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Extraction of *Caesearia Sylvestris* Plant Parts and Characterization for Its Anti Hyperlipidermiac Activity

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A B S T R A C T

The present study is to evaluate the effect of *Caserua sylvestr is* Methanolic Extract and Phenolic Extracts on plasma lipid levels in Triton -X-100 induced hyperlipidemic rats. Major complication of hyperlipidemia is atherosclerotic heart disease, heart attack and heart stroke, but atherosclerosis is primary cause of death. Developing countries are reliant on medicinal plants as their main source of treatment for diseases. As *Caserua sylvestris have* the native habitat the production is more so it is locally available cost effective with no side effects. As *Caserua sylvestris*is cost effective and beneficiary in metabolism of cholesterol, so it has been taken in to consideration in order "To evaluate Anti-hyperlipidemic activity of Methanolic Extract and Phenolic Extracts of *Caserua sylvestris*in triton X -100 induced hyperlipidemic rats". The results concluded that PEBM (500 mg/kg) have definite antihyperlipidemic activity in Triton X-100 induced hyperlipidemic model and which is equipotent activity when compared with Atorvastatin treated groups. Further studies on this extract may lead to identify the possible mechanism of action and isolation of active principle from the same.

Keywords: *Caserua sylvestris,* antihyperlipidemic, Atorvastatin

A R T I C L E I N F O

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1. Introduction

Lipid is the scientific term for fats in the blood. At Normal levels, lipids perform important functions in your body, but can cause health problems if they are present in excess. The term hyperlipidemia means high lipid levels. Hyperlipidemia includes several conditions, but it usually means that you have high cholesterol and high triglyceride levels. Lipid and lipoprotein abnormalities are common in the general population, and are regarded as a modifiable risk factor for cardiovascular disease due to their influence on atherosclerosis In addition, some forms may predispose to acute pancreatitis.

Symptoms and Diagnosis of Hyperlipidemia:[1]

Hyperlipidemia in general has no apparent symptoms and it is discovered and diagnosed during routine examination or evaluation for atherosclerotic cardiovascular disease. The deposits of cholesterol may be formed under the skin in individuals with familial forms of the disorder or in persons with very high levels of cholesterol in the blood. In individuals with hypertriglycemia, several pimple-like lesions may be developed across their bodies. Pancreatitis, a severe inflammation of the pancreas that may be life-threatening can also be developed due to extremely high levels of triglycerides. For diagnosis of hyperlipidemia, levels of total cholesterol, low density lipoprotein cholesterol, high density lipoprotein cholesterol, and triglycerides are measured in a blood sample. It is important to note that the lipid profile should be measured in all adults 20 years and older, and the measurement should be repeated after every 5 years. Food or beverages may increase triglyceride levels temporarily, so people must fast at least 12 hours before giving their blood samples. Special blood tests are carried out to identify the specific disorder when lipid levels in the blood are very high. Specific disorders may include several hereditary disorders, producing different lipid abnormalities and have different risks.

Most blood tests measure levels of LDL (sometimes called "bad") cholesterol, HDL (sometimes called "good") cholesterol, total cholesterol (LDL plus HDL), and triglycerides. To have a low risk of heart disease, desirable lipid levels are:

- LDL less than 130 mg/dL
- HDL greater than 40 mg/dL (men) or 50 mg/dL (women)
- Total cholesterol less than 200 mg/dL
- Triglycerides less than 200 mg/dL

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- **Treatment:**
- The mainstay of treatment for hyperlipidemia is dietary and lifestyle modification, followed by drug therapy, as necessary. Hyperlipidemia should not be considered refractory to dietary treatment if the therapeutic regimen included animal products or more than minimal amounts of vegetable oils. Such diets do not lower LDL cholesterol concentrations as effectively as high-fiber, low-fat diets that exclude animal products
- Regular exercise can improve lipid concentrations. Low to moderate amounts of physical activity such as walking lower triglyceride concentrations by an average of 10 mg/dL, while raising HDL by 5 mg/dL (these numbers are means drawn from large groups). More strenuous activity may have greater effects.[6]
- Patients with familial hypercholesterolemia typically require medication starting in early childhood.
- **HMG CoA reductase inhibitors (statins)** decrease cholesterol production in the liver, and are first-line agents in the treatment of elevated LDL cholesterol. Statins also have important effects on cardiovascular risk aside from their ability to reduce lipid concentrations, and may be indicated for high-risk patients even when lipid targets can be achieved without drug therapy. Potential side effects include myopathy and increased liver enzymes. Some statins may also lower HDL to a below-goal level.
- **Bile acid sequestrants (eg, cholestyramine, colestipol)** are second-line agents for the treatment of elevated LDL cholesterol. These medications can produce gastrointestinal distress, constipation, and impaired absorption of other drugs.
- **Fibrates (eg, gemfibrozil, fenofibrate)** are used as first-line treatment for elevated triglyceride concentrations and may be prescribed in combination with the above drug classes. Gallstones, dyspepsia, and myopathy may occur.

Myopathy risk may be particularly high when fibrates are combined with statins.

- **Nicotinic acid (Niacin)** is a second-line therapy for all lipid disorders. Niacin is often combined with statins, but is also effective as a single agent. Its use is often limited by skin itching or burning. Other side effects include GI distress, hepatotoxicity, hyperglycemia, and gout.
- **Ezetimibe and colesevelam** decrease GI cholesterol absorption, and have emerged as a favored second-line therapy due to their effectiveness, safety, and lack of side effects. They lower LDL and often raise HDL, and are particularly effective when combined with statins (often achieving lipid targets at lower statin doses). Ezetimibe has emerged as the more effective drug.

2. Materials and Methods

Collection and Authentification of Plant Material

The Aerial Parts of *Buteamonosperma* for the study were procured and authenticated

Extraction of Plant Material

The plant is grinded in to a coarse powder with the help of suitable grinder.

Cold Extraction (Methanol Extraction)[38]

In this work the cold extraction process was done with the help of methanol. About 200gms of powdered material was taken in a clean, flat bottomed glass container and soaked in 750 ml of methanol. The container with its contents were sealed and kept for period of 7 days accompanied by continuous shaking with the shaker. The whole mixture then went under a coarse filtration by a piece of a clean, white cotton wool.

Evaporation of Solvent

The filtrates (methanol extract) obtained were evaporated using Rotary evaporator in a porcelain dish. They rendered a gummy concentrate of greenish black. The extract was kept in vaccumdissecator for 7 days.

% Yield value of Methanol Extract from Aerial Parts of *Buteamonosperma* Plant.

Powder taken for extraction = 200gm
Weight of the empty china dish = 53.70gm
Weight of the china dish with extract = 123.24gm
Weight of the extract obtained = (123.24-53.70) gm
= 69.54 gm
% yield of methanol extract = (weight of extract)/(powder taken for extraction) × 100
= 69.54/200 ×100 = 34.7 %.

Acute toxicity studies

The Acute Toxicity Studies was performed using female rats as per OECD Guideline No.423 (Short term toxicity). Male mice were selected of weight around 50 ±10 gm for main test. Single animals are dosed in sequence usually at 48 h intervals. A Dose Progression Factor of 3.2 is used. Using the default dose progression factor, doses would be selected from the sequence (1.75, 5.5, 17.5, 55, 175, 550, 1750, and 5000). However, the time intervals between dosing are determined by the onset, duration, and severity of toxic signs. Treatment of an animal at the next dose should be delayed until one is confident of survival of the previously dosed animal. If the animal survives, the second animal receives a higher dose. If the first animal dies or appears moribund, the second animal receives a lower dose. The toxicological effects were observed in terms of mortality expressed as LD50. The number of animals dying or surviving during a period was noted.

Method of Induction

The systemic administration of the surfactant Triton X-100 to mice results in a biphasic elevation of plasma cholesterol and triglycerides. Hyperlipidemia was induced in Wistar albino rats by single intraperitoneal injection of freshly prepared solution of Triton-X-100 (100 mg/kg) in physiological saline solution after overnight fasting for 18 h.

Bio Chemical Assays for lipids

Estimation Procedures:Plasma Lipid Profile Estimation

Total cholesterol LDL cholesterol, HDL Cholesterol, VLDL cholesterol, Triglycerides levels were measured using commercial kits.

Estimation of Triglycerides. (GPO/PAP Method)

Clinical Significance

Determination of serum Triglycerides concentration is used to assess the possible presence of Increased blood and Serum levels of triglycerides.

Estimation of cholesterol (Total cholesterol) .CHOD/ POD Method[47].

Clinical Significance

Heart disease is often the result of cholesterol deposits on the arteries. While not the only factor for heart disease, serum cholesterol levels are often checked to determine the risk of heart disease on patient.

3. Results and Discussion

Effect of *Caseruasylvestris* Extractson Serum Triglyceride levels.

In the Normal rats the Triglycerides levels were to found be 82.66 ± 2.46 on 0^{th} day respectively. Induction of hyperlipidemia resulted in significantly raised in Triglyceride levels in Group-II, Group-III Group-IV Group-V Group-VI (i.eHyperlipidemic Control, MEBM 500 mg/kg, PEBM 400 mg/kg, PEBM 500 mg/kg, & Standard Atorvostatin 10mg/kg). and the levels were found to be 168.9 ± 5.28, 136.43 ± 7.74, 138.46 ± 1.61, 144.11 ± 7.12, and 148.78 ± 10.23, respectively.

The triglyceride values of hyperlipidemic rats treated with MEBM at dose of 500mg/kg were found to be 117.57 ± 5.25 and PEBM at dose of 400mg/kg and 500mg/kg were 107.93 ± 6.67 and 103.55 ± 4.2. Administration of various doses of the PEBM was able to produce a dose dependant decrease in the triglyceride levels and lowering of triglycerides was dose dependent manner in PEBM. In Standard (Atorvastatin) group the triglycerides was reduced to 102.26 ± 7.68.

Effect of *Caseruasylvestris* Extracts on Serum LDL-C levels: In the Normal rats the LDL-C levels were to found be 8.45 ± 3.43 on 0^{th} day respectively. Treatment with Triton-X-100 caused a significant rise in the levels of LDL-C in Group-II, Group-III, Group-IV, Group-V, Group-VI (i.eHyperlipidemic Control, MEBM 500 mg/kg, PEBM 400 mg/kg, PEBM 500 mg/kg, & Standard Atorvostatin

10mg/kg) and the levels were found to be 136.82 ± 7.00, 122.7 ± 10.93, 132.3 ± 5.05, 139.8 ± 3.44, 130.52 ± 7.98. Administration of various doses of the MEBM &PEBM after the induction of Titon-X-100 resulted in the decreasing of LDL-C levels. The LDL-C levels of groups treated with MEBM at dose of 500mg/kg were 83.58 ± 5.26, and Groups treated with PEBM at dose of 400mg/kg & 500mg/kg were 69.11 ± 10.51 and 59.1 ± 6.89 respectively and lowering of LDL-C was dose dependent manner in PEBM. In Standard (Atorvastatin) group the LDL-C was reduced to 32.91 ± 7.61. The reduction in LDL-C level by MEBM and PEBM was significant at ($p<0.01$).

4. Conclusion

The results concluded that PEBM (500 mg/kg) have definite antihyperlipidemic activity in Triton X-100 induced hyperlipidemic model and which is equipotent activity when compared with Atorvastatin treated groups. Further studies on this extract may lead to identify the possible mechanism of action and isolation of active principle from the same.

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