Focal Segmental Glomerulosclerosis in Malaria

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Introduction

Focal segmental glomerulosclerosis (FSGS) is the most common cause of nephrotic syndrome in American adults, accounting for about 4% of end stage renal disease [1]. Histological variants includes not-otherwise-specified (NOS), perihilar, cellular , tip and collapsing variants which is applicable to both primary and secondary focal segmental glomerulosclerosis [2,3]. Known causes of FSGS includes viral infections (HIV, EBV, CMV), hemophagocytic syndrome, drugs (pamidronate, interferon), and thromboembolic disease of kidneys. Although numerous etiologies have been reported, few cases illustrate the association between acute tubular injury - in this case due to malaria - and FSGS. Most cases are reported in Africa and Southeast Asia where malaria is more prevalent. Due to the rarity of malarial infection in United States, there are very few case reports written in association with the disease. Here we report a case of FSGS with collapsing variant, caused by *Plasmodium falciparum,* in an immigrant from an endemic area.

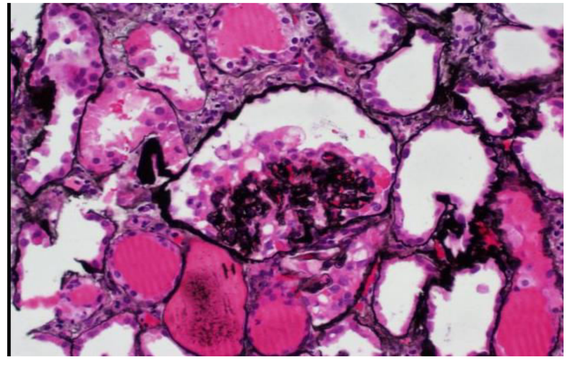
Case

A 56-years-old African male presented directly from the airport with complaints of dark urine, cough, body aches, and fever that began three weeks ago. The patient was travelling from the Ivory Coast where he was undergoing medical treatment for malaria; he had not completed the recommended course of treatment. Prior to arrival, he had intermittent symptoms of myalgia and fatigue for two weeks and was told by his physician that he had a low platelet count.

On examination, the patient was febrile and tachycardic, appeared lethargic and confused, and had icteric conjunctiva. Laboratory investigations were significant for platelet count of 25 (130-400 K/µL), blood urea nitrogen of 27 (9-20 mg/dL), creatinine of 1.6 (0.7-1.3mg/dL), sodium of 126 (137-145 mEq/L), potassium of 2.9 (3.5-5.1 mEq/L), albumin of 2.6 (3.5-5 g/dL), and C-reactive protein of 20.5 (0.7-1 mg/dL). Peripheral blood smear confirmed the presence of *Plasmodium falciparum.* The serological tests for viral hepatitis, dengue, and HIV were negative. The renal biopsy was done (figures 1 and 2) and was consistent with FSGS collapsing glomerulopathy associated with visceral podocyte hypertrophy and acute tubular injury.

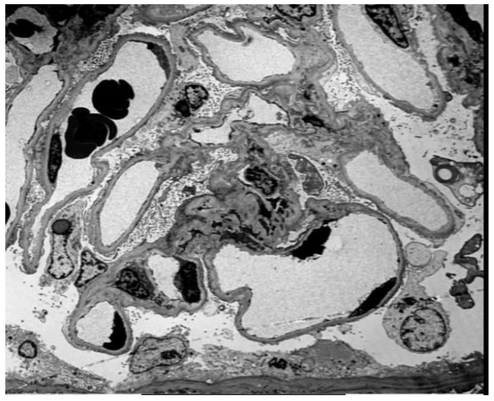
The patient was treated with three days of atovaqone-proguanil. Repeated peripheral smear showed no parasite and an improved platelet count. His renal function improved without the use of steroids or hemodialysis and he was discharged with outpatient follow up.

Figure 1.



Light microscopy of the kidney biopsy. The glomerulus with collapse of the capillary walls and visceral podocyte hypertrophy.

Figure 2.



Electron microscopy of the kidney biopsy showing global effacement of foot processes.

Discussion

The pathogenesis of FSGS in malaria is not known. Albaqumi *et al.* [4] speculates that in the collapse variant, external insult to the glomerular epithelium leads to a proinflammatory, nontolerogenic response by adjacent epithelial cells promoting capillary collapse in the glomerulus, microcystic transformation in the tubulointerstitium, and fibrosis and atrophy of the parenchyma. Regardless of the underlying causative process, management of secondary causes of the FSGS lesion involves treating the underlying condition. Although no specific treatment have been outlined, malaria-induced glomerulopathy should be managed by anti-malarials, supportive therapy, and hemodialysis if necessary. Corticosteriods did not respond in 72% of FSGS cases in a study [4]. The prognosis is very poor in those who develop end-stage renal disease (ESRD). Patients who are black and severely proteinuric, with poor response to treatment, are likely to rapidly progress to ESRD [5]. This case exemplifies how early intervention in treatment of malaria - without the use of steroids or hemodialysis - could indeed be a good prognostic indicator in collapsing glomerulopathy and may avoid progression to end-stage renal disease.

References:
1. Collins, A. J. et al. US Renal Data system 2010 annual data report, AM J. Kidney Dis. 57, e1-e526 (2011)
2. D'Agati VD, Gogo AB, Bruijn JA, Jennette JC. Pathologic classification of focal segmental Glomerulosclerosis: a working proposal. AM J Kidney Dis 2004, 43:368-82
3. Thomas DB, Franceschini N, Hogan SL, et al. Clinical and pathologic characteristics of focal segmental glomerulosclerosis pathologic variants. Kidney int 2006:69:90
4. Albaqumi M., Soos T. J., Barisoni L., Nelson P. J. Collapsing glomerulopathy. *Journal of the American Society of Nephrology*. 2006;17(10):2854–2863. doi: 10.1681/ASN.2006030225.
5. Schwartz MM[1], Evans J, Bain R, Korbet SM. Focal segmental glomerulosclerosis: prognostic implications of the cellular lesion. J Am Soc Nephrol. 1999 Sep;10(9):1900-7.