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REVIEW ARTICLE

HYPERTRIGLYCERIDEMIA: BIOCHEMICAL BASIS AND DIAGNOSIS

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ABSTRACT

Hypertriglyceridemia contribute to increased risk of cardiovascular disease, both directly and indirectly. Hypertriglyceridemia may be primary or secondary in nature. Primary hypertriglyceridemia is the result of various genetic defects and Secondary causes include excessive alcohol intake, metabolic syndrome, untreated Diabetes Mellitus, hypothyroidism, renal or liver disease, pregnancy, autoimmune disorders, and use of certain medications. National cholesterol Education Program Adult Treatment Panel (NCEP ATP) III and the task force members of The Endocrine Society recommended, screening of adults for hypertriglyceridemia as part of a lipid panel [total cholesterol, low-density lipoprotein (LDL), HDL, and triglycerides] on patients beginning at age 20 and repeated at every 5 years. The present review focuses on biochemical findings associated with Hypertriglyceridemia. The data source is from Scopus, PubMed /Medline, EMBASE, science direct, and Google scholar. Mild or moderate hypertriglyceridemia may be a risk factor for cardiovascular disease, whereas severe and very severe hypertriglyceridemia increase the risk of pancreatitis. Therapeutic lifestyle changes (TLC) are the first line of treatment in hypertriglyceridemia before any pharmacologic therapy in the treatment of primary and secondary hypertriglyceridemia

INTRODUCTION

Hypertriglyceridemia (HTG) is defined as an abnormal concentration of triglyceride in the blood [1] . As claimed by the National Cholesterol Education Program Adult Treatment Panel (NCEP ATP III) guidelines, a normal triglyceride level is <150 mg/dl [1]. Elevated plasma triglyceride concentrations contribute to increased risk of cardiovascular disease, both directly and indirectly. The indirect effect is because of the coexisting risk factors like decreased high density lipoproteins, obesity, type 2 diabetes mellitus, metabolic syndrome, proinflammatory and prothrombotic biomarkers [4,5] . Mild-to-moderate hypertriglyceridemia (HTG) is a polygenic disease and severe elevation of Triglyceride (TG) levels can be caused by rare, recessive monogenic disorders [5,6,7]. When a patient's triglyceride level is very high (TG > 885 mg/dl) it is associated with an increased risk of acute pancreatitis, eruptive xanthomas, hepatosplenomegaly and lipemia retinalis [1,4,5,8,9]. The aim of the present review is to focus on biochemical changes that occur in hypertriglyceridemia.

Prevalance

The prevalence of hypertriglyceridemia is increasing in the community. National Health and Nutrition Examination Survey from 2001 to 2006 has found that 32.2% of individuals showed elevated triglyceride levels, including 1.7% with levels considered as severe hypertriglyceridemia (500-2,000 mg/dl) [10,11]. In the United States, the prevalence of hypertriglyceridemia defined as a triglyceride level >150 mg/dl , is ~30% [1-3] . According to epidemiological studies, Indians and migrant South Asians tend to have higher triglyceride levels and lower HDL cholesterol when compared to western populations, while total cholesterol levels are lower than in the US and the UK [12-14]. Prevalence studies from India have also reported greater triglyceride levels in rural and urban populations associated with low HDL cholesterol levels [15].

METHOD

Literature search was carried out over a period of four months from January to June 2018. The literature from 1988 to 2018 was considered for the study. Electronic databases were searched (Scopus, PubMed/Medline, EMBASE, science direct,

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and Google scholar) using the following keywords: "hypertriglyceridemia" and "dyslipidemia". The inclusion criteria for the articles in this review are-

- Studies relevant to the objective of this review.
- Studies with outcome parameters- "hypertriglyceridemia."

All of the articles that met the inclusion criteria were evaluated and selected.

Primary and Secondary Hypertriglyceridemia

Hypertriglyceridemia is classified in to primary or secondary based on the cause [1,4]. Primary hypertriglyceridemia is the result of various genetic defects leading to disordered triglyceride metabolism[1,4]. Secondary causes are acquired causes, such as, high fat diet, obesity, diabetes, hypothyroidism, and certain medications (e.g., thiazides, beta blockers, estrogen, isotretinoin, corticosteroids, bile acid-binding resins, antiretroviral protease inhibitors, immunosuppressants, antipsychotics) [1]. Fredrickson's classification or World Health Organization (WHO) International Classification of Diseases (ICD) hyperlipoproteinemia (HLP) phenotypes(Table 1) is a well-established classification system for hypertriglyeridemia which is based on patterns of lipoprotein fractions[16-21 Familial hypercholesterolemia (FH or HLP type 2A), is the only WHO ICD phenotype that does not have elevated TG levels[16]. The other Five Fedrickson hyperlipidemias contain elevated Triglycerides[16-21].

Table 1. Classification of Hyperlipoproteinemia (Fedrickson's classification) [16].

Name	Primary Lipoprotein Abnormality	Lipid Profile	Clinical manifestations
Familial chylomicronemia (HLP type 1)	Elevated chylomicrons	↑↑↑TG ↑TC	Cutaneous eruptive xanthomata, lipemia retinalis, failure to thrive, recurrent epigastric pain, hepatosplenomegaly, pancreatitis, focal neurologic symptoms
Familial hypercholesterolemia(HLP type2A)	Elevated LDL	↑↑LDL	tendon xanthomata, risk of coronary heart disease
Combined hyperlipidemia (HLP type 2B)	Elevated VLDL, Elevated LDL	↑↑TG ↑↑TC	Physical stigmata such as xanthomas or xanthelasmas are uncommon;
Dysbetalipoproteinemia (HLP type 3)	Elevated IDL, Elevated chylomicron remnants	↑↑TG ↑↑TC	Tuberous and palmar xanthomata Elevations in atherogenic IDL results in increased risk for CVD
Primary simple hypertriglyceridemia (HLP type 4)	Elevated VLDL	↑↑TG ↑TC	Associated with increased risk of CVD, obesity, DM2, hypertension, hyperuricemia, insulin resistance
Primary mixed hyperlipidemia (HLP type 5)	Elevated chylomicrons, Elevated VLDL	↑↑↑TG ↑↑↑TC	Similar clinical manifestations as Type I but develops in adulthood Frequently exacerbated by secondary factors

HLP-hyper lpoproteinemia; TC- total cholesterol; VLDL- very-low-density lipoprotein; LDL- low-density lipoprotein;
IDL- intermediate density lipoprotein; TG-triglyceride.

Biochemical Mechanism Underlying Hypertriglyceridemia

Triglycerides makes up 90% of chylomicrons and 75% of VLDL by weight [16]. During periods of fasting, endogenous triglycerides are secreted by the liver as VLDL and after a meal, over 90% of the circulating triglycerides originate in the intestine and are secreted in chylomicrons [4,22-24]. Lipoprotein lipase is the rate-limiting enzyme in triglyceride metabolism [25]. Lipoprotein lipase is an extracellular enzyme. It is anchored by heparin sulfate to the capillary walls of most tissues predominantly present in those of adipose tissue and cardiac tissue[16]. Both Chylomicrons and VLDL are hydrolyzed by Lipoprotein lipase [16]. So triglycerides are hydrolyzed by Lipoprotein lipase and release non- esterified fatty acids for local use, generating remnant particles within the systemic circulation [11,22-24]. The hepatic receptors, which contain apo (E), recognizes and removes the chylomicrons remnants [26]. The dietary cholesterol from the chylomicron remnant particles is thought to down-regulate the hepatic low-density lipoprotein (LDL) receptors. VLDL remnants, also known as intermediate-density lipoprotein (IDL), contain Apo E and may be removed by the liver through the receptor mediated endocytosis [22] .The increase of triglyceride-rich lipoproteins in plasma results from increased production from the liver and intestine or through decreased peripheral catabolism [4].

Overweight and sedentary life style, are the most general causative factors of hypertriglyceridemia which sequentially lead to insulin resistance, metabolic syndrome (MS) and type 2 diabetes mellitus (T2DM) as well as certain genetic disorders like familial hypertriglyceridemia (FHTG), familial combined hyperlipidemia (FCHL), and familial dysbetalipoproteinemia [1,4,,27,28]. Hypertriglyceridemia triggers the release of free fatty acids (FFA), production of proinflammatory cytokines, fibrinogen, coagulation factors and impairment of fibrinolysis which in turn may also stimulate atherogenesis [27]. Hypertriglyceridemia frequently also leads to reduction in HDL cholesterol and increase in atherogenic small dense LDL cholesterol levels [4,27]. Mild or moderate hypertriglyceridemia may be a precipitate factor for cardiovascular disease where as severe and very severe hypertriglyceridemia intensify the risk for pancreatitis [4,5,8,27, 28]. Types I, IV and V dyslipidemia have also been associated with acute pancreatitis. Types I and V dyslipidemia can cause acute pancreatitis without a predisposing factor, whereas Type IV can do so in the presence of an underlying condition that may increase serum triglyceride levels [25]. Therefore, According to the guidelines of National cholesterol Education Program Adult Treatment Panel (NCEP ATP) III, the task force members of The Endocrine Society recommended, screening of adults for hypertriglyceridemia as part of a lipid panel [total cholesterol, low-density lipoprotein (LDL), HDL, and triglycerides] on patients beginning at age 20 and repeated at every 5 years [27-30]. In healthy asymptomatic patients without risk factors, it is acceptable to obtain a nonfasting total cholesterol and HDL cholesterol level every 5 years [29,30]. On the other hand, for patients with coronary heart disease (CHD), risk factors for CHD or familial dyslipidemia, a fasting lipid panel should be obtained yearly [29,3]. If the triglyceride level is >150 mg/dL, it should be rechecked again after a 12- to 16-hour fast for confirmation. If the triglyceride level is >1000 mg/dl, beta-quantification by ultra centrifugation and electrophoresis can be performed to determine the exact dyslipidemia [29-30].

Investigations

Serum lipid profile is routinely done in fasting state. NCEP and European guidelines also recommended fasting lipid profile for cardiovascular risk assessment which includes estimation of total cholesterol, HDL cholesterol, LDL cholesterol and triglycerides [31-33]. Fasting state refers overnight (12 to 14 hours) complete dietary restriction with the exception of water and medication [33]. Fasting sample is preferred because postprandial triglyceride levels remain elevated for several hours[34], most reference values for serum lipids are

established on fasting blood specimen and the Friedewald's equation, used for calculation of LDL cholesterol (LDL cholesterol = total cholesterol − HDL cholesterol − [triglycerides/5]), uses fasting triglycerides value. If non-fasting triglycerides value is used in this equation the LDL cholesterol, will be underestimated [33]. The Friedewald's equation is not valid for patients with TGs >400 and in patients for type III dyslipoproteinemia [35,36]. So, during the last deacade there was a change from calculated LDL-C to direct measurement of LDL-C (dLDL-C) [36]. But in a study dLDL-C was approximately 10% lower than the corresponding LDL-C results calculated by the Friedewald equation in both men and women, which caused one-third of subjects to be re-classified as having a lower cardiovascular disease risk in relation to recommended LDL-C target values and decision limits. The same discordant pattern was seen in subjects with triglyceride concentrations above 4 mmol/L ($n = 1250$) as for the entire study population [37].

The blood samples in patients with severe hypertriglyceridemia appears lactescent. By undergoing the "refrigerator test", blood samples kept under cool temperatures will result in the separation of chylomicrons (the lipoprotein with the highest concentration of triglycerides) as a milky supernatant [38,39].

NCEP ATP III and the Endocrine Society Guidelines For Classification of HTG

The NCEP ATP III arbitrarily divided fasting serum triglycerides into four different classes (Table 1) [28]. According to this Classification normal fasting TG is <150 mg/dL (<1.7 mmol/L), borderline high TG is 150-199 mg/dL (1.7-2.3 mmol/L), high TG is 200-499 mg/dL (2.3-5.6 mmol/L) and very high TG is ≥500 mg/dL (≥5.6 mmol/L) [23,24]. The Endocrine Society has proposed another system with five clinical strata: normal TG is <150 mg/dL (<1.7 mmol/L), mild HTG is 150-199 mg/dL (1.7-2.3 mmol/L), moderate HTG is 200-999 mg/dL (2.3-11.2 mmol/L), severe HTG is 1000-1999 mg/dL (11.2-22.4 mmol/L) and very severe HTG is ≥2000 mg/dL (≥22.4 mmol/L) [28,29].

Table 2. Criteria proposed for clinical diagnosis of elevated triglyceride levels under fasting conditions[27].

NCEP ATP III			The Endocrine Society 2010[a]		
Normal	<150 mg/dl	<1.7 mmol/liter	Normal	<150 mg/dl	<1.7 mmol/liter
Borderline-high triglycerides	150–199 mg/dl	1.7–2.3 mmol/liter	Mild hypertriglyceridemia	150–199 mg/dl	1.7–2.3 mmol/liter
High triglycerides	200–499 mg/dl	2.3–5.6 mmol/liter	Moderate hypertriglyceridemia	200–999 mg/dl	2.3–11.2 mmol/liter
Very high triglycerides	≥500 mg/dl	≥5.6 mmol/liter	Severe hypertriglyceridemia	1000–1999 mg/dl	11.2–22.4 mmol/liter
			Very severe hypertriglyceridemia	≥2000 mg/dl	≥22.4 mmol/liter

Management

Individuals found to have any elevation of fasting triglycerides should be evaluated for secondary causes of hyperlipidemia such as diabetes mellitus ,hypothyroidism, Metabolic syndrome and medications[1,40,41]. Normally lifestyle modifications are initiated before any pharmacologic therapy in the treatment of primary and secondary dyslipidemia. Therapeutic lifestyle changes (TLC) are the first line of treatment in hypertriglyceridemia. These changes include dietary counseling to achieve appropriate diet composition, alcohol reduction, smoking cessation, and regular aerobic exercise.

A low fat, carbohydrate-controlled diet should be adopted [30]. Saturated fat should not make up more than 7% of total daily calories [42], carbohydrates should be restricted to 50% to 60% of daily calories, and simple sugars like sucrose should be avoided [29,30]. Patients may also consider increasing intake of oily fish (eg, salmon, mackerel, herring) to at least 2 servings per week [43]. High doses of omega-3 fatty acids from fish and fish oil supplements will lower triglyceride levels significantly [28, 43]. Weight reduction in overweight and obese individuals as the initial treatment of mild-to-moderate hypertriglyceridemia [1,28]. A goal of at least 30 minutes of aerobic exercise 5 days a week is greatly beneficial [1,28,29,30]. If TLC and control of secondary medical conditions are not adequate to lower the triglyceride level to <200 mg/dL, then medical therapy is indicated [29,30]. When triglyceride levels range between 200 mg/dL and 500 mg/dL, treatment should be directed primarily toward normalizing the LDL cholesterol. Once the LDL is at goal, a secondary endpoint is the non-HDL cholesterol (total cholesterol-HDL). Non-HDL goals are 30 mg/dL higher than LDL goals. A tertiary treatment goal, particularly in the setting of CHD or CHD risk equivalents, is to raise the HDL to >40 mg/dL[29,30]. In cases of isolated hypertriglyceridemia, fibrates are initially considered [[29-31]. When elevated low-density lipoprotein levels accompany hypertriglyceridemia, 3-hydroxy-3-methylglutaryl coenzyme A (HMG-CoA) reductase inhibitors (statins) are preferred [29-31]. In patients with low HDL levels and hypertriglyceridemia, extended release niacin can be considered. A combination of the medicines may be necessary in recalcitrant cases [30].

CONCLUSION

The prevalence of hypertriglyceridemia is increasing in the community. As there is increased risk for cardiovascular disease and pancreatitis hypertriglyceridemia should be diagnosed early. NCEP ATP III and European guidelines also recommended fasting lipid profile on patients beginning at age 20 and repeated at every 5 years for early detection. Normally lifestyle modifications which includes dietary counseling to achieve appropriate diet composition, alcohol reduction, smoking cessation, and regular aerobic exercise are the first line of treatment in hypertriglyceridemia. Life style intervention is followed by pharmacotherapy.

Conflicts of Interest: There is no conflict of interest.

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