

## Circulating lymphocytes, monocytes and cytokines in renal transplant recipients and healthy controls: a longitudinal analysis

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Renal transplant recipients (RTRs) are at increased risk of cardiovascular disease, infection and malignancy. Surveillance of biomarkers associated with immunity and inflammation would be a valuable tool in patient management to predict events and guide clinical decisions. The aim of this study was to compare immune and inflammatory cells and cytokines in stable RTRs and healthy controls over a longitudinal period of 6 months.

Resting blood samples were taken from stable renal transplant recipients (n = 8, age: 54 ± 12 years, BMI: 25.6 ± 4.7 kg.m<sup>2</sup>, eGFR: 60 ± 12 ml·min<sup>-1</sup>·1.73m<sup>2</sup>) and healthy controls (n = 8, age: 42 ± 19 years, BMI: 24.4 ± 3.5 kg.m<sup>2</sup>, eGFR: 88 ± 2 ml·min<sup>-1</sup>·1.73m<sup>2</sup>) at week 0, week 4, week 8 and week 24. Peripheral blood mononuclear cells were stained with lymphocyte, monocyte, phenotypic and activation markers. EDTA-plasma was analysed for cytokines (IL-6, IL-10 and TNF-α) using affinity ELISA kits.

A Mann-Whitney U test was used to assess between-group differences and Friedman's ANOVA was used to assess within-group differences; any significant findings were followed up using a post-hoc test. There were no differences for circulating lymphocytes or monocyte lineage markers within or between groups over time (P > 0.05). Renal transplant recipients had greater circulating IL-6 at week 0 (P = 0.021) and week 4 (P = 0.011) than healthy controls. TNF-α was greater in renal transplant recipients at week 0 (P = 0.003), week 4 (P = 0.001), week 8 (P = 0.002) and at 6-months (P = 0.029) than healthy controls. No within-group differences were observed over time (P > 0.05).

This is the first study to compare immune and inflammatory cells in renal transplant recipients and healthy controls over a longitudinal period in their habitual states. Our data indicates elevated cytokines in renal transplant recipients that is likely contributing to systemic inflammation.