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A REVIEW: AZADIRACHTA INDICA AS ANTIHYPERLIPIDEMIC ACTIVITY

*** Shalu Baghel[1], Charanjeet Singh[2]**
[1]Department of Pharmacology, Jaipur College of Pharmacy, Jaipur, Rajasthan, India
[2]Principal, Biyani Institute of Pharmaceutical Sciences, Champapura, Jaipur, Rajasthan, India

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Address for Correspondence: Shalu Baghel
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ABSTRACT:
Antihyperlipidemic agents having various pharmacological actions are being tested clinically[3]. Elevated lipid levels result from increased absorption through the gut or enhanced endogenous synthesis and therefore two ways are feasible to reduce hyperlipidemia; to block endogenous synthesis or to decrease absorption. Whole plant of *Azadirachta indica* gives the Antihyperlipidemic activity. Several genetic conditions are known to responsible for primary Hyperlipidemia, such as lipoprotein lipase deficiency, apolipoprotein C-II deficiency etc. In our study we choose cholesterol diet which contains the common ingredients in our daily food. Cholesterol feeding has been often used to elevate serum or tissue cholesterol levels to assess the hypercholesterolemia- related metabolic disturbances in animals. Cholesterol feeding alone however does not affect the serum TG level.
Keywords: *Azadirachta indica,* antihyperlipidemic, antiatherosclerotic activities, Maceration.

1. INTRODUCTION:

Hyperlipidemia is a secondary metabolic dysregulation associated with diabetes. Besides the cause effect relationship with diabetes, elevated serum level of triglycerides, cholesterol and LDL are major risk factors for the premature development of cardiovascular disease like arthrosclerosis, hypertension, coronary heart disease etc[1]. Increased plasma lipid levels mainly total cholesterol, triglycerides and LDL along with decrease in HDL are known to cause hyperlipidemia which is the reason for initiation and progression of atherosclerosis impasse[2].

Antihyperlipidemic agents having various pharmacological actions are being tested clinically[3]. Elevated lipid levels result from increased absorption through the gut or enhanced endogenous synthesis and therefore two ways are feasible to reduce hyperlipidemia; to block endogenous synthesis or to decrease absorption. Both factors can be evaluated in normal animals without artificial diets.

Salacia chinensis (Family:Hippocrateaceae) is a woody climbing plant found in the submontane forests in Sri Lanka and India. The roots and stems of this plant have been extensively used in the treatment of diabetes in the ayurvedic system of Indian traditional medicine. Furthermore, other plant species of the Salacia genus (e.g., S. prinoides, S. reticulata) have been historically used in ayurvedic medicine for their antidiabetic properties[3]. *Salacia chinensis* have been used in India and in other countries as a tonic, blood purifier and to treat amenorrhea and dysmenorrhea[4]. Its root bark is used in treatment of gonorrhoea, rheumatism and skin diseases. Its aqueous extract showed significant hypoglycemic activity. Root bark boiled in oil or as decoction or as powder is used for the treatment of rheumatism, gonorrhoea, itches, asthma, thirst and ear diseases[5].

Patients with Diabetes mellitus DM are at significantly increased risk of CHD compared with non diabetic patients of similar age. DM patients without known CHD appear to have a risk for first myocardial infarction (MI) similar to the risk for recurrent MI of non-DM patients with CHD and a prior coronary event. Patients with type 2 diabetes commonly have other risk factors (hypertension,

high LDL-C, low HDL-C, obesity) that increase risk for cardiac events .

High lipid levels can speed up a process called atherosclerosis, or hardening of the arteries. From inside, arteries are normally smooth and unobstructed , but as increase in age, a sticky substance called plaque forms in the walls of arteries, which is made of lipids and other materials circulating in blood.

As more plaque builds up, arteries can narrow and stiffen. Eventually, enough plaque may build up to reduce blood flow through arteries

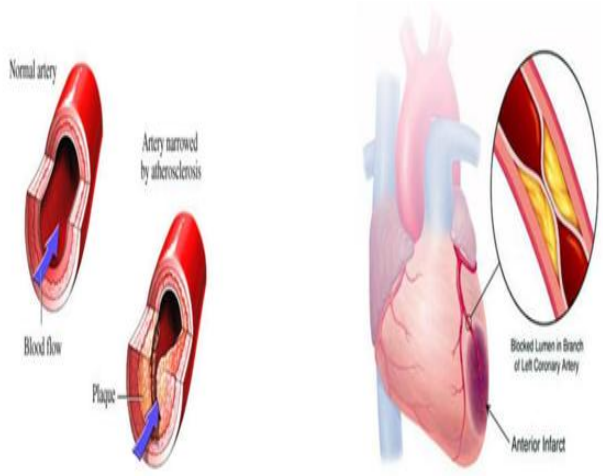

Figure 1:`

Hyperlipidemia is typically asymptomatic and is frequently detected during routine screening.Hyperlipidemia often results from delayed or defective clearance, or overproduction of VLDL by the liver, which is subsequently transformed into LDL. Hypercholesterolemia involves defective hepatic and nonhepatic LDL receptors. Excess intake of saturated fats increases the liver's production of VLDL and triglycerides via a molecular mechanism involving protein activators. Saturated fats are found in animal products, such as meat, whole milk dairy products (milk, cream, cheese), and butter, and tropical oils (palm, palm kernel, and coconut).

High concentrations of total and LDL cholesterol and low levels of high-density lipoprotein (HDL) cholesterol, predicts cardiovascular risk in both men and women. High triglyceride levels have been associated with greater risk in women only. The risk of cardiovascular disease increases by an average of 2%, for each corresponding 1% rise in total cholesterol. Adolescents with high TC or LDL may have a genetic disorder of lipid metabolism such as familial hypercholesterolemia or familial combined hypercholesterolemia. Those with homozygous forms of these disorders can experience myocardial infarction or other events during childhood or early adolescence. Familial hypercholesterolemia is often diagnosed in adolescence and is characterized by high LDL levels that can be refractory to dietary treatment. These patients can present clinically with xanthomas or xanthelasma– cholesterol deposits under the skin on the hands, elbows, knees, heel or eyelids

2. TYPES OF HYPERLIPIDEMIA:

Depending on the complexity of the disease, Hyperlipidemia classified into two types.

a . Primary Hyperlipidemia.

b. Secondary / Acquired Hyperlipidemia.

2.1 Primary Hyperlipidemia:

Several genetic conditions are known to responsible for primary Hyperlipidemia, such as lipoprotein lipase deficiency, apolipoprotein C-II deficiency etc. The primary hyperlipidemia may be treated by anti-lipidemic drugs. Primary Hyperlipidemia are again classified into 5 types.

- Type-I Hyperlipidemia: Severe elevation of chylomicrons (CMs) with resultant elevation of TGs.
- Type-II (A) Hyperlipidemia: Elevations of LDL – C only.
- Type-II (B) Hyperlipidemia: Elevations of both LDL-C and triglycerides (TG's).
- Type-III Hyperlipidemia: It develops due to defect in VLDL remnant Clearance.
- Type-IV Hyperlipidemia: It is characterized by hyper TG's
- Type-V Hyperlipidemia: Characterized by elevated levels of CMs and VLDL.

2.2 Secondary Hyperlipidemia

In this many factors can influence the level of TGs in circulation like diabetes, obesity etc. Secondary Hyperlipidemia demands treatment of original diseases rather than Hyperlipidemia.

3. CAUSES OF SECONDARY HYPERLIPIDEMIA:

- Metabolic influences: Diabetes, obesity, hyperuricemia, glycogen storagediseases.
- Harmonal influences: Insulin, estrogen, thyroxine
- Nutritional influences:-Alcohol, high carbohydrate intake

- Disease states:-Renal diseases, renal failure, nephrotic syndrome
- Drugs: - Diuretics
  Beta-blockers
  Glucocorticoids
  Estrogen replacement

4. CONCLUSION:

It has been well established that nutrition plays an important role in the etiology of hyperlipidimia and atherosclerosis. In our study we choose cholesterol diet which contains the common ingredients in our daily food. Cholesterol feeding has been often used to elevate serum or tissue cholesterol levels to assess the hypercholesterolemia- related metabolic disturbances in animals. Cholesterol feeding alone however does not affect the serum TG level. It is assumed that a high level of saturated fat in addition to cholesterol is required to significantly elevate serum TG level in rat model.

Development of atherosclerotic disease is a complicated process involving accumulation of lipid-containing particles in the walls of coronary arteries other major arteries in the body. Similarly the present study there was a significant weight gain in cholesterol control (toxic), as compared to normal control groups. Treatment with *A. Indica* Leaves extracts significantly reduced the weight gainLowering high cholesterol levels significantly reduce the risk of heart attacks, strokes, and death. A rise in the LDL may cause deposition of cholesterol in arteries and aorta and it is a direct risk factor for CHD.

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