

Research Paper

Aprepitant: an antiemetic drug, contributes to the prevention of acute lung injury with its anti-inflammatory and antioxidant properties

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Abstract

Objectives We investigated, the effects of aprepitant (APRE) on the lung tissues of rats with an experimental polymicrobial sepsis model (CLP: cecal ligation and puncture) biochemically, molecularly and histopathologically.

Methods A total of 40 rats were divided into 5 groups with 8 animals in each group. Group 1 (SHAM), control group; Group 2 (CLP), cecal ligation and puncture; Group 3 (CLP + APRE10), rats were administered CLP + 10 mg/kg aprepitant; Group 4 (CLP + APRE20), rats were administered CLP + 20 mg/kg aprepitant; and Group 5 (CLP + APRE40), rats were administered CLP + 40 mg/kg aprepitant. A polymicrobial sepsis model was induced with CLP. After 16 h, lung tissues were taken for examination. Tumour necrosis factor α (TNF- α) and nuclear factor-kappa b (NFK-b) messenger ribonucleic acid (mRNA) expressions were analysed by real-time PCR (RT-PCR), biochemically antioxidant parameters such as superoxide dismutase (SOD) and glutathione (GSH) and oxidant parameters such as malondialdehyde (MDA) and lung damage histopathologically.

Key findings and conclusions The GSH level and SOD activity increased while the MDA level and the expressions of TNF- α and NFK-b were reduced in the groups treated with APRE, especially in the CLP + APRE40 group. The histopathology results supported the molecular and biochemical results.

Keywords: polymicrobial sepsis; aprepitant; tumour necrosis factor α ; nuclear factor-kappa b

Introduction

Sepsis is one of the leading causes of death in hospitalized patients.^[1] Early diagnosis and treatment are necessary since the mortality due to sepsis is high and so many studies have focused on

the treatment and pathophysiology of sepsis.^[2–4] Sepsis is the last point of inflammation when is out of control, plasma extravasation and increased vascular endothelium permeability occur due to intense inflammation and bacterial translocation and tissue damage