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Effect of interferon free antiviral therapy on glomerular and tubular kidney involvement in HCV child-acirrhosis

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Background: Hepatitis C virus (HCV) infection is associated with an increased risk of renal disease. The correlation between HCV infection and glomerular damage is well recognized, but limited data are available on HCV-mediated tubular damage. Recently, several novel direct antiviral agents (DAAs) have been approved for HCV treatment, but the effects of HCV clearance on renal involvement has not been fully characterized. The aim of this study was to evaluate the effect of viral eradication, by means of DAAs, on renal glomerular (GI) and tubular involvement (TI) in pts with HCV-related cirrhosis.

Material/methods: 94 Child-Pugh A cirrhotic pts treated with DAAs were consecutively enrolled. Estimated glomerular filtration rate (e-GFR) assessed by CKD-EPI equation, urinary albumin to creatinine ratio (ACR), urinary α1-microglobulin to creatinine ratio (α1MCR) and fractional excretion of sodium (FeNa) were evaluated before starting therapy (T0) and six months after treatment withdrawal (FU6). GI was defined as ACR > 30 mg/g and TI was defined as α1MCR > 14 mg/g and/or FeNa > 1%.

Results: Renal involvement (glomerular and/or tubular) occurred in 39 pts (41.5%). GI was found in 19 pts (20.2%), 6 of them (31.6%) had diabetes. TI was detected in 30 pts (31.9%). Pts with renal involvement showed significantly lower e-GFR values than pts without renal involvement (95.2 ± 15.2 mL/min/1.73 m² vs 85.1 ± 15.8 mL/min/1.73 m², p=0.07). In diabetic pts with GI ACR did not change after antiviral treatment (316.2 ± 406.7 mg/g vs 321.3 ± 416.2 mg/g, p=0.92), while a significant reduction of ACR (73.5 ± 138.5 mg/g vs 19.9 ± 12.3 mg/g, p=0.019) occurred in non-diabetic HCV cirrhotic pts and GI resolved in 11/13 (84.6%) pts without diabetes. The proportion of pts with TI decreased significantly after DAAs treatment, since in 14/25 (56%) patients tubular involvement recovered. No significant difference of e-GFR was observed between T0 and FU6 in all pts with any kind of kidney involvement.

Conclusions: Our study confirms a strong relationship between HCV infection and kidney glomerular involvement and underlines significant occurrence of tubular involvement. In HCV cirrhotic pts with diabetes the glomerular damage seems to be mainly driven by the metabolic disorder rather than by HCV infection itself. This is the first report demonstrating a significant improvement of either non-diabetic glomerular, either tubular HCV-induced damage after HCV clearance by interferon free antiviral therapy, emphasizing the importance of antiviral treatment.