Available online on 15.05.2019 at http://jddtonline.info

Journal of Drug Delivery and Therapeutics

Open Access to Pharmaceutical and Medical Research

© 2011-18, publisher and licensee JDDT, This is an Open Access article which permits unrestricted non-commercial use, provided the original work is properly cited

Open Access Research Article

Hepatotoxic effect of Rifampicin as an Anti-Tuberculosis drug on male Albino rat

Maiti Swatilekha[1], Parua Saswati[2], Nandi Dilip Kumar[3], Mondal Keshab Chandra[4], Samanta Saptadip[5]*

[1] Department of Physiology, Garhbeta College, Garhbeta, 721127, Paschim Medinipur West Bengal, India

[2] Department of Physiology, Bajkul Milani Mahavidyalaya, Bajkul, Purba Medinipur, West Bengal, India

[3] Department of Physiology and Nutrition, Raja N.L. Khan Women's College, Midnapore, 721102, West Bengal, India

[4] Department of Microbiology, Vidyasagar University, Midnapore, 721102, West Bengal, India

[5] Department of Physiology, Midnapore College, Midnapore, 721101, Paschim Medinipur, West Bengal, India

ABSTRACT

Tuberculosis is one of the serious airborne infectious diseases. Rifampicin is commonly used as anti-tuberculosis drug which creates drug-induced hepatotoxicity. Physiologically, liver maintains metabolic homeostasis and also regulates the detoxification process. The study of rifampicin mediated hepatotoxicity had been performed on male albino rat after its oral administration with a dose of 50 mg/kg body weight/day for 14 days. Several biochemical markers like serum glutamate pyruvate tranaminase (AST), serum glutamate oxaloacetate transaminase (ALT), alkaline phosphatase (ALP), lactate dehydrogenase (LDH), serum total protein, serum bilirubin, serum cholesterol were considered to evaluate the toxicity. Significant elevation of level of AST (115.89%), ALT (134.40%), ALP (46.15%), serum cholesterol (91%) and bilirubin content (119.44%) had been observed in treated group compared with control group. High level of MDA content as lipid peroxidation marker was also been noticed in drug induced group. Histopathological studies had shown the disintegrated hepatolobular structure with dilated central vein. All these findings indicated that the selected dose of rifampicin is hepatotoxic; proper monitoring and care are essential during the treatment of tuberculosis.

Keywords: rifampicin; hepatoxicity; anti-tuberculosis

Article Info: Received 13 March 2019; Review Completed 18 April 2019; Accepted 22 April 2019; Available online 15 May 2019

Cite this article as:

Maiti S, Parua S, Nandi DK, Mondal KC, Samanta S, Hepatotoxic effect of Rifampicin as an Anti-Tuberculosis drug on male Albino rat, Journal of Drug Delivery and Therapeutics. 2019; 9(3):26-32 **http://dx.doi.org/10.22270/jddt.v9i3.2744**

***Address for Correspondence:**

Dr. Saptadip Samanta, Department of Physiology, Midnapore College, Midnapore, 721101, Paschim Medinipur, West Bengal, India.

Abbreviations

ABCB1: ABC transporter subfamily B member 1; ALP: alkaline phosphatase; ALT: alanine transaminase; AST: aspertate transaminase; CP: continuation phase; DILI: drug-induced liver injury; HRZE: isoniazid, rifampicin (RIF), pyrazinamide, and ethambutol; IP: intensive Phase; LDH: lactate dehydrogenase; LPO: lipid peroxidation; MDA: malondialdehyde; PPARγ: proliferators activated receptor gamma; PXR: pregnane X receptor; RIF: rifampicin; ROS: reactive oxygen species:

INTRODUCTION

Liver is the "metabolic factory" of the body and plays central role to control the metabolism of every nutrient as well as foreign substances including drugs. Hepatic cytochrome P-450 enzyme system is essential for biotransformation of drugs through oxidative pathways followed by conjugation with glucuronide/sulphate/glutathione which convert the molecules to hydrophilic metabolites those are excreted by the kidney or through the gastrointestinal tract.[1] Owing to these properties, liver is the main target of drug toxicity and drug-induced liver injury (DILI) is the most common side effect in clinical.[2] Currently, over 1000 drugs are known to cause DILI, and the list is continuously growing up.[3] Zhou et al.[4] reported that anti-tuberculosis drugs were the leading agents of DILI. Tuberculosis is one of the top curable infectious diseases and creates serious public health problem in developing countries. According to World Health Organization, 9.6 million people were suffering from tuberculosis and 1.5 million had been died in 2014.[5] In developed countries, the incidence of tuberculosis increases due to immunodeficiency disease like HIV (human immunodeficiency virus) infection.[6] Currently, four major pharmacological agents (isoniazid, rifampicin, pyrazinamide,