Objective:

In this experimental study we aimed to investigate

the preconditioning effect of ozone therapy on renal ischemia–reperfusion injury (IR) induced by transient occlusion of infrarenal abdominal aorta (IAA) in rats.

Methods:

Twenty-four Wistar-albino rats were randomized into

three groups (eight per group) as follows: control (sham laparotomy), aortic IR, aortic IR + ozone groups. Aortic IR was achieved by clamping the IAA with microvascular clamp for 60 minutes followed by 60 minutes of reperfusion. Ozone (95% O2 and 5% O3 mix) 1 mg/kg/daily was administered intraperitonealy as a single dose for 10 days. The rats were then decapitated under deep anesthesia and the kidneys were immediately removed for the measurement of tissue levels of malondialdehyde (MDA), superoxide dismutase (SOD), catalase and glutathion peroxidase (GPx) and histopathologic examination.

Results:
Biochemical analysis revealed that, aortic IR caused an increment in tissue levels of SOD and catalase (p < 0.05 vs control).

In the ozone therapy group tissue levels of SOD and catalase were found to be significantly lower than the level in aortic IR group (p < 0.05 vs aortic IR). The tissue levels of the other measured enzymes (MDA and GPx) were also lower in the ozone group as compared to aortic IR group, however the differences were not statistically significant. Histopathologic examination was performed by means of a scoring system based on dilatation of Bowman’s capsule, degeneration of tubular epithelium, tubular dilatation, congestion and interstitial inflammatory infiltration in kidney tissue samples. Pathologic score was observed to be significantly increased in aortic IR (p < 0.05 vs control). Ozone
therapy also lead to an attenuation of ischemia-reperfusion induced tissue damage depicted by a significantly decreased pathologic score in the ozone group as compared to the aortic IR group (p < 0.05).

Conclusions:

The results of this experimental study suggested that ozone therapy ameliorates the renal ischemia-reperfusion injury induced by transient IAA occlusion in rats. These findings may be explained by the preconditioning effect of ozone therapy which decreases the IR induced oxidative stress, however we think that further studies are needed for understanding the underlying mechanisms.