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# Hepatoprotective potential of ethanolic and aqueous extract of flowers of *Sesbania grandiflora* (Linn) induced by $CCl_4$

Ishwer Kale[1*], Mohd Asif Khan[2], Yusufuddin Irfan[1], Veerana Goud A[1]

[1]*Department of Pharmacology, S.C.S. College of Pharmacy, Harapanahalli, Davangere Dist. (Karnataka) 583131. India*
[2]*Department of Pharmacology, Faculty of Pharmacy, VCTE, Bijnor-246701 (U.P.) India*

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ABSTRACT

**Objective:** To investigate the hepatoprotective activity of ethanolic and aqueous extract of *Sesbania grandiflora* (Linn) flower in $CCl_4$ induced hepatotoxicity models in rats. **Methods:** The ethanolic and aqueous extract of *Sesbania grandiflora* (Linn) flower are screened for its hepatoprotective activity in $CCl_4$ (0.5 ml/kg, i.p) induced liver damage in Swiss albino rats at a dose of 200 mg/kg bw. **Results:** The ethanolic and aqueous extract of *Sesbania grandiflora* (Linn) flower significantly ($P<0.001$) decreases the biochemical parameters (SGOT, SGPT, ALP, TP, and TB). Silymarin (25 mg/kg), a known hepatoprotective drug used for comparison exhibited significant activity ($P<0.001$). The extract did not shown any mortality up to a dose of 2000 g/kg bw. These findings suggest that the ethanolic and aqueous extract of *Sesbania grandiflora* (Linn) flower (500mg/kg) was effective in bringing about functional improvement of hepatocytes. The healing effect of this extract was also confirmed by histological observations. **Conclusions:** The ethanolic extract at doses of 250 and 500 mg/kg, p.o. and aqueous extract at a dose of 500 mg/kg, p.o. of *Sesbania grandiflora* (Linn) flower have significant effect on the liver of $CCl_4$ induced hepatotoxicity animal models.

## 1. Introduction

Liver is one of the important organs of the body which plays a major role in the metabolism of proteins, carbohydrates, lipids. It is also having wide range of functions including detoxification, storage of glycogen, vitamin A, D and B12, production of several coagulation factors, growth factors (IGF-1), hormones (angiotensinogen) and biochemicals necessary for digestion (bile). Hepatic damage occurs due to its multi dimensional functions, various xenobiotics and oxidative stress leading to distortion of all of its functions [1]. Liver disease is still a worldwide health problem. Jaundice and hepatitis are two major hepatic disorders that account for high death rate [2]. Unfortunately, conventional or synthetic drugs used in the treatment of liver diseases are inadequate and sometime can have serious side effect

*Sesbania grandiflora* (family Leguminosae) commonly called as Agati in Hindi. The literature survey revealed that libido [3], night blindness [4], anti-ulcer [5], antioxidant [6-7], cardioprotective [8], emollient, astringent,analgesic, antipyretic [9], anti-obesity [10], ozoena, dim vision [11],gout and bronchitis [12], anxiolytic, anticonvulsant [13], hepatoprotective [14] and anthelmintic [15] activity of *Sesbania grandiflora* flower has been already reported. The active ingredients of S. grandiflora are leucocyanidin and cyanidin present in seeds, oleanolic acid and its methyl ester and kaemferol-3-rutinoside which are present in flower. The bark contains tannins and gum. Saponin isolated from the seeds. Sesbanimide isolated from seeds [16]. Based on its diversified pharmacological properties and its used in liver disease, the objective of the present studywas to evaluate the hepatoprotective potential of ethanolic and aqueous extract of flowers of *Sesbania grandiflora* (Linn)

*Corresponding author: Mohd Asif Khan, Assit. Professor, Dept. of Pharmacology, Faculty of Pharmacy VCTE, Bijnor- 246701 (U.P.) India.
Tel.: +91 7376762117
E-mail: asifmpharma@gmail.com

induced by $CCl_4$.

## 2. Material and method

### 2.1 Plant collection and Preparation of extract

*Sesbania grandiflora* flowers was collected from the surrounding fields of Harapanahalli, Davengere, Karnataka, india in the month of July 2009. The plant material was identified and authenticated by Professor K. Prabhu, Department of Pharmacognosy, S.C.S. College of Pharmacy, Harapanahalli, and voucher specimen of the collected sample was deposited in the departmental herbarium for further reference. The flowers were washed with doubled distilled waternto eliminate dirt, then shade dried. The dried materials were powdered and passed through a 10-mesh sieve. The coarsely powered material was extracted by using soxhlet column and extracted with 70% ethanol (60 - 800) and distilled water for 24 hrs. The extract were filtered, pooled and concentrated at 5oC on a rotatory flash evaporator and then dried on hot water bath (70 - 800). The dried extract was stored in airtight container in refrigerator below 100C. Further dilutions were made to obtain different concentration and were used pharmacological investigation.

### 2.2 Experimental animal

The male albino rat wistar stain 150 - 200 g and albino mice 20 - 30 g were procured from Venkateshwara Associates, Bangalore, Karnataka, and kept in conventional cages with free access to water ad libitum and standard rat feed with rodent pellet diet (Gold mohr, Lipton India Ltd.,) at 27 ± 30C, humidity 65 ± 10%, 12 hrs light/dark cycle, respectivily. All the experiments were performed in accordance with the guide for the care and use of laboratory animals, as adopted and promulgaated by the Institutional Animal Ethics Committee (IAEC) [17].

### 2.3 Determination of acute toxicity (LD50)

After acclimization of the animals for 4-5 days, study was carried out on healthy, young adult Albino swiss female mice (20-25 g), nulliporous and non pregnant were used for this study. Food, but not water was with held for 3-4 hr and further 1-2 hr post administration of smple under study. Fixed dose level of 5, 50, 100, 250, 500 mg/kg were initially chosen as dose level that would be expected to allow the identification of dose producing evident toxicity. During the validation procedure, a fixed dose of 2000 mg/kg was added to provide more information on substance of low acute toxicity.

After the administration of extract of formulation, animals were observed individually during the first 30 min and periodically during 24 hr with special attention during the first hours and daily thereafter for a period of 14 days. Once daily animals were observed principally in relation to change in skin, fur, eyes and mucous membrane (nasal) and also autonomic symptoms like sedation, lacrimation, perspiration, pilorection, urinary incontinence and control nervous system (ptosis, drawsiness, gait tremors and convulsions). Fixed dose (OCED Guideline No. 420) method of CPCSEA was adopted for toxicity studies.

### 2.4 Experimental design (Malaya gupta et al., was followed [18])

Animals were divided into seven groups of six rats each. Animals were treated for a period of 10 days. Group I received a single daily dose of CMC (1ml of 1% w/v) for 10 days. Group II received CCl4 (30% in liquid paraffin 1 ml/kg body weight, i.p.) once in every 72 hr. Group III were received daily oral dose of silymarin (25 mg/kg p.o.) once in a day along with CCl4. Group IV and V received ethanolic extract (250 and 500 mg/kg p.o.) and group VI and VII were received aqueous extract of *Sesbania grandiflora* (Linn) flower with dose of (250 and 500 mg/kg p.o.) respectively. In this study the role of silymarin was used as a positive control, as well as the hepatoprotective potential of different doses of *Sesbania grandiflora* (Linn) flower was compared with the effect of silymarin. On 11th day, animal were sacrifed and blood was collected by carotid bleeding for biochemical analysis.

The liver tissue was dissected out for histological investigation and fixed in 10% formalin, dehydrated in gradual ethanol (50-100%), cleared in xyline, and embedded in paraffin. Section were prepared and then stained with hematoxylin and eosin (H-E) dye for photomicroscopic observation, including cell necrosis, fatty changes hyaline generation, ballooning generation. All samples were observed and photographed with microscope.

### 2.5 Determination of biochemical parameters

The collected blood was allowed to clot and centrifuged at 3000 rpm for 15 min to obtain the serum. The biochemical parameters like serum glutamate-pyruate (SGPT), serum glutamate oxaloacetate transaminase (SGOT), serum alkaline phosphatase (ALP), total bilirubin (TB), total protein (TP) [19-21] were estimated using respected assay kits according to the methods described by the manufacturers.

### 2.6 GSH estimation in CCl4 induced hepatotoxicity

Tissue samples were homogenized in ice cold Trichloroacetic acid (500mg tissue plus 5 ml 10% TCA) in an ultra turrax tissue homogenizer. Glutathione measurements were performed using a modification of the Ellamn procedure (Aykae, et. al.,) [22]. Briefly, after centrifugation at 3000 rpm for 10 minutes, 0.5 ml supernatant was added

to 2 ml of 0.3 M disodium hydrogen phosphate solution. A 0.2 ml solution of dithiobisnitrobenzoate (0.4 mg/ml in 1% sodium citrate) was added and the absorbance at 412 nm was measured immediately after mixing. % increase in OD is directly proportional to the increase in the levels of Glutathione. Hence, % increase in OD is calculated.

### 2.7 In vivo lipid peroxidation in CCl4 induced hepatotoxicity

Lipid peroxidation, is accepted to be one of the principle causes of CCl4-induced liver injury, and is mediated by the production of free radical derivatives of CCl4 [23]. The degree of lipid peroxide formation was assayed by monitoring thiobarbituric reactive substance formation [24-25]. Stock solution of TCA-TBA-HCl reagent: 15% w/v trichloroacetic acid; 0.375% w/v thiobarbituric acid; 0.25N hydrochloric acid. This solution may be mildly heated to assist in the dissolution of the thiobarbituric acid. Combine 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-TBA-HCl and mix thoroughly. The solution is heated for 1 hr in a boiling water bath. After cooling, the flocculent precipitate was removed by centrifugation at 1000 rpm for 2 min. The absorbance of the sample is determined at 535 nm against a blank that contains all the reagents minus the lipid (tissue). The malondialdehyde concentration of the sample can be calculated by using an extinction coefficient of $1.56\times105\ M^{-1}\ cm^{-1}$.

### 2.8 Statistical analysis

All the data were presented as means ± SEM. Statistical analyses were performed byn Analysis of Variance (ANOVA) followed by Turkey Kramer test. Difference were considered statistically significant at the value of probability less then <0.001.

## 3. Results

### 3.1 Effect on biochemical markers in CCl4 induced hepatotoxicity

There is a marked increase in SGPT (IU/L) levels were observed in CCl4 treated group (351.37 IU/L). However, the SGPT levels were reversed to near normal levels i.e., (75.74IU/L) and (69.16 IU/L) with the treatment of 500 mg/kg of 70% EEFSG and AQFSG respectively. In addition the standard silymarin 100 mg/kg has restored the SGPT levels significantly i.e. 65.29(IU/L).

Serum SGOT (IU/L) levels were also elevated in the $CCl_4$ treated group (264.48IU/L). Treatment with standard silymarin 100 mg/kg has brought back the SGOT to the near normal levels i.e. 158.43(IU/L). However treatment with the 70% EEFSG and AQFSG restored the SGOT levels in a dose dependent manner at both the tested doses (250 mg/kg and 500 mg/kg) up to (260.75and199.13 IU/L) and (188.33and161.68 IU/L) respectively, which are statistically significant.

In case of total and direct bilirubin there was a noticeable rise (4.41 and 4.75 mg/dl respectively) in serum levels up CCl4 treatment observed. Treatment with 500 mg/kg of 70%EEFSG and AQFSG has reversed the total and direct bilirubin serum levels to (1.67 and 1.19 mg/dl) and (3.68 and 3.72mg/dl) respectively which is statistically significant, when compared with CCl4 treated group. The reversal by standard silymarin 100 mg/kg was also significant i.e. 1.12 and 3.67 mg/dl in case of total and direct bilirubin respectively.

Moreover, rise in ALP IU/L serum levels due to CCl4 challenge was remarkable (295.00 IU/L) and brought back significantly by the 250 mg/kg and 500 mg/kg doses of 70% EEFSG and AQFSG to near normal level i.e.( 214.44 and 195.55 IU/L) and (225.08 and 182.56 IU/L) respectively, whereas standard silymarin 100 mg/kg responded well and restored the ALP levels to 170.40 IU/L Increased wet liver weight and wet liver volume were also significantly prevented by extract or silymarin treatment (table-1 fig . 2-8).

### 3.2 Effect on tissue GSH and in-vivo LPO levels in CCl4 induced hepatotoxicity

There was a marked depletion of GSH levels in CCl4 treated group. Treatment with 100 mg/kg silymarin prevented the depletion of GSH to extend of 69.37%. Treatment with 250 and 500mg/kg of 70% EEFSG and AQFSG extract has

**Table 1**
Effect of EEFSG & AEFSG biochemical and morphological parameter in $CCl_4$ induced hepatotoxicity.

| Groups | SGPT (IU/L) | SGOT (IU/L) | ALP (IU/L) | TB (mg/dl) | DB (mg/dl) | Volume (ml/100g) | Weight (gm/100g) |
|---|---|---|---|---|---|---|---|
| -ve control | 61.79±6.80 | 124.16±4.46 | 173.02±10.19 | 0.98±0.03 | 3.21±0.21 | 3.9±0.12 | 3.1±0.10 |
| +ve control | 351.37±11.25 | 264.48±4.92 | 295.00±24.11 | 4.41±0.18 | 4.75±0.10 | 5.18±0.19 | 4.76±0.22 |
| Standard silymarin 100 mg/kg | 65.29±7.18*** | 158.43±3.96*** | 170.4±6.53*** | 1.12±0.09*** | 3.67±0.15*** | 4.05±0.14*** | 3.58±0.17*** |
| EEFSG 250 mg/kg | 97.84±7.02*** | 260.75±22.35* | 214.44±12.22** | 2.801±0.16*** | 3.97±0.20* | 4.63±0.08* | 3.9±0.07** |
| EEFSG 500 mg/kg | 75.74±5.82*** | 199.13±16.08** | 195.55 ±6.26* | 1.67 ±0.13*** | 3.68±0.16*** | 4.11±0.11*** | 3.68±0.12*** |
| AQFSG 250 mg/kg | 95.04±19.69*** | 188.33±5.27*** | 225.08±5.47*** | 1.91±0.13*** | 3.82±0.14** | 4.56±0.14* | 3.75±0.17*** |
| AQFSG 500 mg/kg | 69.16±3.74*** | 161.68±6.65*** | 182.56±11.06*** | 1.19±0.07*** | 3.72±0.11*** | 4.35±0.11*** | 3.63±0.13*** |

Values are mean±SEM ($n$=6).
* $P$<0.05, ** $P$<0.01, *** $P$<0.001 as compared to positive control.

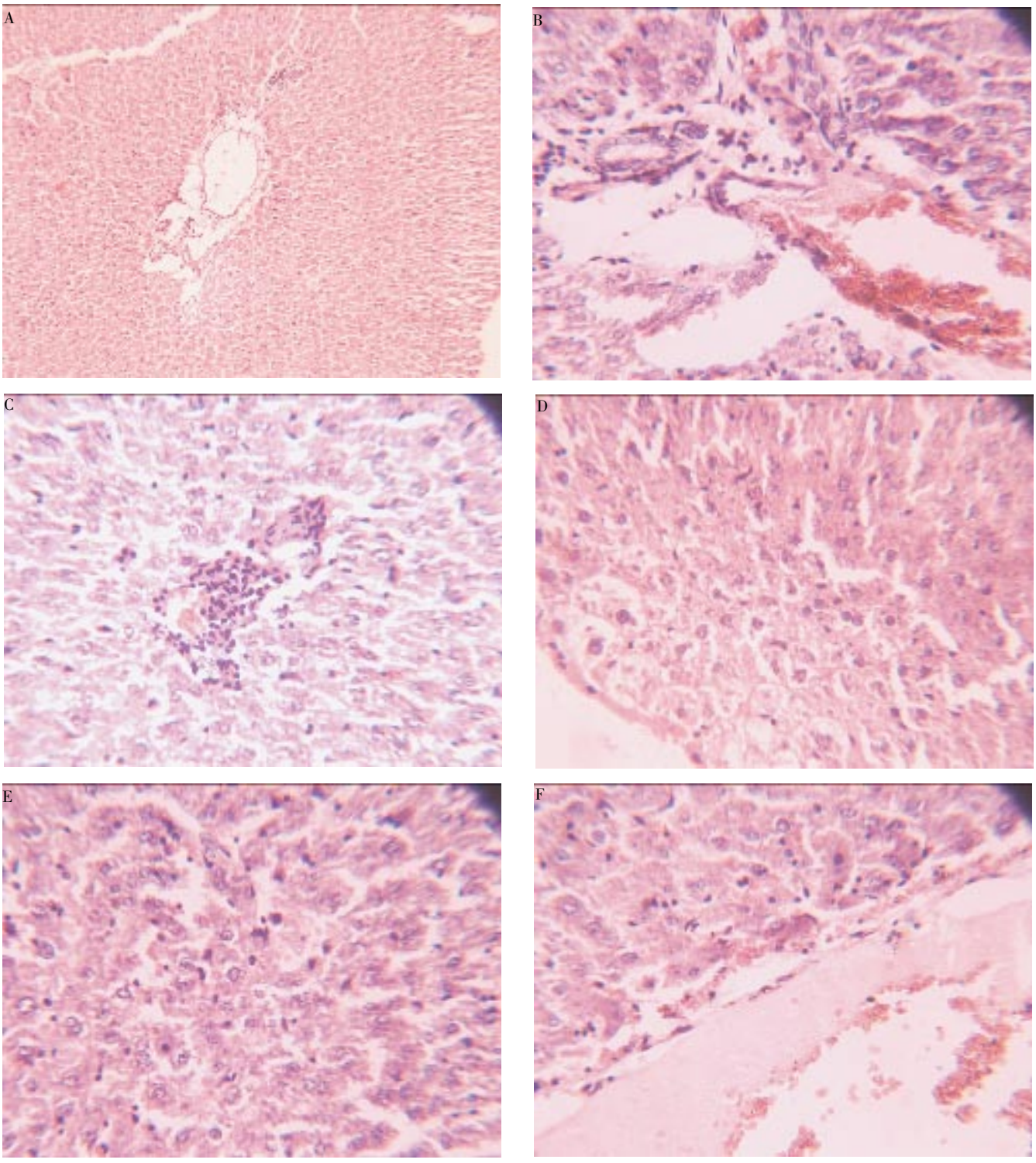
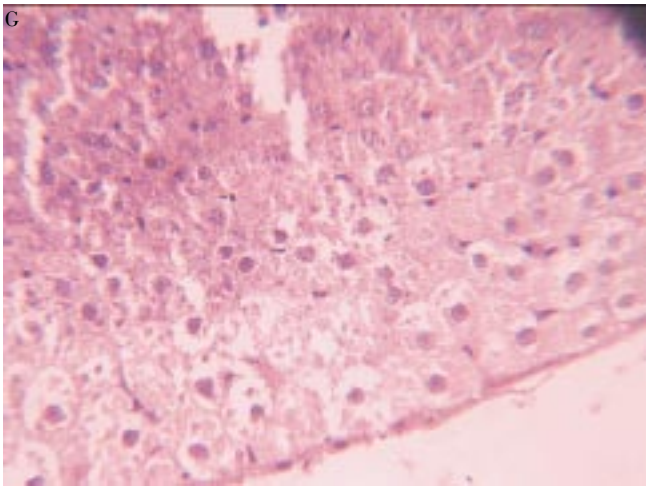



**Figure 1.** Effect of Sesbenia grandiflora flower extract on liver architecture in normal and CCl4 treated rats. [A: liver architecture of negative control, B: liver architecture of positive control after CCl4 challenge (ballooning of hepatocytes, fatty cysts), C: liver architecture after Silymarin + CCl4 challenge (lymphocytic infiltration); D: liver architecture after EEDRF (100 mg/kg) + CCl4 challenge (lymphocytic infiltration, hepatocytes); E: liver architecture after EEDRF (250 mg/kg) + CCl4 challenge (lymphocytic infiltration); F: liver architecture after AEDRF (100 mg/kg) (lymphocytic infiltration, hepatocyte) + CCl4 challenge; G: liver architecture after AEDRF (250 mg/kg) + CCl4 challenge (lymphocytic infiltration, normal hepatic architecture]




brought back the decreased GSH levels in a dose dependant manner to a near normal level. The test extracts was found to be statistically significant at both lower and higher doses in normalizing tissue GSH levels. However, test extracts at 500 mg/kg (highest dose studied) was found to be near to standard (silymarin).

There was a dose dependent inhibition of in-vivo lipid peroxidation by both the doses (250 mg/kg and 500 mg/kg) of 70% ethanolic and aqeous extract of *Sesbania grandiflora* (Linn.) Silymarin 100mg/kg showed 60.46 % inhibition whereas both the extracts at a dose of 500 mg/kg has 47.16% and 51.95% inhibition (Table-2 and figure no.9, 10).

### 3.3 Histopathological Studies in CCl4 induced hepatytoxicity

Histopathological profile of liver from CCL4 (-ve control) rats reveals hepatic globular structure, central vein, portal tract and kuffer cells look normal. CCL4 (+ve control) intoxicated architecture disrupted, hepatic cells has shown various degree of fatty degeneration like ballooning of hepatocytes, fatty cyst, infiltration of

**Table 2**

Effect of ethanolic and aqeous aqueous extract of flowers of *Sesbinia grandiflora* (Linn.) on tissue GSH levels and in vivo lipid peroxidation in $CCl_4$ induced hepatotoxicity.

| Treamant | GSH levels | | in vivo lipid peroxidation | |
|---|---|---|---|---|
| | Absorbance Mean±SEM | % inhibition | Absorbance Mean±SEM | % inhibition |
| Negative Control (1ml vehicle) | 0.783±0.057 | -- | 0.218±0.008 | -- |
| Positive Control CCl4 + Liq. Paraffin (1:1) (2 ml/kg s.c.) | 0.215±0.032 | -- | 0.564±0.112 | -- |
| CCl4 + Standard (Silymarin) (2 ml/kg s.c. + 100 mg/kg p.o.) | 0.702±0.035*** | 69.37 | 0.223±0.006*** | 60.46 |
| CCl4 + EEFSG (2 ml/kgs.c. + 250 mg/kg p.o.) | 0.493±0.028*** | 56.38 | 0.411±0.024*** | 27.12 |
| CCl4 + 70% EEFSG (2 ml/kg s.c.+ 500 mg/kg p.o.) | 0.642±0.073*** | 66.51 | 0.298±0.012*** | 47.16 |
| CCl4 + AQFSG (2 ml/kg s.c.+ 250 mg/kg p.o.) | 0.689±0.34*** | 68.79 | 0.436±.007*** | 22.69 |
| CCl4 + AQFSG (2 ml/kg s.c.+ 500 mg/kg p.o.) | 0.700±0.058*** | 69.28 | 0.271±0.008*** | 51.95 |

The values are mean±SEM ($n$=6) ***$P$<0.001 Vs +ve control.

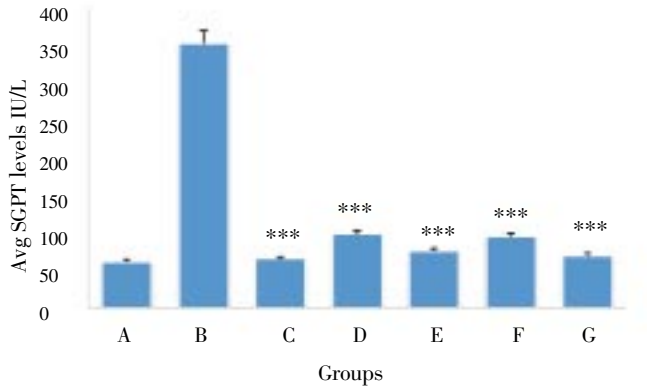



**Figure 2.** Histogram showing SGPT levels of ethanolic and aqueous extract of Sesbenia grandiflora flower on CCl4 induced hepatotoxicity on rats. [A= negative control; B= positive control (CCl4 challenge); C= Standard (Silymarin + CCl4 challenge); D= EEFSG (250 mg/kg p.o.) + CCl4 challenge; E= EEFSG (500 mg/kg p.o.) + CCl4 challenge; F= AEFSG (250 mg/kg p.o.) + CCl4 challenge; G= AEFSG (500 mg/kg p.o.) + CCl4 challenge].

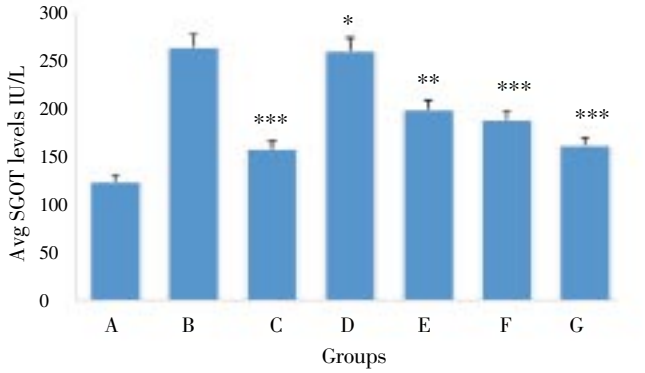



**Figure 3.** Histogram showing SGOT levels of ethanolic and aqueous extract of Sesbenia grandiflora flower on CCl4 induced hepatotoxicity on rats. [A= negative control; B= positive control (CCl4 challenge); C= Standard (Silymarin + CCl4 challenge); D= EEFSG (250 mg/kg p.o.) + CCl4 challenge; E= EEFSG (500 mg/kg p.o.) + CCl4 challenge; F= AEFSG (250 mg/kg p.o.) + CCl4 challenge; G= AEFSG (500 mg/kg p.o.) + CCl4 challenge].

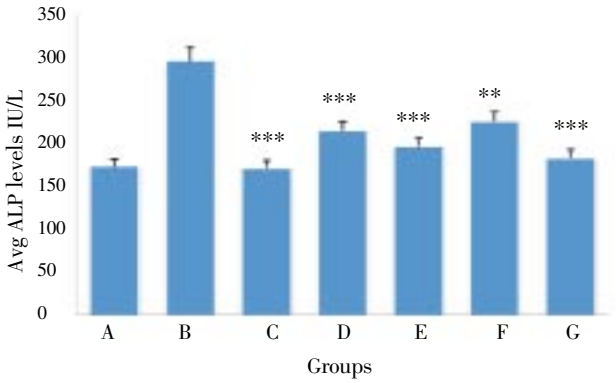



**Figure 4.** Histogram showing ALP levels of ethanolic and aqueous extract of Sesbenia grandiflora flower on CCl4 induced hepatotoxicity on rats. [A= negative control; B= positive control (CCl4 challenge); C= Standard (Silymarin + CCl4 challenge); D= EEFSG (250 mg/kg p.o.) + CCl4 challenge; E= EEFSG (500 mg/kg p.o.) + CCl4 challenge; F= AEFSG (250 mg/kg p.o.) + CCl4 challenge; G= AEFSG (500 mg/kg p.o.) + CCl4 challenge].

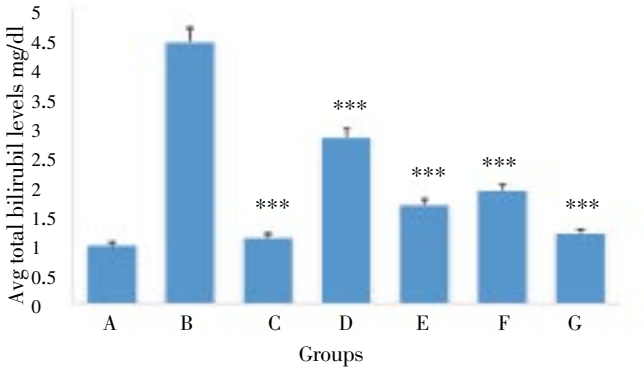



**Figure 5.** Histogram showing total bilirubin levels of ethanolic and aqueous extract of Sesbenia grandiflora flower on CCl4 induced hepatotoxicity on rats. [A= negative control; B= positive control (CCl4 challenge); C= Standard (Silymarin + CCl4 challenge); D= EEFSG (250 mg/kg p.o.) + CCl4 challenge; E= EEFSG (500 mg/kg p.o.) + CCl4 challenge; F= AEFSG (250 mg/kg p.o.) + CCl4 challenge; G= AEFSG (500 mg/kg p.o.) + CCl4 challenge]

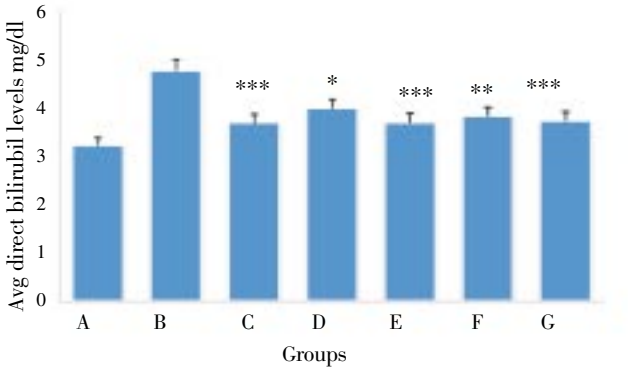



**Figure 6.** Histogram showing direct bilirubin levels of ethanolic and aqueous extract of Sesbenia grandiflora flower on CCl4 induced hepatotoxicity on rats. [A= negative control; B= positive control (CCl4 challenge); C= Standard (Silymarin + CCl4 challenge); D= EEFSG (250 mg/kg p.o.) + CCl4 challenge; E= EEFSG (500 mg/kg p.o.) + CCl4 challenge; F= AEFSG (250 mg/kg p.o.) + CCl4 challenge; G= AEFSG (500 mg/kg p.o.) + CCl4 challenge].

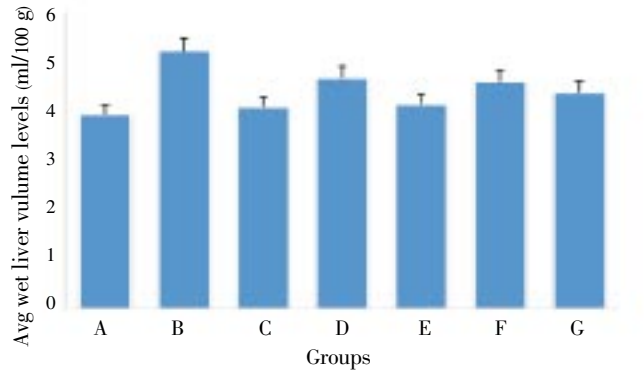



**Figure 7.** Histogram showing wet liver volume levels of ethanolic and aqueous extract of Sesbenia grandiflora flower on CCl4 induced hepatotoxicity on rats. [A= negative control; B= positive control (CCl4 challenge); C= Standard (Silymarin + CCl4 challenge); D= EEFSG (250 mg/kg p.o.) + CCl4 challenge; E= EEFSG (500 mg/kg p.o.) + CCl4 challenge; F= AEFSG (250 mg/kg p.o.) + CCl4 challenge; G= AEFSG (500 mg/kg p.o.) + CCl4 challenge].

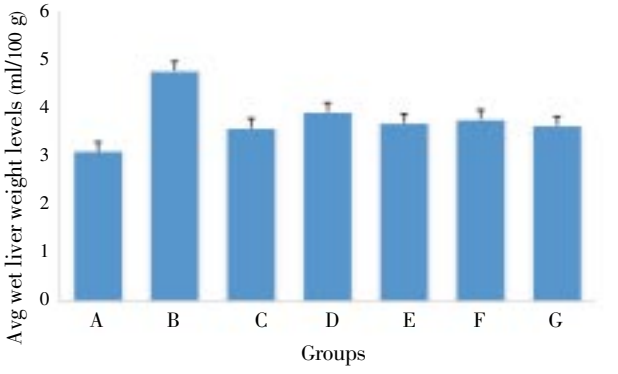



**Figure 8.** Histogram showing wet liver weight levels of ethanolic and aqueous extract of Sesbenia grandiflora flower on CCl4 induced hepatotoxicity on rats. [A= negative control; B= positive control (CCl4 challenge); C= Standard (Silymarin + CCl4 challenge); D= EEFSG (250 mg/kg p.o.) + CCl4 challenge; E= EEFSG (500 mg/kg p.o.) + CCl4 challenge; F= AEFSG (250 mg/kg p.o.) + CCl4 challenge; G= AEFSG (500 mg/kg p.o.) + CCl4 challenge]

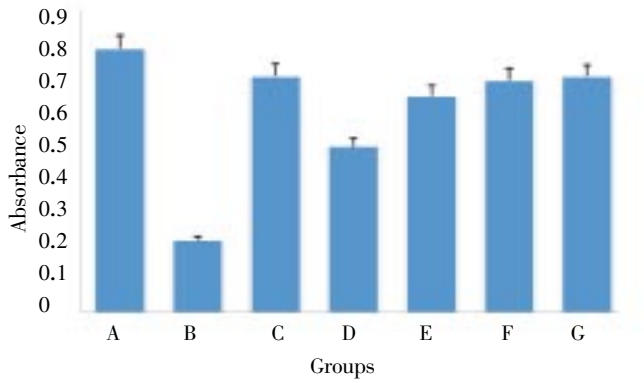



**Figure 9.** Histogram showing GSH levels of ethanolic and aqueous extract of Sesbenia grandiflora flower on CCl4 induced hepatotoxicity on rats. [A= negative control; B= positive control (CCl4 challenge); C= Standard (Silymarin + CCl4 challenge); D= EEFSG (250 mg/kg p.o.) + CCl4 challenge; E= EEFSG (500 mg/kg p.o.) + CCl4 challenge; F= AEFSG (250 mg/kg p.o.) + CCl4 challenge; G= AEFSG (500 mg/kg p.o.) + CCl4 challenge].

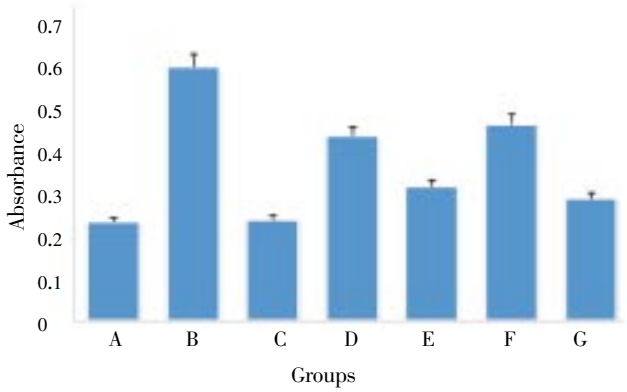



**Figure 10.** Histogram showing Lipid peroxidase levels of ethanolic and aqueous extract of Sesbenia grandiflora flower on CCl4 induced hepatotoxicity on rats. [A= negative control; B= positive control (CCl4 challenge); C= Standard (Silymarin + CCl4 challenge); D= EEFSG (250 mg/kg p.o.) + CCl4 challenge; E= EEFSG (500 mg/kg p.o.) + CCl4 challenge; F= AEFSG (250 mg/kg p.o.) + CCl4 challenge; G= AEFSG (500 mg/kg p.o.) + CCl4 challenge]

lymphocytes and proliferation of kuffer cells. Congestion of liver sinusoids. Protective effect of test extract was confirmed by histopathological examination of liver section. Administration of test extract at the dose of 250 mg/kg and 500mg/kg that is showed a significant improvement of the hepatic architecture and areas of Kuffer cell proliferation and sinusoid appeared normal.

## 4. Discussion

Hepatic system of an organism is involved in metabolic activities of it. In this process it is exposed to various challenges and hence, hepatic system is not only evolved to perform its function but also to protect itself to various challenges like exposure to antibiotics/xenobiotics, chemicals etc. Liver is such an organ that its physiological role and its self protective mechanism are well developed and orchestrated. Inspite of such balanced internal mileiu, hepatic aberration, damage and necrosis commonly occurring due to over exposure to hepatotoxic causes to such an extent that it over powers the mechanism. Inspite of ultra modern advances in medical science, pharmaco-therapeutics treatment with synthetic drugs is not yet realized. However there are several herbs and herbal formulation which are found to be/claimed for treating hepatic disorders.

In the present study one of the local available plant *Sesbania grandiflora* (Linn) were selected on the basis of native practitioners and available phytochemical profile of the plant. In the present study the extract was subjected phytochemical screening, it is observed that the extract showed a marked presence of carbohydrate, flavanoids, tannins, glycoside, and steroids. Liver damage induced by CCl4 are commonly used models for the screening of hepatoprotective drugs [26-27].

Hepatic damage induced by CCl4 causes instability of liver cell metabolism, including triacylglycerol accumulation, change in serum transaminases activities and depression of protein synthesis, these are the indicators of liver damage [28-29]. Present study demonstrate a significant increase in the activity of SGPT, SGOT, ALP and serum bilirubin levels, that indicates increased permeability, severe damage to tissue membrane and necrosis of hepatocytes after exposing with CCl4. Administration of extract at different dose levels prevented the rise in the level of above serum enzymes, alkaline phosphatase and serum bilirubin levels. It indicate a possible stabilization of plasma membrane as well as repair of hepatic tissue damage caused by CCl4 exposure. Hepatoprotective activity is associated with antioxidant activity, since it is free radical mediated damage [30]. Elevated levels of MDA reflects an enhanced lipid peroxidation leading to tissue damage and failure of antioxidant defence mechanisms to prevent formation of excessive free redicals [31]. The enzymatic antioxidant defence system are the natural protecter against lipid peroxidation. SOD, CAT enzyme are impostant scavengers of superoxide ions and hydrogen peroxide. These enzyme prevent generation of hydroxylradical and protect the cellular constituent from oxidative damage [32].

We conclude that a reductionin the activities of antioxidant enzyme (SOD and CAT) in CCl4 treated group as compared to the control group. After extract administration, the above changes were reversed as compared to the group of rats which received only CCl4. Those finding showed extract can scavenge reactive free radicals that could lead to the decrease in severity of damage in the liver.

The probable mechanism by which *Sesbania grandiflora* (Linn) exerts its protective action against CCl4 induced hepatotoxicities could be the stimulation of hepatic regeneration through an improved synthesis of proteins, or with interference with the liberation of microsomal activation to toxicants.

Moreover it is reported that flavanoids and tannins were reported to posses' variety of pharmacological activity including hepatoprotective activity. In the present investigation also preliminary phytochemical investigation on *Sesbania grandiflora* (Linn) gave positive tests for flavanoids and tannins this could be the reason for significant hepatoprotective property of the test extract.

## Conflict of interest statement

We declare that we have no conflict of interest.

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