HEPATOPROTECTIVE AND ANTI-HEPATOTOXIC ACTIVITIES OF AQUEOUS LEAF EXTRACT OF *TACAZZEA BARTERI* AGAINST CARBON TETRACHLORIDE INDUCED HEPATOTOXICITY IN ALBINO RATS

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ABSTRACT

The hepatoprotective and anti hepatotoxic effect of *Tacazzea barteri* was evaluated using carbon tetrachloride (CCL₄) induced hepatotoxicity. For the hepatoprotective study, the animals were treated with 200, 400 and 600 mg/kg of *T. barteri* extract for 7 days prior to the intra peritoneal injection of 1 ml/kg of carbon tetrachloride while for the anti hepatotoxic study; 1 ml/kg of carbon tetrachloride was administered via intra peritoneal route before treatment (2 days). Blood samples were collected from the animals before and after treatment and serum biochemical parameters (AST, ALT, ALP, Bilirubin and Albumin) were determined. The extract showed significant ($p < 0.01$) reduction in the carbon tetrachloride induced elevation of ALT, AST, ALP and Bilirubin and a significant ($p < 0.01$) increase in carbon tetrachloride induced suppression of Albumin induction in both models. Histological photomicrographs of liver sections of the untreated groups showed high cellular damage while no visible histological damages were noticed in the extract treated groups in the hepatoprotective model while mild cellular damage was noticed in the anti-hepatotoxic model. The extract not only showed protection of the liver cells against CCL₄ hepatotoxin but also showed reversal of hepatic damage cause by CCL₄. This study justifies the folkloric use of *T. barteri* in the treatment of liver diseases.

Keyword: *Tacazzea barteri*, Hepatoprotective, Anti hepatotoxic, Carbon tetrachloride.

INTRODUCTION

The liver plays a vital role in the maintenance and regulation of homeostasis in the body¹. Some of these functions include carbohydrate, protein and fat metabolism, bile secretion, urea formation, excretion and detoxification of wastes and foreign chemicals². A healthy liver is therefore crucial for human growth, development, fertility and overall health and well-being. Unfortunately, the prominent position of the liver in the circulatory system makes it vulnerable to potential toxicants at their highest concentrations³. Also, being the primary organ for biotransformation and detoxification, it receives the brunt effects of toxic reactive chemicals and their metabolites⁴. These factors make the liver unique as a target organ for toxicity. Liver diseases remain one of the serious health problems⁵. Drug treatment of most liver diseases is also problematic owing to their side effects that tend to worsen the disease condition in most cases⁶. The efficacy, safety and minimal side effects of natural products have led to increased interest of researchers in investigating their pharmacological bases and optimization of their effectiveness through standardization and quality control⁷. To this effect, there have been over reliance on natural products for the treatment of liver diseases⁶. The leaf extract of *Tacazzea barteri*, is very popular among traditional herbal practitioners for the treatment of liver diseases, yet no scientific studies have been done to authenticate this folkloric claim. We therefore investigated the hepatoprotective and hepatotoxic effect of the aqueous extract of *T. barteri* using CCL₄ induced hepatotoxicity model.

MATERIALS AND METHOD

Plant collection and identification

The leaves of *Tacazzea barteri* were collected on December, 2012 from Orlu in Imo State, identified and authenticated by

a taxonomist, Mr. Ugwuozo of the Department of Botany, Nnamdi Azikiwe University, Awka, Nigeria. The leaves were cleaned; air dried at room temperature and pulverized. A 180 g portion of the powdered leaf was cold macerated in 1.5 L of distilled water for 48 h with intermittent shaking and filtered using white muslin cloth. The filtrate was concentrated by lyophilization at -40°C using freeze dryer and stored at 2 – 8°C in the refrigerator. Aqueous extract was chosen to mimic ethno pharmacological (folkloric) method of preparation considering the likely contributions of other chemical solvents to liver toxicity.

Animals

Swiss albino mice (25 – 28 g) and rats (230 - 240 g) were obtained from the animal house of the Department of Pharmacology and Toxicology, Faculty of Pharmaceutical Sciences, Nnamdi Azikiwe University, Awka. They were maintained in standard animal house conditions of 12 hours light, 25 – 30°C, cross ventilation and were allowed free access to food and water *ad libitum*. All the animal experiment were conducted in compliance with the principle of laboratory animal care (NIH publication No. 85-23, reversed 1985) as approved by the institutional ethical committee on the use of laboratory animals (ref. 087/2013/Bpharm/Phcol).

Phytochemical tests

The phytochemical analysis of the leave extract was carried out using standard methods^{8,9}.

Acute Toxicity Studies

This was done using Lorke's method¹⁰. A total of 26 animals (mice and rats in equal ratio) were used for the study. The study was divided in two phases. In the first phase nine

animals each were used. They were grouped into three groups of three animals each and were given oral administration of 10, 100 and 1000 mg/kg of the extract. The animals were observed for signs and symptoms of toxicity and mortality over a period of 24 h. In the second phase, four mice were given oral administered of 2000, 3000, 4000 and 5000 mg/kg of the extract respectively. The animals were also observed for 24 hours post treatment for obvious toxicity and mortality.

Hepatoprotective Activity

The ability of the extract to protect the liver against CCL₄ induced hepatotoxicity was evaluated using 20 albino rats. The animals were divided into four groups of five animals each. Groups 1 – 3 received oral administration of 200, 400 and 600 mg/kg of the extract respectively for 7 days while group 4 served as the control and received 0.4 ml of normal saline. Intra peritoneal injection of 1 ml/kg of CCL₄ in 10 % tween 80 was given to the animals and blood samples collected through retro-orbital plexus 24 hours later. The blood samples were allowed to coagulate for 30 minutes and the clear serum was separated by centrifuging at 3000 rpm for 10 minutes and was then used for analysis of biochemical hepatic markers- aspartate aminotransferase (AST)¹¹, alanine aminotransferase (ALT)¹¹, alkaline phosphatase (ALP)¹², total bilirubin¹³ and albumin¹⁴. Histopathological studies

were done on liver isolates from different groups of the animals. Pre-treatment blood samples were also collected for the analysis of biochemical hepatic markers.

Anti Hepatotoxic Activity

To evaluate the recovery rate of the liver from hepatotoxic effect CCL₄ on albino rats, liver damage was induced using 1 ml/kg of CCL₄ in 10 % tween 80 prior to administration of the extract. Twenty-four hours after CCL₄ administration, blood samples were collected from the animals through retro-orbital plexus and serum liver biochemical parameters carried out to ascertain induction of liver damage. The animals in groups 1 – 3 were then given oral administration of 200, 400 and 600 mg/kg of the extract for 2 days while the control group (group 4) received 0.4 ml of normal saline. The extent of recovery was evaluated using biochemical hepatic markers as discussed above. Histological examination of the liver from each group was also evaluated.

Statistical Analysis

Data were expressed as the Mean $\pm$ SEM. Means were analyzed using a one-way analysis of variance (ANOVA) and Student's t-test. All the statistical analyses were done using SPSS, Version 17.0. P values < 0.05 and 0.01 were used to quantify levels of significance.

Table 1: Phytochemical analysis of aqueous leaf extract of *T. barteri*

Phytocompounds	Degree of Occurrence
Alkaloids	++
Flavonoids	+++
Terpenoids	+++
Tannins	++
Saponins	++
Glycosides	+++

++ = moderately Abundant +++ = Abundant

Table 2: Acute toxicity study of aqueous leaf extract of *T. Barteri*

Phases	Dose (mg/kg)	Mortality
Phase 1	10	0/3
	100	0/3
	1000	0/3
Phase 2	2000	0/3
	4000	0/3
	5000	0/3

Table 3: The effect of aqueous leaf extract of *T. barteri* on serum Alkaline Transaminase

Treatment	Dose (mg/kg)	Mean Serum ALT (U/I) $\pm$ SEM		Percentage Reduction (%)
		Pre-treatment	Post-treatment	
N. saline	0.4 ml	28.0 $\pm$ 0.2	100.0 $\pm$ 0.5	-
Extract	200	26.5 $\pm$ 0.4	**78.5 $\pm$ 0.3	21.5
Extract	400	28.0 $\pm$ 0.2	**58.0 $\pm$ 0.6	42
Extract	600	31.5 $\pm$ 0.2	**45.5 $\pm$ 0.4	54.5

N = 5; ** p < 0.01 compound with control

Table 4: The effect of aqueous extract of *T. barteri* on serum Aspartate Transaminase

Treatment	Dose (mg/kg)	Mean Serum AST (U/I) $\pm$ SEM		Percentage Reduction (%)
		Pre-treatment	Post-treatment	
N. saline	0.4 ml	54.0 $\pm$ 0.8	94.0 $\pm$ 0.8	-
Extract	200	52.0 $\pm$ 0.7	*72.0 $\pm$ 0.4	23.4
Extract	400	53.0 $\pm$ 0.8	**33.5 $\pm$ 0.6	64.4
Extract	600	53.0 $\pm$ 0.5	**31.0 $\pm$ 0.6	67.0

N = 5; *p < 0.05; **p < 0.01 compared with control

Table 5: The effect of aqueous extract of *T. barteri* on serum Alkaline Phosphate

Treatment	Dose (mg/kg)	Mean Serum ALP (IU/I) $\pm$ SEM		Percentage Reduction (%)
		Pre-treatment	Post-treatment	
N. saline	0.4 ml	43.3 $\pm$ 0.5	100.0 $\pm$ 0.8	-
Extract	200	46.7 $\pm$ 0.6	*86.7 $\pm$ 0.5	13.3
Extract	400	43.3 $\pm$ 0.5	**73.3 $\pm$ 0.6	26.7
Extract	600	43.3 $\pm$ 0.5	**66.7 $\pm$ 0.5	33.3

N = 5; *p < 0.05; **p < 0.01 compared with control

Table 6: The effect of aqueous leaf extract *T. barteri* on serum Albumin

Treatment	Dose (mg/kg)	Mean Serum Albumin (g/dl) $\pm$ SEM		Percentage Reduction (%)
		Pre-treatment	Post-treatment	
N. saline	0.4 ml	3.4 $\pm$ 0.4	1.6 $\pm$ 0.3	52.9
Extract	200	3.1 $\pm$ 0.3	**2.4 $\pm$ 0.6	23.9
Extract	400	3.4 $\pm$ 0.3	**2.8 $\pm$ 0.5	17.6
Extract	600	3.4 $\pm$ 0.2	**2.8 $\pm$ 0.4	17.6

N = 5; **p < 0.01 compared with pre treatment

Table 7: The effect of aqueous leaf extract of *T. barteri* on serum Total Bilirubin

Treatment	Dose (mg/kg)	Mean Serum Total BIL (mg/dl) $\pm$ SEM		Percentage Reduction (%)
		Pre-treatment	Post-treatment	
N. saline	0.4 ml	3.2 $\pm$ 0.7	1.9 $\pm$ 0.4	-
Extract	200	0.42 $\pm$ 0.5	*1.6 $\pm$ 0.2	15.8
Extract	400	0.42 $\pm$ 0.8	**1.4 $\pm$ 0.4	26.3
Extract	600	0.32 $\pm$ 0.6	**1.1 $\pm$ 0.8	42.1

N = 5; *p < 0.05; **p < 0.01 compared with control

Table 8: The effect of aqueous leaf extract of *T. barteri* on serum Alkaline Transaminase

Treatment	Dose (mg/kg)	Mean Serum ALT (U/I) $\pm$ SEM		Percentage Reduction (%)
		Pre-treatment	Post-treatment	
N. saline	0.4 ml	34.0 $\pm$ 0.2	78.5 $\pm$ 0.4	-
Extract	200	28.0 $\pm$ 0.2	**49.5 $\pm$ 0.3	37.6
Extract	400	29.0 $\pm$ 0.4	**41.0 $\pm$ 0.7	47.8
Extract	600	28.0 $\pm$ 0.5	**39.0 $\pm$ 0.6	50.3

N = 5; **p < 0.01 compared with control

Table 9: The effect of aqueous extract of *T. barteri* on serum Aspartate Transaminase

Treatment	Dose (mg/kg)	Mean Serum AST (U/I) $\pm$ SEM		Percentage Reduction (%)
		Pre-treatment	Post-treatment	
N. saline	0.4 ml	59.0 $\pm$ 0.9	134.0 $\pm$ 0.8	-
Extract	200	59.0 $\pm$ 0.6	**72.0 $\pm$ 0.6	46.3
Extract	400	52.0 $\pm$ 0.8	**66.0 $\pm$ 0.6	50.8
Extract	600	54.0 $\pm$ 0.6	**59.0 $\pm$ 0.5	56.0

N = 5; *p < 0.05; **p < 0.01 compared with control

Table 10: The effect of aqueous leaf extract of *T. barteri* on serum Alkaline Phosphatase

Treatment	Dose (mg/kg)	Mean Serum ALP (U/I) $\pm$ SEM		Percentage Reduction (%)
		Pre-treatment	Post-treatment	
N. saline	0.4 ml	42.1 $\pm$ 0.6	76.3 $\pm$ 0.8	-
Extract	200	39.5 $\pm$ 0.8	*63.2 $\pm$ 0.6	17.2
Extract	400	36.8 $\pm$ 0.5	**57.9 $\pm$ 0.6	24.2
Extract	600	34.2 $\pm$ 0.6	**57.9 $\pm$ 0.5	24.2

N = 5; *p < 0.05; **p < 0.01 compared with control

Table 11: The effect of aqueous leaf extract *T. barteri* on serum Albumin

Treatment	Dose (mg/kg)	Mean Serum Albumin (g/dl) $\pm$ SEM		Percentage Reduction (%)
		Pre-treatment	Post-treatment	
N. saline	0.4 ml	4.4 $\pm$ 0.6	3.1 $\pm$ 0.5	30.5
Extract	200	4.6 $\pm$ 0.4	3.6 $\pm$ 0.6	20.7
Extract	400	4.8 $\pm$ 0.5	*3.8 $\pm$ 0.4	19.9
Extract	600	4.5 $\pm$ 0.4	**4.4 $\pm$ 0.4	2.6

N = 5; *p < 0.05; **p < 0.01 compared with pre treatment

Table 12: The effect of aqueous extract of *T. barteri* on serum Total Bilirubin

Treatment	Dose (mg/kg)	Mean Serum Total BIL (mg/dl) $\pm$ SEM		Percentage Reduction (%)
		Pre-treatment	Post-treatment	
N. saline	0.4 ml	0.32 $\pm$ 0.4	2.7 $\pm$ 0.4	-
Extract	200	0.32 $\pm$ 0.4	**1.8 $\pm$ 0.2	21.9
Extract	400	0.32 $\pm$ 0.6	**1.2 $\pm$ 0.4	39.3
Extract	600	0.32 $\pm$ 0.4	**0.4 $\pm$ 0.8	60.7

N = 5; **p < 0.01 compared with control

Figure 1: Photomicrograph of liver section from hepato-protective control group showing portal oedema and cellular infiltration (portal hepatitis). See arrows. Hand E X40

Figure 2: Photomicrograph of liver section from the 200 mg/kg treated group of hepato-protective model. No remarkable change is seen. H and E X40

Figure 3: Photomicrograph of liver section from the 400 mg/kg treated group of hepato-protective model. No remarkable change is seen. H and E X40

Figure 4: Photomicrograph of liver section from the 600 mg/kg treated group of hepato-protective model. No remarkable change is seen. Hand E X40

Figure 5: Photomicrograph of liver section from the anti-hepatotoxic control group showing high cellular infiltration of portal area (P). Hand E X40

Figure 6: Photomicrograph of liver section from the 200 mg/kg treated group of anti-hepatotoxic model showing portal oedema and mild cellular infiltration (portal hepatitis). See arrows. H and E X40

Figure 7: Photomicrograph of liver section from the 400mg/kg treated group of anti-hepatotoxic model showing mild cellular infiltration around the bile duct (arrow). H and E X40

Figure 8: Photomicrograph of liver section from the 600mg/kg treated group of anti-hepatotoxic model. No visible histological change is seen H and E X40

RESULTS

Phytochemical Analysis

The phytochemicals present in the aqueous extract of *T. barteri* is as shown in Table 1. All the phytochemicals were present in appreciable quantities.

Acute Toxicity Study

The extract did not produce any obvious toxic symptoms or mortality up to 5000 mg/kg body weight in both mice and rats species (Table 2).

Hepatoprotective Activity

The results of hepatoprotective effects of aqueous leaf extract of *T. barteri* against CCL₄ induced hepato-toxicity were as shown in Tables 3 – 7. The significant acute hepato cellular damage, impaired hepatic function and biliary obstruction in the CCL₄ treated rats were indicated by the elevated levels of ALT, AST, ALP and Bilirubin with decrease in Albumin level. Percentage reduction in elevated serum ALT, AST, ALP and Bilirubin were calculated against control post-treatment while that of Albumin was calculated against pre-treatments.

Anti-hepatotoxic Activity

The results of effect of aqueous extract of *T. barteri* on CCL₄ induced hepato-toxicity were as shown in Tables 8 – 12. There were elevated ALT, AST, ALP, Albumin and Bilirubin in CCL₄ treated rats. Significant ($p < 0.01$) reduction in elevated ALT, AST, ALP and Bilirubin were observed in the extract treated groups compared with control while significant ($p < 0.01$) elevation in reduced Albumin was observed as well in the extract treated groups compared with control.

Histopathological Examination

Histo-photomicrographs of liver sections of the untreated groups of hepatoprotective and anti hepatotoxic models (Figure 1 and 5) showed disarrangements of normal hepatic cells with high cellular infiltration and portal oedema. No visible histological changes were noticed in groups treated with 200 mg, 400 mg and 600 mg of the aqueous extract in the hepato-protective model (Figure 2 - 4). In anti-hepatotoxic model, mild cellular infiltrations were noticed in groups treated with 200 mg and 400 mg of the extract (Figure 6 and 7) while group treated with 600 mg of the extract showed no remarkable change (Figure 8).

DISCUSSION

Phytochemical analysis of the aqueous leaf extract of *T. barteri* showed that Phenolic compounds (flavonoids, terpenoids) were present in abundant quantities. Phenolic compounds have long been established to exhibit hepato-friendly effects acting mainly through anti-oxidation and free radical scavenging¹⁵. Alkaloids, saponins and tannins have as well been isolated from natural products known to exhibit hepato-protective effect. No mortality and obvious toxic symptoms were observed from the acute toxicity study, an indication that the plant may be safe when taken orally. Amino transferases (ALT and AST) and Alkaline Phosphatase are present in high concentration in the liver¹⁶. Their estimation in the serum is a useful quantitative marker for the extent and type of hepatocellular damage¹⁷. The elevation of these marker liver enzymes in the CCL₄ treated rats is attributed to damage in structural integrity of the liver because these enzymes are cytoplasmic in location and increase in circulation following cellular damages¹⁸ indicate development of hepatotoxicity. The elevation of these marker enzymes is consistent with other works on CCL₄ induced hepatotoxicity¹⁹⁻²¹. The tendency of these enzymes to return to near normalcy in the extract treated groups is a clear manifestation of hepato-protective and anti-hepatotoxic effects of the extract. Also, the significant ($p < 0.01$) reduction in ALP and bilirubin levels in the extract treated groups compared with control suggests the stability of the biliary function after injury with CCL₄²². The levels of albumin were reduced in the CCL₄ treated rats. The reduction may be attributed to damage by CCL₄ affecting endoplasmic reticulum (ER) resulting in reduced protein synthesis²³. Serum albumin levels were raised in extract treated animals in both hepatoprotective and Antihepatotoxic models suggesting the stabilization of ER leading to improved protein synthesis. The histological photomicrograph is in agreement with the result obtained from the biochemical markers. There were no visible histological changes in the liver of the extract treated groups compared with the control group that had high cellular infiltration, an evidence of the hepato-protective effect of the extract. Also, the improved repair in injury inflicted by CCL₄ by the extract is an indication of anti-hepatotoxic effect of the extract.

CONCLUSION

Aqueous extract of *T. barteri* not only showed hepato-protective effect against CCL₄ but also anti-hepatotoxic

effect. This study justifies the folkloric claim of *T. barteri* use in the treatment of liver disorders.

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