

Improvement in dasatinib-induced proteinuria after switching to nilotinib: a case report

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Background: Chronic myelogenous leukemia (CML) develops as a result of the production of an activated tyrosine kinase. Imatinib is a first-generation tyrosine kinase inhibitor (TKI) that has been used to treat CML, whereas second generation TKIs, such as dasatinib and nilotinib, are used in cases with side effects or poor responses to imatinib. The adverse effects of these TKIs vary according to the inhibition site of the signaling pathways. In the present case report, we describe a case of proteinuria induced by dasatinib therapy. **Case presentation:** A 56-year-old Korean woman was referred to our clinic for thrombocytosis. Initial complete blood cell count examinations indicated a white blood cell count of $13.2 \times 10^3/\mu\text{L}$, hemoglobin level of 11.0 g/dL, and platelet count of $741 \times 10^3/\mu\text{L}$. She was diagnosed with CML based on the findings of a bone marrow biopsy. Dasatinib therapy (100 mg/day) was then initiated, and the patient exhibited complete hematologic remission within 6 months of drug initiation. However, after 3 years, she developed hypertension and proteinuria. Treatment with losartan (25 mg/day) was initiated, and the dose was gradually increased to 100 mg/day. As this treatment had no effect, a kidney biopsy was performed, which showed mildly enlarged glomeruli, leukocytic infiltration, and glomerular capillary walls that were mildly thickened with segmental corrugation and double contouring. Thereafter, dasatinib treatment was switched to nilotinib (300 mg, twice a day). After 1 month, the spot urine protein creatinine ratio was found to have decreased from 2985.0 mg/g to 237.8 mg/g. **Conclusion:** This is the first case of heavy proteinuria that developed after long-term TKI treatment (3 years) and improved after switching to another TKI agent. Although proteinuria appeared as a delayed renal complication of dasatinib, it rapidly improved after switching from dasatinib to another TKI (nilotinib) followed by complete remission of the proteinuria.

Comparison of tenofovir and entecavir in kidney transplanted patients with chronic hepatitis B

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Background: Nucleotide reverse transcriptase inhibitor is used for the treatment of chronic hepatitis B (CHB). Preemptive antiviral therapy can improve the survival of HBsAg-positive renal allograft recipients. But there are concerns about the potential risk of nephrotoxicity with long-term use. This study aims to assess the nephrotoxicity and efficacy of tenofovir and entecavir in kidney transplant recipients. **Methods:** We performed a single center based, retrospective study of 55 patients with CHB treated with tenofovir (n=34) and entecavir (n=21) after kidney transplantation (KT). Patients with an estimated glomerular filtration rate (eGFR) decrease <20% were recorded, calculated using 4-variable Modification of Diet in Renal Disease Study equation. Treatment efficacy was assessed by HBV DNA levels and virological breakthrough. **Results:** Tenofovir was associated with a decrease in eGFR at 1 year from initiation of treatment (unadjusted odds ratio, 10.313; 95% confidence interval, 1.111-95.762; $p=0.026$; number of patients with decreased eGFR, 5/21 vs. 1/34). Mean eGFR at 1 year was 55.6 ml/min/1.73 m² and 64.0 ml/min/1.73 m² in tenofovir and entecavir group. There was no significant change in eGFR between the tenofovir group and the entecavir group after a mean of 45 months (mean eGFR, 62.7 ml/min/1.73 m² vs. 56.5 ml/min/1.73 m²). And there was no difference in rate of virological breakthrough between the tenofovir group and the entecavir group at the end of treatment (number of patients with virological breakthrough, 5/21 vs. 6/34; $p=0.731$). By multivariate analysis, the significant factor associated with a decrease in eGFR at 1 year from treatment were tenofovir (adjusted odds ratio, 18.477; 95% confidence interval, 1.123-275.953; $p=0.034$). **Conclusions:** Patients who received KT and treated with tenofovir were likely to have decline in renal function than patients who received KT and treated with entecavir at 1 year from treatment. Tenofovir was independently associated with decrease in eGFR at 1 year from treatment. There was no significant difference in an efficacy between tenofovir and entecavir group at the end of treatment.