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Naringin as preventive effect on a rat model of Renal Ischemia-Reperfusion Injury.

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Objectives:

In a rat model of I/R renal ischemic reperfusion injury we investigated the mechanism of action of Naringin to determine its role in treating I/R injury.

Methods:

The male rat was randomly divided in 28 groups: control; ischemia-reperfusion (I/R); naringin+ ischemia-reperfusion (naringin+ I/R). In rats control, an abdominal incision was made and the bilateral renal pedicle dissected without further arterial cross-clamping. Naringin+I/R rats were given 50 mg/kg naringin per day for 7 consecutive days before surgery, while 0.9% salinity + 0.1% (v/v) ethanol per day for 7 consecutive days prior to surgery were administered.Renal ischemia to Naringin+I/R and I/R mice caused acute kidney injury after 45-minutes of blockage of bilateral renal arteries. Serum creatinine and blood urea levels of nitrogen have been measured. Periodic staining of acid-ship (PAS) was carried out to detect damaged renal tissue. The activity of myeloperoxidase (MPO) and the immunofluorescent CD68 expression detection were performed for each group. ELISA monitored inflammatory cytokines secreted by renal tissue.

Results:

In the naringin+ I/R and I/R groups, serum creatinine and blood urea nitrogen levels were high, compared to rats ($p<0.05$), while the I/R group was considerably higher than the I/R Group ($p<0.05$).

There was a significantly lower number of damaged renal tubular cells and apoptotic cells in control and I/R groups of Naringin + as in I/R rats. Inflammatory cytokines have been reduced to activity and secretion of MPOs in naringin+ I/R and I/R groups ($p < 0.05$) and CD68 in naringin+ I/R rats was lower than in I/R rats ($p < 0.05$). The expressions of Bax and cleaved-caspase-3 in the I/R group were considerably higher than in a rat control, whereas Bcl-2 was less than in I/R rat naringin+ ($p<0.05$).

Conclusions: Naringin may inhibit I/R renal injury by inhibiting the release and regulation of inflammatory factors.