

### Tenofovir as a first line option for prophylaxis in a patient with hepatitis B virus associated nephrotic syndrome

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To the Editor,

The relation between HBV and glomerulonephritis including membranous and mesangio-proliferative glomerulonephritis has been well documented.<sup>1</sup> However, only six cases of HBV associated focal segmental glomerulosclerosis (FSGS) were reported previously.<sup>2-5</sup> Reduction of HBV DNA titers is important for remission of HBV-associated glomerulopathies. We herein represent a case of HBV-associated FSGS who relapsed under lamivudine therapy and improved with tenofovir treatment. We reviewed the pertinent literature regarding HBV-associated FSGS.

A 26 year-old man was admitted to our clinic with severe edema. He had a history of HBV infection for eight years. During follow up for HBV, his liver enzymes were within normal limits and HBV DNA was lower than 2,000 IU/mL hence did not need any treatment. Physical examination revealed 4+ pretibial edema. In his laboratory examination; BUN: 26 mg/dL, serum creatinine: 0.8 mg/dL, AST: 20 IU/L, ALT: 46 IU/L, Hbe Ag negative, anti-Hbe positive, HBV DNA: 1254 IU/mL, serum albumin: 2.5 g/dL and total protein excretion was 14.2 g/day in 24 h urine samples.

Percutaneous kidney biopsy was performed. The renal biopsy specimen was consistent with minimal change disease. Thereafter 30 mg/day prednisolone was started and tapered to 4 mg/day with 100 mg/day lamivudine treatment and angiotensin converting enzyme inhibitor (2.5 mg/day ramipril) because

of nephrotic syndrome. Proteinuria decreased to 2 g/day and partial remission achieved. After 18 months of treatment, proteinuria relapsed and increased to 5.7 g/day and HBV DNA elevated to 8454 IU/mL. Because of the recurrence of nephrotic syndrome under steroid therapy, renal biopsy was re-performed. The second renal biopsy was showed FSGS. Prednisolone was continued 4 mg/day, lamivudine discontinued and tenofovir 240 mg/day started because of increasing HBV DNA titers under lamivudine therapy. After 3 months, serum HBV DNA turned undetectable level and proteinuria decreased to 1.3 g/day. The partial remission has been continued during 15 months of treatment.

The duration of follow up was maximally 12 months in previous reported cases with FSGS.<sup>2-5</sup> The common treatment strategy includes steroid and lamivudine therapy in those cases. Remission was achieved in four patients and partial remission in one.<sup>2,3,5</sup> One patient had resistant to steroid plus lamivudine therapy.<sup>4</sup> The presence of remission was correlated with reducing titers of HBV DNA in all patients with remission by lamivudine therapy.<sup>2-4</sup>

This condition was consistent with other HBV related nephropathies.<sup>6</sup> Long term remission usually could not be achieved with lamivudine therapy because of increasing resistance to this drug by years.

Lai, *et al.*, emphasized increasing resistance of lamivudine and reported relapse of membranous glomerulonephritis after two years while the patients was under lamivudine therapy.<sup>7</sup> Similarly, nephrotic syndrome was relapsed in our patient after 18 month of lamivudine treatment with elevation of HBV DNA titers. These findings put emphasis on correlation between decreasing of HBV DNA titers and improvement of proteinuria and this relation is independent from antiviral agents.

In conclusion, tenofovir may be preferred as a first option for preemptive treatment in patients

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with HBV-associated focal segmental glomerulosclerosis who would use immune suppressive drugs.

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