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A Review on Causes and Risk Factors of Hyperlipidemia



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ABSTRACT

Hyperlipidemia is a condition of elevated lipid level in blood. Hyperlipidemia may be defined by serum total cholesterol >240mg/dL and/or LDL cholesterol >160mg/dL and/or total cholesterol: HDL ratio >5.7 and/or total triglycerides >150mg/dL in adults. Hyperlipidemia is a major cause of atherosclerosis and atherosclerosis related conditions like coronary heart disease (CHD), ischemic cerebrovascular disease, peripheral vascular disease and pancreatitis¹⁻². The increase in lipids like low density lipoproteins (LDL), cholesterol (esters derivatives) and triglycerides are mainly responsible for this condition. Many inter-related causes and risk factors are considered for the development of hyperlipidemia and other chronic illnesses of metabolic syndrome (diabetes and hypertension). Exogenous factors, such as dietary intake (fat, cholesterol), alcohol, use of contraceptives and other pharmacologic agents are indicated as the main secondary causes and risk factors of hyperlipidemia in adults. Other secondary causes of hyperlipidemia (diabetes mellitus, alcohol intake, weight gain, physical activity and drugs) need to be included in avoiding hyperlipidemia and its effects/complications. This review article mainly focuses on the causes and risk factors of hyperlipidemia.



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INTRODUCTION

Hyperlipidemia is too much cholesterol in the blood. Cholesterol is a waxy, fat protein produced by the liver and is essential for healthy cell membranes, hormone production, and **vitamin** storage [1]

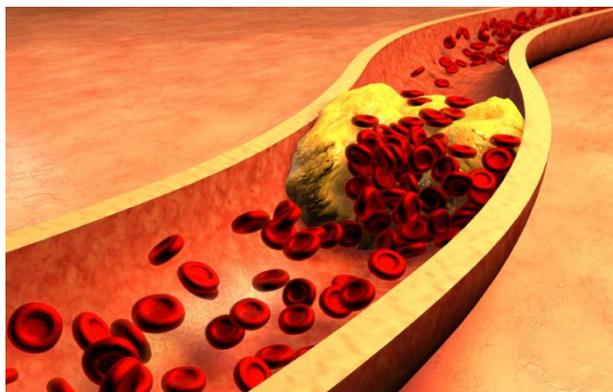


Fig. 1: Hyperlipidemia

The term hyperlipidemia means high lipid levels. Hyperlipidemia includes several conditions, but it usually means that you have high cholesterol and high triglyceride levels. Cholesterol is essential for the proper functioning of the brain. Cholesterol becomes a problem when too much of the bad kind is ingested through regular eating of unhealthy foods. Cholesterol is carried through the blood to cells by lipoproteins that are either low density (LDL) or high density (HDL). The lipoprotein as the vehicle and cholesterol as the passenger. HDL is the good lipoprotein because it carries extra cholesterol back to the liver where it can be eliminated. LDL is bad, it will build up excess cholesterol in the blood. Triglycerides are another type of fat in the blood, are different from cholesterol, but because of their strong association with heart disease, triglycerides are measured as well. Both the LDL and triglycerides that are elevated in hyperlipidemia. In the fasting state, most plasma triglycerides are present in VLDL. In the non-fasting state, chylomicrons appear transiently and contribute significantly to total plasma triglycerides level. LDL contains about 70% of total plasma cholesterol and HDL contains about 20% to 30% of plasma cholesterol. [2]

Causes and Risk Factors of Hyperlipidemia

Dietary Causes Dietary Fats and Fatty Acids: Dietary fatty acids are divided into three major classes (saturated, monounsaturated and polyunsaturated fatty acids). The foods that contribute to saturated fatty acids (e.g. myristic acid, palmitic acid, stearic acid, etc) meats

(e.g. beef, pork, processed meat products, poultry), 2) milk and other dairy products (e.g. butter, cheese, ice cream, yoghurt), 3) tropical fats (e.g. coconut, palm oils) and 4) egg (contain proportionately less saturated fat compared to other animal food sources). Monounsaturated fatty acids are present as oleic acid in olive oil, avocado, animal fats, etc. Polyunsaturated fatty acids are the omega-3 fatty acids (e.g. linoleic acid) and omega-6 fatty acids (e.g. linolenic acid). [2]

Food choices made by individuals can influence intake of the different saturated fatty acids. Selecting leaner cuts of meat are high in palmitic acid and limiting the amount of lean meat would help in lowering saturated fat intake [3]. Milk and other dairy products are high in myristic acid content. Substituting skim milk and non-fat dairy products for whole milk products will result in a reduction of saturated fat such as myristic acid intake.

Dietary Cholesterol: Like other sterols, cholesterol is a sterol i.e. a combination of steroid and alcohol) and lipid (a type of fat). It is found in foods such as eggs and dairy products and is also manufactured in the body, especially the liver. Cholesterol also stabilizes a cell against temperature changes. It is a major part of the membranes of the nervous system, the brain, the spinal cord and the peripheral nerves. In particular, it is incorporated into the myelin sheath that insulates the nerves from the surrounding tissue. Cholesterol is also the forerunner of important hormones such as the female sex hormone, oestradiol and the male sex hormone, testosterone and of vitamin D. Cholesterol is also used to produce the bile which is required to digest the fats in food. Nearly most of the body tissues are capable of making cholesterol, but the liver and intestines make the most.

The dietary cholesterol is responsible for both the development of hypercholesterolemia and atherosclerosis has been the focus of many investigators. Many studies in rabbits (and other animal models) and in human diet and epidemiologic investigations indicated the importance of dietary cholesterol on serum cholesterol levels and its associated effects [3]. However, other investigations have come to opposite conclusions after reviewing numerous human feeding studies (although many continue to support the view that dietary cholesterol is the major hypercholesterolemic and atherogenic nutrient in the diet) [4].

Other Dietary Factors

Carbohydrates:

Dietary recommendations to lower the total fat intake include increasing dietary carbohydrate intake because favorable plasma lipid and lipoprotein levels have been reported for populations and individuals whose habitual diet is rich in carbohydrates. High carbohydrate consumption being associated with a decrease in HDL cholesterol levels. Plasma triglyceride levels are not elevated in these individuals, possibly because obesity is rare [5].

Fiber: Studies have shown that only water-soluble fiber plays a role in lipoprotein metabolism in humans. A meta-analysis of 20 studies found that intake of oat products reduces serum cholesterol levels [5]. The mechanism by which dietary fiber affects plasma lipid levels is unknown. Insoluble fibers in wheat and vegetables do not to reduce cholesterol, but they do have other beneficial effects.

Protein: Soy protein also lowers serum cholesterol levels in animals and in hypercholesterolemic individuals when compared with casein (a dairy protein) and beef proteins. The mechanism underlying these changes is unknown but it has been stated that soy protein affects cholesterol absorption, bile acid absorption, the insulin-glucagon ratio, serum thyroxine levels and hepatic LDL-receptor activity.

Obesity: For a given level of body mass index (BMI), obesity is associated with hyperlipidemia, insulin resistance and hypertension and independent predictor of coronary artery disease (CAD). A meta-analysis of 70 studies indicated that weight reduction was related to increases in HDL cholesterol levels and significant decreases in total, LDL and VLDL cholesterol and triglyceride levels [6]. Although they are not always coincident, obesity is also often accompanied by hyperlipidemia. Both obesity and hyperlipidemia are independently associated with atherosclerosis, non-alcoholic fatty liver disease and insulin resistance [7]

Diabetes and Insulin Resistance: Insulin resistance (type II diabetes) is associated with a number of lipid and lipoprotein abnormalities [8]. The lipid abnormality is associated with insulin resistance and hyperinsulinemia is hypertriglyceridemia. VLDL and total triglycerides are elevated in individuals with type II diabetes although the exact roles of insulin resistance and hypertriglyceridemia are disputed.

Physical Exercise/Activity: Sedentary lifestyles contribute to the development and maintenance of obesity [8]. Diet can also change in plasma lipoprotein concentrations that occur with exercise.

Alcohol Intake: Low dose ethanol consumption in healthy volunteers modestly activates hepatic de novo lipogenesis and that the major quantitative fate of ethanol is acetate produced in the liver. The acetate released into the plasma which inhibits lipolysis in peripheral tissues by 53% and whole body lipid oxidation is decreased by 73%.

Alcohol intake is second only to diabetes mellitus as a cause of hyperlipidemia in the population, about 25% of hospitalized alcoholics have fasting blood triacylglycerol concentrations above normal limits and 17% have concentrations >3 mmol/L. Hypertriglyceridemia is seen mostly in patients with fatty liver and rarely in cirrhosis patients. Patients with cirrhosis have a lower capacity to produce blood lipids than do subjects without liver injury when challenged with diet and alcohol experimentally.

Contraceptives and Other Pharmacologic Agents: Premenopausal women, using oral contraceptives containing a relatively low dose of estrogen combined with a medium or high dose of progestin had a 24 % higher median concentration of LDL cholesterol than who are not using hormones. Glucocorticoids and estrogens elevate triglycerides and raise levels of HDL cholesterol [9].

Antihypertensives have variable effects on lipids and lipoproteins. Although short-term use of thiazide raises cholesterol, triglycerides and LDL cholesterol, long-term usage is not associated with significant alterations in lipid levels. [10]

CONCLUSION

Although food rich in saturated fats and cholesterol are most common cause of hyperlipidemia seen in our society, alcohol excess, diabetes mellitus, weight gain, physical exercise and genetic factors can explain this metabolic syndrome. Several classes of drugs (including contraceptives) to be considered as common causes of altered lipid profiles. A relationship of cause and effect appears to exist among the known chronic metabolic illnesses (diabetes, obesity, hypertension and hyperlipidemia) which show a growing burden on our society. In high cholesterol levels or hyperlipidemia, lipid deposits and damage caused by

reduced blood flow are not limited to major arteries and vessels only. The effect may also occur around the eye and in other parts of the body as well.

REFERENCES

1. Brutis, C.A., E.R. Ashood and D.E. Burns, 2006. Tietz Textbook of Clinical and Molecular Diagnostics. 4th ed. USA. pp: 903-981.
2. Fauci, A.S., E. Braunwald, D.L. Kasper, S.L. Hauser, D.L. Longo, J.L. Jameson and J. Loscalzo, 2008. Harrison's Principles of Internal Medicine. 7 ed. Th.
3. Sereday, M.S., C. Gonzalez, D. Giorgini, L.De Lored, J. Braguinsky, C. Cobeñas, C.Libman, C.Tesone, 2004 Prevalence of diabetes, obesity, hypertension and hyperlipidemia in the central area of Argentina. *Metab.* 30(4): 335-9.
4. Ruixing, Y., C.S. Yuming and P. Shangling, 2006, Comparison of lipid levels, hyperlipidemia prevalence and its risk factors between Guangxi Hei Yi Zhuang and Han populations. *Archives of Medical Research.* 37(6): 787-93.
5. Charney, P., 1999. Coronary Artery disease in women. 2 ed. Philadelphia, USA. pp: 101-159. Nd.
6. Woollett, L.A., D.K. Spady and J.M. Dietschy 1992. Saturated and unsaturated fatty acids independent entry regulate LDL receptor activity and production rate. *J. Lipid Res.*, 33: 77-88.
7. Cortse, C., Y. Levy and E.D. Janus, 1983, Modes of action of lipid-lowering drugs in man; Studies of apo-B kinetics in relation to fat consumption and dietary fatty acid composition. *Eur. J. Clin Invest.* 13: 79-85.
8. Keys, A., J.T. Anderson and F. Grande, 1985. Serum cholesterol response to changes in the diet, Part IV; Particular saturated fatty acids in the diet. *Metabolism.* 14: 776-87.
9. Hegsted, D.M., R.B. McGandy, M.I. Myers and F.J. Stare, 1985. Quantitative effects of dietary fat on serum cholesterol in man. *Am J. Clin Nutr.*, 17: 281-95.
10. Bananome, A. and S.M. Grundy, 1988. Effect of dietary stearic acid on plasma cholesterol and lipoprotein levels. *N. Engl. J. Med.*, 318: 1244-8.

