ABSTRACT

“The Effect of Urinary Tract Infection (UTI) by *Escherichia coli* on the Expression of Osteopontin as Urinary Stone Formation Promotor”

**Background:** The effect of urinary tract infection (UTI) on the mucosal damage and promotor protein production which promote urinary stone formation has not been elucidated. **Objective:** To demonstrate that expression of TLR-4, JNK, mRNA OPN, OPN, TNFR-1, iNOS, HMGB-1, and apoptosis process are higher than normal at tubulus cells due to urinary tract infection by *Escherichia coli*. **Material & Method:** A randomized post test only controlled group design study was performed. Thirty New Zealand strain rabbits were divided in 3 groups. The first group acted as controls, the second group underwent ligation of right ureter, the third group underwent ligation of right ureter and injection of *Escherichia coli* $10^5$/ml proximal to ligation. Nephrectomy and histological examination were performed after 5 days. Kidney specimens from all groups were HE stained to examine mucosal damage, and specific monoclonal antibodies for TLR-4, JNK, mRNA OPN, OPN, TNFR-1, iNOS and HMGB-1. Apoptotic nuclei were demonstrated using TUNEL method. Statistical calculations were performed using Anova test, with $p<0.05$ considered significant. **Results:** The findings confirmed the hypothesis that infection of urinary tract by *Escherichia coli* demonstrated higher expression of TLR-4, JNK, mRNA OPN, OPN, TNFR-1, iNOS, HMGB-1, apoptosis process and mucosal damage than normal. **Conclusions:** Infection of urinary tract by *Escherichia coli* caused higher than normal expression of promotor protein osteopontin and mucosal damage at kidney tubulus. These findings suggest that urinary infection may promote stone formation by mucosal damage and elevate promotor protein osteopontin at tubulus cell, allowing easier crystal retention and nucleation.

**Keywords:** UTI, osteopontin, apoptosis, necrosis, stone formation.
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