

Inebriated liver causing mesangial turmoil

Abstract

IgA nephropathy is the most common primary glomerular disease worldwide, diagnosed 10% of renal biopsies in the United States, up to 20% in Europe and approximately 40% in Asia, the latter being the leading cause of ESRD in Asia. Multiple studies have shown the relationship between cirrhosis and the development of glomerular lesions leading IgA deposition in the mesangium causing IgAN. Here we present an unusual case of a 64-year-old female presenting with IgA nephropathy induced by liver cirrhosis.

Keywords: glomerular lesions, IgA nephropathy, hepatocellular carcinoma

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Case presentation

A 64-year-old female presented to the emergency department (ED) after a one week history of unexplained rash in the lower extremities. The rash began as a red, non palpable lesion located in the ankles bilaterally, that later on progressed to a purple lesion extending towards the mid portion of both thighs. Rash was associated with fatigue, generalized sharp abdominal pain, 3/10 in intensity that progressed to 7/10 in the last 3 days, also reported abdominal distention with non-exertional shortness of breath. The past medical history was significant for Hepatocellular carcinoma, alcohol induced cirrhosis undergoing liver transplant evaluation. On evaluation vital signs: Blood pressure 100/56mmhg, Heart rate 126, Respiratory rate 16, saturating 97% on room air. Physical exam was significant for purple macular, non-tender rash located in the ankles, extending to the mid-portion of the inner thigh bilaterally and hypogastric area, and non-tender abdominal distention with dry mucous membranes. The initial laboratory evaluation at the hospital revealed an elevated creatinine level at 2.39, a BUN of 22, total bilirubin 1.9 and INR 1.5, with normal transaminase, decreased complement C3, C4 levels at 51 and 11 respectively. Abdominal ultrasound done in the ED reported moderate ascites, therapeutic paracentesis was done significantly improving abdominal distention. Albumin was administered no improvement of renal function. Skin biopsy was performed and reported early lesion of leukocytoclastic vasculitis. Due to high suspicion of Henoch-Schonlein Purpura, 60mg of prednisone was started and significant improvement of renal function was noted. Renal biopsy showed deposition of IgA in the glomerulus confirming the etiology suspected.¹⁻⁸

Discussion

Glomerular deposits of IgA have been reported in 35% to 90% on cirrhotic patients. It has been previously stated that the majority of IgA synthesis in humans is mucosal, polymeric IgA with two isotypes, IgA1 and IgA2. Limited polymeric IgA reaches to human mucosal. Polymeric IgA2 is mainly eliminated through the hepatic asialoglycoprotein receptor as compared to Polymeric IgA1, this explains the reason why a higher percentage of polymeric IgA2 has been noted in the circulation of patients with chronic liver disease.⁹

Liver cirrhosis has been associated with the development of IgA nephropathy, pathogenesis remains uncertain, but is thought that inadequate liver IgA circulating immune complex metabolism leads to tissue deposition, and subsequently developing IgA nephritis. Nonetheless, It hasn't been established the degree of liver involvement associated with deposition of IgA immune complexes.¹⁰ In the present case, after confirming the etiology with skin and renal biopsy IgA nephropathy was diagnosed and was suspected to be associated with liver cirrhosis. Our approach with prednisone 60mg showed significant improvement of renal function after the first week of treatment.

Conflicts of interest

The author declares no conflicts of interest.

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