THYROXINE FOR ACUTE KIDNEY INJURY IN HUMANS: A SYSTEMATIC REVIEW

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In experimental models, thyroxine has been shown to improve recovery from toxic and ischemic acute kidney injury (AKI) by enhancing EGF/EGFR-mediated epithelial recovery. However, clinical trials involving thyroxine have shown discouraging results. We aimed to systematically review the randomized controlled trials (RCTs) to ascertain the use of thyroxine in treatment of AKI.

We searched the Cochrane Renal Group's specialized register, Cochrane Central Register of Controlled Trials, MEDLINE, EMBASE and reference lists of retrieved articles. Two reviewers independently identified relevant trials, abstracted patient data, treatment characteristics, and outcomes. Statistical analyses were performed using the random effects model and results expressed as relative risks (RR) for dichotomous outcomes and weighted mean difference for continuous outcomes, with 95% confidence intervals (CI).

Our search identified 4 trials out of which 2 satisfied our study eligibility criteria. There were no significant differences between thyroxine and control arms in terms of renal replacement therapy (RRT) requirement (2 RCTs, 79 patients, RR 1.67, 95% CI 0.68-4.10), RRT duration (2 RCTs, 79 patients, WMD 3.34 days, 95% CI -0.53 to 7.20), and mortality (2 RCTs, 79 patients, RR 1.84, 95% CI 0.51 to 6.61) [Figures 1 and 2].There were no significant differences in peak and end-of-treatment serum creatinine values between the two arms. None of the studies reported increased incidence of adverse effects with thyroxine.

In contrast to the beneficial effects seen in experimental AKI, we found that thyroxine has no effect on the course and mortality of AKI in humans. Sample sizes of the included studies are small and involved patients in these studies had severe AKI. Role of thyroxine in milder forms of AKI and as a prevention strategy remains unknown and an appropriately powered trial is needed to look at these roles.