■ S-473 ■

A case of IgA nephropathy combined with polycythemia vera

[1]Division of Nephrology, Departiment of Internal Medicine, Korea University Ansan Hospital, Ansan,
[2]Department of Internal Medicine, Ilsan Paik Hospital, Inje University College of Medicine, Goyang, Korea
[3]Department of Pathology, Korea University Ansan Hospital, Ansan, Korea

*S. Y. Kim[1], Y. S. Kang[1], D. R. Cha[1], S. Y. Han[2] and N. H. Won[3]

Polycythemia vera (PV) is a myeloproliferative disorder which is characerized by excessive production of erythrocytes as well as myeloid and megakaryocytic proliferation. IgA nephropathy is one of the most common form of glomerulonephritis. However, the relation of IgA nephropathy and polycythemia has rarely been described. We experienced the case of IgAN that is characteristic of heavy proteinuria and combined with polycythemia vera. Polycythemia vera was diagnosed via bone marrow biopsy and the polycythemia Vera Study Group (PVSG) criteria. Renal biopsy showed dominantly mesangial IgA, C3 deposition in a granular pattern and diffuse distribution. The patient was started with hydroxyurea and presented a good response to hydroxyurea with normal hemoglobin level.

■ S-474 ■

PRES during the recovery phase of AKI following hepatitis A

Nephrology Division, Department of Medicine, Samsung Medical Center, Sungkyunkwan University School of Medicine

*Soo Min Kim, Heejung Choi, Hye Ryoun Jang, Jung Eun Lee, Wooseong Huh, MD, Dae Joong Kim, Ha Young Oh, Yoon-Goo Kim

We report a case of posterior reversible encephalopathy syndrome (PRES) during the recovery phase of acute kidney injury (AKI) following fulminant hepatitis A. Although the pathogenesis of PRES remains unclear, autoregulatory failure and endothelial dysfunction were suggested as possible mechanisms. The patient was a 25-year-old healthy male. He suffered from fever, chill, and abdominal pain for 5 days prior to the hospital visit and was diagnosed with acute hepatitis A. At admission, he presented with anuric AKI and hepatic encephalopathy. He underwent continuous renal replacement therapy (CRRT) followed by regular hemodialysis. Hemolytic anemia, bilateral proximal thigh intermuscular hematoma, both radial artery pseudoaneurysms, and gastric ulcer bleeding were accompanied. At hospital day 30, the patient's urine output started increasing to more than 1000 cc/day which increased to 1610 cc/day at hospital day 31. The blood pressure had been well controlled with the use of a calcium channel blocker. At hospital day 32, headache and visual disturbance sudden developed and convulsion occurred 3 times followed by post-ictal confusion and high fever. T2 and fluid-attenuated inversion recovery (FLAIR) images of brain MRI revealed hyperintense signal alterations on bilateral subcortical regions of the temporo-parietal and occipital lobes, consistent with PRES. Spinal tapping showed increased intracranial pressure (IICP) but no evidence of CNS infection. Mental status was fully recovered after 7 hours of conservative treatment including CRRT and mannitol. Headache, visual disturbance, and fever were gradually improved. CRRT was stopped after 12 hours because urine volume increased to more than 150 cc/hour and azotemia was improved. Dialysis was not required thereafter. At hospital day 56, the patient was discharged with no neurologic sequelae. The patient's renal function was recovered to 60 mL/min/1.73m2 of eGFR 40 days after discharge. Our case suggests that PRES can develop during the recovery phase of severe AKI possibly through autoregulatory failure. Early diagnosis and lowering IICP with supportive treatment are important.