Introduction:
Ischemia-Reperfusion (IR) causes renal dysfunction and damage. IR induces renal tubular injury triggered by hypoxia and hyperoxia, mediated by oxidative stress and inflammation. Furosemide inhibits Na\(^+\)-K\(^+\)-2Cl\(^-\) cotransporter in the thick ascending limb of the renal medulla to decrease Na\(^+\) reabsorption, reducing oxygen consumption. We investigated if furosemide could improve renal oxygenation, function and damage by reducing O\(_2\) consumption and oxidative stress after IR.

Methods:
24 Wistar albino rats were divided into 4 groups, with 6 in each group; Sham-operated Control (C), Control + Furosemide (C+F), IR and IR+F. After anaesthesia (BL), 45 min supra-aortic occlusion was applied to IR and IR+F groups followed by 15 min (T1) and 2 hours of reperfusion (T2). Furosemide 50\(\mu\)g/kg/h infusion was simultaneously administered to C+F and IR+F after ischemia. Systemic hemodynamic, Renal Blood Flow (RBF), Renal Vascular Resistance (RVR), Renal Oxygen Delivery (DO\(_{2\text{ren}}\)), Renal Oxygen Consumption (VO\(_{2\text{ren}}\)), Creatinine clearance (Ccr), Sodium handling, Urine Output (UO), Cortical (C\(\mu\)O\(_2\)) and Medullar (M\(\mu\)O\(_2\)) microvascular oxygenation were measured.

Results:
RBF was reduced in IR (2.1\(\pm\)1) and IR+F (2.3\(\pm\)1) at T1 (p<0.05) but it was further reduced in IR+F (1.9\(\pm\)1) (p<0.05) at T2 compared to C and C+F. RVR was increased in IR (5338\(\pm\)2860) and IR+F (5123\(\pm\)2517) at T1 compared to C. RVR was normalized in IR (2198\(\pm\)879) but not in IR+F (4232\(\pm\)2636) at T2 compared to C (p<0.05). C\(\mu\)O\(_2\) and M\(\mu\)O\(_2\) did not differ between groups after IR insults. Tissue O\(_2\) was reduced at the medulla, but not at the cortex in IR+F group compared to IR. DO\(_{2\text{ren}}\) and VO\(_{2\text{ren}}\) were reduced in IR (56\(\pm\)17 and 26\(\pm\)12 ml/min) and IR+F (34\(\pm\)20 and 21\(\pm\)14) at T2 (p<0.05). PC was higher in IR+F (37.33\(\pm\)4.27) compared to IR 29.67\(\pm\)3.39 (p<0.05). VO\(_2\)/TNa\(^+\) was increased in IR+F compared to IR. No change in Ccr and UO was observed.

Conclusion:
Furosemide after IR causes further impairment of renal perfusion, energy utilization and renal oxygenation resulting in renal damage.

Image:
Cortical and Medullar Oxygenation. C: Control, C+F: C+Furosemide, IR: Ischemia Reperfusion, IR+F: IR + Furosemide *: p<0.05 vs BL +: p<0.05 vs T1 #: p<0.05 vs C