# EFFECT OF ANTI-TUMOR NECROSIS FACTOR THERAPY ON KIDNEY FUNCTIONS IN PATIENTS WITH INFLAMMATORY ARTHRITIS

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**INTRODUCTION AND AIMS:** Anti-tumor necrosis factor-alpha (TNF-α) inhibitors provide fast and effective resolution of inflammation in patients with rheumatoid arthritis (RA) and ankylosing spondylitis. However, new adverse events are being reported with the increased use of TNF-α therapy. The effect of anti-TNF-α therapy on kidney functions remains unclear. We aimed to investigate the effect of etanercept treatment on kidney functions, namely proximal tubular and glomerular functions.

**METHODS:** 11 RA and 8 AS subjects who were unresponsive to standard first line therapy and planned to begin etanercept treatment were included to this prospective observational study. Demographic data were recorded. All subjects received etanercept at a dose of 25 mg subcutaneously twice per week. Inflammatory markers, serum creatinine, blood urea nitrogen, creatinine clearance (CCR), plasma cystatin C, urinary N-acetyl-ß-glucosaminidase, microalbumin excretion and ß2-microglobulin were measured at baseline before treatment and at 4$^{th}$ and 16$^{th}$ weeks. Disease activity scores were evaluated with disease activity score 28-erythrocyte sedimentation rate (DAS-ESR) in RA patients and with Bath AS disease activity index (BASDAI) in AS patients. Statistical analyses were performed using the Wilcoxon signed-rank and Fisher exact tests.

**RESULTS:** The mean ages of the patients with RA and AS were 50.8±12.2 and 35.1±8.7 respectively. Duration of disease was 9.3±5.1 years in RA patients and 4.2±5 years in AS patients. Inflammatory markers and disease activity scores were significantly improved in both groups after TNF-α therapy. Biochemical parameters evaluating renal functions were unchanged after treatment compared to baseline levels (Table 1, p>0.05 for all comparisons).

**Table 1: Summary Of Disease Activity And Kidney Function Parameters**

| | Baseline | 4$^{th}$ week | 16$^{th}$ week |
|---|---|---|---|
| Urea (mg/dl) | 28±10 | 30±10 | 27±8 |
| Creatinine (mg/dl) | 0.84±0.24 | 0.8±0.14 | 0.82±0.14 |
| CCR (ml/min) | 96±31 | 100±28 | 93±29 |
| Cystatin-C (mg/l) | 1.03±0.25 | 1.05±0.24 | 1.04±0.18 |
| Urine NAG (U/L) | 6.31±4.16 | 5.42±2.94 | 7.16±4.01 |
| Urine NAG (U/24 hours) | 12.87±8.45 | 9.93±4.65 | 12.57±6.12 |
| Microalbuminuria (mg/24 hours) | 16.9±14.8 | 31±69.8 | 27.4±53.2 |
| β-2 microglobulin (µg/l) | 1795±381 | 1754±461 | 1889±432 |

CCR: Creatinine clearance, NAG: N-acetyl-ß-D-glucosaminidase

**CONCLUSIONS:** Our data suggest that TNF-α therapy does not have adverse effects on the proximal tubule and glomerular structure in short-term. However, longitudinal studies are needed to further explore the changes in kidney function with different anti-TNF-α agents.

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