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SECONDARY IGA GLOMERULONEPHRITIS AS INITIAL PRESENTATION FOR CIRRHOSIS- A CASE REPORT

Bipneet Singh	Henry Ford Jackson, Michigan, USA
Palak Grover	Henry Ford Jackson, Michigan, USA
Jahnavi Ethakota	Henry Ford Jackson, Michigan, USA
Iman Ekfeki	Henry Ford Jackson, Michigan, USA
Sakshi Bai	Henry Ford Jackson, Michigan, USA
Gurleen Kaur	GMC Amritsar, India

ABSTRACT IgA disease can be primary or secondary. Primary is autoimmune due to impaired glycosylation, which produces kidney disease, whereas secondary disease with different kinds of impaired glycosylation rarely produces kidney disease due to an unknown mechanism. The case shows rare case of IgA nephropathy in a cirrhotic and further dwells into pathophysiology of the disease.

KEYWORDS : IgA nephropathy, Budesonide, Kupffer cells

INTRODUCTION

IgA nephropathy causes a myriad of pathology in kidneys. However, a subset of IgA disease, called secondary IgA nephropathy, is caused in the setting of cirrhosis. This likely occurs in the setting of impaired Kupfer cell clearance. This leads to impaired glycosylation, which is slightly different than primary IgA disease. However, secondary IgA disease rarely produces glomerular disease. However, our patient presented with IgA-induced glomerulonephritis due to secondary disease.

Case

A 61-year-old male was admitted to the intensive care unit 6 months ago with acute renal failure requiring continuous renal replacement therapy. The patient underwent a renal biopsy during the hospitalization given he had no prior history of renal disease. Renal biopsy results were consistent with IgA deposition. Hospital stay at the time was complicated with new onset atrial fibrillation and associated heart failure. The patient was started on Budesonide 16 mg and had an improvement in renal function. Further patient underwent TEE-guided cardioversion in an outpatient setting leading to improvement in ejection fraction from 25% to 65%.

A month after the initial presentation, the patient started developing abdominal distention and confusion and was taken to primary care. Suspecting possible ascites from cirrhosis, ordered chronic liver workup, which was unremarkable including anti smooth muscle antibody, ceruloplasmin, iron saturation, hepatitis-B hepatitis-C. The patient was deemed to have progress to cirrhosis more likely in the setting of IgA nephropathy and was started on rifaximin for encephalopathy and Lasix 40 mg and Aldactone 50 mg for new onset ascites.

Finally, 6 months after initial presentation, patient developed worsening confusion over one week followed by episodes of lethargy and fatigue. He was brought to the emergency department. He was hemodynamically stable with heart rate 64, blood pressure 110/58 mm of Hg, afebrile. The patient was alert but oriented to just place and person, he had asterixis on examination. He had abdominal distension with dullness on percussion. His lab work including white blood cell count electrolytes and renal functions were within normal limits. Hepatology was consulted and looking at imaging from previous years, the patient had steatosis likely in the setting of underlying obesity. It was theorized that the patient had developed cirrhosis in the setting of underlying metabolic steatosis, and that had further led to IgA nephropathy.

Suspecting worsening encephalopathy, work-up for exacerbating factors was initiated. Blood cultures, chest X-ray, urinalysis was ordered. CT head was obtained to rule out any bleeding or stroke. All the work-up was non remarkable. Patient was started on Lactulose and Rifaximin regimen without much improvement over next 2-3 days. At this point patient underwent an EEG showing patterns consistent with generalized neuropathy. Suspecting autoimmune neuritis, patient underwent a lumbar puncture showing IgG antibodies to West Nile Virus. Infectious diseases were consulted at this point and given absence of prior exposures; it was deemed a cross reactive antibody. Rheumatology then reviewed the patient and suggested steroid induced psychosis rather than an autoimmune disease.

Patient was started on a steroid taper from Budesonide 16 mg to 8 mg with gradual improvement in mentation.

Discussion

Secondary IgA nephropathy (IgAN) is a well-known co-occurring complication of liver cirrhosis (LC)[1,2]. The hepatic asialoglycoprotein receptor (ASGR) on Kupffer cells, is the primary mechanism via which polymeric IgA type 2 is removed [3]. Further cirrhotic patients have impaired motility and increased permeability which favors bacterial overgrowth, subsequent increased IgA production [4]. This leads to an increased circulating IgA against gut flora in patients with chronic liver disease [4]. Up to 90 percent of cirrhotic individuals, glomerular deposits of IgA have been observed [1,2].

Like in this case, there have been discussions about whether IgA nephropathy contributes to cirrhosis or whether people with chronic liver disease or cirrhosis are at higher risk for IgA related disease. However when cirrhotic patients' renal profiles improved with improved portal pressures after starting propranolol, there was some indication that the liver was initially involved[5], Nakamura et al [6]. In yet another distinct case of non-cirrhotic PH and IgA-induced Nephropathy, portacaval anastomosis decreased portal pressure, led to complete resolution of hematuria and proteinuria [7]. Further Babbs et al described a patient with IgA nephropathy due to portal hypertension in the setting of splenic artery aneurysm which improved with aneurysm resection and splenectomy[8].

Porto-caval shunting sounds counterproductive but hypothesis is shunting rather improves backflow to liver

leading to better metabolism of IgA. Further making the association interesting IgAN was found a few months after a combined liver and kidney transplant for some patients pointing IgA disease could have been primary to begin with [5].

CONCLUSION

In any cases with IgA nephropathy, cirrhosis should be ruled out given rare cases with secondary IgA disease.

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