KSN 2022
SEOUL, KOREA MAY 26 - 29

Abstract Submission No. : 1089

Urinary cytokines/chemokines for differential diagnosis of acute interstitial nephritis and acute tubular necrosis

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Objectives: Acute interstitial nephritis (AIN) accounts for 5-27% of unexplained acute kidney injury (AKI) and acute tubular necrosis (ATN) is the leading cause of AKI among hospitalized patients. Definitive diagnostic method to differentiate AIN from ATN is kidney biopsy. However, kidney biopsy carries the risk of bleeding and may not be feasible in some patients. We aimed to elucidate noninvasive urinary biomarkers for differential diagnosis of AIN and ATN as well as predicting response to steroid treatment in AIN.

Methods: Adult patients who underwent kidney biopsy from 2001 to 2020 at Samsung Medical Center were included. Patients with kidney transplantation and other pathological findings such as glomerulonephritis, diabetic nephropathy, and hypertensive nephrosclerosis in the biopsy report were excluded. Serum and urine samples were collected on the day of kidney biopsy. TNF-α, IL-9, TGF-β, RANTES, VEGF, and MCP-1 were analyzed. Urinary cytokines/chemokines levels were adjusted with urinary creatinine levels. A semi-quantitative analysis of immunohistochemistry staining for CD3, CD20, CD45, and Ki-67 was also performed with QuPath.

Results: A total of 89 patients (34 in ATN, 55 in AIN) were included in the final analysis. Serum TNF-α, IL-9, TGF-β, RANTES, VEGF, and MCP-1 were comparable in both groups. Urinary RANTES/creatinine was significantly higher in the AIN group (ATN vs. AIN, pg/mg, 13.8 [0-23.3] vs. 63.5 [12.5-296.5], p=0.022), whereas urinary TNF-α, IL-9, TGF-β, VEGF, and MCP-1 were comparable between groups. Semi-quantitative analyses showed that the percentage of intrarenal CD3, CD20, CD45, and Ki-67 positive cells was significantly higher in the AIN group compared to the ATN group.

Conclusions: Our study suggests the clinical relevance of urinary RANTES/creatinine as a noninvasive biomarker for differential diagnosis of AIN and ATN.