



Original Research Article

## Hepatoprotective Activity of Methanolic Extract of Whole Plant of *Rhynchosia beddomei* in Wistar Rats

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### ABSTRACT

In the present study, the methanolic extract of *Rhynchosia beddomei* leaves was evaluated for its hepatoprotective effect against Ranitidine and Paracetamol induced hepatic injury in rats. Alteration in the levels of biochemical markers of hepatic damage like SGOT, SGPT, ALP and total proteins were evaluated. Paracetamol (500 mg/kg) and Ranitidine (150 mg/kg) induces hepatotoxicity in 7 and 21 days respectively and enhances the SGPT, SGOT, ALP, liver weight and reduced total proteins. Treatment with methanolic extract of *Rhynchosia beddomei* (150 mg/kg and 300 mg/kg) has brought back the altered levels of biochemical markers significantly to the near normal levels in the dose dependent manner. The results were supported by histopathological studies of liver tissue. Phytochemical analysis of *Rhynchosia beddomei* indicated the presence of alkaloids, phenolics, saponins, flavonoids and polysaccharides and its hepatoprotective potential may be due to the presence of flavonoids.

**Keyword:** *Rhynchosia beddomei*; Biochemical markers; SGOT; SGPT; ALP

### INTRODUCTION

The liver is the vital organ of paramount importance involved in the maintenance of metabolic function and detoxification from the exogenous and endogenous challenges, like xenobiotic, drugs, viral infection and chronic

alcoholism [1]. If during all such exposures to the above mentioned challenges the natural protective mechanisms of the liver are overpowered, the result is hepatic injury. Liver damage is always associated with cellular

necrosis, increase in tissue lipid peroxidation and depletion in the tissue GSH levels. In addition serum levels of many biochemical markers like SGOT, SGPT, ALP and bilirubin are elevated [2].

Herbs play a major role in the management of various liver disorders along with other system associated diseases. Hepatotoxicity is very common ailment resulting in serious debilities ranging from severe metabolic disorders to even mortality [3]. Plant derived natural products such as flavonoids, terpenoids and steroids have received considerable attention in recent years due to their diverse pharmacological properties including antioxidant and hepatoprotective activity [4,5]. Herbal medicines are known to play an important role in the treatment of various elements including liver disorders and many traditional practitioners have claimed that numerous medicinal plants can be extensively used for the alleviation of different types of liver disorders [6]. In spite of phenomenal growth of modern medicine there are no synthetic drugs available for the treatment of hepatic disorders. However there are several herbs/herbal formulation claimed to possess beneficial activity in treating hepatic disorders. *Rhynchosia beddomei* Baker, is a plant of natural origin belonging to the family Fabaceae is a rare and endemic plant restricted to Seshachalam (Gogarbham hills) of Eastern Ghats of Andhra Pradesh, India. Different parts of *R. beddomei* plant have been found to possess medicinal properties; it possesses wound healing [7], diuretic [8], anti-inflammatory, carcinoma, antioxidant [9] and antidiabetic activity [10]. *R. beddomei* contains volatile oils which were first reported. The phytochemical properties of the leaves and reported the presence of alkaloids, indole alkaloids, anthracene glycosides, anthraquinones, carotenoids, coumarins, dihydrochalcones, fatty acids, flavonoids, flavones, flavonols, steroids and triterpenoids [11]. So in the present investigation whole plant

of *R. beddomei* was selected and screened for hepatoprotective activity.

## MATERIALS AND METHODS

### Plant material and preparation of extract

*R. beddomei* whole plant was collected from Seshachalam hills and authenticated by Dr. K. Madhava Chetty, Assistant Professor in Department of Botany, Sri Venkateshwara University, Tirupati, Chittoor district, Andhra Pradesh. The crude plant material was dried under shade and powdered mechanically to coarse powder. The coarsely powdered plant material (500 gm) was subjected to extraction with methanol using simple distillation. The extract was evaporated to semisolid mass and subjected to preliminary phytochemical investigation.

### Animals Studies

Healthy adult Wistar Albino rats of 180-250 gm were selected for the study. The animals were obtained from Gentox laboratories, Hyderabad. The animals were housed according to CPCSEA guidelines (under standard temperature condition). They were given a pellet diet and water ad libitum. The ethical clearance was obtained from Institutional Animal Ethics Committee (IAEC) before the experiment (Reg. no.1175/ac/08/CPCSEA).

### Acute toxicity studies (OECD 423)

An acute toxicity study was performed on methanol extract following OECD guidelines (423). The dosage for the pharmacological studies was selected as 1/10<sup>th</sup> of the highest dose (3000 mg/kg) administered.

### Experimental design

#### *Ranitidine induced hepatotoxicity*

In this study Male Wistar Albino rats of 150 – 200 gm are taken. Total of 30 animals are involved. Animals are divided into six groups as Group I as (Control) which receives normal distilled water, Group II as (Negative control)

which receives Ranitidine at a dose of 150 mg/kg bd.wt for 21 days through *i.p* route, Group III as (Test I) which receives Ranitidine for 21 days and MERB at a dose of 150 mg/kg bd.wt for 10 days through oral route, Group IV as (Test II) which receives Ranitidine for 21 days and along with it MERB at a dose of 300 mg/kg for 10 days through oral route, and Group V as (Standard) which receives Ranitidine for 21 days and the standard drug Liv-52 at a dose of 5 ml/kg bd.wt through oral route [12,13]. On the 11th day, 18 h after the dose of drugs, all the animals were anaesthetized under light ether anesthesia and the blood was collected from retro orbital sinus using a heparinized capillary tube for the study of biochemical parameters.

#### **Paracetamol induced Hepatotoxicity**

In this study Male Wistar Albino rats of 150 – 200 gm are taken. Total of 30 animals are involved. Animals are divided in to six groups as Group I as (Control) which receives normal distilled water, Group II as (Negative control) which receives Paracetamol at a dose of 500 mg/kg bd.wt for 7 days through *i.p* route, Group III as (Test I) which receives Paracetamol for 7 days [6,14] and MERB at a dose of 150 mg/kg bd.wt for 7 days through oral route, Group IV as (Test II) which receives Paracetamol for 7 days and along with it MERB at a dose of 300 mg/kg for 7 days through oral route, and Group V as (Standard) which receives Paracetamol for 7 days and the standard drug Liv-52 at a dose of 5 ml/kg bd.wt through oral route. On the 8th day, 18 h after the dose of drugs, all the animals were anaesthetized under light ether anesthesia and the blood was collected from retro orbital sinus using a heparinized capillary tube for the study of biochemical parameters.

#### **In vivo Antioxidant studies**

Antioxidant studies such as superoxide scavenging activity, Lipid peroxidation (LPO) assay, GSH (glutathione) assay and Catalase assay were evaluated. [6]

#### **Histopathological studies**

A portion of liver tissue (liver slices) of normal control, ranitidine control, paracetamol control and treated groups of rats with Liv-52 and MERB (150 mg/kg) and MERB (300 mg/kg) were stored in containers for 12 hours in 10% formalin solution and subjected to histopathological studies. Observed microscopically for histopathological changes i.e., normal liver, damaged and recovered liver was studied and compared. [15] The results were shown in Figure 1 & Figure 2.

#### **Statistical analysis**

All the values were expressed as mean  $\pm$ SEM. The data's were statistically analyzed by one way ANOVA followed by Dunnett's t-test and values  $p < 0.05$  was considered to be significant.

## **RESULTS**

#### **Acute toxicity studies**

Administration of *Rhynchosia beddomei* bark extracts in the doses of 50, 300 & 2000 mg/kg resulted in no mortalities or evidence of adverse effects implying that *Rhynchosia beddomei* is nontoxic. Throughout 14 days of the treatment no changes in behavioral pattern, clinical signs and body weight of mice in both control and treatment groups were observed. This shows that *Rhynchosia beddomei* was safe up to a dose of 3000 mg/kg.

**Table 1: Phytochemical investigation of *Rhynchosia beddomei* methanolic extract**

Sl.No	Constituents	Report
1	Carbohydrates	+
2	Steroids	++
3	Alkaloids	+
4	Flavonoids	++
5	Glycosides	+
6	Proteins	-
7	Tannins	+
8	Volatile oils	-

'+' = Present, '-' = Absent

**Hepatoprotective activity**

The results of ranitidine and paracetamol induced hepatotoxicity were shown in table 2, 3. In the control group, the significant acute hepatocellular damage and biliary obstruction was indicated by the elevated level of SGPT, SGOT, ALP and decreased levels of total proteins. But the group which received the test drug of methanolic extract at the dose of 150 mg/kg and 300 mg/kg bd.wt p.o showed a

significant decrease in the elevated levels of SGPT, SGOT, ALP, and significant increase in the reduced levels of total proteins, these biochemical parameters are comparable with the standard Liv-52 hepatoprotective drug. Therefore, the Liv-52 and the methanolic extract restored the altered level of enzymes significantly.

**Table 2: Effect of methanolic extract of *Rhynchosia beddomei* on Ranitidine induced Hepatotoxicity**

Groups	SOPT U/L	SGPT U/L	ALP U/L	Total protein
Normal Control	62.17±1.26	52.33±0.60	41.17±1.74	7.16±0.70
Ranitidine Control	146.3±12.4 <sup>b,**,B</sup>	100.7±10.6 <sup>a,**,A</sup>	107.1±9.37 <sup>b,**,B</sup>	13.02±0.41 <sup>a,**,B</sup>
MERB (150 mg/kg)	123.2±9.54 <sup>b,**,B</sup>	82.76±8.45 <sup>a,**,A</sup>	93.5±6.42 <sup>b,**,B</sup>	5.06±0.45 <sup>a,**,B</sup>
MERB (300 mg/kg)	98.3±6.71 <sup>a,**,B</sup>	68.8±5.45 <sup>a,**,B</sup>	78.7±4.87 <sup>a,**,B</sup>	6.8±0.49 <sup>a,**,B</sup>
Standard Liv-52 (5ml/kg)	70.0±5.09 <sup>a,**,B</sup>	48.50±5.9 <sup>a,**,B</sup>	43.43±2.19 <sup>a,**,B</sup>	5.25±1.36 <sup>a,**,B</sup>

Values were represented as mean ± SEM. All the data were statistically analyzed by one way ANOVA followed by Dunnett's test and the significant values are expressed as control (a = p<0.001, b = p<0.05) Ranitidine control (\*\* = p<0.001, \* = p<0.05) Standard (A = p<0.01, B = p<0.05).

**Table 3: Effect of methanolic extract of *Rhynchosia beddomei* on Paracetamol induced Hepatotoxicity**

Groups	SGPT U/L	SGOT U/L	ALP U/L	Total protein
Normal Control	62.17±1.26	52.33±0.60	41.17±1.74	7.16±0.70
Paracetamol Control	235.50±10.1 <sup>b,**,A</sup>	228.52±9.4 <sup>b,**,A</sup>	109.81±7.8 <sup>a,**,A</sup>	12.21±0.8 <sup>a,**,A</sup>
MERB (150 mg/kg)	126.27±8.8 <sup>b,**,A</sup>	180.35±8.85 <sup>b,**,A</sup>	80.9±9.04 <sup>a,**,A</sup>	6.84±0.41 <sup>a,**,B</sup>
MERB (300 mg/kg)	80.4±16.2 <sup>b,**,B</sup>	98.2±9.08 <sup>a,**,B</sup>	68.4±6.74 <sup>b,**,B</sup>	7.05±0.47 <sup>a,**,B</sup>
Standard Liv-52 (5 ml/kg)	70.0±5.09 <sup>b,**,B</sup>	48.50±5.9 <sup>a,**,B</sup>	43.43±2.19 <sup>b,**,B</sup>	5.25±1.36 <sup>a,**,B</sup>

Values were represented as mean ± SEM. All the data were statistically analyzed by one way ANOVA followed by Dunnett's test and the significant values are expressed as control (a = p<0.001, b = p<0.05) Paracetamol control (\*\* = p<0.001, \* = p<0.05) Standard (A = p<0.01, B = p<0.05).

**In vivo Antioxidant studies****Ranitidine induced Hepatotoxicity****Effect on Antioxidant parameters**

After treatment with MERB at a doses of 150 mg/kg & 300 mg/kg has increased the Superoxide dismutase levels, GSH levels,

catalase levels and decreased the LPO levels significantly ( $p < 0.01$ ) and standard Group Liv - 52 at a dose of 5 ml/kg showed significant ( $p < 0.01$ ) increase in Superoxide dismutase levels when compared to that of hepatotoxic rats (Table 4).

**Table 4: Effect of MERB on Antioxidant parameters in Ranitidine induced hepatotoxicity**

Groups	Treatment	SOD Levels (mg/protein)	GSH Levels ( $\mu\text{g}/\text{mg}$ )	CAT Levels ( $\mu\text{M}/\text{min}/\text{mg}$ )	LPO Levels (nm/mg)
I	Normal Control	0.78 $\pm$ 0.017	0.94 $\pm$ 0.017	7.94 $\pm$ 0.2	5.94 $\pm$ 0.09
II	Ranitidine (150 mg/kg)	0.30 $\pm$ 0.005 <sup>a,**,A</sup>	0.084 $\pm$ 0.01 <sup>b,**,B</sup>	2.09 $\pm$ 0.04 <sup>a,**,B</sup>	11.27 $\pm$ 0.09 <sup>b,**,A</sup>
III	MERB (150 mg/kg)	0.44 $\pm$ 0.02 <sup>a,**,A</sup>	0.63 $\pm$ 0.01 <sup>a,**,B</sup>	3.23 $\pm$ 0.07 <sup>a,**,A</sup>	3.43 $\pm$ 0.07 <sup>a,**,A</sup>
IV	MERB (300 mg/kg)	0.61 $\pm$ 0.01 <sup>a,**,A</sup>	0.78 $\pm$ 0.01 <sup>a,**,B</sup>	5.18 $\pm$ 0.06 <sup>a,**,A</sup>	5.18 $\pm$ 0.06 <sup>a,**,A</sup>
V	Standard Liv-52 (5 ml/kg)	0.73 $\pm$ 0.01 <sup>a,**,A</sup>	0.83 $\pm$ 0.017 <sup>a,**,B</sup>	6.71 $\pm$ 0.17 <sup>a,**,A</sup>	5.8 $\pm$ 0.17 <sup>a,**,A</sup>

Values were represented as mean  $\pm$  SEM. All the data were statistically analyzed by one way ANOVA followed by Dunnett's test and the significant values are expressed as control ( $a = p < 0.001$ ,  $b = p < 0.05$ ) ranitidine control (\*\* =  $p < 0.001$ , \* =  $p < 0.05$ ) Standard (A =  $p < 0.01$ , B =  $p < 0.05$ ).

**Paracetamol induced Hepatotoxicity****Effect on Antioxidant parameters**

After treatment with MERB at a doses of 150 mg/kg & 300 mg/kg has increased the Superoxide dismutase levels, GSH levels, Catalase levels and decreased the LPO levels

significantly ( $p < 0.01$ ) and standard Group Liv - 52 at a dose of 5 ml/kg showed significant ( $p < 0.01$ ) increase in Superoxide dismutase levels when compared to that of hepatotoxic rats shown in Table 5.

**Table 5: Effect of MERB on Antioxidant parameters in Paracetamol induced Hepatotoxicity:**

Groups	Treatment	SOD Levels (mg/protein)	GSH Levels ( $\mu\text{g}/\text{mg}$ )	CAT Levels ( $\mu\text{M}/\text{min}/\text{mg}$ )	LPO Levels (nM/mg)
I	Normal Control	0.54 $\pm$ 0.012	9.04 $\pm$ 0.017	0.934 $\pm$ 0.017	11.04 $\pm$ 0.09
II	Paracetamol (500 mg/kg)	0.27 $\pm$ 0.08 <sup>b,*,B</sup>	2.27 $\pm$ 0.01 <sup>b,*,A</sup>	0.108 $\pm$ 0.01 <sup>a,*,A</sup>	18.54 $\pm$ 0.09 <sup>b,**,A</sup>
III	MERB (150 mg/kg)	0.37 $\pm$ 0.01 <sup>a,*,B</sup>	6.57 $\pm$ 0.019 <sup>a,*,A</sup>	0.46 $\pm$ 0.019 <sup>a,*,A</sup>	7.03 $\pm$ 0.07 <sup>a,**,A</sup>
IV	MERB (300 mg/kg)	0.46 $\pm$ 0.06 <sup>a,*,B</sup>	8.76 $\pm$ 0.018 <sup>a,*,A</sup>	0.52 $\pm$ 0.018 <sup>a,*,A</sup>	5.48 $\pm$ 0.06 <sup>a,**,A</sup>
V	Standard Liv-52 (5 ml/kg)	0.54 $\pm$ 0.02 <sup>a,*,B</sup>	7.24 $\pm$ 0.026 <sup>a,*,A</sup>	0.74 $\pm$ 0.026 <sup>a,*,A</sup>	9.03 $\pm$ 0.17 <sup>a,**,A</sup>

Values are expressed as Mean  $\pm$  SEM ( $n=6$ ). All the groups were compared with control group, Paracetamol control group and standard group. Significant values are expressed as control group

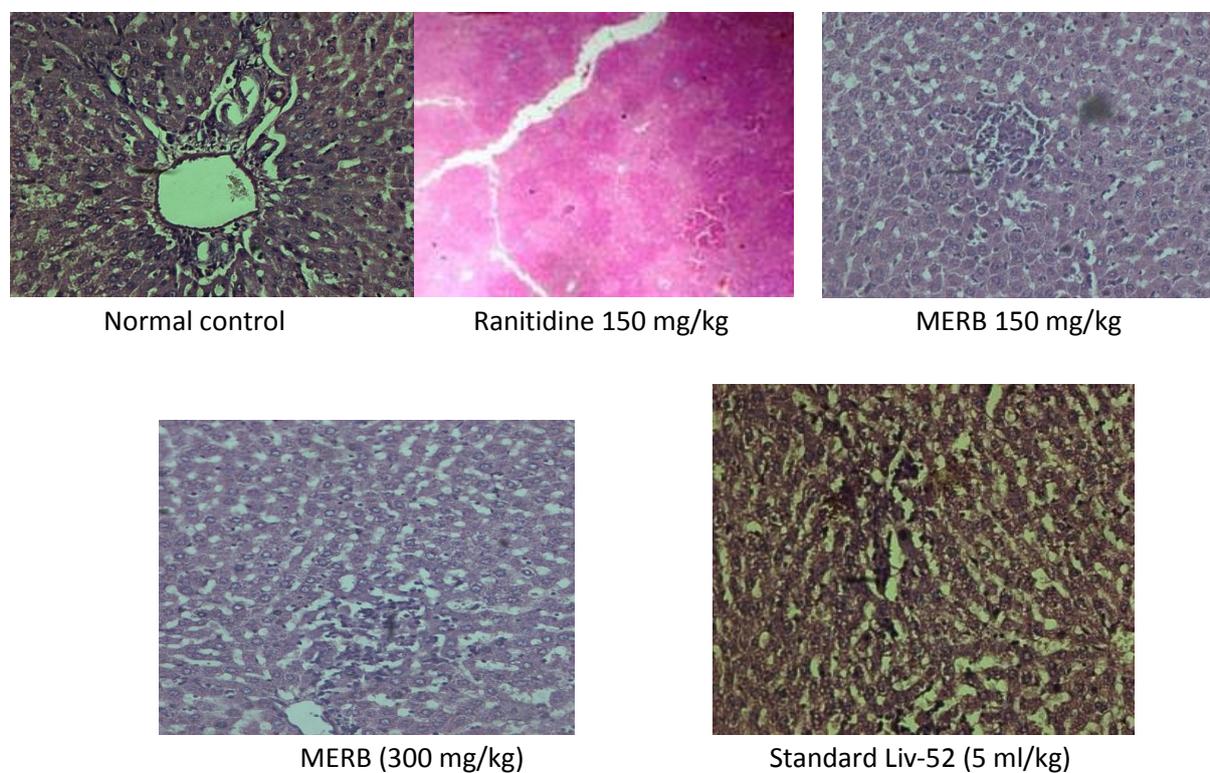
( $\alpha=p<0.01$ ,  $b=p<0.05$ ), paracetamol control (\*\*=  $p<0.01$ , \*=  $p<0.05$ ) and standard (A =  $p < 0.01$ , B =  $p < 0.05$ )

### Histopathology

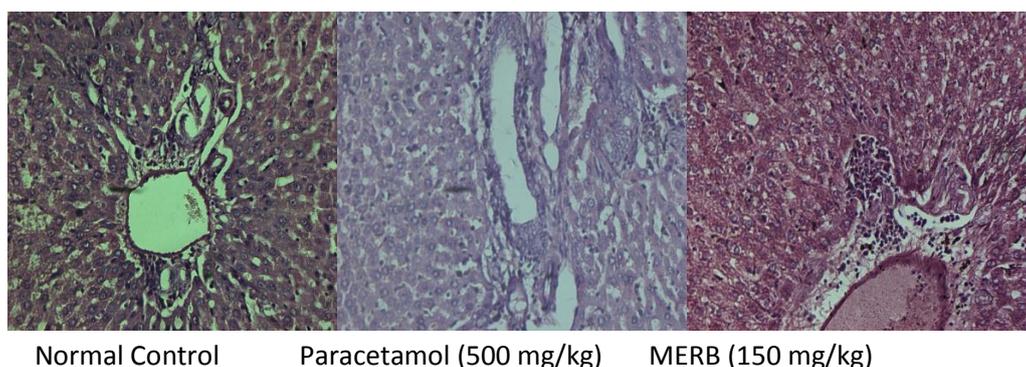
The histopathological study showed recovery of the damaged liver cells in the drug treated group. The reputed cells of the intoxicated liver were reformed. The degree of vascularization was also reduced as compare to hepatotoxic group. Multiple foci of inflammation and necrosis noticed in centrilobular region of liver.

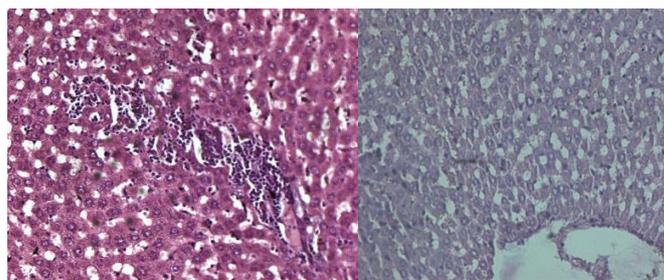
Also infiltration of inflammatory cells noticed in the inflammatory region of liver. In Ranitidine model mild to moderate sinusoidal space dilatation along with hemorrhages noticed in the sinusoidal space of liver & multiple foci of inflammation along with infiltration of inflammatory cells particularly lymphocytes noticed in the centri lobular region of liver.

**Fig. 1: Histopathology of liver in Ranitidine induced hepatotoxic model**



**Fig. 2: Histopathology of liver in Paracetamol induced hepatotoxic model**





MERB (300 mg/kg)

Standard Liv-52 (5 ml/kg)

In normal rat liver bile duct appeared normal, portal triad appeared normal & no inflammation or fibrosis noticed surrounding the portal region of liver. In Paracetamol control rat liver mild to moderate bile duct hyperplasia or bile duct proliferation noticed in surrounding the portal region of liver. In MERB (150 mg/kg) treated rat liver a small foci of periportal inflammation with infiltration of inflammatory cells noticed in the liver. In MERB (300 mg/kg) rat liver multiple small foci of inflammation along with infiltration of inflammatory cells noticed in the centrilobular region of liver. In standard Liv 52 (5 ml/kg) rat liver hepatocytes appeared normal, periportal and centrilobular region appeared normal but mild sinusoidal space dilatation noticed in the periportal region of liver.

## DISCUSSION

Preliminary phytochemical investigation of methanol extract was found to contain carbohydrates, flavonoids, phenolic compounds and tannins. Alkaloids, flavonoids and saponins are known to possess hepatoprotective activity. Acute toxicity studies of methanolic extract at the dose of 3000 mg/kg showed no toxic symptoms or death in any of the animals up to one week and till the end of the study. Thus the drug was considered to be safe.

Liver injury caused by ranitidine is due to its metabolite which leads to the hepatic oxidative damage generating immunoallergic reactions. Severe inflammatory changes with collagenous septa beginning to form after pronounced

centrilobular and bridging necrosis. In the parenchyma there was focal liver cell necrosis with some accumulation of histolytic elements and slight steatosis and cholestasis. Portal tract shows fibrosis, bile duct proliferation and infiltrate consisting of lymphocytes plasma cells, polymorphs and eosinophils. Liver injury is manifested in terms of increase in levels of serum aminotransferases, modest hepatic infiltration by both lymphocytes and eosinophil's and slight focal hepatocellular necrosis also causes liver cholestasis associated with increased plasma bilirubin and alkaline phosphatase [15]. Ranitidine specifically inhibits the cytochrome P-450 (CYP 450) mixed function oxidase (MFO) system. Serum SGOT, SGPT and ALP parameters were increased in all groups injected with ranitidine relative to the control group. After treatment with the extract it decreases serum SGOT, SGPT and ALP levels.

Paracetamol (acetaminophen) is a commonly and widely used analgesic and antipyretic agent. Hepatotoxic doses of acetaminophen deplete the normal levels of hepatic glutathione, when NAPQI covalently binds to cysteine groups on proteins to form 3-(cysteine-S-yl) acetaminophen adducts. The glutathione protects hepatocytes by combining with the reactive metabolite of paracetamol thus preventing their covalent binding to liver proteins [16].

Amino transferases SGPT and SGOT catalyze the interconversion of amino acids and  $\alpha$ -keto acids by the transfer of an amino group. These enzymes are very sensitive and are reliable indices for hepatoprotective or curative effects

of various compounds. Elevated levels of SGPT, SGOT, ALP and bilirubin were observed in positive control group and were reduced significantly in all drug treated groups. Liver cells synthesize various proteins like albumin, fibrinogen, hepatoglobin, transferrin and antitrypsin. The blood levels of these proteins are decreased in extensive liver damage. Serum proteins levels were found to decrease in positive control group which was reversed in extract treated group. Serum enzyme levels are not a direct measure of hepatic injury, but elevated levels are indicative of cellular leakage and loss of integrity of cell membrane. Thus lowering of enzyme content in serum is a definite indication of hepatoprotection of the drug.

The marker enzyme levels in different group of animals are measured. The serum levels of SGPT, SGOT and ALP were increased significantly. In our study, the administration of methanolic extract at doses of 150 mg/kg & 300 mg/kg, *p.o* showed significantly reduced levels of SGPT, SGOT and ALP whereas the total protein levels were increased significantly in the extract treated group. The results clearly indicated that extracts were capable of lowering the serum levels of SGPT, SGOT and ALP.

#### **Antioxidant activity**

The enzyme antioxidant defense system is the natural protector against lipid peroxidation. SOD, CAT and GSH enzymes are important scavengers of superoxide ion and hydrogen peroxide. These enzymes prevent generation of hydroxyl radical and protect the cellular constituents from oxidative damage [17]. Catalase (CAT) is an enzymatic antioxidant widely distributed in all animal tissues and the highest activity is found in the red cells and in the liver. CAT decomposes hydrogen peroxide and protects the tissue from highly reactive hydroxyl radicals. Therefore the reduction in the activity of these enzymes may result in a

number of deleterious effects due to the accrual of superoxide radicals and hydrogen peroxide. Lipid peroxidation has been suggested to the vicious process of liver injury due to acetaminophen administration. In the present study the elevations in the levels of end products of lipid peroxidation in the liver of rat treated with ranitidine and paracetamol were observed. The increase in malondialdehyde (MDA) levels in liver suggests enhanced lipid peroxidation leading to tissue damage and failure of antioxidant defense mechanisms to prevent formation of excessive free radicals [18].

#### **Histopathology studies**

Liver photomicrographs of different groups were shown normal liver control showed normal hepatic architecture with portal tracts, central veins, hepatocytes and sinusoids. Positive control group showed loss of normal liver architecture with degenerative hepatocytes, fibrosis, sinusoidal spaces with inflammatory cells, ballooning of cells and centri lobular necrosis. Liver photomicrograph of drug extract (150 mg/kg) showed mild fibrosis, light hepatocyte regeneration and ballooning of hepatocytes, whereas drug extract (300 mg/kg) showed minimal fibrosis, regeneration of hepatocytes and ballooning of hepatocytes. Treatment with standard Liv-52 (5 ml/kg) showed almost normal liver architecture.

#### **CONCLUSION**

In conclusion the results of the present study clearly demonstrate with histopathological evidence of hepatoprotective property of MERB. The phytochemical study revealed the presence of flavonoids, tannins phenols. The flavonoids showed the protective effect of liver in liver injury caused by paracetamol and ranitidine in rats. The above compounds may contribute to presence of hepatoprotective activity. The activity was found to be dose

dependent and further studies are required to isolate, characterize and find out the mechanism of action of the active compounds in MERB responsible for hepatoprotective activity.

#### CONFLICT OF INTEREST STATEMENT

The authors declare that they have no competing interests.

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