Research Article

Evaluation of Hepatoprotective Potential of Ethanolic Extract of Morinda citrifolia Root

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ABSTRACT

To evaluate the hepatoprotective potential of ethanolic extract of the plant *Morinda citrifolia* root. against paracetamol and corbon tetrachloride(CCl_4) induced hepatotoxicity in rats. Two doses 200mg/kg and 400mg/kg b.w p.o of the extract were subjected for the evaluation of Hepatoprotective potential against PCM (2g/kg) and CCl_4 (0.7mg/kg) induced liver injury. Silymarin (100mg/kg) was used as a standard drug. The parameters like SGPT, SGOT, ALP, Total Bilirubin and endogenous enzymes were estimated to assess the liver functions. In addition histopathological study was also carried out. Both the lower (200mg/kg) and higher dose (400mg/kg) of Morinda citrifolia root extract showed dose dependent significant decrease in SGPT, SGOT, ALP and TB levels when compared with toxic control. Both extracts showed decrease in LPO and increase in GSH, SOD and CAT levels. Hepatoprotective effect was also confirmed by histopathological analysis of liver which showed less damage in extract treated rats. The results obtained were comparable with that of the standard. The present study concluded that *Morinda citrifolia*. Plant root were found to be effective against hepatotoxicity induced by Alcohol and Paracetamol.

Keywords: Hepatoprotective; Silymarin; PCM; CCl_4; *Morinda citrifolia.*

INTRODUCTION

Liver diseases have become one of the major causes of morbidity and mortality all over world. However we do not have satisfactory liver protective drugs in allopathic medical practice for serious liver disorders. There are numerous plants and traditional formulations available for the treatment of liver diseases. About 600 commercial herbal formulations with claimed hepatoprotective activity are being sold all over the world. 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 ALT, AST, triglycerides, cholesterol, billirubin and alkaline phosphatase are elevated[1]. It has been estimated that there are some 200 million chronic carriers of the hepatitis B virus of which 40% are expected eventually due to hepatocellular carcinoma and 15% of cirrhosis.

In recent years in vivo and in vitro test models have been developed for the evaluation of plants for their antihepatotoxic activities[2]. The testing may be carried out by causing liver damage in experimental animals and estimating beneficial effects of treatment as measured by liver function test or by removing part of the liver and measuring the rate of regeneration. In such liver damage, the serum level of the liver enzymes, particularly serum glutamic-oxaloacetic transaminase and serum glutamic-pyruvic transaminase is raised and extend of its control by the antihepatotoxic drug under test is used as a basis for estimation[2]. It has been reported that about 170 phytoconstituents isolated from 110 plants belonging to 55 families were stated to possess liver protective activity. Conventional medicine does not provide many remedies for hepatitis, cirrhosis, liver damage by toxins or for biliary tract disorders[3].

In absence of a reliable liver protective drug in the modern system of medicine, a number of medicinal preparations in Ayurveda, the Indian system of medicine, are recommended for the treatment of liver disorders[4]. "*Morinda citrifoia*" is one of the potential plants for the liver disorders. It also possesses antioxidant, hypolipidemic and free radical scavenging activity, which will be beneficial properties in the treatment of hepato toxicity. The bark of "*morinda citrifolia*" plant used for the treatment of the liver diseases like jaundice by the tribal healers. Previous reports have proved that fruits of *Morinda citrifoia* possessed antidiabetic , anti hepatotoxic, hypolipidemic activities and are involved in lowering the level of blood sugar and total cholesterol[5].However, there is paucity of scientific data for the hepato protective activities of the whole plant. Hence, the present study is designed to evaluate the efficacy of morinda citrifolia' plant root extract on experimental models of hepato toxicity in rats.

MATERIALS AND METHODS

Experimental animals

Healthy Wistar albino rats of either sex weighing 150-200 g were used. Animals used in the study were procured from registered breeder. The animal care and handling was carried out according to CPCSEA guidelines. Animals were acclimatized to the animal quarantine for one week prior to the experiment under controlled conditions of temperature (27 ± 2°C) and were housed in sterile polypropylene cages containing paddy husk as bedding material with maximum of six animals in each cage. The rats were fed on standard food pellets and water *ad libitum*. The studies conducted were approved by the Institutional Animal Ethical Committee, Srinivas College of Pharmacy, Mangalore, Karnataka (Approval No.: SCP/IACE/F150/P103/2016).

Collection of plant material and preparation of extract[6]

The *Morinda citrifolia* plant root of one year-old was collected locally and authenticated by a Taxonomist. The whole plant was dried in the shade, segregated, pulverized by a mechanical grinder and passed through a 40 mesh sieve. The powder was extracted by ethanol in Soxhlet apparatus by continuous hot percolation method. After filtration through Whatmann filter paper No 40, the filtrate was vaccum dried at 35 to 40^{0}C. The extracts were stored in screw cap vials at 40^{0}C until further use. The ethanolic extract of *morinda citrifolia* was subjected to preliminary phytochemical screening to find out the presence of active principles. The collected *morinda citrifolia* ethanolic root extract (DBME) was suspended in 50% sucrose solution for oral administration.

Preliminary phytochemical evaluation

The ethanolic extract obtained from *Morinda citrifolia* root were subjected to qualitative phytochemical investigation for identification of various constituents. The extract of *Morinda citrifolia* obtained was dissolved in ethanol which is used to test the presence of various phytochemicals according to standard procedure.

1. **PHARMACOLOGICAL ACTIVITIES**
   1. Determination of acute toxicity (LD_{50})
   2. Hepatoprotective activity

Determination of Acute toxicity (LD_{50})[7]

The animals were kept fasting for overnight with sufficient water. The suspension of extract prepared in 50% sucrose solution, was administered orally for one animal at the limit dose of 2000mg/kg and was observed for 14 days (with special attention for the first 4hr of administration followed by next 20hr) for mortality, general behavior and signs of discomfort. If the animal survived, another four animals were dosed sequentially so that total five animals were tested.

EVALUATION OF HEPATOPROTECTIVE ACTIVITY

1. PARACETAMOL (PCM) INDUCED LIVER TOXICITY[8]:

Experimental design

In this experiment, Wistar albino rats were randomly assigned into five groups of six each. The different groups are:

Group I : Vehicle control (distilled water S1 ml/kg)
Group II : Hepatotoxic control (Paracetamol 2g/kg)
Group III : Reference standard (Paracetamol 2g/kg + Silymarin 25mg/kg)
Group IV : *Morinda citrifolia* root extract (200mg/kg + Paracetamol 2g/kg)
Group V : *Morinda citrifolia* root extract (400mg/kg + Paracetamol 2g/kg)

2. CORBON TETRACHORIDE (CCl_4) INDUCED LIVER TOXICITY[9]

Experimental design

The Wistar albino rats (150-200 g) of either sex were randomly divided into five groups of six each. The different groups were assigned as follows.

Group I: Vehicle control (1% Tween 80, 1ml/kg)
Group II: Hepatotoxic control (CCl_4 0.7 mi/kg, i.p)
Group III: Reference standard (Silymarin 100mg/kg) +(CCl_4 0.7ml/kg, i.p)
Group IV: *M.citrifolia* plant root extract (200mg/kg) +(CCL_4 0.7ml/kg,i.p)
Group V: *M.citrifolia* plant root extract (400mg/kg) + (CCL_4 0.7 ml/kg,i.p)

Group I and II were treated with veicle. Group III animals were treated with silymarin 100mg/kg and group IV and V were treated with *M.citrifolia* plant root extract 200mg/kg and 400mg/kg respectively

seven days.All the animals expect Group I will be intoxicated by the administration of CCL_4 (1:1 of CCL_4in olive oil) on 2nd, 4th and 6th day of treatment.

EVALUATION

On seventh day blood was collected through retro orbital puncture and analysed for various biochemical parameters. Blood was allowed to clot at room temperature for 30 min,subjected to centrifugation (3000 rpm for 15 min.) and estimation of biochemical parameters. Liver was dissected out and subjected for morphological study such as wet liver weight and volume of each animal.Further the liver was placed in 10% formalin solution for histopathological study. Serum enzymes, which were assessed, include Serum gluataic oxaloacetate transaminase (SGOT) and Serum glutamic pyruvic tranaminase (SGPT) (Retiman and Frankel, 1957), total bilirubin and direct bilirubin (Malloy and Evelyn, 1937), andAlkaline phosphate (ALP) content. Tissue glutathione measurements were performed using a modification of the Ellman procedure (GeorgeEllman, 1959; Aykae, 1985). Tissue samples were homogenized in ice cold trichloroacetate (TCA) (l gm tissue plus 10 ml 10% TCA) in a homogeniser. Briefly, after centrifugation at 3000 rpm for 10 min, 0.5 ml supernatant was added to 2 ml of 0.3 M disodium hydrogen phosphate solution. A 0.2 ml solution of 5, 5 Dithio-bis 2- nitrobenzoic acid (DTNB), (0.4 mg in 1 ml of 1% Sodium nitrate) was added and the absorbance at 412 nm was measured immediately after mixing. Extent of lipid peroxidation was done by combining 1.0 ml of biological sample (0.1 – 2.0 mg of membrane protein or 0.1–0.2 μmol of lipid phosphate) with 2.0 ml of TCA- tribromoanisole (TBA)-hydrochloric acid (HCl) and thoroughly mixed. The solution was heated for 15 min in a boiling water bath. After cooling, the flocculent precipitate was removed by centrifugation at 1000 rpm for 10 min. The absorbance of the sample is determined at 535 nm against blank that contains all the reagents without the lipid (John and Steven, 1978).

RESULTS AND DISCUSSION

Preliminary phytochemical screening

Preliminary phytochemical analysis of extract is shown in Table No.1 revealed the presence of following phytochemicals: Alkaloids, Tannins, Terpenoids, and Sterols.

Table 1: Preliminary phytochemical screening

S. No.	Test	Result
1	Alkaloids	+
2	Carbohydrates	-
3	Sterols	+
4	Glycosides	+
5	Saponins	_
6	Tannins	_
7	Flavonoids	+
8	Terpenoids	_

(+ = Present in test, - = Absence in test)

ACUTE TOXICITY STUDY (LD_{50})

Ethanolic extract of *Morinda citrifolia* plant roots was studied for acute toxicity at the dose level of 2000mg/kg, p.o. according to OECD guideline Annexure 425. It was found to be safe up to 2000mg/kg body weight by oral route. There was no mortality and no signs of toxicity and extract were found to be safe. So two dose levels i.e., 200mg/kg ($1/10^{th}$), and 400mg/kg ($1/5^{th}$) body weight were selected for the present study.

PHARMACOLOGICAL ACTIVITIES

Evaluation of hepatoprotective activity of *M.citrifolia* on PCM induced hepatic damage in rats

The effects of extract on serum transaminase, alkaline phosphatase, bilirubin and total protein levels in paracetamol-induced liver damage in rats are summarized in Table-1. The paracetamol induced group showed elevation in SGPT, SGOT, ALP and Total Bilirubin levels up to 288.3 ± 1.47, 382.7 ± 3.38, 388 ± 2.49 and 2.23 ± 0.02 respectively when compared to the control group. In the groups treated with 100 mg/kg and 200 mg/kg of the *M.citrifolia* plant root extract, the above biochemical markers of hepatotoxicity were found to be decreased when compared to CCl4 treated control group. Evidently, the hepatoprotective effects of higher dose of *M.citrifolia* plant root extract (400 mg/kg) were near to that of standard i.e. Silymarin (100 mg/kg). Both the doses of *M.citrifolia* plant root extract used in the study showed significant protective property than control.
Table No.3 shows the effects of extracts of *M.citrifolia* on LPO, SOD, GSH and CAT concentrations in rat liver after challenging with paracetamol. It was observed that animals treated with PCM developed a hepatic damage, increase in LPO and decrease in GSH, CAT & SOD when compared to normal

control. Animals treated with standard (Silymarin) showed extremely significant (P<0.001) increase in GSH, CAT & SOD and decrease in LPO. *M.citrifolia* (200mg/kg) treated animals showed significant (P<0.05) decrease in LPO and significant (P<0.05) increase in GSH, CAT & SOD. *M.citrifolia* (400mg/kg) treated animals showed moderately significant (P<0.01) decrease in LPO and moderately significant (P<0.01) increase in GSH, SOD & CAT.

Table 2: Effect of Silymarin and *M.citrifolia* root extract on SGOT, SGPT, ALP and Total Bilirubin in PCM induced liver toxicity

Groups	Treatment	ALP (U/l)	SGOT (U/l)	SGPT (U/l)	TB (mg/dl)
Vehicle control	Distilled water 1ml/ Kg	141.0±2.01	110.03±1.35	89.0±1.15	0.73±0.02
Toxic control	PCM 2g/Kg p.o	388.1±2.49[a]	382.7±3.38[a]	288.3±1.47[a]	2.23±0.02[a]
Standard	Silymarin 25mg/Kg, p.o	152.3±2.12***	123.8±2.71***	97.83±1.66***	0.85±0.02***
Low dose	*M.citrifolia* 200mg/Kg, p.o	294.1±1.27*	285.2±1.30*	195.2±1.13*	1.74±0.01*
High dose	*M.citrifolia* 400mg/Kg, p.o	192.0±1.18**	182.5±0.76**	134.5±1.12**	1.15±0.01**

All the values are Mean±SEM, n=6. One way ANOVA followed by Dunnett's t test. [a]p< 0.001 when compared with vehicle treated control group.*p<0.05, **p<0.01, ***p<0.001 when compared with toxic control.

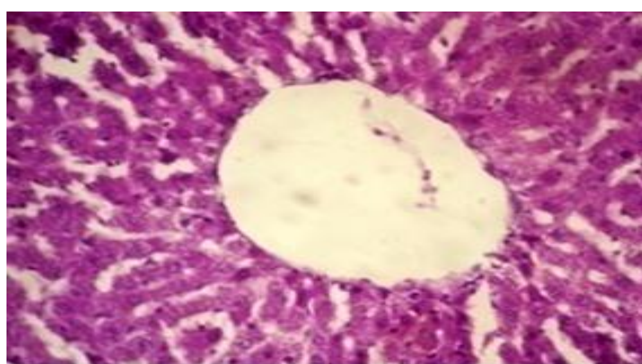
Table 3: Effect of Silymarin and *M.citrifolia* on LPO, SOD, GSH, and CAT in PCM induced liver toxicity

Groups	Treatment	LPO (Abs at 535 nm)	SOD (Abs at 560 nm)	GSH (Abs at 412nm)	CAT (Abs at 620 nm)
Normal Control	Distilled water ml/kg	2.96± 0.89	21.81± 1.25	28.0± 1.01	43.12± 0.971
Toxic control	PCM 2g/Kg .	16.71± 3.23[a]	13.18± 3.12[a]	18.19± 4.14[a]	27.15± 4.95[a]
Standard	Silymarin 100mg/Kg p.o	5.31± 1.41***	20.22± 2.59***	23.15± 1.81***	39.43± 2.04***
Low dose	*M.citrifolia* 200mg/Kg p.o	11.12± 2.21*	14.56± 3.81*	19.30± 2.14*	32.01± 3.01*
High dose	*M.citrifola* 400mg/Kg p.o	8.12± 1.19**	16.20± 2.65**	21.14± 1.81**	35.33± 3.51**

All the values are Mean±SEM, n=6. One way ANOVA followed by Dunnett's t test, [a]p<0.001 when compared with vehicle treated control group. *p<0.05, **p<0.01, ***p<0.001 when compared with toxic control.

I. Histopathological studies of the liver

The histopathological evaluation of PCM toxicity in all the groups was examined and shown in Fig.5. Liver section of normal group shows liver parenchyma with intact architecture. Most hepatocytes appear normal. In toxic control group shows inflammation, centrilobular degeneration and necrosis. Treatment with *M.citrifolia* (200mg/kg & 400mg/kg) found to reduce inflammation, centrilobular and bridging necrosis. Liver section of this group shows normal hepatocytes with significant reduction in areas of necrosis when compared to toxic group. These changes show protective effect of the drug against hepatic damage induced by PCM.



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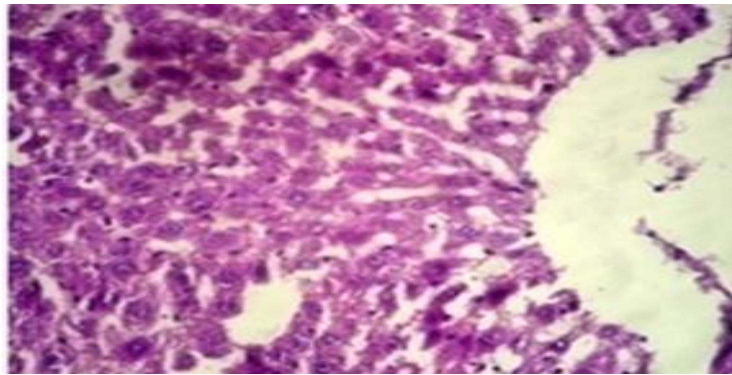

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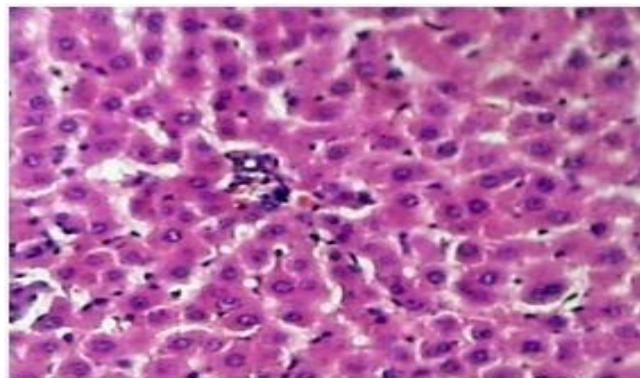

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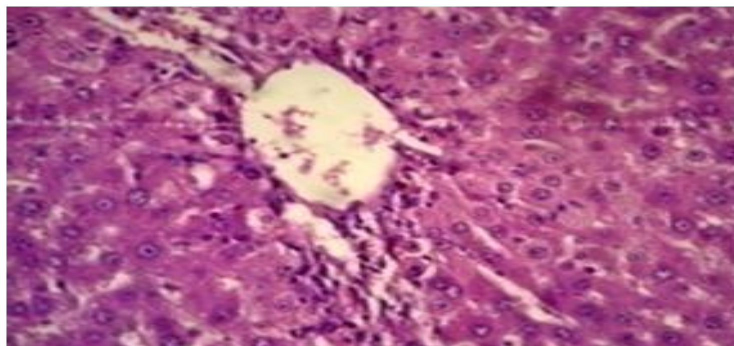

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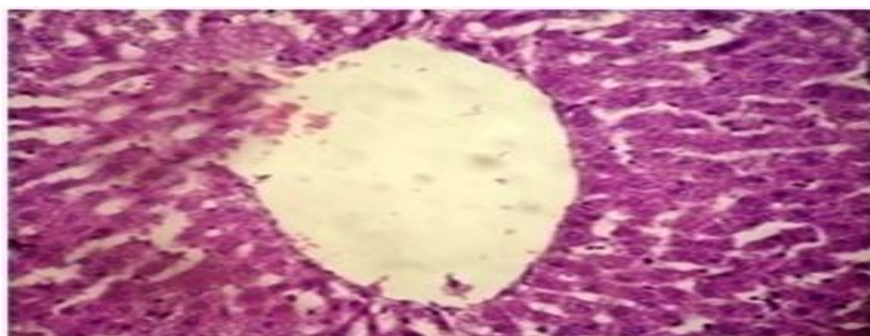

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Fig. 1: Effect of *M.citrifolia* on liver histology in PCM induced liver toxicity.
A: Liver of normal rat; B: Liver of PCM induced rat; C: Liver of Silymarin treated rat; D: Liver of *M.citrifolia* 200 mg/kg treated rat; E: Liver of *M.citrifolia* 400mg/kg treated rat.

Evaluation of hepatoprotective activity of *M.citrifolia* on CCl_4 induced hepatic damage in rats

The results of antioxidant potential of plumieride in CCl4-intoxicated rats are shown in Tables 4 and 5. It was observed that, the activities of serum SGOT, SGPT, ALP & TB were increased markedly in CCl_4 fed animals as compared to normal control group. The administration of *Morinda citrifolia* extract 200mg/kg and 400mg/kg lowered the ccl_4 induced elevation of serum parameters. The Standard (Silymarin) treatment showed extremely significant (P<0.001) reduction in SGPT, SGOT, ALP, and TB. *Morinda citrifolia* (200mg/kg) treated animals showed significant (P<0.05) reduction in SGPT, SGOT, ALP & TB levels as compared to toxic control group. *Morinda citrifolia* extract 400mg/kg treated animals showed moderately significant (P<0.01) reduction in SGOT, SGPT, ALP & TB level as compared to toxic control group.

Table No.5 shows the effects of extracts of *Morinda citrifolia* on SOD, CAT, GSH and lipid peroxidation concentrations in rat liver after challenging with CCl_4. It was observed that animals treated with CCl_4 developed a hepatic damage observed as increase in LPO and decrease in GSH, CAT & SOD when compared to normal control. Animals treated with standard (Silymarin) showed extremely significant (P<0.001) increase in GSH, CAT & SOD and decrease in LPO. *M.citrifolia* root extract (200mg/kg) treated animals showed significant (P<0.05) decrease in LPO and significant (P<0.05) increase in SOD, GSH & CAT as compared to toxic control. *Morinda citrifolia* root extract (400mg/kg) treated animals showed moderately significant (P<0.01) decrease in LPO and moderately significant (P<0.01) increase in GSH, SOD & CAT.

Table 4: Effect of Silymarin and *Morinda citrifolia* on SGOT, SGPT, ALP& TB in CCl_4 induced liver toxicity

Groups	Treatment	ALP(U/l)	SGOT(U/l)	SGPT(U/l)	TB(mg/dl)
Normal control	Saline	145.7± 2.12	1080.01± 1.16	83.9± 1.28	0.78± 0.08
Toxic control	CCL_4 0.7 mg/kg i.p	289.91± 2.05	259.51± 1.59	250.01± 1.61	2.89± 0.12
Standard	Silymarin 100mg/Kg, p.o	183.0± 2.17***	132.1± 2.13***	98.83± 1.51***	0.95± 0.01***
Low dose	*Morinda citrifolia* root extract 200 mg/Kg, p.o	237.11± 1.67*	201.09± 1.97*	160.5± 2.09*	1.81± 0.02*
High dose	*Morinda citrifolia* root extract 400 mg/Kg, p.o	198.48± 1.16**	190.31± 1.48**	110.21± 1.21**	1.21±0.03**

All the values are Mean±SEM, n=6. One way ANOVA followed by Dunnett's t test. [a]p< 0.001 when compared with vehicle treated control group.*p<0.05, **p<0.01, ***p<0.001 when compared with toxic control.

Table 5: Effect of *Morinda citrifolia* on LPO, SOD, GSH, and CAT in CCl_4 induced liver toxicity

Groups	Treatment	LPO (Abs at 535 nm)	SOD (Abs at 560 nm)	GSH (Abs at 412nm)	CAT (Abs at 620 nm)
Normal Control	Saline	3.81± 0.90	23.12± 2021	32.15± 1.12	45.71± 0.99
Toxic control	CCl_4 0.7 ml/kg i.p	18.81± 4.01[a]	14.91± 3.92[a]	19.06± 4.01[a]	27.14± 3.89[a]
Standard	Silymarin 100mg/kg	6.28± 1058***	22.15± 2.31***	25.81± 1.56***	35.01± 2.11***
Low dose	*Morinda citrifolia* root extract 200mg/kg	10.86± 2.38*	12.81± 3.23*	17.21±2.45*	25.51± 3.34*
High dose	*Morinda citrifolia* root extract 400mg/kg	8.73± 1.01**	15.78± 2.19**	21.54±1.78**	32.3±3.61 **

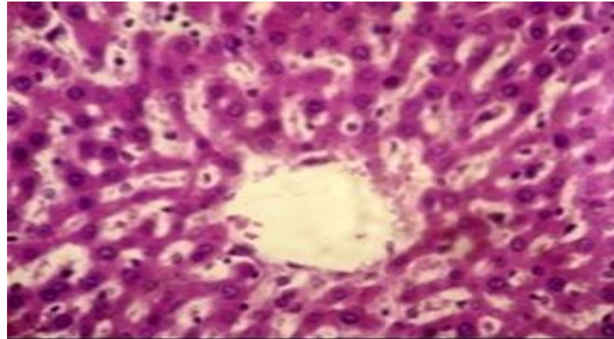
All the values are Mean±SEM, n=6. One way ANOVA followed by Dunnett's t test. [a]p< 0.001 when compared with vehicle treated control group. *p<0.05, **p<0.01, ***p<0.001 when compared with toxic control.

III. Histopathological studies of the liver

The histopathological evaluation of ccl_4 toxicity in all the groups was examined and shown in Fig.2.

Liver section of normal group shows liver parenchyma with intact architecture. Most hepatocytes appear normal.

In toxic control group shows inflammation, centrilobular degeneration and necrosis. Treatment with *Morinda citrifolia* root extract (200mg/kg & 400mg/kg) found to reduce inflammation, centrilobular and bridging necrosis. Liver section of this group shows normal hepatocytes with significant reduction in areas of necrosis when compared to toxic group. These changes show protective effect of the drug against hepatic damage induced by CCl_4.



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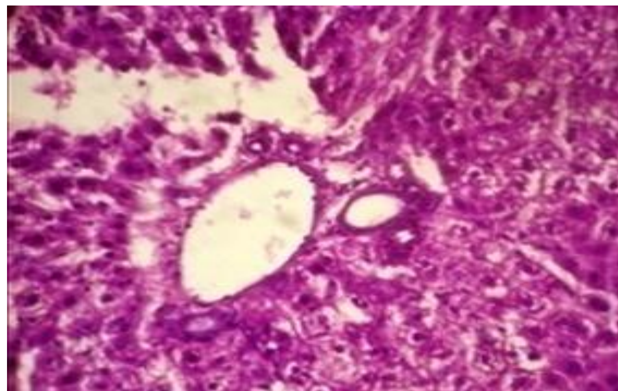

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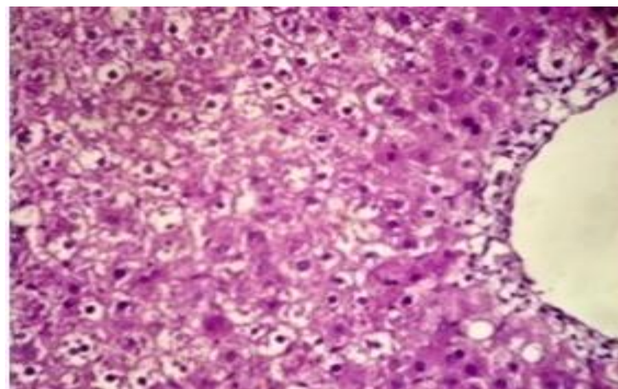

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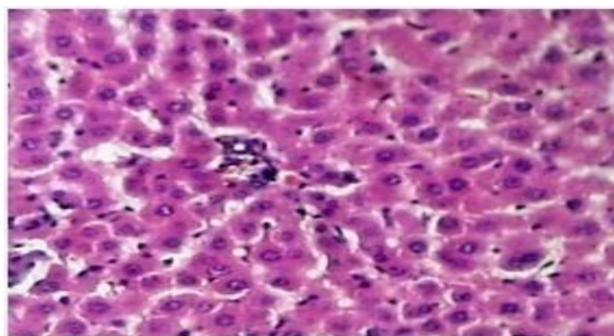

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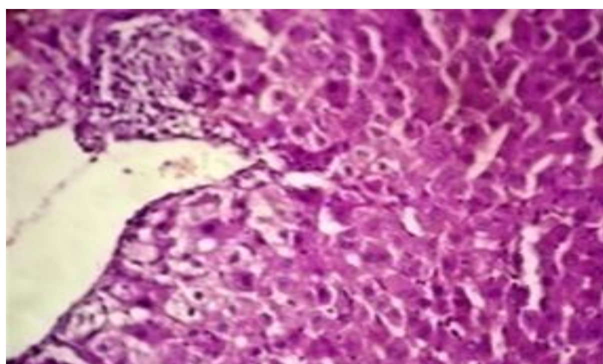

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Fig. 2: Effect of *Morinda citrifolia* on liver histology in CCl_4 induced liver toxicity
A: Liver of normal rat; B: Liver of CCl_4 induced rat; C: Liver of Silymarin treated rat; D: Liver of *Morinda citrifolia* 200mg/kg treated rat; E: Liver of *Morinda citrifolia* 400mg/kgtreated rat

DISCUSSION

The present study was under taken to evaluate the hepatoprotective activity of *Morinda citrifolia* plant root extract. The study was conducted by using two models i.e., paracetamol and ccl_4 induced liver damage model. The parameters used for the assessment of hepatoprotective activity were serum enzyme estimations like SGPT, SGOT, ALP, Total billirubin, serum CAT, SOD, GSH level and histopathological studies.

Administration of paracetamol elevated the serum levels of SGPT, SGOT, ALP and Total billirubin significantly. This is due to its bioactivation to a toxic electrophile, N-acetyl-p-benzoquine-imine. Paracetamol is normally eliminated mainly as sulphate and glucuronide. Only 5% of the paracetamol is converted into N- acetyl-p-benzoquinineimine. However, upon administration of toxic doses of paracetamol, the sulfation and glucuronidation routes become saturated and hence, higher percentage of paracetamol molecules are oxidized to highly reactive N-acetyl-p-benzoquineimine (NAPQI) by cytochrome-450 enzymes. A semi Quinone radical, obtained by one electron reduction of NAPQI, can covalently binds to macromolecules of cellular membrane and increases the lipid peroxidation resulting in the tissue damage[10].

The hepatotoxic effect of paracetamol was also confirmed by histopathological studies which showed portal tract inflammation.

Pre-treatment with *Morinda citrifolia* plant root extract was able to prevent the elevation of SGPT, SGOT, ALP and Total billirubin by paracetamol. These biochemical effects may be due to the inhibitory effects on cytochrome P450 and /or promotion of its glucuronidation[11]. This was also confirmed by histopathological studies which showed almost normal hepatocytes.

CCl4-induced hypofunctions of the hepatic cell membrane due to hepatic injury are associated with the peroxidation of lipids and reduced antioxidant levels through the production of toxic species—trichloromethyl free radical (CCl3•) or/and trichloromethyl peroxy radical (CCl3OO•) which in turn alter the hepatic metabolism via oxidative stress leading to hepatotoxicity[12,13].

The lipid peroxidative degradation of biomembranes is one of the principle causes of hepatotoxicity induced by CCl4. This is evidenced by the elevation of serum marker enzymes such as AST, ALT, GGT, ALP along with the total bilirubin and reduction in the level of total protein. The decrease in activity of antioxidant enzymes like superoxide dismutase, glutathione peroxidase are speculated to be due to the damaging effects of free radicals produced following CCl_4exposure or alternatively could be due to a direct effect of acetaldehyde, formed by oxidation of ethanol[13].

Elevated levels of serum biomarkers in CCl_4 treated animals are indicative of cellular leakage & loss of functional integrity of hepatic cell membranes implying hepatocellular damage. The plant extract *M.citrifolia* (200mg/kg and 400mg/kg) showed dose dependent decrease in the elevated serum biomarkers (SGPT, SGOT, ALP and bilirubin), lipid peroxidation, and significant increase in endogenous enzymes (GSH, SOD, CAT). Hence it might be possible that the mechanism of hepatoprotection by *Morinda citrifolia* plant is due to its antioxidant potential.

Histopathological studies of liver, treated with CCl_4 alone revealed the affected architecture of liver parenchyma with damaged hepatocytes. Treatment with *M.citrifolia* plant root extract (200mg/kg & 400 mg/kg) revealed significant improvement in architecture of liver parenchyma towards normal and

regenerating hepatocytes indicating hepatoprotection. The standard drug Silymarin showed extremely significant reduction in serum biomarkers and endogenous enzyme level.
Recent studies have shown that many flavonoids and related polyphenols contribute significantly to the total antioxidant activity of many plant extracts[14]. Natural antioxidants from the plant extracts provide a measure of production of radical scavengers that slows the process of oxidative damage; further previous report indicates that the *M.citrifolia* plant root extract proved for its potential antioxidant properties. The present study revealed that the *Morinda citrifolia* plant root extract have proved its synergistic antioxidant effects of bioactive constituents for observed hepatoprotective activity.

CONCLUSION

The present study was undertaken to assess the hepatoprotective activity of *Morinda citrifolia* plant root extract. The extract found to have significant hepatoprotective activity in both; paracetamol and carbon tetrachloride induced hepatic injury models. Biochemical and histopathological studies have revealed that this fruit have comparable hepatoprotective activity with that of Silymarin. It leads to the conclusion that the *Morinda citrifolia* plant root extract can be utilized for its hepatoprotective activity. Further studies are needed to isolate and characterize the active principles and to find out the mechanism responsible for its hepatoprotective activity.

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