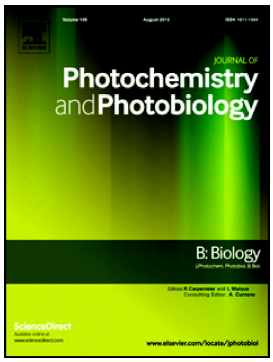

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Ameliorative Effects of Rutin on Hepatic Encephalopathy- induced by Thioacetamide or Gamma irradiation

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Abstract

Hepatic encephalopathy (HE) is a syndrome resulting from acute or chronic liver failure. This study was designed to evaluate the effect of rutin on thioacetamide (TAA) or γ- radiation-induced HE model. Animals were received with TAA (200 mg/kg, i.p.) twice weekly for four weeks or exposed to 6 Gy of γ- radiation to induce HE then groups orally treated with Rutin (50 mg/kg b.wt.) for four weeks. At the end of experiment, blood, liver and brain samples were collected to assess biochemical and biophysical markers as well histopathological investigations. TAA or γ-radiation exposed rats experienced increases in serum activities of ALT, AST, ALP and ammonia level. Also an alteration in relative permeability and conductivity of erythrocytes was observed. Furthermore, cytokines levels and AChE activity were induced whereas the activities of HO-1 and neurotransmitters contents were depleted. TAA or γ- radiation caused distortion of hepatic and brain architecture as shown by histopathological examination. Treatment with Rutin resulted in improvement in liver function by the decline in serum AST and ALT activities and reduction in serum ammonia level. In addition, the administration of Rutin significantly modulated the alteration in cytokines levels and neurotransmitters content. Histopathological examinations of liver and brain tissues showed that administration of rutin has attenuate TAA or radiation - induced damage and improve tissue architecture. Consequently, Rutin has been a powerful hepatoprotective effect to combat hepatic encephalopathy associated hyperammonemia and its consequential damage in liver and brain.

Key words: Hepatic encephalopathy, TAA, γ-irradiation, Rutin, neurotransmitters, electrical properties

1. Introduction

Hepatic encephalopathy (HE) is a neuropsychiatric disorder resulting from acute or chronic liver failure [1]; it influences a considerable number of patients worldwide with a mortality index ranging from 50 to 90% [2]. This condition can bring about an extensive variety of clinical manifestations which incorporate psychomotor dysfunctions, sensory abnormalities, poor concentration, impaired memory and expanded response time. In its most severe form, patients may develop stupor, coma and death [3]. Although, the exact mechanism of hepatic encephalopathy not fully understood; many researchers suggested that ammonia play the main role in hepatic encephalopathy pathogenesis. In light of International Society for hepatic encephalopathy and Nitrogen Metabolism recommendations, a toxin model of hepatic encephalopathy was chosen to utilized thioacetamide (TAA). The model is the same as human acutely progressive hepatic disorders with the parallel involvement of the brain [4].