Original Article

Steroidal saponin from *Chlorophytum nimonii* (Grah) with lipid-lowering and antioxidant activity

Abstract

Aim: Since drugs used these days to lower the lipids are all synthetic drugs, they have some or the other side effects, therefore in search of cheaper lipid-lowering drugs with no side effects, we have conducted a study on *Chlorophytum nimonii* for its lipid-lowering and antioxidant properties. **Materials and Methods:** Chloragin and Gemfibrozil both caused a significant decrease in the serum level of lipids in triton-induced hyperlipidemic rats, and this model has been successfully used for the evaluation of lipid-lowering activity of chloragin in the rats. **Results and Discussion:** The lipid-lowering action of steroidal saponin and chloragin of the *C. nimonii* has been studied in triton model (in cholesterol-fed hyperlipidemic rats) *in vivo* and antioxidant activity *in vitro* model. Serum lipids were found to be lowered by the steroidal saponin (100 mg/kg body weight) in triton WR-1339-induced hyperlipidemia. Chronic feeding of this compound (50 mg/kg) in animals simultaneously fed with high-fat diet for 30 days caused lowering in the lipid and lipoproteins levels of low-density lipoproteins in experimental animals. **Conclusion:** Chloragin activates lipolytic enzymes in plasma and liver. Chloragin is mediated through inhibition of hepatic lipids, increased fecal bile acid excretion, and enhanced plasma lecithin cholesterol acyl transferase activities. Chloragin from the *C. nimonii* showed potent antioxidant activity as well.

Key words:
Chlorophytum nimonii, hyperlipidemic, steroidal saponin, triton-WR-1339

Introduction

Atherosclerosis is a condition in which an artery wall thickens as a result of a buildup of fatty materials such as cholesterol. It is a syndrome effecting arterial blood vessels, a chronic inflammatory response in the walls of arteries, in large part due to the accumulation of macrophage white blood cells and promoted by low-density lipoproteins without adequate removal of fats and cholesterol from the macrophages by functional high-density lipoproteins (HDL). It is commonly referred to as a hardening or furring of the arteries. It is caused by the formation of multiple plaques within the arteries.

Atherosclerosis and its associated complications are now the major cause of myocardial morbidity and mortality worldwide. Elevated levels of cholesterol concentration and lipoproteins especially low-density lipoprotein (LDL) and triglyceride (TG) along with free radicals oxidative stress are recognized as the leading cause in the development of atherosclerosis and coronary heart diseases. In general, oxidative damage takes place in the LDL of plasma by the hydroxyl radicals (OH) generated by metal ions present in the serum due to the alterations with their oxidation states. It has been demonstrated that oxidative damaged LDL are relatively more atherogenic than the native LD. Currently, several drugs are being used in the treatment of dyslipidemia. The drug can intervene by lowering cholesterol (LDL and total cholesterol) or by lowering TGs' level in the plasma. Treatment of hyperlipidemia using statins has lowered the serum levels of cholesterol and TGs. However, their side effects such as myositis are well known. Therefore, to develop therapeutics for the treatment of hyperlipidemia is extremely urgent.

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Vijai Lakshmi, Abbas Ali Mahdi[1], Santosh Kumar Agarwal[2], Ashok Kumar Khanna[3]

Medicinal and Process Chemistry Division, Central Drug Research Institute, [1]Biochemistry Division, Lucknow C.S.M. Medical University, [2]Phytochemistry Division, CIMAP, [3]Biochemistry Division, Central Drug Research Institute, Lucknow, Uttar Pradesh, India

Address for correspondence:
Dr. Vijai Lakshmi,
Department of Biochemistry, CSM, Medical University,
Lucknow, Uttar Pradesh, India.
E-mail: vijlakshmius@yahoo.com

Antioxidants are compounds that act as inhibitors of the oxidation process and are found to inhibit oxidant chain reactions[1] in small concentrations and thereby eliminate the threat of pathological processes. Phenolic compounds present in the medicinal plants have been reported to posses powerful antioxidant activity[2] and found to have a potential role in prevention of various diseases through their antioxidant activity.

The genus *Chlorophytum* (family Antheraceae) consists of at least 200 species distributed mainly in the tropical and subtropical countries.[3,4] About 15 species are found in India, mainly in peninsular India. Most of the species are herbs with short stalk-fasciled roots, which are often thick, fleshy, and tuberous. This plant exhibited a high order of antihyperglycemic and antihyperlipidemic activities. Previous chemical investigations have revealed that major chemical constituents of this genus are spiroketal steroidal saponins, sapogenins phenolic glycosides, and fatty acids.[5-9] This study deals with the isolation and characterization of a novel spiroketal saponin with antidyslipidemic and antioxidant activity from the aerial part of the plant, designated as chloragin [Figure 1].[10] Lipid-lowering activity of of *Chlorophytum nimonii* was done by the methods described in the literature.[11-13]

Materials and Methods

Plant materials

Aerial part of the plant was collected from Kerala state of India in May 1998 by the Botany Division of the Institute, and voucher specimen has been preserved in the herbarium of the Institute with the botany serial number 6949.

Extraction and isolation

Air-dried and powdered aerial part (1.0 kg) of the plant was percolated in 80% aqueous ethanol for 24 h and was boiled for 10 h. The aqueous ethanol extract was filtered in hot and was concentrated under reduced pressure to 200 ml volume. It was kept in refrigerator overnight to get white shinning crystals (1.2 g); the crystals thus obtained were purified by chromatography over a column of silica gel using eluent $CHCl_3$–MeOH-H_2O (8:2:0.2 to 7:3:0.5, v/v) and finally purified by HPLC using acetonitrile–water (30:70, v/v) to give chloragin [Figure 1] (200 mg). The structure was confirmed by spectral data [Tables 1 and 2].

Lipid-lowering activity of *C. nimonii*

The lipid-lowering activity of chloragin[1] was evaluated in triton-treated hyperlipidemic male Charler foster rats (150–200 g) which were divided into control, hyperlipidemic, and hyperlipidemic with chloragin-treated containing six animals in each group. Hyperlipidemia was induced by the administration of triton WR-1339 (400 mg/kg i.p.). All the animals were maintained on a standard pellet diet and water *ad libitum*. Chloragin[1] and standard drug genifibrozil were macerated with 0.2% aqueous gum acacia suspension. The suspension was fed orally at the dose of 100 and 200 mg/kg p.o. simultaneously with triton in drug-treated group. The animals of the control group received the same amount of gum acacia suspension. At the end of experiment after 18 h, blood was withdrawn from retro-orbital plexus and plasma Was separated and used for the estimation of total cholestrol (TC), phospholipid (PL), triglyceride (TG) and

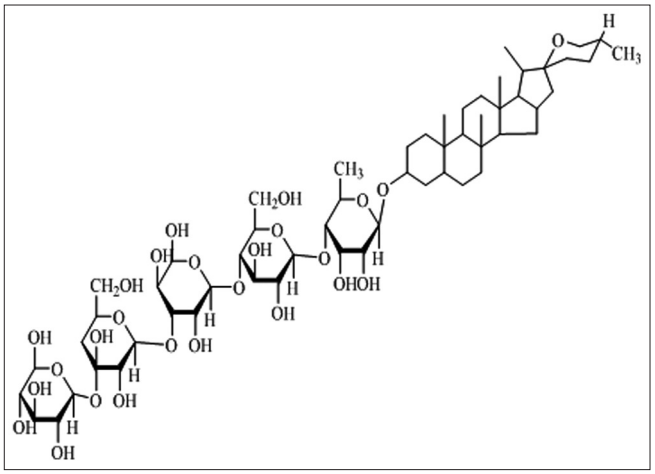

Figure 1: Chloragin

Table 1: ^1H and ^{13}C NMR for aglycon moiety of chloragin[1]

Carbon No.	δ_C [a]	δ_H multiplicity [b] (J values in Hz)
C-1	36.6	0.88 m; 1.25 m
C-2	29.4	1.65 m; 1.81 m
C-3	76.6	3.89 m
C-4	33.3	1.62 m; 1.89 m
C-5	44.0	0.90 m
C-6	29.1	1.20 m ; 0.88 m
C-7	31.9	1.65 m ; 8.85 m
C-8	34.7	0.85 m
C-9	53.8	1.01 m
C-10	35.4	–
C-11	20.7	1.28 m
C-12	39.6	1.35 m
C-13	40.3	–
C-14	55.7	2.00 m
C-15	31.8	1.58 m ; 2.05 m
C-16	80.4	4.38 m
C-17	61.8	2.26 m
C-18	16.0	0.86 s
C-19	12.0	0.82 s
C-20	41.6	2.24 m
C-21	14.5	1.14 d 6.7
C-22	109.0	–
C-23	30.8	1.28 m;1.60 m
C-24	28.9	1.58 m ; 1.62 m
C-25	30.6	1.56 m
C-26	65.9	3.51 dd (10.2, 10.3) 3.62 dd (10.3, 3.2)
C-27	17.2	0.70 d (6.1)

[a]Recorded at 75.0 MHz in DMSO-d_6. [b]Recorded at 300 MHz in DMSO-d_6

proteinby standard spectrophotometric methods using kit (16) [Table1].

High-fat diet-induced hyperlipidemia

Hyperlipidemia was induced by feeding of high-fat diet once a day for 30 days. Drugs were administered orally (100 mg/kg, p.o.) simultaneously with high-fat diet in drug-treated groups. Control animals received the same amount of normal saline or groundnut oil. At the end of the experiments, rats were fasted overnight and blood was withdrawn. The animals were killed and the liver was excised immediately.

Biochemical analysis of plasma/serum

Plasma lecithin cholesterol acyl transferase (LCAT) activity[12] and post-heparin lipolytic activity (PHLA) were assayed[13]. Serum was fractionated into very low-density lipoprotein (VLDL) and LDL.

Results

Effect of chloragin in triton-induced hyperlipidemia

The acute administration of triton WR-1339 caused a marked increase in serum levels of TC (4.37 F), PL (3.0 F), TG (2.98 F), and protein (1.52 F). Treatment with these compounds caused reversal in these levels of TC (-24%) together with a decrease in PL (-23%), TG (-23%), and protein (-20%) [Table 3]. The lipid-lowering activity of these drugs with hyperlipidemic rats was comparable to that of gemfibrozil.

Effect of chloragin on lipid composition in serum lipoproteins and liver

The data in [Table 4] show that the administration of cholesterol in rats increased their serum levels of TC, PL TG, and protein +2.37 F, +2.24 F, +2.63 F, and +2.10 F, respectively. Feeding with chloragin and gemfibrozil reversed the levels of the serum lipids of TC, PL TG, and protein (26, 23, 24, and 25%) in cholesterol- and drug-treated animals. The analysis of hyperlipidemic serum showed a marked increase in the level of lipids and apoproteins constituting lipoprotein, and these effects were pronounced for VLDL-TG (+2.22 F) and LDL-TC (+4.15 F). Treatment with chloragin and gemfibrozil significantly reduced these levels of VLDL lipids (-22, -23%) as well as LDL-TC (-25%), PL (-21%), TG (-26%), and apo-LDL (-19%), respectively, in hyperlipidemic rats. At the same time, the decreased levels of HDL-lipids and apo-HDL in these animals were partially recovered [Table 4]. The increased levels of TC, PL, TG, and protein in liver (-21, 24, 22, and 23%) of cholesterol fed rats were observed to be lowered by their treatment with drugs [Table 5].

Effect of lipolytic enzymes

Cholesterol feeding caused the inhibition of plasma LCAT (27%) and PHLA (20%), respectively [Table 2] and total

Table 2: ^{1}H and ^{13}C data of the sugar moieties of chloragin[1]

Carbons/sugar unit	δ_C [a]	δ_H multiplicity [b] (J values in Hz)
Rh-1	99.9	5.12 d (3.6)
-2	70.6	3,66 m
-3	70.6	3.39 dd (8.8,3.0)
-4	80.2	3.18 dd (8.8, 3.0)
-5	68.1	3.95 dq (9.6,6.2)
-6	17.7	1.70 d (6.2)
Glc-1	104.0	4.60 d (7.8)
-2	71.4	3.31 dd (7.6, 8.3)
-3	85.7	3.60 dd (8.3, 9.0)
-4	69.0	3.09 m
-5	75.8	3.22 m
-6	59.9	3.71 m, 3.35 m
Xyl-1	103.5	4.48 d (7.9)
-2	69.5	3.03 dd (7.9, 9.1)
-3	70.6	3.11 m
-4	74.4	3.49 m
-5	63.9	3.09 dd (11.0,5.4) 3.76 dd (11.0, 11.1)
Glc-1	98.4	4.29 d (7.6)
-2	72.6	3.31 dd (7.6, 8.5)
-3	73.5	3.33 m
-4	79.5	3.76 m
-5	75.8	3.34 m
-6	61.5	3.70 m, 3.34 m
Xyl-1	103.6	4.36 d (7.4)
-2	73.5	3.03 dd (7.4, 9.2)
-3	76.6	3.39 m
-4	74.4	3.50 m
-5	63.9	3.90 dd (11.2, 5.6) 3.40 dd (11.2, 11.1)

[a]Recorded at 75.0 MHz in DMSO-d_6; Multiplicity by DEPT experiments; [b]Recorded at 300 MHz in DMSO-d_6

Table 3: Lipid-lowering activity of chloragin in triton-treated hyperlipemic rats

Experimental schedule	Total cholesterol[a]	Phospholipid[a]	Triglyceride[a]	Protein[b]
Contol	87.39±6.14	80.27±5.62	85.53±6.17	6.12±0.17
Triton treated	382.48±20.44*** (+4.37F)	242.77±16.30*** (+3.0F)	255.17±18.00*** (2.98F)	9.32±0.28*** (1.52F)
Triton+chloragin	300.17±16.66*** (–24)	182.62±12.04*** (–23)	195.62±13.62*** (–23)	7.44±0.48*** (–20)
Triton+gemfibrozil (standard drug)	260.12±13.37*** (–32)	163.34±14.24*** (–33)	170.30±11.88*** (–33)	6.80±0.27** (–27)

Unit: [a]mg/dl; [b] g/dl serum. Values are mean±SD from six animals ***$P<0.001$. Triton group compared with control, triton, and drug treated with triton

Table 4: Effect of chloragin and gemfibrozil on blood lipids and lipolytic enzymes in hyperlipidemic rats

Parameters	Control	Cholesterol treated	Cholesterol and chloragin treated	Cholesterol and gemfibrozil treated
Serum				
Total cholesterol[a]	88.32±6.14	210.66±14.73*** (+2.37F)	154.25±10.62*** (–26)	138.37±11.00*** (–34)
Phospholipid[a]	80.44±6.00	180.72±14.40*** (+2.24F)	138.89±9.80*** (–23)	118.66±7.94*** (–34)
Triglyceride[a]	85.37±5.73	225.11±18.14*** (+2.63F)	170.21±13.14*** (–24)	150.62±10.89*** (–33)
Protein[b]	6.11±0.37	12.84±0.71*** (+2.10F)	9.58±0.41*** (–25)	9.14±0.49*** (–29)
VLDL				
Total cholesterol[a]	8.44±0.51	33.82±3.00*** (+4.00F)	25.82±1.14*** (–23)	23.11±1.62*** (–31)
Phospholipid[a]	15.16±0.47	32.22±2.18*** (2.12F)	24.79±1.72*** (–23)	22.87±1.00*** (–30)
Triglyceride[a]	39.79±2.84	88.39±6.17*** (+2.22F)	68.88±4.00*** (–24)	62.66±3.18*** (–25)
Apoprotein[b]	7.00±0.38	13.88±1.98*** (+1.98F)	10.88±0.47** (–22)	9.88±0.43*** (–28)
LDL				
T cholesterol[a]	14.48±1.13	60.17±4.32*** (+4.15F)	44.77±2.48** (–25)	42.88±2.00*** (–29)
Phospholipid[a]	13.33±0.79	39.66±1.89*** (+2.97F)	31.22±1.64** (–21)	20.81±1.69*** (–27)
Triglyceride[a]	18.00±1.17	38.81±3.12*** (+2.15F)	28.68±2.11*** (–26)	26.71±1.88*** (–31)
Apoprotein[b]	19.14±1.08	30.33±2.17*** (+1.58F)	24.60±2.00* (–19)	22.14±1.11*** (–27)
HDL				
T cholesterol [a]	47.39±3.72	36.28±2.14*** (–23)	43.33±3.14* (+16)	44.89±3.55* (+19)
Phospholipid [a]	38.37±3.00	26.37±1.82*** (–31)	32.77±2.10** (+20)	34.66±2.68*** (+24)
Triglyceride [a]	16.65±1.00	12.68±0.77*** (–24)	15.32±1.10* (+17)	15.66±0.82* (+19)
Apoprotein[b]	170.38±12.22	123.30±12.00*** (–28)	142.77±11.66* (+14)	149.89±12.64* (+18)
Plasma LCAT activity[c]	68.70±4.80	36.14±2.63*** (–47)	49.99±3.75*** (+27)	51.66±4.00*** (+30)
PHLA[d]	18.11±1.00	11.33±0.68*** (–37)	14.17±0.38** (+20)	15.18±1.10*** (+25)

Unit: [a]mg/dl serum; [b]g/dl serum; [c]n mol cholesterol released /h/l plasma; [d]n mol free fatty acid formed /h/ml plasma. Values are mean±SD from six animals; ***$P<0.001$, **$P<0.01$, *$P<0.05$; cholesterol-treated compared with control, cholesterol-, and drug-treated compared with cholesterol

lipolytic activity (+12%) in liver [Table 5]. Treatment with chloragin and gemfibrozil partially reactivated these lipolytic activities in plasma and liver of hyperlipidemic rats.

Effects on fecal excretion of bile acids

It was observed that feeding with cholesterol decreased significantly in the fecal excretion of cholic acid (-40%) and deoxycholic acid (-55%) which were recovered by the treatment with chloragin (+27 and +37%) and gemfibrozil (+25 and 26%) in the experimental animals.

Antioxidant activity of chloragin and effect of these chloragin on oxygen free radical generation *in vitro*

The scavenging potential of these saponin at 100 and 200/μg/ml against formation of o^{-2} and OH in non-enzymatic system was also studied [Table 6]. The significant inhibition of superoxide anions by (-35%), hydroxyl radicals (-44%), and microsomal lipid peroxidation (-40%), respectively, was also studied.

Discussion

Chloragin and gemfibrozil both caused a significant decrease in the serum level of lipids in triton-induced hyperlipidemic rats, and this model has been successfully used for the evaluation of lipid-lowering activity of chloragin in the rats.[15,16] The present investigation with cholesterol-fed hyperlipidemic animals shows that chloragin could increase the level of HDL by increasing the activity of LCAT, which plays a key role in lipoprotein metabolism. The increase of lipolytic activity in

Table 5: Effect of choragin and gemfibrozil on hepatic lipids and fecal bile acid excretion in hyperglycemic rats

Parameters	Control	Cholesterol treated	Cholesterol and chloragin treated	Cholesterol and gemfibrozil treated
Liver				
LPL activity[a]	132.64±1.10	73.22±5.17*** (–45)	83.37±6.12 (+12)	87.77±8.00*** (+16)
Total cholesterol[b]	6.82±5.17	11.23±0.72*** (+1.64F)	8.80±0.32** (–21)	8.10±0.33*** (–28)
Phospholipid[b]	24.14±2.00	38.17±3.11*** (+36%)	28.66±1.62*** (–24)	15.88±1.12*** (–32)
Triglyceride[b]	11.00±0.72	16.37±1.00*** (+32%)	12.80±0.68** (–22)	11.38±0.62*** (–30)
Protein[b]	145.82±12.11	220.00±16.12*** (+34%)	170.37±11.82*** (–23)	160.37±12.82*** (–27)
Fecal bile acids				
Cholic acid[c]	82.39±6.11	49.38±3.00*** (–40)	60.14±4.12*** (+27%)	55.30±4.12* (+10)
Deoxycholic acid[c]	54.44±4.37	24.34±2.11*** (–55)	38.66±2.41*** (+37)	33.00±2.38*** (+26)

Unit: [a]n mole free fatty acid formed/h/mg protein; [b]mg/g; [c]µg/g. Values are mean±SD of six animals; ***P<0.001, **P<0.01, cholesterol-treated group compared with control and cholesterol plus drug-treated group compared with cholesterol-treated group

Table 6: Effect of natural products (3764) on the generation of superoxide anions, hydroxyl radicals, and lipid peroxidation in microsomes

Test compound	Conc. of compounds (µg/ml)	Superoxide anions[a](O_2^-)	Hydroxyl radicals[b] (OH*)	Microsomal lipid peroxidation[b]
Control		120.22±9.77	Control 75.77±	Control 85.66±
Chloragin	100	Exp 90.33±6.89*** (–25)	Exp 57.32±2.79*** (–24)	Exp 65.50±500*** (–23)
	200	Exp 78.17±5.42*** (–35)	Exp 42.14±3.00*** (–44)	Exp 51.23±4.11*** (–40)
Standard drug (gemfibrozil)	200	50.78±3.62*** (–57) (Alloperinol)	40.39±2.8*** (–47) (Manitol)	38.27±2.60*** (–55) (a tocopherol)

Units: [a]n mol formazone formed/min; [b]n mol MDA formed/h/mg protein. Each value is mean±SD of six values ***P<0.001. Experimental data compared with control experiment

liver and the level of blood HDL-TC followed by the decrease of B-lipoprotein lipids and the suppression of hepatic lipids by these drug are of great utility for regressing atherosclerosis. The stimulation of LDL catabolism by the compounds in hyperlipidemic animals may be due to a significant decrease in the level of serum and tissue lipids. The drug may also enhance the synthesis of LDL apoprotein (Apo B) and receptor protein to accelerate the turnover of cholesterol. Increased synthesis of receptor protein decreased the rate of hepatic cholesterol biosynthesis and inhibition of oxidative modification in LDL may regulate cholesterol level in the body.[15,16] It has been reported that the hypolipidemic activity of natural products such as *Phyllanthus nirori*[14] may be linked with increased fecal bile acid excretion and with the inhibition of cholesterol biosynthesis by steroidal saponins are of great utility for representing atherosclerosis. The stimulation of LDL catabolism by these saponins in hyperlipidemic animals may be due to a significant decrease in the level of serum and tissue lipids. This drug may also enhance the synthesis of LDL apoprotein (Apo B) and receptor protein to accelerate the turnover of cholesterol. Increased synthesis of receptor protein decreased the rate of hepatic cholesterol biogenesis and inhibition of oxidative modification in LDL may regulate the cholesterol level of lipid peroxidation product in the liver membrane of treated animals. It has been reported that the hypolipidemic activity of natural products such as *Achyranthus aspera*, *Terminalia Chebula*, *Terminalia arjuna*, *Phyllanthus niruri*, and Picroliv may be linked with increased fecal bile acid excretion and with the inhibition of cholesterol biosynthesis.

The potentially reactive derivatives of oxygen ascribed as ROS such as superoxide anions, hydroxyl radicals, and hydrogen peroxide are continuously generated inside the human body as a consequence of exposure to exogenous chemicals and/or a number of endogenous metabolic processes involving redox enzymes and bioenergetics electron transfer. Owing to the ROS overproduction and/or inadequate antioxidant defense, there is upsurge of ROS and this culminates in oxidative stress. It is quite interesting to note that plants have good antioxidant ability and are safer than the synthetic antioxidants. The antioxidant activity can be attributed to various mechanisms like prevention of chain initiation, binding of transition metal ion catalyst,

decomposition of peroxide, reductive capacity, and radical scavenging activity.

In this study, steroidal saponin (chloragin) of *C. nimonii* has shown promising antihyperlipidemic and antioxidant activity. The lipid-lowering action of saponin may be due to activation of LCAT and tissue lipolytic enzymes, enhanced catabolism of LDL, and increased fecal bile acid excretion, and some of these effects were comparable to that of gemifibrozil. In antioxidant activity, i.e., superoxide anions, hydroxyl radicals and lipid peroxidation were comparable to eloperinal, manitol, and tocopherol.

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