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Antihyperlipidemic activity of Lagenaria siceraria in Albino Rats

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Abstract

Coronary artery disease (CAD) is an atherosclerotic condition marked by the development of lesions called atherosclerosis plaques in the walls of coronary arteries. Hyperlipidemia, also known as lipidemia, is the excess of lipids that form groups of fat with a lipoprotein material counterpart in the blood. Lagenaria siceraria, often known as the bottle gourd (syn. calabash, doodhi, lauki, or kadoo), is a recognized member of the Indian Ayurvedic Pharmacopoeia that contains all of the vital components needed for human health. It is used as a purgative, cooling agent, aphrodisiac, antidote to poison, general tonic, diuretic, ulcer, fever, pectoral cough, asthma, bronchial diseases, and scorpion strings. Fruit juices that can be made by chopping fresh fruits in a blender and drying them in an oven at 40 to 50^0C are employed in the current investigation. The parent dry extract was fractionated using chloroform, acetic acid, methanol, pyridine, and water, in ascending order of polarity. This study showed that the administration of L. siceraria juice dramatically decreased the raised blood cholesterol, triglycerides, LDL, and decreased HDL that occurs in hyperlipidemia.

Keyword

Atherosclerosis, Hyperlipidemia, Lagenaria siceraria

Introduction

Atherosclerosis, coronary artery disease, and brain vascular disorders are all strongly predicative risk factors for hyperlipidemia. The generalized disease of the arterial network known as atherosclerosis (sclero-hardening of arteries) is also known as coronary artery disease (CAD). It is characterized by the development of lesions called atherosclerosis plaques in the walls of large and or medium-sized coronary arteries and reduces blood flow to the myocardium. There is a lot of evidence connecting hyperlipidemia to atherosclerosis. Clinical trials proved beyond a reasonable doubt that reducing blood cholesterol lowers CAD-related morbidity and death in people with established CAD and also lowers mortality and new CAD events in patients without established CAD.[3] According to Ayurveda, several condiments, medicinal plants, and fruits used in daily meal preparation in Indian kitchens are hypolipidemic.[4] Lagenaria siceraria, which has its official name in the Ayurvedic Pharmacopoeia of India, is also known as the bottle gourd, calabash, doodhi, and lauki in Hindi, and kadoo in Marathi.[5] The fruits of L. siceraria are traditionally used for their cooling, diuretic, aphrodisiac, antidote to several poisons, cardioprotective, cardiotonic, general tonic, and aphrodisiac effects. It treats chest cough, asthma, and other bronchial problems as well as discomfort, ulcers, and fever.[6,7]

MATERIALS AND METHODS

Collection and Authentication of Plant Material

The fresh fruits of L. siceraria were procured from local market between the months of August and December and verified by the authority of the botany department.

Extraction of Plant Material

The fresh fruits of L. siceraria were crushed in a mixer (juicer) to create the fruit juice, which was then dried at a temperature between 40 and 50^{o} C.

Fractional Extraction of Parent Extract

The parent dried juice extract was then fractionated by using the solvents according to polarity in ascending order i.e. by using chloroform: acetic acid, methanol, pyridine, and water
[Table 1]. Each fraction was dried in oven at 40-50^{0}C.

Isolation and Purification of Methanolic Extract by Column Chromatography

1. Glass column

2. Dimensions: 45 cm in length and 3 cm in diameter

3. Silica gel, the stationary phase

4. Illustration: Ethanolic extract

5. The gradient elution phase

The parent extract's methanolic fraction was submitted to isolation over a silica gel column with a mesh size of 100–200.

Earlier, the mobile phase was used to create the silica gel slurry. The mobile phase washed the column for an adequate amount of time. Methanolic fraction was then applied on top of the silica gel. Continuously passing the mobile phase at a constant flow rate of 10 ml/min. The fractions were gathered at regular intervals, evaporated at a temperature of between 40 and 50^{0} C, and then tested for antihyperlipidemic activity.

Isolation of Compounds by Thin Layer Chromatography

The column chromatography active fraction was employed for additional TLC isolation. The solvent mixture (6:2:2) of n-butanol, methaol, and water was produced by trial and error. It was possible to obtain four seats, which were designated LSN-I, LSN-II, LSN-III, and LSN-IV. Prepared TLC was used to gather isolated spots.

Evaluation of the Isolated Compound

Diagnostic Kit's Antihyperlipidemic Activity Merck Ltd. provided the diagnostic kits that were utilized to estimate triglyceride, cholesterol, and HDL-C levels.

Hyperlipidemia Inducer-

Hyperlipidemia Triton-X-100 was employed as an inducer to cause hyperlipidemia in test rats.

Animals and Treatments

The Adult male albino rats raised in the animal house. Weighing 180–200g Normal Healthy Rats With a 12-hour day/night cycle, the animals were kept in polypropylene cages at room temperature. Animals were given a healthy feed and unlimited access to water throughout the whole study period.

Induction of Hyperlipidemia

After an overnight fast of 18 hours, Wistar albino rats were given a single intraperitoneal injection of a newly made solution of Triton-X-100 (100 mg/kg) in physiological saline solution. [8, 9]

Dose Preparation and Administration of Extracts

After giving Triton X-100 intravenously, the isolated L. siceraria compounds LSN I, LSN II, LSN III, and LSN IV were given orally to the animals via stomach intubation three times a day with a 3-hour interval.

Protocol for Antihyperlipidaemic Activity

Out of 42 rats Total 36 hyperlipidemia rats and 6 control rats were used in the study. Seven groups of six rats each were formed from the group of rats.

Group I: - Regularly treated distilled water.

Group II: - Triton-X-100 (100 mg/kg) intravenous,

Group III: Lovastatin (10 mg/kg) p.o. and Triton-X-100 (100 mg/kg) i.p.

Group IV: LSN I p.o. and Triton-X-100 (100 mg/kg) i.p.

Group V: LSN II p.o. and Triton-X-100 (100 mg/kg) i.p.

Table 1: Fractionation of parent extracts

Solvent	Polarity Index	Fractionation	% Yield
Chloroform + Glacial acetic acid (1:1)	5.13	Soxhelation	4.3
Methanol	5.0	Soxhelation	49
Pyridine	5.2	Maceration	12
Water	9.0	Maceration	27.5

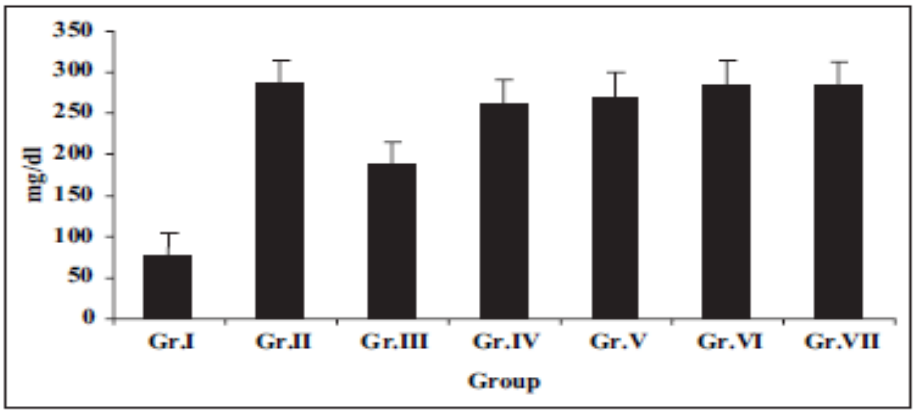

Figure 1: Effect of isolated compounds (LSN) on Cholesterol level of Triton-induced hyperlipidemia in rats

Table 2: Effect of isolated compounds (LSN) on total cholesterol level of triton-induced hyperlipidemia in rats

Groups	Treatments	Cholesterol(mg/dl)
I	Normal Saline	74.31 ± 0.87
II	Triton control (100mg/kg)	284.55 ± 1.82
III	Triton + standard (10mg/kg)	185.49 ± 1.23
IV	Triton + LSN I (10mg/kg)	261.81 ± 1.02**
V	Triton + LSN II (10mg/kg)	270.071 ± 0.91**
VI	Triton + LSN III (10mg/kg)	283.00 ± 1.77*
VII	Triton + LSN IV (10mg/kg)	283.66 ± 1.75ns

***P* < 0.01, very significant; **P* < 0.05, significant; and ns *P* > 0.05, non-significant One-way ANOVA followed by Dunnett's test

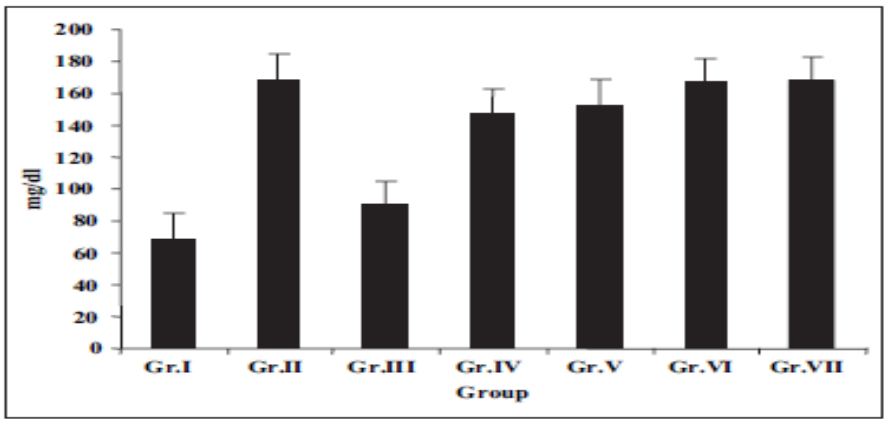

Figure 2: Effect of isolated compounds (LSN) on Triglyceride level of Triton-induced hyperlipidemia in rats

Table 3: Effect of isolated compounds (LSN) on Triglyceride level of Triton-induced hyperlipidemia in rats

Groups	Treatments	Triglycerides (mg/dl)
I	Normal Saline	69.07 ± 0.93
II	Triton control (100mg/kg)	169.20 ± 0.70
III	Triton + standard (10mg/kg)	89.53 ± 0.86
IV	Triton + LSN I (10mg/kg)	146.19 ± 2.52**
V	Triton + LSN II (10mg/kg)	152.24 ± 2.00**
VI	Triton + LSN III (10mg/kg)	165.72 ± 0.89**
VII	Triton + LSN IV (10mg/kg)	166. 73 ± 1.02ns

** $P < 0.01$, very significant and ns $P > 0.05$, non-significant. One-way ANOVA followed by Dunnett's test

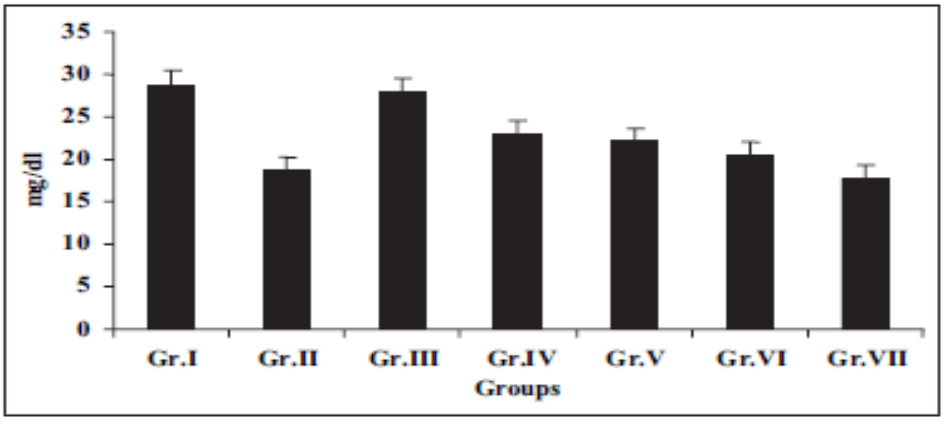

Figure 3: Effect of isolated compounds (LSN) on HDL-c level of Triton-induced hyperlipidemia in rats

Table 4: Effect of isolated compounds (LSN) on HDL- C level of Triton-induced hyperlipidemia in rats

Groups	Treatments	Triglycerides (mg/dl)
I	Normal Saline	27.73 ± 1.46
II	Triton control (100mg/kg)	18.65 ± 1.19
III	Triton + standard (10mg/kg)	27.93 ± 1.42
IV	Triton + LSN I (10mg/kg)	22.99 ± 0.63**
V	Triton + LSN II (10mg/kg)	21.93 ± 0.67**
VI	Triton + LSN III (10mg/kg)	20.35 ± 0.78**
VII	Triton + LSN IV (10mg/kg)	17.79 ± 0.80^{ns}

** $P < 0.01$, very significant and ns $P > 0.05$, non-significant. One-way ANOVA followed by Dunnett's test

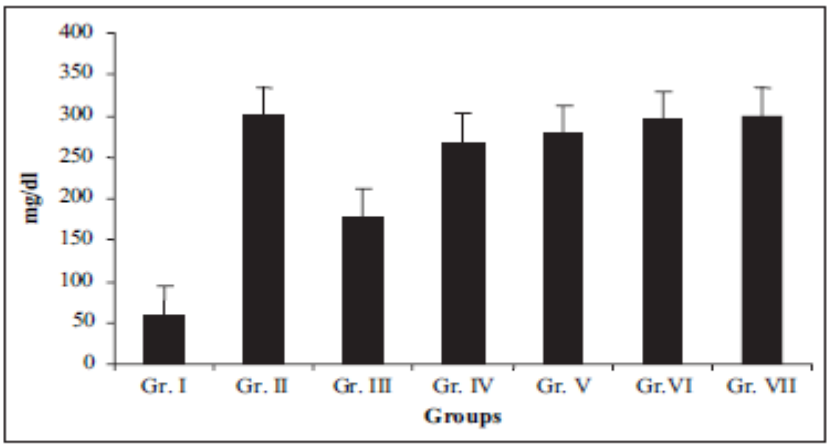

Figure 4: Effect of isolated compounds (LSN) on LDL level of Triton-induced hyperlipidemia in rats

The Triton control was compared with normal. The experimental results were compared with Triton control.

Table 5: Effect of isolated compounds (LSN) on LDL level of Triton-induced hyperlipidemia in rats

Groups	Treatments	Triglycerides (mg/dl)
I	Normal Saline	59.58 ± 2.24
II	Triton control (100mg/kg)	299.73 ± 2.23
III	Triton + standard (10mg/kg)	175.45 ± 1.40
IV	Triton + LSN I (10mg/kg)	267.26 ± 1.67**
V	Triton + LSN II (10mg/kg)	279.44 ± 1.30**
VI	Triton + LSN III (10mg/kg)	295.98 ± 2.08**
VII	Triton + LSN IV (10mg/kg)	298.74 ± 2.13^{ns}

** $P < 0.01$, very significant and ns $P > 0.05$, non-significant. One-way ANOVA followed By Dunnett's test

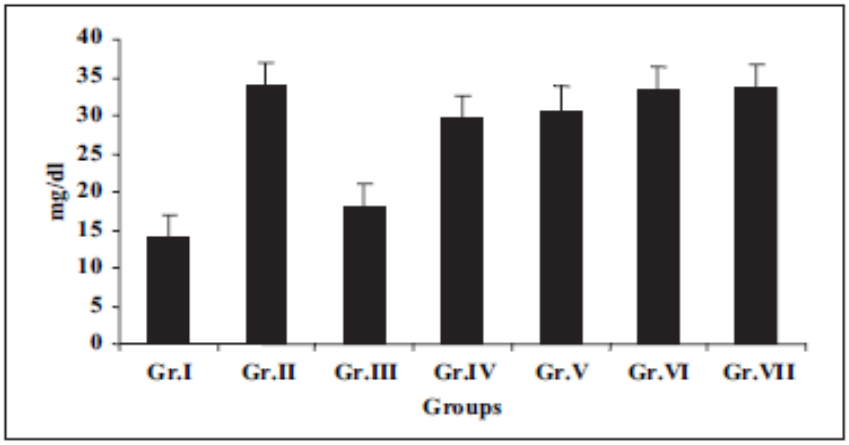

Figure 5: Effect of isolated compounds (LSN) on VLDL level of Triton-induced hyperlipidemia in rats

Table 6: Effect of isolated compounds (LSN) on VLDL level of Triton-induced hyperlipidemia in rats

Groups	Treatments	Triglycerides (mg/dl)
I	Normal Saline	13.81 ± 0.18
II	Triton control (100mg/kg)	33.83 ± •0.14
III	Triton + standard (10mg/kg)	17.90 •± 0.17
IV	Triton + LSN I (10mg/kg)	29.43 •± 0.50**
V	Triton + LSN II (10mg/kg)	30.64 •± 0.4**
VI	Triton + LSN III (10mg/kg)	33.35 ± 0.15*
VII	Triton + LSN IV (10mg/kg)	33.54 •± 0.20ns

** $P < 0.01$, very significant; *$P < 0.05$, significant; and ns $P > 0.05$, non-significant. Onaway ANOVA followed by Dunnett's test.

DISCUSSION

The goal of the study was to assess the antihyperlipidemic potential of several isolated L. siceraria fruit components. These substances were separated from the column chromatography and TLC, the active methanolic fraction of the parent extract of L. siceraria fruit juice is produced. LSN-I, LSN-II, and LSN-III showed significant results when the isolated compounds' antihyperlipidemic action was examined [Figs. 1–5 and Tables 2–6]. The study showed that the administration of fractions of L. siceraria fruit juice significantly decreased the raised blood cholesterol, triglycerides, LDL, and decreased HDL that occur in hyperlipidemia. DISCUSSION

This discovery offers some biochemical support for the use of fruit, fruit juice, or fruit extracts in the treatment of hyperlipidemic patients.
The L. siceraria (Mol.) Stand fruit is now added to the list of antihyperlipidemic medicines and has the potential to become a common household medication with both preventive and therapeutic effects against hyperlipidemia.

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