of cFSGS, it poses an unforeseen problem when identifying the etiology in a rapidly declining renal function with confirmed IgM kappa light chain disease.
• There is limited research on the pathogenesis of non-HIV associated cFSGS, and similarly, a lack of clinical trials to support an optimal treatment plan.
• Uncovering a better treatment model may prevent this in the future.

Conclusion

We presented a patient in a small community hospital with collapsing subtype FSGS and coexisting paraproteinemia who improved following prednisone and hemodialysis, amongst other medical intervention. While further studies are needed to investigate the pathogenesis of non-HIV related disease, and identify a gold standard treatment, there are still ways to attack the disease from other angles such as blood pressure control and minimization of proteinuria.

Reference

• Reference #2: Up to date image