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Captopril ameliorates L-arginine induced acute pancreatitis via downregulation of iNOS and elevation of glutathione

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Acute pancreatitis (AP) is a common inflammatory disease mediated by damage in acinar cells and subsequent pancreatic inflammation with infiltration of leukocytes. The pancreatic renin-angiotensin system may play an important role in the pathogenesis of AP. The present study aimed to investigate the possible role of captopril (CAP), an angiotensin-converting enzyme inhibitor, in attenuating L-arginine-induced AP in a rat model and to elucidate the underlying molecular mechanisms. Forty-eight adult male Wister rats were divided into four equal groups: control group (rats received vehicle orally for 10 days), AP group (3 g/kg L-arginine, single i.p.) on 10th day of the experiment, CAP group (50 mg/kg captopril, orally, once daily) and MP group (30 mg/kg methylprednisolone, orally, once daily). CAP and MP were administered for 10 days prior to L-arginine injection. Then rats were sacrificed 24 hours after L-arginine injection. Inflammatory biomarkers; pancreatic tumor necrosis factor-alpha (TNF-α) concentration, myeloperoxidase (MPO) activity and inducible nitric oxide synthase (iNOS) gene expression were determined. Oxidative stress biomarkers; nitric oxide (NO) and reduced glutathione (GSH) concentrations were assayed. In addition, serum α-amylase and lipase activities were measured and histopathological studies of the pancreas were done. CAP treatment significantly reduced TNF-α, MPO activity, NO and downregulated iNOS gene expression compared to AP group. CAP treatment significantly, increased pancreatic GSH and ameliorated the histological changes of AP. Captopril treatment may have a protective role in AP rat model which is comparable to MP treatment.

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