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A Review on Risk Factors/Indicators and Effects of Hyperlipidemia

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Abstract: Hyperlipidemia or hyperlipoproteinemia is the condition of abnormally elevated levels of any or all lipids and/or lipoproteins in the 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. Objective: The objective of this article is to review up-to-date findings (risk factors/indicators, metabolic effects/complications and prevention/control mechanisms) for such an increasing aspect of metabolic syndrome illnesses in most parts of the world population. Results: Many inter-related causes and risk factors are considered for the development of hyperlipidemia and other chronic illnesses of metabolic syndrome (diabetes and hypertension). Many current studies reveal that there are significant differences in blood lipid levels and the prevalence of hyperlipidemia between ethnic groups, different dietary habits, life style and level of physical activity, as well as genetic background. 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. Once developed, hyperlipidemia results in various effects/complications to the body including atherosclerosis, cholilithiasis and others. Conclusion and Recommendations: An intricate relationship of cause and effect appears to exist among the known chronic metabolic illnesses (diabetes mellitus, obesity, hypertension and hyperlipidemia) which show a growing burden on our today's society in particular. Multiple strategies (including dietary measures) have to be considered to maintain normal blood lipid levels. 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.

Key words: Lipids · Lipoproteins · Hyperlipidemia · Risk factors/indicators · Complications

INTRODUCTION

Hyperlipidemia or hyperlipoproteinemia is the condition of abnormally elevated levels of any or all lipids and/or lipoproteins in the blood [1,2].

Lipids including fatty acids, cholesterol, phospholipids and others are found from exogenous sources (e.g. diet) or synthesized endogenously in the liver and intestines. Lipids have to be transported to the various tissues to accomplish their metabolic functions. Because of their insolubility, they are transported in the plasma in macromolecule complexes with proteins called lipoproteins. The categories of lipoproteins include 1) chylomicrons (Cm), 2)very low density lipoproteis (VLDL), 3) intermediate density lipoproteins (IDL), 4) low-density lipoproteins (LDL), 5) high-density lipoproteins (HDL) and 6) lipoprotein (a)(Lp(a)). 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 triglyceride level. LDL carries about 70% of total plasma cholesterol but very little triglycerides. HDL contains about 20% to 30% of plasma cholesterol. Generally, hyperlipidemia is 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 [1].

There is little information available about the prevalence of chronic metabolic diseases (diabetes, obesity, hypertension and hyperlipidemia) in many countries (especially developing countries). In a study conducted in the central region of Argentina [3], the prevalence of these four diseases in the population aged

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20 years and over indicated; diabetes mellitus was 6% to 8%, obesity was close to 26% and hypertension and hyperlipidemia almost affected one third of the population.

Many causes and risk factors are considered for the development of hyperlipidemia which results in various effects to the body. Many current studies reveal that there are significant differences in blood lipid levels and the prevalence of hyperlipidemia between ethnic groups, different dietary habits, life style and level of physical activity, as well as genetic background [4]. Exogenous factors, such as dietary intake (fat, cholesterol), alcohol, use of contraceptives and other pharmacologic agents are the main secondary causes of hyperlipidemia in adults [1].

Once developed, hyperlipidemia results in various associated effects/ complications including atherosclerosis, cholilithiasis and others. Hence, the objective of this article is to review the intricate inter-related causes/risk factors of hyperlipidemia and its effects/complications to the body.

Causes and Risk Factors of Hyperlipidemia

Dietary Causes

***Dietary Fats and Fatty Acids*:** Dietary fatty acids are divided in to three major classess (saturated, monounsaturated and polyunsaturated fatty acids) (*Table 1*). The foods that contribute to saturated fatty acids (e.g. *myristic acid, palmitic acid, stearic acid*, etc) include 1) 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 mainly 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*) [5].

Several classic studies demonstrated that increase in the percent of calories from saturated fat predicted increases in total plasma cholesterol levels. The mechanisms by which saturated fatty acids raise LDL cholesterol levels have been investigated intensively [6]. In a variety of animal models, down regulation of the LDL receptors, coupled with increased production of cholesterol carrying liporproteins from liver, accounts for the rise in plasma LDL levels. In primary hepatocytes from rats fed saturated fat, levels of LDL receptors mRNA are depressed; similar effects have been observed in other rodents and non-human primates.Other investigations found that saturated fats (in human studies) increased the number of VLDL secreted by the liver and the conversion of VLDL to LDL [7].

Table 1: Major dietary fatty acids by class [5]

Major classes of fatty acids	Examples with their carbon numbers and double bonds
Saturated Fatty Acids	*Lauric acid (C12)*
	Myristic acid (C14)
	Palmitic acid (C16)
	Stearic acid (C18)
Monounsaturated Fatty Acids	*Oleic acid (C18; 1 ^{9})*
Polyunsaturated Fatty Acids	*Linoleic acid (C18;2 ^{9,12})*
Omega-6	*Linolenic acid (C18; 3 ^{9,12,15})*
Omega-3	*Arachidonic acid (C20; 4 ^{5,8,11,14})*

Data from the Keys and Hegsted trials suggested that *myristic acid* may be more cholesterolemic (both total and LDL cholesterol) than *palmitic acid* [8,9]. Both studies assigned a neutral role to *stearic acid*, a role confirmed by more recent studies [10]. The lack of cholesterol raising effect of from stearic acid is due in part to its desaturation to oleic acid and shortly after absorption as well as its higher incorporation in to phosphatidyl choline (versus triglyceride and cholesteryl esters) compared with palmitic acid. Saturated fatty acids, with the exception of stearic acid, suppress clearance of receptor-dependant LDL cholesterol from the circulation.

Food choices made by individuals can impact intake of the different saturated fatty acids. Selecting leaner cuts of meat which are high in *palmitic acid* and limiting the amount of lean meat would help in lowering saturated fat intake. 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 [5].

In both the Keys and Hegsted studies above [8,9], monounsaturated fats specifically *cis-oleic acid* (Figure1) were found to have cholesterol-raising effects. The addition of moderate amounts of *cis-oleic acid* to the diet (in the range of 5%-10% of total calories) is unlikely to have a discernible effect on total and LDL cholesterol.A unique but commercially important monounsaturated fatty acid is *elaidic acid*, the trans-isomer of cis-oleic acid (Figure 1). Individuals receive some trans-fatty acids from dairy foods and ruminant meats, but most people obtain them from products containing commercially hydrogenated vegetable oils (e.g. margarine, shortenings and baked goods). Mensink and Katan [11] published a paper indicating that trans-fatty acids might behave more like saturated fatty acids (i.e cholesterol-raising effect and LDL levels increased).

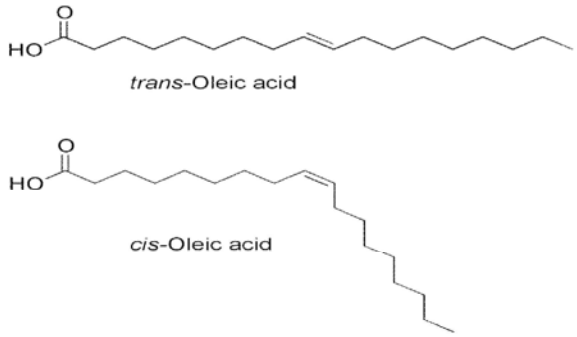

Fig. 1: Comparison of the trans-isomer (top) Elaidic acid and the cis-isomer (bottom) Oleic acid [5].

However, the Joint Task Force of the American Society for Clinical Nutrition and the American Institute of Nutrition conducted that the effects of trans-fatty acids are less adverse than those of saturated fats and trans-fats are reasonable substitutes for saturated fats but not for polyunsaturated fats [12].

Both the Keys and Hegsted studies [8,9] estimated negative regression coefficients for the effects of polyunsaturated classes of fatty acids. When individuals were fed diets high in polyunsaturated fatty acids, their total cholesterol levels were reduced and LDL cholesterol levels fell in other studies as well [11]. The mechanisms for lowering LDL levels during consumption of diets high in polyunsaturated fats are the opposite of those demonstrated for saturates; increased LDL-receptor function and reduced lipoprotein-cholesterol secretion from the liver.

In reports by Mensink and Katan [11] and more recently by Yu *et al* [3], saturated, monounsaturated and poluunsaturated fatty acids were all found to actually have raised HDL cholesterol levels; however, the relative potency of these classes was saturated > monounsaturated >> polyunsaturated. Thus, if monounsaturated fatty acids were used as a replacement for saturated fats, the fall in HDL levels was very slight but was statistically insignificant in most of the small diet studies. If polyunsaturated fats replaced saturated fats, the fall in HDL cholesterol levels was greater (polyunsaturated fats raise HDL about 40% as much as saturated fats) and was statistically significant in most previous studies.

In general, saturated fatty acids increase plasma triglyceride levels moderately, where as polyunsaturated fatty acids reduce them to a similar degree. An exception to the moderate effects of polyunsaturated fatty acids on plasma triglycerides are those observed when large amounts of omega-3 fatty acids (4-8gm/day) are consumed [14].

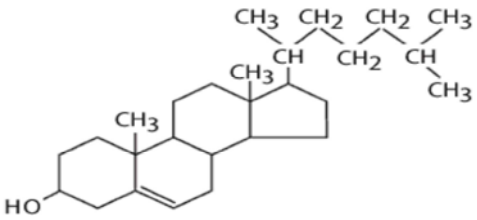

Fig. 2: Structure of Cholesterol [1]

Table 2: Classification of LDL, total and HDL cholesterol (mg/dL) in adult life [1]

Classification of cholesterol	Blood profile (mg/dL)	Level considered
LDL cholesterol	<100	Optimum
	100-129	Near or above optimum
	130-159	High
	≥190	Very high
Total cholesterol	<200	Desirable
	200-239	Borderline high
	≥240	High
HDL cholesterol	<40	Low
	≥60	High

Dietary Cholesterol: Like other sterols, cholesterol is a sterol (a combination of steroid and alcohol) and lipid (a type of fat). It is of high molecular weight and the molecule contains 27-arbon atoms with a molecular formula of $C_{27}H_{46}O$ (Figure 2). Virtually all cells and body fluids contain some cholesterol. It's found in foods such as eggs and dairy products and is also manufactured in the body, especially the liver [1].

Cholesterol is not a life-threatening toxin, but a medium-sized molecule that is really a building block for important parts of the body. In particular it is an essential component of cell membranes. 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 all body tissues are capable of making cholesterol, but the liver and intestines make the most.

Animal products- especially meat, egg yolk, sea food and whole fat dairy products-provide the bulk of dietary cholesterol. On the average, 30% to 60% of dietary and intestinal cholesterol is absorbed daily. With increments in dietary cholesterol, additional cholesterol is absorbed to a maximum of 1gm/day when oral intake reaches 3gm/day. Increased amounts of fat in the diet may also result in expansion of mixed micelles, which in turn allows for more cholesterol to be solubilized and absorbed. In addition to animal cholesterol, approximately 200 to 300 mg of plant sterols are ingested daily. Of the total cholesterol synthesized daily, about 300 to 1000mg is derived from dietary intake. Cholesterol is also synthesized endogenously by the liver and other tissues from simple molecules, particularly acetate [1]. However, although cholesterol is an important molecule, it is associated with various disorders if its blood concentration rises above the normal limits.

The role of dietary cholesterol in 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 [15]. 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) [5].

A reduction in consumption of eggs, a concentrated source of cholesterol (one yolk produces about 215mg cholesterol), had been widely recommended in an effort to lower blood cholesterol. However, studies conducted by Njike *et al* [16] showed that daily consumption of eggs did not unfavourably influence total serum cholesterol or other measures of serum cholesterol. Despite all these conflicting observations, meanwhile the recommended intake of daily dietary cholesterol continues to be 300mg/day or less for healthy adults and less than 200mg/day for individuals with elevated cholesterol or heart disease. Many studies showed (in humans) controversies partially due to individual differences (cholesterol absorption, hepatic synthesis of cholesterol and degradation of cholesterol to bile acids) in serum cholesterol responses to dietary cholesterol. In addition, poor control over nutrient intake other than cholesterol is also responsible for the differences found [5].

Other Dietary Factors

Carbohydrates: Dietary recommendations to lower total fat intake include increasing dietary carbohydrate intake because favourable plasma lipid and lipoprotein levels have been reported for populations and individuals whose habitual diet is rich in carbohydrates. There is, however, concern over reports of high carbohydrate consumption being associated with a decrease in HDL cholesterol levels. Plasma triglyceride levels are not significantly 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 [17]. The mechanism by which dietary fiber affects plasma lipid levels has not been established. Insoluble fibers in wheat and vegetables do not seem to reduce cholesterol, but they do have other beneficial effects.

Protein: Soy protein has been shown to lower 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 not clear but it has been stated that soy protein may affect cholesterol absorption, bile acid absorption, the insulin-glucagon ratio, serum thyroxine levels and hepatic LDL-receptor activity [5].

Obesity: For a given level of body mass index (BMI), obesity is associated with hyperlipidemia, insulin resistance and hypertension and is a significant and independent predictor of coronary artery disease (CAD). A meta-analysis of 70 studies indicated that weight reduction was associated with increases in HDL cholesterol levels and significant decreases in total, LDL and VLDL cholesterol and triglyceride levels [18]. Although they are not always coincident, obesity is often accompanied by hyperlipidemia. Both obesity and hyperlipidemia are independently associated with atherosclerosis, nonalcoholic fatty liver disease and insulin resistance [19]

Diabetes and Insulin Resistance: Insulin resistance (type II diabetes) is related to a number of lipid and lipoprotein abnormalities [20]. The lipid abnormality most closely 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.Generally, insulin resistance is associated with hyperlipidemia characterized by high plasma VLDL and triglyceride concentrations, low HDL cholesterol concentrations and an increase in small, dense cholesterol concentrations [5].

Physical Exercise/Activity: Sedentary lifestyles contribute to the development and maintainance of obesity [5]. Diet can influence the changes in plasma lipoprotein concentrations that occur with exercise. Whether exercise or diet exerts the dominant effect on lipids and lipoproteins depends largely on the level of exercise achieved and the total energy balance [21].

Alcohol Intake: Siler *et al* [22] report that 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 inhibits lipolysis in peripheral tissues by 53% and whole-body lipid oxidation is decreased by 73%. However, chronic liver damage (cirrhosis) caused by the chronic effects of higher doses of alcohol consumption diminishes the liver's capacity to synthesize and export lipids. The interaction of ethanol and lipid metabolism is relevant to the effect of alcohol consumption on body weight and body composition, to the pathogenesis of alcoholic fatty liver and hyperlipidemia. Men and women who drink alcohol tend to have a stable body weight over a decade of observation compared with their nondrinking counterparts, whose weight increases [23]. Energy wastage when ethanol is metabolized by the microsomal ethanol oxidizing system is one reason for relatively low body weight. Another reason is the mitochondrial inefficiency in fatty acid oxidation secondary to chronic ethanol consumption and acetaldehyde toxicity [24].

The pathogenesis of alcoholic fatty liver and alcoholic hyperlipidemia has been known for a long time to be due mainly to a combination of decreased fatty acid oxidation in mitochondria and to increased glycerolipid synthesis. The quantitative data of Siler *et al* [22] from an elegant experiment are consistent and support this generally held view of pathogenesis. An increase in the ratio of NADH to NAD favors conversion of dihydroxyacetone phosphate to glycerol-3-phosphate and glycerolipids. Other factors, derived from alcohol consumption, favor diminished fatty acid oxidation by hepatic mitochondria, including impaired ß-oxidation of fatty acids and the lesser availability of NAD, which diminishes citric acid cycle activity. Both of these processes are also lessened by chronic ethanol consumption, illustrating the problem of extrapolating findings from acute paradigms to chronic situations.

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 (2 mmol/L) and 17% have concentrations >3 mmol/L. Hypertriglyceridemia is seen mostly in patients with fatty liver and rarely in patients with cirrhosis. Patients with cirrhosis have a lower capacity to produce blood lipids than do subjects without liver injury when challenged with diet and alcohol experimentally [22]. Increasing alcohol consumption beyond moderation is associated with increasing heart disease and abstinence from alcohol consumption is associated with more heart disease than is low-to-moderate alcohol consumption (2 drinks/d in humans). The principal change in serum lipids in moderate drinkers is an increase in HDL cholesterol which has a protective role. This change is more persistent than is the increase in triacylglycerols (have been identified as an independent risk factor for cardiovascular disease) and it occurs at a lower level of alcohol consumption.

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 (*Norlestrin*, *Ovral*, or *Demulen*) had a 24 % higher median concentration of LDL cholesterol than did those not using hormones. Women using oral contraceptives that are high in estrogen and low in progestin (*Enovid* or *Oracon*) had significantly higher concentrations of HDL cholesterol than did nonusers; those using *Ovra*l, a low-estrogen and high-progestin formulation, had significantly lower levels of HDL cholesterol [25]

Glucocorticoids and estrogens elevate triglycerides and raise levels of HDL cholesterol [26]. Anabolic steroids taken orally markedly reduce levels of HDL cholesterol in contrast to injectable testosterone, which does not adversely affect the LDL-to-HDL ratio. Oral contraceptives affect atherosclerotic risk depending on the kind and doses of progestin/estrogen. In those with an underlying primary hypertriglyceridemia and

associated obesity, estrogenic medications can depress triglyceride removal mechanisms, leading to the chylomicronemia syndrome and pancreatitis.

Antihypertensives have variable effects on lipids and lipoproteins. Although short-term thiazide usage raises cholesterol, triglycerides and LDL cholesterol, long-term usage is not necessarily associated with significant alterations in lipid levels. Alpha blockers may cause an increase in HDL cholesterol, whereas beta blockers raise triglycerides and lower HDL cholesterol. Sympatholytics, angiotensin converting enzyme inhibitors and calcium channel blockers are essentially lipid neutral. Retinoids can be associated with increased LDL-to-HDL ratios and occasionally striking elevations in triglycerides. Cyclosporine raises LDL cholesterol and lp(a). Classes of drugs that may raise HDL cholesterol include cimetidine, antiepileptic drugs and tamoxifen, but the effect may be seen primarily in women[5].

Genetic Factors: Hyperlipidemia is associated with genetic disorders. Most hereditary lipid disorders are common among generations of families with obesity problems. Some familial lipid disorders can directly result in over production of cholesterol by the body. Another condition called *Familial Combined Hyperlipidemia* (FCHL) can lead to high cholesterol levels including high triglyceride levels. Another hereditary condition called Familial Defective Apolipoprotein B-100, can cause the LDL blood cholesterol (also called the "bad" cholesterol) to increase and also raise total cholesterol levels [27].

Families in which there was a predominance of elevated cholesterol or triglyceride levels were assigned to groups termed familial hypercholesterolemia and familial hypertriglyceridemia, respectively. Pedigrees containing members with hypercholesterolemia and hypertriglyceridemia were said to have Familial Combined Hyperlipidemia (FCHL). The core FCHL lipid profile comprises high serum cholesterol and/or triglyceride levels, elevated apolipoprotein (apoB) levels and increased numbers of small-dense LDL particles [27,28,29]. Patients may also have cholesterol-enriched VLDL and/or reduced HDL-cholesterol and this may be associated with an enrichment of the HDL2 fraction with triglyceride. These changes in lipoprotein composition, which often reflect patients' serum cholesterol and triglyceride levels, may vary over time[30]. In turn, serum cholesterol and triglyceride levels are affected by many factors, including gender, body mass index (BMI), diet (i.e. high carbohydrate and/or fat) and insulin-mediated uptake of glucose by the liver, muscle and adipose tissue.

FCHL is caused by multiple genes and environmental factors as well as by their interactions. Because the genetic background of this common lipid disorder is largely unknown, a significant number of affected individuals are not receiving proper preventative care and are exposed to premature coronary disease. The discovery of the genetic basis for FCHL and its component traits will thus help develop more accurate diagnostic and preventive tools [31].

Effects/Complications of Hyperlipidemia: Coronary artery Disease (CAD) and other cardiovascular disorders (Atherosclerosis)

Hyperlipidemia is associated with the development of CAD in both women and men; however, cholesterol and lipoprotein levels change differently with aging in women and men. Elevated cholesterol, LDL cholesterol, triglyceride levels and low HDL cholesterol levels are predictors of CAD [5].

Atherosclerosis (*Figure 2*) is hardening of a blood vessel from a build up of plaque. Plaque is made of fatty deposits, cholesterol and calcium which builds on the inside lining of large and medium-sized arteries. This causes the artery to narrow and harden. As plaque builds up it can slow and even stop blood flow. This means the tissue supplied by the affected artery is cut off from its blood supply. This often leads to pain or decreased function and this condition can cause a number of serious health problems. Depending on the location of the blockage, it can cause: 1) CAD-loss of blood to areas of the heart and causes the occurrence of angina pectoris or myocardial infarction, 2) stroke-loss of blood to areas of the brain, 3) kidney problems - which loses its function, 4) peripheral vascular disease-characterized by leg pain with walking.

It is well known that elevated blood cholesterol, especially elevated LDL cholesterol, is an important risk factor for cardiovascular disease [32].

Cholelithiasis/Cholesterol Gallstones: Cholesterol cholelithiasis is an extremely common clinical problem in both Europe and North America. Recent estimations indicate that in 1% of adults living in these areas cholesterol gall stones will form and produce symptoms. Cholesterol is a water-insoluble lipid, believed to be dissolved in normal bile by incorporation into molecular aggregates (micelles) of bile acids and lecithin. Current concepts of the pathogenesis of cholesterol gallstones include the assumption that supersaturation of the cholesterol-solubilizing micellar system and consequent

precipitation of cholesterol crystals are necessary for stone formation. Accordingly, much effort has been directed towards determining the circumstances in which supersaturation occurs and ascertaining whether the supersaturation is due to a deficiency of lecithin or bile acids, or both of these solubilizing substances, or to an excess of cholesterol [33].

Bile acid pool size has been measured by isotope dilution techniques and it is found to be reduced in patients with cholesterol gallstones [34].

Effects on Eyes: Corneal arcus is a whitish, gray or yellowish deposit around the circumference of the cornea which is associated with high cholesterol, particularly among those individuals with extremely high levels of cholesterol and those with FCHL. Retinal vein occlusion is also a vascular disease of the eye is a concern in individuals with high cholesterol. Individuals with high cholesterol levels had an approximately 2.5-fold higher risk of retinal vein occlusion, a condition in which a blockage of blood supply from the retina leads to vision loss. Xanthomas which are fatty deposits that occur beneath the skin and produce soft, yellowish bumps on the skin's surface may also be associated with hyperlipidemia [35].

CONCLUSION

Although diets rich in saturated fats and cholesterol are a common cause of t hyperlipidemia seen in our society, alcohol excess, diabetes mellitus, weight gain, physical exercise and genetic factors can explain much of the tendency toward this metabolic syndrome. Several classes of drugs (including contraceptives) need to be considered as common causes of altered lipid profiles. An intricate 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.

In Line with These, the Following Points Are Forwarded:

- Because diets are a mixture of different fatty acids and cholesterol in varying amounts, multiple strategies have to be included to lower intake of saturated fatty acids and cholesterol that raise serum triglyceride and cholesterol levels.
- Other secondary causes of hyperlipidemia (diabetes, alcohol, weight gain, physical activity and drugs) need to be considered in avoiding hyperlipidemia and its effects.
- Lifestyle changes (including feeding habits towards fruits and vegetables) seems to help a lot in combating the growing burden of hyperlipidemia and other chronic illnesses.

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