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Evaluation of cardioprotective effects of *Parkia biglobosa* (Jacq. Benth) Mimosaceae stem bark

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ABSTRACT

Parkia biglobosa is one of the main medicinal plants used in traditional medicine to treat cardiovascular diseases in Africa. We investigated cardioprotective effects of stem bark hydro-alcoholic extract (HAE) of *P. biglobosa* in isoproterenol (ISO) induced myocardial infarction (MI). Four groups of five Wistar rats of each were used. Group I, control group, received per os (p.o) saline solution (5ml kg^{-1} b.w.) as vehicle daily for 15 days and was injected subcutaneously with the vehicle (5ml kg^{-1} b.w.) at an interval of 24 hours, on day 14 and 15. Group II referred to as infarcted rats, received vehicle p.o and was injected subcutaneously with isoproterenol (125 mg kg^{-1} b.w.) as in group I. Groups III and IV were pre-treated p.o with HAE (90 and 60 mg kg^{-1} b.w. respectively) daily for 15 days and was injected subcutaneously Isoproterenol (125 mg kg^{-1} b.w.). ISO has induced MI, characterized by significant increased ($p < 0.001$) of MDA, biochemical alterations, heart hypertrophy and histological changes in infarcted rats. HAE, prevented significantly ($p < 0.001$) induction of all these anomalies in pre-treated rats. These results suggested cardioprotective effects of *P.biglobosa* against ISO induced MI.

INTRODUCTION

Myocardial infarction is one of the main causes of death from cardiovascular disease. In 2002, World Health Organization (WHO) estimated that 7.2 millions of deaths worldwide were from ischemic heart (Sakande**,** 2009). To treat these affections, conventional medicine uses some well-known drugs. In traditional practice, many medicinal plants like *Parkia biglobosa* (Jacq) Benth are widely used in Africa to treat various cardiovascular diseases as hypertension, cardiac failure and cardiac disturbances. *P. biglobsa* (Mimosaceae), is a widespread savanna tree over the sahelo-sudanian region (Adjanohoun *et al.*, 1991) and largely prescribed in traditionalmedicine because of it multiple medicinal virtues. It was also reported that, the stem bark of this plant is successfully used for the treatment of: infectious, dental caries, pneumonia, bronchitis, diarrhea, wounds, otitis, dermatosis, bilharziosis, leprosies, ankylosis tracheitis, conjunctivitis, chest pains, jaundice, hemorrhoids and hypertension (Millogo-Kone *et al*., 2008).

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Hence, previous ethnopharmacological investigations reported many biological activities of *P. biglobosa*. Adi *et al.* (2006) have shown that, the fermented seeds of *P. biglobosa* had anti-inflammatory and antimicrobial activities. Bonnah *et al*. (1998) reported that hydro alcoholic and aqueous extracts of the seed had hypotensive and cardiohinibitory activity. Kouadio *et al*. (2000) suggested that, the hexane extract from the bark of *P. biglobosa* had anti inflammatory and analgesic effects. Azuzu *et al.* (2003) reported that methanolic extract of *P. biglobosa* showed significant protection against the neurotoxic, hemotoxic and cytotoxic effect poisonous snake venom. Stem bark chemical studies identified long chain ester of trans-ferulic acid, a mixture of long chain cis-ferulates and different kinds of catechins (Tringali *et al.*, 2000). It's well-known that catechins and ferulates are antioxidants, and their antioxidant proprieties may be responsible for some of the medicinal effects seen for this plant. The present study aimed to find effects of *P. biglobosa* stem bark on myocardial infarction induced by isoproterenol in rats, in order to justified it use in traditional medicine.

MATERIALS AND METHODS

Plant material

Stem barks of *Parkia biglobosa* was collected in Kara region in the north of Togo in December 2009 and authenticated by the Department of Botany of Faculty of Sciences of university of Lomé, were a voucher specimen was deposited under No: Togo12674. The material was washed and cut in to small pieces and shade-dried at 22°C for one week.

Animals

All the experiments were carried out with male albinos Wistar rats, weighing b*et*ween 150-200g and obtained from the Animal House of Faculty of Sciences of University of Lomé. They were housed in polypropylene cages (47 x 34 x 20cm) under standard conditions. The rats were feed on standard diet and water was provided ad libitum.

Extraction preparation

450 g of stem bark were extracted with 1500 ml of ethanol-water (1:1, v/v) by maceration for 72 hours. The supernatant was filtered using filter paper. The solvent was evaporated under vacuum at 45° C with evaporator, (BUCHI Model R- 210) to obtain a concentrated hydro alcoholic extract (EHA) which is water soluble. The yield of the dry extract was 14.03%.

Experimental design

The experimental designs were carried out according the method of Ponnian *et al*. (2008) modified. The rats were divided in to 4 groups of 6 animals: Group I: negative control group, received 9‰ of saline solution (5 mg kg^{-1} body weight) p.o daily for 15 days and was injected subcutaneously with the same solution (2 ml kg^{-1} body weight) at an interval of 24 hours on day 14 and 15. Group II: referred to as infarcted group or positive control group, received p.o saline solution (5 mg kg^{-1} body weight) daily for 15 days and was injected subcutaneously with Isoproterenol (Sigma, St. Louis, MO. USA) (125mg kg^{-1} body weight) dissolved in saline solution at an interval of 24 hours on day 14 and 15).

Group III and IV: received p.o HAE (90mg kg^{-1} and 60 mg kg^{-1} body weight respectively), daily for 15 days and was injected subcutaneously with isoproterenol at an interval of 24 hours on day 14 and 15. On day 15, 12 hours after the isoproterenol injection, all the rats were weighed and anesthetized using anesthetic ether before blood was collected via retro-orbital puncture and centrifuged at 2500 rpm for 10 minutes with electric centrifuge (SPN- 400 Shimadzu Scientific corporation, Tokyo, Japan). Then serum was separated and used for estimation of various biochemical parameters. After blood collection, all the rats were sacrificed by cervical dislocation. The heart was dissected out, washed immediately in ice-chilled saline, blotted, weighed and conserved at 0°C for lipid and Malondialdehyde (MDA) assay. Heart from 2 rats in each of the following groups: negative control group, positive control group and the pretreated with HAE (90 mg kg^{-1}) group were randomly selected for histopathology examinations.

Estimation of hart relative weight

In each group, heart relative weight was determined by the equations:

Hart relative weight = hart weight / animal body weight x 100 (Arvinkudkmar *et al*., 2009)

Serum parameters estimation

Total cholesterol, triglycerides, high density lipoprotein (HDL) were measured using an automated spectrophotometric (Genitek Biochemistry analyzer, Model: GB 300 PLUS.Version 3.8, Germany) and Labkits regents (Chmlex, D. A. Barcelona). The serum activities of lactate dehydrogenase (LDH) and creatin kinase (CK) were estimated by using Standard SPRINREACT kits regents, (S. A. U. Centra Coloma).

Serum low density lipoproteins (LDL) and very low density lipoproteins (VLDL) were calculated with Fridewald formula (Friedewal *et al*., 1972):

VLDL = triglycerides/5; LDL = total cholesterol – (HDL cholesterol + VLDL cholesterol) respectively.

Tissue lipid estimation

100 mg of heart tissue was homogenized in 2.5 ml of 0.1M Tris-HCl (Ph7.4) buffer solution. The homogenate was centrifuged at 3000 rpm for 10 minutes and the supernatant was used for the estimation tissue total cholesterol and triglycerides using the same assay for estimation of serum lipid.

Lipid peroxidation (MDA) assay

150 mg of heart tissue was homogenized in 1ml of 0.1M Tris-HCl (Ph7.4) buffer solution. Lipid peroxides were estimated by measurement of thiobarbituric acid reactive substances in the homogenate (Satoch, 1978). Color absorbance was measured using an ultraviolet-visible spectrophotometer (Model spectra Max Molecular Service, Sunyal Corporation, California. USA).

Histological studies

Histological evaluation was performed on the apical portion of the heart tissue. Fresh heart tissue were excised and then fixed in 10% formalin for 24 hours. The fixative was removed by washing through running tape water overnight. After dehydration through graded series of alcohols (70°, 90°, 95° and 100°) the tissues were cleaned in methyl benzoate, embedded in paraffin wax. Section were cut into 5µm thickness and stained with hematoxylin and eosin. The sections were mounted and observed under light microscope with magnification of 100X for histological changes.

Statistical Analysis

The result was express as mean ± standard deviation for six rats for each group. The statistical analysis was performed using Graph Pad Prism software version 4.00. Analysis was made

using one way analysis (ANOVA) followed by Duncan's multiple comparison tests. A P value < 0.05 was considered statistically significant.

RESULTS AND DISCUSSION

Previous studies after myocardial infarction in human reported: cardiac hypertrophy (Vlasov *et al*., 2004), increased of lipid peroxidation (Kasap *et al*., 2007; Moselhy *et al*., 2004 and Aznar *et al*., 1983), elevated of cardiac enzymes activity in serum (Damourou *et al*., 2005) and high concentration of serum lipids (Pasupathi *et al*., 2009). Experimental studies on rats (Rajadurai *et al*., 2005; Arvindkumar *et al*., 2009; Panda *et al*., 2009; Ponnian *et al*., 2008) indicated that, all these pathophysiological abnormalities observed in humans after an acute myocardial infarction were similar to abnormalities exhibited in rats after isoproterenol administration. Isoproterenol (ISO) is a synthetic catecholamine derivative of noradrenaline which does not exist in the body (Rang *et al*., 2003; Rajadurai *et al*., 2005). It is a β- adrenergic agonist. Administration of ISO causes oxidative stress mainly via the β-1 adrenergic receptor stimulation (Arvindkumar *et al*., 2009). Stimulation of these receptors β-1 especially present in the heart muscle (Rang *et al*., 2003) is responsible for the acute positive inotropic and chronotropic effects on heart, causing an imbalance between energy intake by the blood flow and increased oxygen demand (Prabhu *et al*., 2006) leading to ischemia. If ischemia is severe and prolonged, it can induce cardiac cells death (necrosis or myocardial infarction), and the release of their enzyme content in the extra cellular medium. Ischemic muscle quickly generates the radical species derived from oxygen (ROS) when the capacity of cellular antioxidant enzymes decreased (Arvindkumar *et al*., 2009) Free radical-mediated lipid peroxidation and consequent changes in membrane permeability are primary factor of ISO cardio-toxicity. In addition it causes lipolysis in the heart, and promotes lipolysis in the myocardium resulting in the release of cardiac lipids bloodstream (Ponnian *et al*., 2008). In the present sturdy, heart relative weight (6.97±0.44 %) in positive control group rats was higher significantly (P < 0.001), compared with negative control rats (3.73±0.09%). Heart relative weight increase identifies heart hypertrophy (Table 1). Hypertrophy of the heart is an adaptive response to any intrinsic or extrinsic stimuli or during the remodelling that occurs in the evolution of ischemic heart disease (Choukroun *et al*., 2002; Arvindkumar *et al*., 2009). Hypertrophy observed is a compensatory response to necrosis of the heart muscle, caused by the severe stress of the heart induced by administration of ISO (Ennis *et al*., 2003).

Hart relative weight increase may be due to the increase in overall protein biosynthesis during development of hypertrophy accompanied by oedema or overexpression of genes encoding proteins involved in the contractile unit (Heather *et al*., 2009; Choukroun *et al*., 2002). The elevation of cytoplasmic calcium during MI could also be a factor responsible for the activation of intracellular signals governing the hypertrophic response of cardiac cells (Choukroun *et al*., 2002).

The induction of cardiac hypertrophy and cardiac toxicity is a standard model used to study the beneficial effect of many products on cardiac function (Choulhary *et al*., 2006; Rathore *et al*., 1998). HAE pre-treated groups (groups III and IV), showed significantly (P < 0.001) lower heart relative weight when compared to positive control group (Table 1). The extract was therefore able to protect heart cells against stress caused by ISO. LDH (4301.00±25.41 UI/L) and CK (1576.83±72.69 UI/L) activities in positive control group were significantly increased (p < 0.001) by 138% and 160% respectively when compared to negative control group (1802.33±10.31 UI/L and 609.83±44.08 UI/L respectively). HAE pre-treatment decreased significantly (p < 0.001) their activities compared to infracted rats group (Table 1).

Table. 1: Effect of hydro alcoholic extract on heart relative weight, LDH and CK.

Groups	Heart relative weight (%)	LDH (UI/L)	CK (UI/L)
Control	3.73±0.09	1802.33±10.31	609.83±44.08
ISO (125 mg kg^{-1})	6.97±0.44***	4301.00±25.41***	1576.83±72.69***
HAE (60mg kg^{-1})+ ISO	4.78±0.02# # #	2623.00±29.56 # # #	1300.33±60.59$^{\Leftrightarrow}$
HAE (90mg kg^{-1})+ ISO	4.41±0.11# # #	1773.66 ±14.12 # # #	870.33±48.79 # # #

HAE was administered to rats daily for period of 15 days. Values are mean ± S .D for 6 rats in each group. *** P < 0.001 *vs* control, # # # P < 0.001and $^{\Leftrightarrow}$p < 0.05 *vs* ISO

Table. 2: Effect of HAE pre-treatment on isoproterenol induced changes in serum lipid **.**

Groups	Cholesterol (mg/dl)	HDL (mg/dl)	LDL (mg/dl)	VLDL (mg/dl)	Triglycerides (mg/dl)
Control	72.43±4.15	40.31±0.64	21.69±5.05	8.798±0.73	43.99±3.65
ISO (125 mg kg^{-1})	139.31±14.57^{a}	30.48±1.174^{a}	93.13±14.29	15.64±1.40^{a}	79.69±6.53^{a}
HAE (60mg kg^{-1})+ISO	99.88±6.01^{c}	33.44±1.44	55.51±6.19	11.52±0.32^{c}	57.45±1.72^{b}
HAE (90mg kg^{-1})+ISO	77.48±6.34^{b}	37.71±1.9^{c}	29.62±7.65^{b}	9.71±1.03^{b}	48.57±5.15^{b}

HAE was administered to rats daily for period of 15 days. Values are mean ± S .D for 6 rats in each group. ^{a}p < 0.001 *vs* control, ^{b}p < 0.001 *vs* ISO , ^{c} p < 0.05 vs ISO

Table. 3: Effect of EHA on total cholesterol, triglycerides and MDA in heart tissue.

Groups	Total cholesterol (mg/g)	Triglycerides (mg/g)	MDA (n M /L)
Control	3.72±0.72	17.23±0.92	369.50±26.60
ISO (125 mg kg^{-1})	6.32± 3.60* *	32.31±3.60**	742.83±38.29***
HAE (60mg kg^{-1})+ ISO	4.04±0.60$^{\#}$	25.14±2.69	447.66±12.37 ###
HAE (90mg kg^{-1})+ ISO	3.87±0.31 $^{\# \#}$	21.88±2.55$^{\#}$	353.66±19.79###

HAE was administered to rats daily for period of 15 days. Values are mean ± S .D for 6 rats in each group. ***P < 0.001,** P < 0.01 *vs* control; # # # P < 0.001 # # P < 0.01 and #p < 0.05 *vs* ISO

Cardiomyocytes contain large amounts of CK and LDH (Prabhu *et al*., 2006, Sathish *et al.,* 2003). Higher activity of LDH and CK in serum of infarcted rats is indicative of the extent of cellular injury due to excessive formation of free radicals, responsible for the impairment of cardiac cells membrane permeability by ISO and subsequent cardiac cells content released into bloodstream (Panda *et al*., 2009; Niv*ethe*tha *et al*., 2009). Pre-treatment with hydro-alcoholic extract (60mg kg^{-1} and 90mg kg^{-1}) reduced significantly ($p < 0.001$) the activity of LDH and CK. These results suggested cardioprotective activity of *P. biglobosa* EHA, and it possible ability to maintain myocardial cells integrity, by preventing lipid peroxidation induced myocardial damage and consequently cardiac cells death. Table 2 showed that myocardial tissue MDA concentration (742.83±38.29 nmol /mg of tissue) in infarcted rats (group II) was significantly ($P < 0.001$) increased compared to negative control group (369.50±26.60 nmol/ mg). Our results were in line with previous results (Panda *et al*., 2009; Prabhu *et al*., 2006). MDA concentration in groups III and group IV (447.66±12.37 nmol /mg and 353.66±19.79 nmol /mg of tissue respectively) were significantly ($P < 0.001$) lower when compared to infracted rats group. ISO-induced myocardial infarction is often characterized by lipid peroxidation (Rajadurai *et al*., 2005). MDA is the only stable indicator marker of installation process of lipid peroxidation (Aznar *et al*., 1983). Increased of MDA concentration (50.26%) in infarcted rats compared to negative control is a consequence of oxidative stress due to the alteration of the mechanism of dynamic balance b*et*ween pro-oxidants and antioxidants (Dhalla *et al*., 1996) and excessive ROS formation, responsible for lipid peroxidation and irreversible destruction of heart cells induced by ISO (Pasupathi *et al*., 2009; Prabhu *et al*., 2006). Hence, MDA decreased in pre-treated groups suggested that, HAE probably may prevent the formation of lipid peroxides in the myocardium. It is also possible that *P. biglobosa* extract cardioprotective effects may pass by inhibiting cells membrane permeability disturbance or reinforcing myocardium antioxidant activities. Antioxidants prevent cells membrane alterations and protect them from the negatives effects of frees radicals. Tringal *et al*. (2008) identified from the stem bark of *P.biglobosa* catechins and ferulates and suggested that their antioxidant properties may be responsible for same of the medical effects of this plant. Bonnah *et al*. (1998) showed that HAE possessed negative inotropic and chronotropic effect on the heart. Therefore HAE properties may prevent cardiac ischemia and then excessive free radicals generation. Lipid profile study on cardiac tissue showed moderate increase ($p<0.01$) of total cholesterol (69.89%) and triglycerides (46.67%) concentrations (Table 2). Our results showed that ISO injection increased significantly ($p<0.001$) serum total cholesterol (92%) and triglycerides (81.15%) concentrations in infarcted rats when compared to negative control group rats. ISO injection increased also significantly ($p<0.001$) LDL (329%) and VLDL (77%) levels, while HDL level was decreased (24.38%) compared to negative control group rats. These results corroborated previous studies (Ponnian *et al*., 2008; Rajadurai *et al*., 2005).

These changes in lipids profile might be due to enhanced lipids biosynthesis by cardiac cyclic adenosine monophosphate (Prince *et al*., 2005 Rajadurai *et al*., 2005). Increased cholesterol level, could be due to HDL decease since, its known that HDL inhibit the uptake of LDL by the arterial wall and facilitates the transport of cholesterol from tissue to liver where it is catabolised and excr*et*ed from body (Ponnian *et al*., 2008 ; Rajadurai *et al*., 2005 ; Rang *et al*., 2003). Increased triglycerides level was due to the inhibition of protein lipase activity and therefore their transport into the circulation. Previous studies (Pasupathi *et al*., 2009) have shown that high concentrations of total cholesterol, triglycerides, LDL, VLDL have positive correlation with myocardial infarction while, low level of HDL have negative correlation with myocardial infarction and commonly contribute to the development of cardiovascular diseases. Conversely, HDL increase, protects heart against cardiovascular diseases risks (Rang *et al*., 2003; Rajadurai *et al*., 2005). Increased oxidative stress and excessive formation of free radicals resulting from the conversion of LDL in oxidized LDL is involved in the development of atherosclerotic lesions in the vascular endothelium (Pasupathi and *al*., 2009).

High cholesterol is a major factor in the development of atherosclerosis. EHA pre-treatment prevented significantly ($p < 0.001$) lipid profile alteration in serum and cardiac tissue (Table 2 and Table 3) in rats pre-treated when compared to positive control rats. This suggests that, HAE may stimulates the synthesis of HDL or enhance the activity of the protein lipase. Therefore *P.biglobosa* EHA may protect the heart from ISO induced damage. The antihyperlipidemic effect of the extract could represent a protective mechanism against the development of atherosclerosis in human. Microscopic examination of negative control rat heart tissue showed clear integrity of normal myocardial cells architecture (fig. 1A). Positive control rats, heart tissue showed severe necrosis and massive infiltrations of inflammatory cells (fig. 1B) and slightly necrosis for pre-treated rat (fig. 1C). These results were in concord with previous studies of Panda *et al*. (2009) and Heather *et al*. (2009) and confirmed the adverse effects of ISO mentioned above.

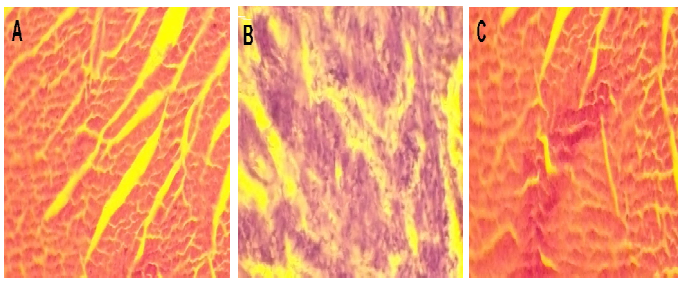

Fig. 1: Hematoxylin and Eosin staining of heart tissue 10x X 10x = 100X
A. Negative control: Clear integrity of myocardial tissue.
B. Infarcted rats (ISO 125mg kg^{-1} body weight): Tissue necrosis and infiltration of inflammatory cells.
C. Pre-treated with HAE (90 mg kg-1 body weights): Traces of necrosis in myocardium tissue

CONCLUSION

The present investigation evaluated the cardioprotective effect of *P. biglobosa* stem bark used in traditional medicine, on isoproterenol (ISO) induced myocardial infarction in rats. EHA ameliorated positively biochemical alterations, prevented oxidative stress and histological and morphological changes induced by isoproterenol. Therefore *P. biglobosa* stem bark has cardioprotective effects. Therapeutic success of *P. biglobosa* in traditional medicine may be due to its antioxidant, antilipoperoxidative and antihyperlipidemic activities.

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