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(57) Abstract :
6. ABSTRACT OF THE INVENTION: Ammonia is an important source of nitrogen and is required for amino acid synthesis. It is also necessary for normal acid-base balance. When present in high concentrations, ammonia is toxic. Endogenous ammonia intoxication can occur when there is impaired capacity of the body to excrete nitrogenous waste, as seen with congenital enzymatic deficiencies. Hyperammonemia is a major contributing factor to neurological abnormalities observed in hepatic encephalopathy and in congenital defects of ammonia detoxication. Ammonia toxicity results in lipid peroxidation and free radical generation, which cause hepatic dysfunction and failure and significantly increase the number of brain peripheral benzodiazepine receptors and could increase the affinity of ligands for these receptors that might enhance GABA (gamma-amino butyric acid) adrenegergic neurotransmission. Naringin, plant bioflavonoid extracted mainly from grapefruit and other related citrus species. This study was designed to assess the neuroprotective effect of naringin on ammonium chloride (NH4C1) induced hyperammonemic rats. Naringin administration drastically restored the levels of blood ammonia, plasma urea, nitric oxide (NO), glutamate, glutamine, lipid peroxidation, lipid profile, activities of liver marker enzymes, antioxidant status and sodium/potassium-ATPase (Na+/ K+-ATPase). Hence, this study suggested that nargingin exhibited their protective effect against NH4C1 induced toxicity via enhancing the activities of antioxidant enzymes and inhibiting the lipid peroxidation process. Take together, this study provides data that naringin effectively reduced neurotoxicity by attenuating hyperammonemia, suggesting that naringin act as a potential therapeutic agent to treat hyperammonemic rats.

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