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RESEARCH ARTICLE

Effects of Methanolic Extracts of *Quisqualis indica* (Aerial Parts) on Passive Smoking Induced Hyperlipidemia in Rats

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ABSTRACT:
In the present study the hypolipidemic activity of methanolic extracts of aerial parts of *Quisqualis indica* (QI) including flowers on passive smoking (PS) induced hyperlipidemia in rats had been evaluated. Hyperlipidemia was induced by passive smoking in a closed chamber having 1 burning ciggrette inside it. The hypolipidemic activity was analysed by reading the blood serum level in UV at 505 nm after treated with reagent present in auto span diagnostic kit. Dose of Methanolic extracts of QI had been prepared by using distilled water i.e. 200 mg/kg p.o. Methanolic extracts of QI significantly reduce the harmful lipid layer in blood serum at varying concentration and dose dependent manner which shows that the plant carries the hypolipidemic properties. It reduces the LDL, VLDL, cholesterol, triglyceride and raise HDL level in blood serum upto certain extent which was may be due to the inhibition of lipid peroxidation as the plants contain some active ingredients acting as antioxidants. Our result shows that the plants extracts recover the disorders in lipid metabolism noted in hyperlipidemic state.

KEY WORDS: Hypolipidemic, Hypocholesterolemic, Passive smoking (PS), Hyperlipidemia, *Quisqualis indica*, Coronary heart disease.

1. INTRODUCTION:
Hyperlipidemia refers to elevated levels of lipids and cholesterol in the blood, and is also identified as dyslipidemia, to describe the manifestations of different disorders of lipoprotein metabolism (Jacobson MS, 1998) and a major risk factor in the initiation and progression of atherosclerotic lesions, conditions such as coronary heart disease, ischemic cerebrovascular disease and peripheral vascular disease. This leads to high mortality and morbidity rate in developed countries. Hyperlipidemia also has an indirect role by stimulating the production of oxygen free radicals from polymorphonuclear leukocytes and monocytes .It is considered as one of the five leading causes of the death in the world (NCEP Report, 2002; Crowther MA, 2005). Diet modification is the cornerstone of therapy for mild to moderate hyperlipidemia. Modifying the diet is also recommended along with pharmacologic therapy in people at higher risk of CAD (NCEP Report 2001).

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Passive smoking causes approximately 50 000 deaths annually in the United States, with the vast majority of these deaths due to heart disease (California Environmental Protection Agency: Air Resources Board, 2005). The effects of secondhand smoke on many pathophysiological mediators of coronary artery disease are nearly as large as those of active smoking, including impaired platelet function, damage to vascular endothelium and its associated repair mechanisms, a rise in inflammatory molecules and dysfunctional lipid metabolism by forming free radicals (Barnoya and Glantz, 2005).

Cell membranes are made of unsaturated lipids and these unsaturated lipid molecules of cell membranes are particularly susceptible to free radicals. Oxidative damage can direct to a breakdown or even hardening of lipids, which composition of all cell walls. Breakdown or hardening is due to lipid peroxidation leads to death of cell or it becomes unfeasible for the cell to properly get its nutrients or get signals to achieve another. Epidemiological studies suggest that increased dietary intake of antioxidants

reduces the risk of coronary artery disease (Kushi et al., 1996; Rimm et al., 1993).

Many herbal medicinal products reported to have potential to reduce lipid and cholesterol in body and encourages safety profile (Devi and Sharma, 2004; Patil et al., 2004; Shukla et al., 2004). *Quisqualis indica Linn* is an evergreen plant growing all over the countries as ornamental plants showing various pharmacological activities such as anti-inflammatory activity, immunomodulatory activity, anti-staphylococcal activity, anthelmintic activity, antioxidants etc due to its presence of various active constituents all over the parts of plants which had been reported. (Yadav et al., 2011a, 2011b; Singh et al., 2010; Jahan et al., 2009; Kaisar et al.,2009; Wetwitayaklung et al.,2007.)

In the present study, the methanolic extracts of aerial parts of *Quisqualis indica* (QI) including flowers was investigated for hypolipidemic activity on passive smoking induced hyperlipidemia in rats.

2. MATERIALS AND METHOD:

2.1 Plant Material

The mature aerial parts of *Quisqualis indica* were collected in the morning from Bhopal, Madhya Pradesh, India, in the month of January 2012. Identification and authentication of herb by Dr. Zia ul Hassan, Professor of Botany, Safia College of Science, Bhopal, Madhya Pradesh, India (Voucher. No 323/Bot/Safia/2010).The collected parts were washed with a normal tap water so that the sticked dirt particle had been washed and then dried in a shed area, after dried it had been crushed into small pieces for extraction process. About 80 gm of dry powder was taken in a soxhlet apparatus and extracted with 400 ml methanol for about 8 days at 10-15 degree centigrade. The marc left was dried under room temperature to get a dry mass i.e. free of solvent. The final obtained extract was weighed and stored in air tight glass container at cool place.

2.2 Phytochemical analysis

Preliminary Phytochemical studies of methanolic *extract of Quisqualis indica* was performed for major classes of constituents like alkaloids, carbohydrates, protein and amino acid, Saponins, glycosides, steroids, tannins, flavonoid and phenolic compounds according to published standard methods. The dose limits were selected on the basis of previously performed oral acute toxicity studies in albino mice, in accordance with the OECD (423) guidelines.

2.3 Animals and animal diet

Albino wistar rats (100-200 gm) of either sex had been taken which were obtained from Sapience Bio Analytical Research laboratory, Bhopal (M.P.) animal house (Reg. No. 1413/A/11 CPCSEA) and housed 6 animals per cage made up of polypropylene, habituated at laboratory condition for 2 days prior to experiment procedure which were maintained at environment [(25^0 C± 2) temperature,30-50% humidity and 12 hr light and dark condition alternately)]. The animals were fed with standard pellet diet and water ad libitum and provided with ciggrette smoke twice a daily i.e. 1 cigarettes provided to a group of 6 animals at morning and evening by the use of smoking chambers having 1 ventilacle hole at both sides throughout the experiments except control group. The animals were divided into 3 groups containing 6 animals in each group i.e. Control Group, Passive smoking induced group and Passive smoking + Test Methanolic extract (200 mg/kg p.o.) group. After 28 days rats were fasted for 10-12 hours and then they were anaesthetized with mild chloroform, blood sample was collected by retro orbital sinus puncture. Collected blood was poured slightly into tubes marked and immediately centrifuged for 2000 rpm for 15 minutes to obtain clear serum. The amount of blood parameters was calculated in mg/dl.

2.4 Biochemical analysis

The blood serum were assayed for total cholesterol (TC), triglycerides (TG), high-density lipoprotein (HDL), low-density lipoprotein (LDL), very low-density lipoprotein (VLDL) using Span diagnostic kit having standard kit formula. The amount of LDL-Cholesterol and VLDL-Cholesterol were calculated using Friedewald's equation. LDL-Cholesterol =Total Cholesterol - Triglycerides/5-HDL -Cholesterol

VLDL-Cholesterol = Triglycerides/5
(Kaplan and Szabo, 1983).

2.5 Statistical analysis

The results were expressed as mean ± S.E (Standard Error). Statistical analysis was carried out by using ANOVA followed by Tukey's multiple comparison tests using Graph pad PRISM software version 5.04 (2010).P values < 0.05 were considered as statistically significant.

3. RESULTS:

The present investigation showed that the passive smoking induced hyperlipidemia in rats by raising the lipid level i.e. LDL, VLDL and TC and TG and lowering of HDL shown in table I. The results were discussed mainly under lipid layer as in hyperlipidemia lipid level were increased. Results shows that the passive smoking also raises total cholesterol 81.42% and triglyceride 74.55 % compare to control group. When passive smoking along with extracts had given it was noted the total cholesterol (TC), triglycerides (TG), low-density lipoprotein (LDL) and very low-density lipoprotein (VLDL) lowering and increases the high-density lipoprotein (HDL) level upto certain extent which was may be due to inhibition of lipid peroxidation. By the results it had been concluded that the extracts is acting as antihyperlipidemic drugs at the dose level of 200 mg/kg by lowering the harmful effects of lipid level in a dose dependent manner.

Table I: Effect of Methanolic extract of *Quisqualis indica* on TC, TG, HDL, LDL and VLDL in blood Serum of Control and Experimental Rats

Groups	TC	TG	HDL	LDL	VLDL
Control	52.15±0.39	41.37±1.37	21.65±0.44	24.66±1.03	8.275±0.27
PS	64.05±0.89***	55.49±0.52***	12.45±0.40***	40.50±0.49***	11.10±0.10***
QI (Meth ext) 200 mg/kg.	58.57±0.37**	47.94±0.78***	16.49±0.61***	32.88±0.27***	9.588±0.16***

Values are in mean ± SE; Number of animals in each group = 6;
*** Significant differences are shown by $P < 0.05$ analysis of variance (Statistical).
Where, **TC-** Total cholesterol**, TG-** Triglyceride**, HDL-** High density lipoprotein**, LDL**- Low density lipoprotein and **VLDL-** Very low density lipoprotein

4. DISCUSSION:

The present results shows that the extracts of *Quisqualis indica* linn produce a significant reduction in harmful lipids and raised the HDL level which is good cholesterol and thus it act as hypolipidemic and hypocholesterolemic. Active smoking is associated with reduced HDL cholesterol levels in adults and young persons. In adults, passive smoking had been reported to have a similar effect. It is well known that HDL-Cholesterol levels have a protective role in Coronary artery disease (Wilson et al, 1988). The increased level of HDL- cholesterol and decreased cholesterol level along with its LDL and VLDL fraction which is evident from the results could be due to inhibition of lipid peroxidation so that it cannot breakdown to form plaque and block the artery, thus atherosclerosis doesn't exists. By decreasing the harmful lipid levels and cholesterol in the body under the influence of plant *Quisqualis indica* linn strongly strengthen the hypolipidemic activity of the plant at dose dependent otherwise it may produce toxic effects. Overdosage of the herb will cause hiccups, dizziness, vertigo and vomiting. Taking this herb with hot tea can also cause hiccups (Takeatsu Kimura, 1996).

Environmental tobacco smoke consists of approximately 85 percent sidestream smoke (from the burning ends of cigarettes) and 15 percent exhaled mainstream smoke. (Taylor et al., 1992). Since cigarettes burn at higher temperatures during inhalation, combustion is more complete and some toxic components of tobacco smoke are broken down or filtered out before inhalation. Consequently, many toxic constituents, such as carbon monoxide and benzopyrene, are found in higher concentrations in sidestream than in inhaled smoke and more than 4000 chemicals are contained in environmental tobacco smoke (Glantz and Parmley, 1991). One or more of these compounds may be injurious to the arterial wall; in laboratory animals, exposure to environmental tobacco smoke is associated with endothelial dysfunction and with accelerated atherosclerosis. (Penn et al., 1994, Zhu et al., 1993). This dose-dependent relation between passive smoking and endothelial dysfunction is similar to that between active smoking and arterial injury (Celermajer et al., 1993). Cigarette smoking causes harmful cardiovascular and atherogenic effects resulting from changes in lipid metabolism (Mjos, 1988).

Adverse effects of smoking on the cardiovascular system include a reduction in high-density lipoprotein (HDL) cholesterol, an increase in platelet reactivity and an increase in fibrinogen concentrations. These effects on systemic and coronary hemodynamics, lipid metabolism and hemostasis may contribute to the long-term adverse consequences of smoking (Winniford 1990). Habitual smoking increases plasma levels of glycerol as well as nor adrenaline, which is the main stimulating hormone of adipose tissue lipolysis (Meyer et al., 2005).. In addition, a recent study in Japan showed a measurable decrease in the elasticity of the coronary arteries of nonsmokers after just 30 minutes of exposure to second hand smoke (Otsuka et al, 2001).

Cell membranes are made of unsaturated lipids and these unsaturated lipid molecules of cell membranes are particularly susceptible to free radicals (Langseth., 1996). Oxidative damage can direct to a breakdown or even hardening of lipids is due to lipid peroxidation leads to death of cell or it becomes unfeasible for the cell to properly get its nutrients or get signals to achieve another (Davies, 1991). Antioxidants cause protective effect by neutralizing free radicals, toxic byproducts of natural cell metabolism which inhibit the lipid peroxidation or lipolysis (Vadnere et al, 2011).

The active ingredients present in extracts help in recovering the disorderness in lipid metabolism noted in hyperlipidemic state which was may be due to inhibition of lipid peroxidation. Thus, our present study showed that the administration of Methanolic extracts at the dose 200 mg/kg of *Quisqualis indica* was effective to manage hyperlipidemia.

5. CONCLUSION:

The present study established that the passive smoking raise the lipid and cholesterol level with reducing the HDL level which cause hyperlipidemia as well as hypercholesterolemia existing heart disease such as heart attack, heart stroke etc in future. Epidemiological studies suggest that increased dietary intake of antioxidants reduces the risk of coronary artery disease and the plant extracts showing positive *indica*tion that it contains flavonoids and phenolic compounds helpful in CVD. The present investigation shows that the methanolic extracts of aerial parts of QI had markly reduced the raised lipid level LDL, VLDL and cholesterol due to passive smoking induced, thus it acts as hypolipidemic at dose dependent manner.

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