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Inflammatory and tubular damage markers in children with chronic kidney disease

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Description

Increased immunocompetent cell migration to the sites of inflammation and consequent renal fibrosis are features of Chronic Kidney Disease (CKD). The latter is a crucial component of tubular injury that is gradual and permanent. The aforementioned CKD-related problems are influenced by a variety of chemotactic drugs. Early stages of cell migration activity, such as the migration of monocytes to the sites of inflammation and their conversion to macrophages, are governed by Monocyte Chemoattractant Protein-1 (MCP-1) and Macrophage Colony-Stimulating Factor (MCSF).

Therefore, the intensity of the inflammatory process, the activity of the monocytes and macrophages, as well as the cell damage during chemotactic migration and transition, may be treated instead of the MCP-1 and MCSF activity. MCP-1's profibrotic activity and tubular cell localisation have been demonstrated in animal experiments.

Children with focal segmental glomerulosclerosis and adults receiving hemodialysis have both had their MCP-1 gene (MCP-1-2518 A/G) polymorphism examined. MCSF was evaluated in grownups receiving hemodialysis. MCP-1 or MCSF, however, have never been the centre of attention for the development of chronic renal disease in children. Neopterin was the lone exception in this group because it is produced as a result of increased cell migration and continuing inflammation rather than as a cause. Neopterin is specifically excreted by monocytes and macrophages in response to stimulation. Consequently, this chemical might be a sign of cellular immunity. Adults with nephrotic syndrome, those in late stages of CKD, and those receiving hemodialysis all had higher serum and urine amounts of neopterin. Adults with mesangial proliferative glomerulonephritis also showed higher neopterin excretion with urine. Neopterin served as a helpful risk indicator of renal transplant rejection. The aforementioned variables have never been examined in children with CKD as indicators of inflammation or macrophage activity, though.

Fractional Excretion (FE) study has only been done on phosphate metabolism in clinical practise as a replacement for tubular dysfunction in CKD patients. Heat shock protein (Hsp27) in adults and apoptotic markers in youngsters from our prior studies were the focus of the scientific approach to FE. The results of that experiment supported the value of FE in illustrating tubular damage and the epithelial-mesenchymal transition in CKD patients. However, neither MCP-1, MCSF, nor neopterin have been investigated as potential indicators of tubular damage in chronic kidney disease in light of fractional excretion. By measuring the levels of MCP-1, MCSF, and neopterin in the blood and urine of children with CKD and of controls, this study sought to evaluate

the components involved in monocyte activation, migration, and their conversion into macrophages. As potential indicators of inflammation, monocyte-macrophage interaction, and tubular damage in the course of CKD, we also assessed the use of Fractional Excretion (FE) of MCP-1, MCSF, and neopterin.

In CKD stages 1-2, the MCP-1 concentrations in serum and urine had already increased dramatically. The interpretation of these results was difficult because there are no pertinent data on MCP-1 levels in individuals with CKD receiving conservative treatment. Since there is no correlation between any of the data and eGFR, the source of the serum elevation was more likely overproduction than accumulation, although enhanced excretion of urine inferred the activation of monocytes in the renal interstitium. This could imply that MCP-1, specifically monocyte activity, is a helpful indicator of inflammation in both serum and urine. Additionally, because kidney MCP-1 is primarily found in the proximal tubules, urine MCP-1 levels may reflect how well they are functioning.

Conclusion

In the course of chronic kidney disease, measuring tubular damage and inflammation may be possible using fractional excretion of the studied indicators. According to the FE MCP-1 levels, tubular dysfunction is preceded by an inflammatory process. New markers of renal parenchyma increasing deterioration in children with CKD may include FE MCSF and FE neopterin.