Comment on "Hepatic-associated Immunoglobulin A Nephropathy in a Child with Liver Cirrhosis and Portal Hypertension"

Sir,

We read with great interest the case report entitled "Hepatic-associated immunoglobulin-A nephropathy in a child with liver cirrhosis and portal hypertension," published recently in your esteemed journal.[1] The authors presented a 14-year-old boy with cryptogenic liver cirrhosis and portal hypertension (PH), who also had hematuria and proteinuria associated with histologic changes of IgA nephropathy (IgAN). Following a kidney biopsy, they found mesangial expansion and hypercellularity and glomerular basement membrane thickening on light microscopy (LM). Immunofluorescence (IF) study of the biopsy revealed diffuse granular deposits predominantly of IgA (2+), IgM (1+), IgG (1+), and C3c (1+). The ultrastructural examination using electron microscopy (EM) showed mesangial and endocapillary proliferation and focal para-mesangial dense deposits, consistent with IgAN. They attributed the kidney disease as secondary to chronic liver disease (CLD).

We take this opportunity to emphasize a few points relevant to the case.

- The authors have simply attributed IgAN diagnosis as secondary to liver disease without concrete proof of this causation. The treatment and ultimate outcome of both the liver and kidney disease in this child are also lacking. In the absence of such data, it can only be speculated about the association of one disease with the other. These may well represent just co-incidence of two separate and quite common in the adolescent age group
- While checking for the etiology of liver cirrhosis, the authors missed the serology of human delta virus (HDV). A few cases of CLD with this virus infection and no current evidence of hepatitis B virus infection (HBV)are reported
- To better explain the morphologic lesions by LM, it is indispensable to use the Oxford classification of IgAN, the international yardstick for reporting the pathological lesions of IgAN.[2-4] Although the latter scheme was primarily formulated on cases of primary IgAN, it is time to check its relevance in secondary cases also
- The exact definition of IgAN requires also absence of C1q deposits on immunostaining, as the mentioned morphologic lesions on LM may be presented by other systemic diseases, especially systemic lupus erythematosus (SLE)
- Additionally, the authors should have provided the extent of interstitial fibrosis/tubular atrophy (IFTA) and the proportion of globally sclerotic glomeruli, given the mild renal failure in the case.[5-7]

Hamid Nasri, Muhammed Mubarak[1]

Department of Nephrology, Division of Nephropathology, Isfahan University of Medical Sciences, Isfahan, Iran, [1]Department of Histopathology, Sindh Institute of Urology and Transplantation, Karachi, Pakistan
E-mail: drmubaraksiut@yahoo.com

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