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PROTECTIVE EFFECT OF TAURINE ON INDOMETHACIN -INDUCED GASTRIC MUCOSAL INJURY

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It has been suggested that oxygen-derived free radicals have an important role in the pathophysiology of acute gastric ulceration induced by NSAIDs and ischemia-reperfusion. Taurine is hypothetized to exert its protective effect on NSAIDs-induced gastric injury by its antioxidant properties. Protective effect of taurine on indomethacin-induced gastric mucosal lesion and its protective mechanism were investigated. Intragastric administration of 25 mg/kg of indomethacin induced hemorrhagic lesions on the glandular stomach in rats. Pretreatment with 0.25 g/kg of taurine for 3 days significantly reduced the gastric lesion formation and inhibited the elevation of lipid peroxide level in gastric mucosa. Both resting and FMLP-induced luminol-dependent chemiluminescence of rat peritoneal neutrophils increased immediately after treatment of indomethacin. 5-20mM of taurine inhibited chemiluminescence of neutrophils activated by indomethacin and/or FMLP. Human neutrophils (polymorphonuclear leukocytes) significantly adhered to confluent monolayer of human umbilical vein endothelial cells(HUVEC) after coincubation with aspirin or indomethacin. Also taurine prevented neutrophil adhesion induced by these drugs to HUVEC in dose-dependent manner. These results indicate that the protective effect of taurine against NSAIDs-induced gastric mucosal injury is due to its antioxidant effect, which inhibits lipid peroxidation and neutrophil activation.