INTERNATIONAL RESEARCH JOURNAL OF PHARMACY
www.irjponline.com
ISSN 2230 – 8407

Research Article

HEPATOPROTECTIVE ACTIVITY OF *BAUHINIA RACEMOSA* ROOTS EXTRACTS

P. Aravanan *, S. Jayakumari
School of Pharmaceutical Sciences, Vels institute of Science, Technology and Advanced Studies (VISTAS), Pallavaram, Chennai, India
*Corresponding Author Email: p_aravanan2011@yahoo.com

Article Received on: 25/11/20 Approved for publication: 18/01/21

DOI: 10.7897/2230-8407.1201114

ABSTRACT

Of all types of plants, we come across everyday every plant is useful some more than other. Our predecessors left an ocean of knowledge regarding the usage of natural sources for medicinal purposes all that we need is, to work on the molecular level to better understand how to avoid negative effects. In the present study Chloroform and Ethanolic extracts of *Bauhinia racemosa* obtained by soxhlation were used in the determination of Hepatoprotective activity using Paracetamol induced hepatotoxicity method. Oral administration of *Bauhinia racemosa* Chloroform extract and *Bauhinia racemosa* Ethanolic Extract showed significant decrease in biochemical parameters such as the ALT, AST, ALP. Animals treated with Ethanolic extract at dose level of 200 mg/kg & 400 mg/kg b.w, p.o in Hepatotoxicity induced rats exhibited a significant reduction of ALP (178.2±1.65 & 169.1±1.88), AST (123.5±1.87 & 115.7±1.25), ALT (115.2±0.94 & 111.4±1.29) respectively. Extensive literature review showed that *Bauhinia racemosa* is capable of several pharmacological activities and the present study sheds light on the hepatoprotective nature of the selected plant.

KEY WORDS: Hepatoprotective activity, Paracetamol, *Bauhinia racemosa*, Liver Necrosis, Silymarin, Histopathological Studies.

INTRODUCTION

Liver is the key organ regulating homeostasis in the body. It is involved with almost all the biochemical pathways related to growth, fight against disease, nutrient supply, energy production and reproduction. Because of its unique metabolism and relationship to the gastrointestinal tract, the liver is an important target of the toxicity of drugs, xenobiotics and oxidative stress.[1] More than 900 drugs, toxins and herbs have been reported to cause liver injury and drugs account for 20% - 40% of all instances of fulminant liver failure.[2] The clinical consequences of liver diseases are hepatic dysfunction in the form of jaundice, hypoalbuminemia, hyperammonemia, hyperglycemia, hepatitis, palmar erythema, spider angiomas, hypogonadism, gynecomastia, weight loss, muscle wasting, and portal hypertension from cirrhosis if left untreated will lead to life threatening complications like hepatic failure in the form of hepatic encephalopathy, hepatorenal-syndrome; or portal hypertension from cirrhosis. Paracetamol, a widely used analgesic and antipyretic drug, produces acute liver damage in high doses causing necrosis of the centrilobular hepatocytes characterized by nuclear pyknosis and eosinophilic cytoplasm followed by large excessive hepatic lesion.[3] The covalent binding of N-acetyl-P-benzoquinoneimine, an oxidative product of Paracetamol to Sulphydryl groups of protein, result in lipid peroxidative degradation of glutathione level and thereby, produces cell necrosis in the liver.[4,5]

The present study using different extracts of *Bauhinia racemosa* roots was performed according to the OECD guidelines 423 methods after Animal Ethical Committee clearance. (Approval No.CPCSEA/IAEC/EXP/25/50/2019/EXP-05). In this method the Hepatoprotective activity of Chloroform (BRCE) and Ethanol (BREE) extracts of roots of *Bauhinia racemosa* was tested using Paracetamol Induced Hepatotoxicity Model. The test dose of 200 mg/kg and 400 mg/kg was selected after a careful step wise Oral toxicity study.

Wistar Albino rats (150-200mg) of either sex were used in this investigation. They were maintained at standard housing condition of 12 hrs day and 12 hrs night cycle at a temperature of 24^0 C and fed with commercial rat chow diet (Hindustan lever ltd., Bangalore), water ad libitum during the experiment. The Institutional Animal Ethical Committee permitted the study, according to the acute toxic classic methods as per OECD guidelines 14.

MATERIALS AND METHODS

All the materials used for experiment were of pharmacopoeial grade. Paracetamol and Silymarin were purchased from the local supplier. Diagnostic kits for the estimation of serum ALT, serum ALP, Serum ASP were purchased from local supplier manufactured by Crest Biosystems, a division of coral clinical systems, Goa.

EXPERIMENTAL PROCEDURE

500 Gm of coarsely powdered *Bauhinia racemosa* root were extracted successively with, Chloroform and Ethanol using Soxhlet apparatus. Extraction was carried out till extractive becomes colourless. The extract was filtered through a cotton plug, followed by Whattman filter paper (no-1). The extract was evaporated under reduced pressure using rotovac evaporator. Thus, obtained extracts are stored in airtight container and used appropriately.

Group I Normal group received 0.5% CMC-Na
Group II Negative control received Paracetamol 900mg/kg b.wt
Group III Positive control received Paracetamol 900mg/kg and Silymarin 50mg/kg
Pretreatment groups

a. Group IV received BRCE 200mg/kg
b. Group V received BRCE 400 mg/kg
c. Group VI received BREE 200 mg/kg
d. Group VII received BRCE 400 mg/kg

After habituation all the animals were fasted for 24 hrs and later administered with Paracetamol (900mg/kg b.wt), Silymarin (50mg/kg b.wt) and extracts BRCE (200,400 mg/kg b.wt), BREE (200, 400 mg/kg b.wt). On 28th day samples were collected from overnight fasted rats by retro orbital puncture under mild anesthesia. Serum is separated by centrifuge (3000rpm) under cooling (2-4^{0}C) for ten minutes. The serum biochemical parameters such as Alanine amino transferase (ALP), Alkaline Phosphatase (ALP) and Aspartate aminotransferase (AST) were estimated.[7,8] Animals were sacrificed by cervical dislocation and livers were isolated for histopathological examination. They are stored in 10 % formalin and further dehydrated in ethanol and sectioned using microtome and processed for detailed study of the liver tissue using eosin dye.

RESULTS & DISCUSSION

Effect of BRCE & BREE extracts on Paracetamol Induced Hepatoxicity in rats

Biochemical parameters like ALT (U/L), ALP (U/L), AST (U/L), Total Bilirubin (mg/dl) and Total Protein (gm/dl) of group I to VII animals were estimated quantitatively and tabulated in Table 1.

The comparative efficacy of the BRCE & BREE extracts tested for their Hepatoprotective nature in rats, along with the relationship between dose and hepatoprotective activity were depicted in the form of a bar diagram as shown in Figure 1, 2 & 3.

Table 1: Effect of BRCE and BREE extracts on Paracetamol Induced Hepatotoxicity

Groups	ALP	AST	ALT
Normal control	107.7± 1.54	92.17± 1.07	86±1.29
Hepatotoxic control	223.5±2.44*	129.5±1.74*	120.7±1.16*
Standard (Silymarin)	109.7±1.88**	94± 1.18**	88.3± 0.88 **
BREE 200 mg/kg	178.2±1.65**	123.5±1.87**	115.2±0.94**
BREE 400 mg/kg	169.1±1.88**	115.7±1.25*	111.4±1.29*
BRCE 200 mg/kg	175.1±1.02*	110.5±1.34*	109.4±1.22*
BRCE 400 mg/kg	164.3±1.14*	102.4±1.12**	104.8±1.75*

*P< 0.05 Significant, **P< 0.001 highly significant.

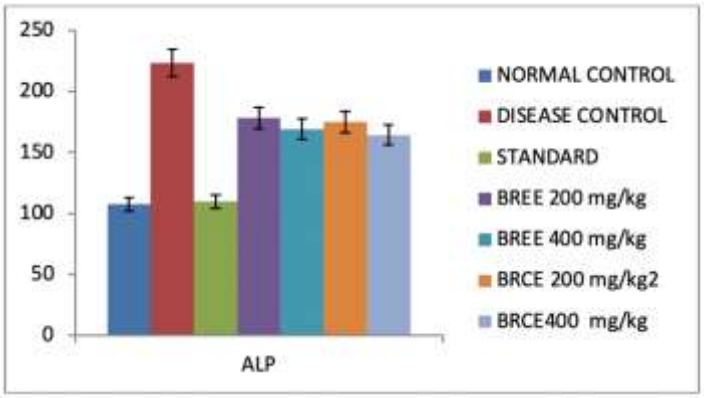

Figure 1: Effect of BRCE & BREE extracts on ALP

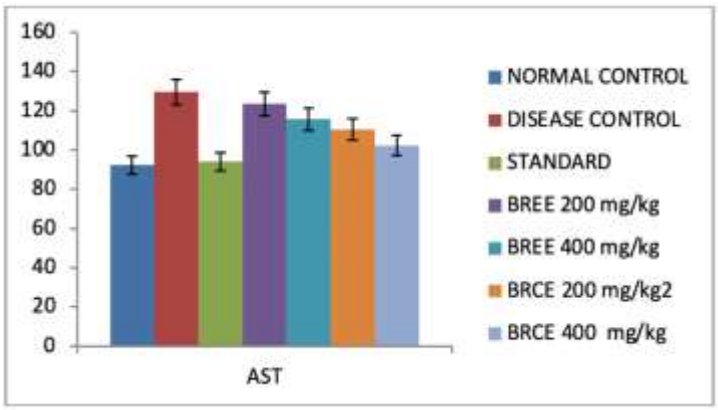

Figure 2: Effect of BRCE & BREE extracts on AST

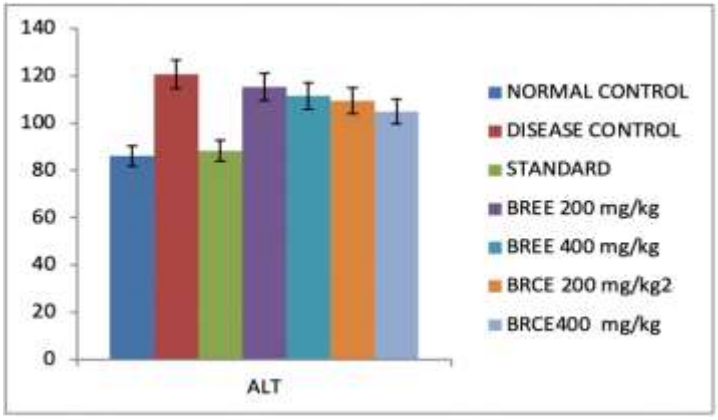

Figure 3: Effect of BRL & BRS extracts on ALT

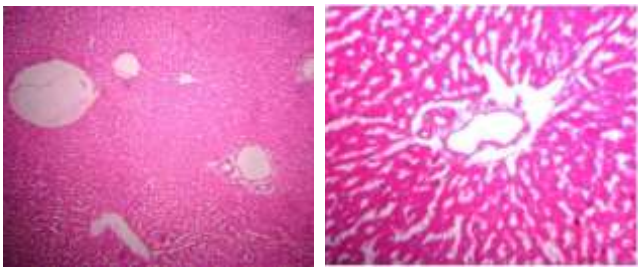

Figure 4: Images of (a) normal rat liver and (b) liver treated with toxic control group treated with Paracetamol showing central vein necrosis and sinusoid dilation respectively.

Figure 5: Standard group treated with Silymarin showing normal Histopathological appearance with no evidence of necrosis

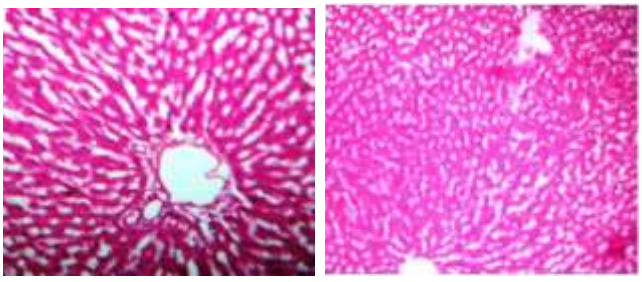

Figure 6: Test group treated with BREE 200, 400 mg/kg showing normal Histopathology and least evidence of necrosis

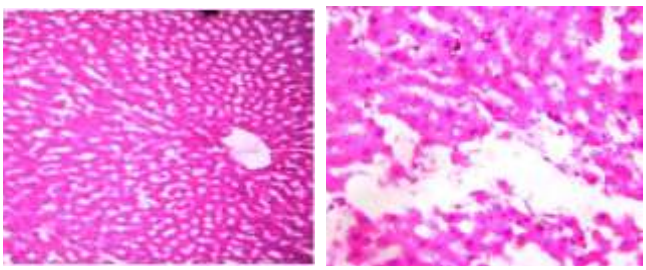

Figure 7: Test group treated with BRCE 200, 400 mg/kg showing normal Histopathology and least evidence of necrosis

HISTOPATHOLOGICAL STUDIES

Comparative Histopathological study of liver tissue from different groups of rats was conducted and the result confirmed the hepatotoxicity of Paracetamol and the preventive effect of extracts. (Figure 4)

Histological observation of liver tissue supported the results obtained from serum enzyme assays. The liver of Paracetamol administered rat showed massive fatty changes, gross necrosis and broad infiltration of lymphocytes and loss of cellular boundaries. Degeneration in centrilobular fatty section was revealed. Sinusoids were inflamed and flooded with inflammatory cells. In centribular area degeneration in fatty component were present moderately. Dilated and congested blood cells and fatty acid changes were observed. Lobular inflammation, changes in lobular architect, extensive areas of patchy and confluent hepatocytes necrosis and lobular inflammation was also observed. (Figure 5)

Recovery against Paracetamol induced necrosis was observed to have decreased in magnitude when compared to the Paracetamol treated animals. They were found to be normal shape and size. (Figure 6, 7)

DISCUSSION

Oral administration of BRCE & BREE at different concentrations significantly decreased AST, ALP and ALT. These extracts contain myriad number of compounds like tannins, phenolic compounds and alkaloids, among them methyl tiglate in both extracts may be responsible for Hepatoprotective activity. In treated group significant protection was observed as a sign of necrosis disappeared although few cells were found to be inflamed with sign of infiltration of macrophages. Extracts were found to be much effective in protecting hepatocytes at selected dose. It was quite difficult to ascertain any type of differences between the histology of liver of different animals with BREE and BRCE. Thus, it can be said that at histological level no significant variation was present between sample treated groups.

These results indicate that BRCE & BREE extracts produce considerable Hepatoprotective activity and that BREE is more effective than that of BRCE. Thus, it is safe to say that the plant has a high scope as an active drug in hepatotoxic treatment upon further study.

REFERENCES

1. Jaeschke H, Gores GJ, Cederbaum AI, Hinson JA, Pessayre D, Lemasters JJ. Mechanisms of hepatotoxicity. Toxicol Sci. 2002 Feb;65(2):166-76. doi: 10.1093/toxsci/65.2.166. PMID: 11812920.
2. Anusha M, Venkateswarlu M, Prabhakaran V, Taj S S, Kumari B P, Ranganayakulu D. Hepatoprotective activity of aqueous extract of *Portulaca oleracea* in combination with lycopene in rats. Indian J Pharmacol [serial online] 2011 [cited 2018 Jan 22]; 43:563-7. Available from: https://www.ijp-online.com/text.asp?2011/43/5/563/ 84973
3. De-Giorgio F, Lodise M, Chiarotti M, d'Aloja E, Carbone A, Valerio L. Possible fatal acetaminophen intoxication with atypical clinical presentation. J Forensic Sci. 2013 Sep;58(5):1397-400. doi: 10.1111/1556-4029.12205. Epub 2013 Jul 3. PMID: 23822653.
4. Tittarelli R, Pellegrini M, Scarpellini MG, Marinelli E, Bruti V, di Luca NM, Busardò FP, Zaami S. Hepatotoxicity of paracetamol and related fatalities. Eur Rev Med Pharmacol Sci. 2017 Mar;21(1 Suppl):95-101. PMID: 28379590.
5. Jaeschke H, Gores GJ, Cederbaum AI, Hinson JA, Pessayre D, Lemasters JJ. Mechanisms of hepatotoxicity. Toxicol Sci. 2002 Feb;65(2):166-76. doi: 10.1093/toxsci/65.2.166. PMID: 11812920.
6. Luper S. A review of plants used in the treatment of liver disease: part two. Altern Med Rev. 1999 Jun;4(3):178-88. PMID: 10383482.
7. Mallhi, T. H., M. I. Qadir, Y. H. Khan, and M. Ali. Hepatoprotective Activity of Aqueous Methanolic Extract of *Morus nigra* Against Paracetamol-Induced Hepatotoxicity in Mice. *Bangladesh Journal of Pharmacology*, 2014; 9(1) :60-66. doi:10.3329/bjp.v9i1.17337.
8. Patel, Bhavik & Patel, Jignesh & Raval, Bhuvan & Tejal, Gandhi & Patel, Kirti & Patel, Paresh. (2010). Hepatoprotective activity of *Saccharum officinarum* against ethyl alcohol induced hepatotoxicity in rats. *Der Pharmacia Lettre*, 2010: 2 (1) 94-101.

Cite this article as:

P. Aravanan and S. Jayakumari. Paracetamol hepatoprotective activity of *Bauhinia racemosa* roots extracts. Int. Res. J. Pharm. 2021;12(1):29-32.
http://dx.doi.org/10.7897/2230-8407.1201114

Source of support: Nil, Conflict of interest: None Declared

Disclaimer: IRJP is solely owned by Moksha Publishing House - A non-profit publishing house, dedicated to publishing quality research, while every effort has been taken to verify the accuracy of the content published in our Journal. IRJP cannot accept any responsibility or liability for the site content and articles published. The views expressed in articles by our contributing authors are not necessarily those of IRJP editor or editorial board members.