Thesis Title : MECHANISMS OF RENAL FAILURE AFTER UNILATERAL URETERAL OBSTRUCTION

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ABSTRACT

Renal blood flow (RBF) and glomerular filtration rate (GFR) were significantly decreased after release of 24 hours of unilateral ureteral obstruction (UUO) in rats. The mechanisms responsible for these reduction are still unclear. The present studies were performed to investigate the role of prostaglandin and thromboxane on the reduction of glomerular filtration rate, especially after renal blood flow was normalized by iso-oncotic volume loading. Furthermore, the effect of calcium blocker, verapamil on renal function after UUO was also investigated.

Inhibition of thromboxane synthesis by imidazole did not improve the depressed RBF or GFR after UUO, but with simultaneous iso-oncotic loading RBF was normalized whereas GFR was only 50% of control. With prostaglandin inhibition by indomethacin, GFR was slightly elevated in the absence of any change in RBF. Simultaneous iso-oncotic loading in this group resulted in normalization of both RBF and GFR.

Interestingly, verapamil infusion resulted in both improvement of GFR and RBF by about 110% and 42% of pretreatment

values, respectively. Furthermore, iso-oncotic loading in this group also normalized both RBF and GFR.

These results suggest that thromboxane may not have any effect on renal function or hemodynamic after UUO, whereas, indomethacin induced a marginal increase in GFR possibly via an increment in Kf and/or an elevation in glomerular proximal hydrostatic pressure. Lastly, both vasodilation and an increment in Kf may contribute to the elevation of both GFR and RPF, induced by verapamil.