Expansion and characterization of regulatory T cell populations from acute kidney injury patients

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Background: The mechanism of acute renal injury is complex. Previous studies have shown that the immune inflammatory response plays an important role in acute kidney injury. Regulatory T cells, one of the CD4-positive T cells, express IL-2 receptor (CD25). Foxp3 (Forkhead Box P3), a transcription factor that regulates immunosuppressive activity, and FoxP3-positive regulatory T cells are the drugs normal kidney monocytes, which accounts for 2%. Recent studies have shown that regulatory T cells play a role in protecting the kidney and are the expected immunotherapy target in acute renal injury. In this study, regulatory T cells were isolated and proliferated from patients with acute renal injury to confirm the kidney prognosis. Regulatory T cells that proliferated in vitro were re-administered to the same patient to reduce kidney damage, prevent chronic kidney disease, and reduce the occurrence of end-stage renal disease.

Methods: Thirty patients with acute kidney injury between March and December 2020 were enrolled in this study. Differentiation and expansion of Tregs were determined using flow cytometry to compare Treg subpopulations. Tregs were defined as CD4⁺CD25^{high}CD127^{low}/FoxP3⁺ cells.

Results: In patients with acute renal injury, the number of regulatory T cells increased immediately after the decrease in renal function, and the number of regulatory T cells was normalized after the renal function was restored.

Conclusions: In patients with acute renal injury, the number of regulatory T cells increased immediately after the decrease in renal function, and the number of regulatory T cells was normalized after the renal function was restored. In the future, it is expected to reduce kidney damage, the occurrence of chronic kidney disease, and end-stage kidney disease by re-administering regulatory T cells grown in vitro to the same patient.

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