Risk of Dis-lipids Syndrome in Modern Society

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Abstract

Aim of this work was to emphasis the preclinical evaluation of dis-lipids syndromes types at the patients which were presented to a routine control for checking health status, in the hospital ambulatory.

Material and Method:

Were analyzed 60 patients, registered in Clinical Laboratory, assessing by running on the Hitachi 912 Analyzer, the principal biochemical parameters of lipid metabolism: Cholesterol, Triglycerides and fractions of Cholesterol, HDL and LDL. From the total of 60 patients 35 were females and 25 males.

Results

The persons with an alarm signal of atherosclerotic process were in 28 % and persons with low HDL was in 17%. The cases with atherosclerotic index, report-LDL/HDL>3.5 for men and 2.5 for women were in 14 % , the cases with predictive value with coronary risk, report-CO/HDL>5 were presented in 5 % and the cases with dis-lipid syndrome type 2- 4, with high Cholesterol and Triglycerides, were presented in 30% percent.

Conclusions

Lipids controls, and its fractions, are necessary to be prevented atherosclerotic process in the incipient status of ill.

Key words: low density lipoproteins, very low density lipoproteins, intermediate density lipoproteins, cholesterol, triglycerides, chylo-microns.

Introduction

In the all developed countries, the numbers of obese persons, diagnosed with insulin resistance (IR) have increased rapidly to >40% in recent years. IR is a patho-physiological state characterized by a subnormal physiological response to insulin concentrations. This state precedes the development of metabolic syndrome (MS). Insulin resistance is often considered a pre-diabetic condition [1].

MS consists of multiple, interrelated risk factors of metabolic origin that appear to promote the development of atherosclerotic cardiovascular disease (ASCVD) and which are strongly associated with type 2 diabetes mellitus or the risk for this condition [2].

The metabolic risk factors consist of atherogenetic dyslipidemia (elevated triglycerides and apolipoprotein B, small LDL particles, low HDL cholesterol [HDL-C] concentrations), elevated blood pressure, elevated plasma glucose, a prothrombotic state, and a proinflammatory state [3].

The World Health Organization (WHO) has slightly different criteria for metabolic syndrome, including high insulin levels, elevated fasting blood glucose, or elevated post-meal glucose alone with at least two of the following criteria: abdominal obesity (defined as a waist-to-hip ratio greater than 0.9), a body mass index of ≥30 kg/m2 or a waist measurement >37 inches, a lipid panel showing a triglyceride level ≥150 mg/dl or an HDL cholesterol >35 mg/dl an blood pressure ≥ 140/90, (or receiving treatment for high blood pressure), [4].

Dis-lipids syndromes mean the metabolic statuses with qualitative and quantitative modifies of lipoproteins. Hyper-lipids syndromes means increases concentration of triglycerides and or cholesterol past acceptable limits in function of age, sex and health status. Hyper-cholesterol isolate status means high concentration of fraction low density lipoproteins (LDL), hyper-triglyceride status means high concentration of very low lipoproteins, (VLDL) and seldom chylomicroms and hyper-lipids mixed syndromes mean together high LDL and VLDL increased, seldom intermediate density lipoproteins (IIDL), [5].

Lack of receptors for LDL to fibroblasts cells and adipose cells will develop familial high-cholesterol with increases in serum of LDL-oxizing, together with cholesterol and triglycerides which will determine premature atherosclerosis [6].

The rapport: Total Cholesterol/ HDL (high density lipoproteins) have the predictive value for coronary risk if its value is higher or equal with 5. The report LDL/ HDL, with predictive value past >3.5 for men and past>2.5 to women, mean atherosclerotic incipient index [7].

The diet and exercise combination presented in this study proved to be an effective treatment for MS, and this or a similar regimen should be recommended by physicians to patients who show signs of MS. For patients who show limited results from exercising on a stationary bicycle (or other exercising methods), drug treatment, such as specific treatment for dyslipid disorder, administration of leptins, leptin genes, or promoter drugs may be necessary, in addition to physical exercise and maintenance of an appropriate diet [8].

Further studies, which should include an appropriate control group, are necessary to confirm these findings, which can be regarded at present as only a hypothesis derived from the signs and symptoms of MS.

Scope

The aim of this work was to emphasis the preclinical evaluation of dis-lipids syndromes types at the un-treated patients for any disease, which were presented to a routine control for checking heath status in ambulatory.

Material and Method:

In our study we have analyzed 60 patients, registered in Clinical Laboratory**,** assessing by running on the Hitachi 912 Analyzer, with the principal biochemical parameters of lipid metabolism: Cholesterol, Triglycerides and fractions of Cholesterol, HDL and LDL.

Cholesterol was determined enzymatic using cholesterol esterase and cholesterol oxidase reagents. Cholesterol esters is cleaved by the action of cholesterol esterase to yield free cholesterol and fatty acid; Cholesterol esters + H2O > cholesterol > cholesterol + RCOOH; Cholesterol is converted by oxygen with the aid of cholesterol oxidase to cholesten-4, one and hydrogen peroxide. The hydrogen peroxide created forms a red dyes-tuff by reacting with 4 amino-phenazone and phenol under the catalytic action of peroxidase. The color intensity is directly proportional to the concentration of cholesterol and can be determined photometrical

Triglycerides are esters of the triglyceride alcohol glycerol with 3 long-chain fatty acids. To glycerol followed by oxidation to di-hidroxy-acetone-phosphate and hydrogen peroxide. The hydrogen peroxide produced then reacts with 4-aminophenazone and chlorophenol under the catalytic action of peroxidase to form a red dyestuff, (Trinder endpoint reaction).

The cholesterol concentration of **HDL-cholesterol** was determined enzymatic by cholesterol esterase and cholesterol oxidase coupled with polyethylene, (PEG) to the amino groups (approx 40 %). Cholesterol esters are broken down quantitatively into free cholesterol and fatty acids by cholesterol esterase. In the presence of oxygen, cholesterol is oxidized by cholesterol oxidase to Δ4-; cholestenone and hydrogen peroxide. The color intensity of this of dye is directly proportional with to the cholesterol concentration and is measured photometrical.

LDL Cholesterol was measured as direct method by Analyzer Hitachi 912 with the specific reagents. Cholesterol esters are broken down quantitatively into free cholesterol and fatty acids by cholesterol esterase. LDL-cholesterol esters>> Detergent >> Cholesterol + free fatty acids; LDL cholesterol + O2 Cholesterol oxidase → Δ4 – cholestenone+H2O2; 2H2O2 + 4 aminoantipyrine + Peroxidase purple blue pigment+5H2O, In the presence of oxygen, cholesterol is oxidized by cholesterol oxidase to cholestenone and hydrogen peroxide.

From the total of 60 patients 35 were females and 25 males. (Mean age for females was 27-45 years females and 37-52 years for males). All patients have not been in evidence with known cardiovascular, chronic, metabolic or endocrine diseases. All our results obtained

were in validation after that was accomplished calibrations, internal controls and reference interval established on 120 known apparent health patients, analyzed in clinical laboratory, on each parameter measured. Interval References**:** Cholesterol = 114-225 mg/dl), Triglyceride =53-145 mg/dl), HDL=36-60 mg/dl) and LD=48-130 mg/dl.

Results:

By processing of results from upper table we registered following synthesis:, the persons with metabolic lipid health, were registered in 23% percent, the patients which has presented isolate high LDL as an alarm signal of start, atherosclerotic process, were in 28 % percent in time that low HDL was in 17% percent.

The cases with atherosclerotic index, Report-LDL/HDL>3.5 for men and 2.5 for women were in 14 % percent, the cases with predictive value with coronary risk, Report-CO/HDL>5 were presented in 5 % percent, the cases with dis-lipid syndrome type 2- 4, with high Cholesterol and Triglycerides were presented in 30% percent.

Discussion:

Cholesterol is a steroid with a secondary hydroxyl group in the C3 position. It is synthesized in many types of tissue, but particularly in the liver and intestinal wall. Approximately three quarters of cholesterol is newly synthesized and a quarter originates from dietary intake. Cholesterol esters are elevated by the action of cholesterol esterase to yield free cholesterol and fatty acids [9].

The determination of triglycerides is utilized, nephrosis, liver obstruction, lipid metabolism disorders and numerous other endocrine diseases. Triglycerides: Reference of reagent = 50-150 mg/dl, expected range :< 2.26 mmol/L (<2 00mg/dL), properly interval of reference:=53-145 mg/dl [10], [Table 1].

HDL is responsible for the reverse transport of cholesterol from the peripheral cells to the liver. Here, cholesterol is transformed to bile acids which are excreted into the intestine via the biliary tract. Monitoring of HDL – cholesterol in serum is of clinical importance since an inverse correlation exists between serum HDL – cholesterol concentrations and the risk of atherosclerotic disease [11].

Elevated HDL- cholesterol concentrations are protective against coronary heart disease, while reduced HDL- cholesterol concentrations, in conjunction with elevated triglycerides, increase the cardiovascular risk. HDL cholesterol is affected by a number of factors e.g. smoking, exercises, hormones, sex and age.

Education Program (NCEP) National Cholesterol guideline; shows low HDL cholesterol (major risk factor for CHD), ≥60mg/dL (1.56 mmol /L): high HDL cholesterol (negative risk factor for CH [Table 2], [12].

HDL-CO and Cardiovascular Risck

The cholesterol content of HDL is conventional used to assess the multivarious antithrombotic and immune related function of HDL particles. The reliance on HDL-CO in clinical practice partly derives from its use at a principal component of the friedwald equation used to estimate LDL cholesterol.in multivariable models ajusted for both nonlipid and lipid triglycerides and non-HDL colesterol risk factor, HDL colesterol is in early associated with CHD events [13].

For every 0.39 mmolL (15 mg/dl), increase in HDL cholesterol concentration, the risk of CHD was reduced by 22%, (95% CI, 18%-26%). Low concentrations of HDL cholesterol predict CHD mortality equaly well in nondiabetic and diabetic patients. HDL cholesterol can be separated on the basis of size and charges, by ultracentifugation and MALDI-TOFFI. The concentration of these particles are expressed in miligrams per liter of apolipoprotein A-1, (Apo A-1), and as perecentage of total plasma A1 concentration.

Pre-beta (pre B-1), HDL particles mobility, are most efficient in interacting with ATP binding cassete transporter A-1(abc a-1), to promote cholesterol efflux from cells, whereas large alpha -1 HDL are most efficient in interacting with the scavenger receptor B1 for delivery of cholesterol to the liver. When the concentration of apo A1 in alpha-1 HDL is less than 140 mg/dl, the individual is at increased risk of developing CHD [14].

LDL plays a key role in causing and influencing the progression of atherosclerosis and in particular, coronary sclerosis. The LDL is derived from VLDL rich in triglycerides by the action of various lipolytic enzymes and which are synthesized in the liver. The elimination of LDL from plasma takes place mainly by liver parenchymal cells via specific LDL receptors, [15].

Elevated LDL concentrations in blood and an increase in their residence time coupled with an increase in the biological modification rate results in the destruction of the endothelial function and a higher LDL-cholesterol uptake in the monocytes/machropages system as well as by smooth muscle cells in vessel walls. The majority of cholesterol stored in atherosclerotic plaques originates from LDL [16].

The LDL – cholesterol value is the most powerful clinical predictor among all of the single parameters with respect to coronary atherosclerosis. Therefore, therapies focusing on lipid reduction primarily target the reduction of LDL-oxidated – cholesterol, which is then expressed in an improvement of the endothelial function, prevention of atherosclerosis and reducing its progression as well as preventing plaque rupture. Expected: Reference of Reagent= 39-120 mg/dl [Table 3].

Weight loss is generally good for you if you are seriously overweight, but bone loss can cause significant problems later in life. In the study, rats who received leptin had a weight reduction of about 20 percent, but they did not have any bone loss. The rats that lost weight were able to maintain that weight loss. They also had large reductions of abdominal fat, also known as "bad" fat, which is known to contribute to weight-related health problems [17].

Leptin is required for normal skeletal growth and maintenance. The hormone also plays a significant role in the body's ability to maintain weight, telling your brain how much fat you have and if fat stores are sufficient. But people appear to develop leptin resistance with weight gain, and the brain no longer receives accurate messages.

Conclusions

Lipids controls, on fractions of Cholesterol, LDL, HDL**,** Triglycerides are necessary to be prevented atherosclerotic process in incipient status of ill. Familial dis-lipids syndromes must be analyzed at young ages, to different, apparent health persons, with ages past 35 years. Further information must to be accomplished by electrophoresis lipid, ultracentrifugation in density gradient, and measurement of apolipoprotein-B, by ELISA equipment [Table 4]

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CHOLESTEROL and TRIGLYCERIDES

CLINICAL INTERPRETATION OF RESULTS:

According to the recommendations of the European Atherosclerosis Society:

		mg/dL	Lipid metabolism disorder
Cholesterol **Triglycerides**		**<200** **<200**	**44 case = 26.4%** **No**
Cholesterol		**200 – 300**	**11 cases = 6.6%** **Yes if HDL – cholesterol <45 mg/dL for women and < 35% for men**
Cholesterol **Triglycerides**		**>300** **>200**	**5 cases=3% Yes**

Table 1.Results of investigated cases for Cholesterol and Tryglicerides

Desirable cholesterol level : < 5.2 mmol/L (<200 mg/dL), borderline high cholesterol: <5.2-6.2 mmol/L (200 – 239 mg/dL), high cholesterol: ≥6.2 mmol/L (≥240 mg/dL), cholesterol reference reagent = 180-240 mg/dl.

HDL- CLINICAL INTERPRETATION OF RESULTS

	No risk	Moderate risk	High risk
Men	Men	Men	Men
Mg/dL	>55	35-55	<35
Women	Women	Women	Women
mg/dL	>65	45-65	<45

Table 2. ‾Expected values: for HDL-Co

National Cholesterol Education Program (NCEP) guidelines: < 40mg/dL (1.04mmol dL): low HDL cholesterol (major risk factor for CHD), ≥60mg/dL (1.56 mmol /L): high HDL cholesterol (negative risk factor for CHD)

LDL- INTERNAL QUALITY CONTROL ON HITACHI ANALYZER

LDL-Cholesterol	Within-run LDL-CO			Between-run		
Sample		MEAN	cv %	mean	mean	cv %
	Mg/d/L			Mg/d/L		
Human serum1	50	65	0.71	74.9		1.20
Human serum 2	160.3		0.81	179.5		1.16
Precinorm L	89-121	105	1.22	88.0		0.96
Precipath HDL/LDL-C	18.2		0.64	179.5		0.87

Table 3. LDL Co, expected values: optimal =<100 mg/dl, 60-99 mg/dl, near optimal= 100-129mg/dl, borderline High=160-189, very High >190, properly reference interval =48-130 mg/dl.

METABOLISM OF LIPIDS FRACTIONS

Type	Electrophoresis Aspect	Lipo- protein composition	Causes	Mechanism
IV VLDL	Composition	Triglycerides 60%	Ester cholesterol endogenous 12%	Phospho-lipids 15% and major Apo-protein B100.
II a LDL	Composition	Cholesterol esters, 40%, Trygliceride, 10% and Apo B100, 20%	Family high - cholesterol	Deficit of receptor LDL
II b LDL and VLDL	Increasing level of plasmatic fraction pre-beta and beta	VLDL and LDL	High-lipids family with combination	Amplification of synthesis Hepatic apo B.
III IDL	Band beta large	VLDL-→ LPL→ =IDL with low triglycerides.	High lipids with increasing IDL	Abnormal APO -E.
IV	Increasing of level plasmatic al fraction Pre-beta	VLDL	Familial high-triglycerides	Increasing of synthesis hepatic apo- B and triglycerides.
V	Increasing of level plasmatic fraction pre-beta	VLDL and chilomicroms	Familial high triglycerides	Amplification of synthesis hepatic triglycerides.

Table 4. The combination of fractions cholesterol in dis-lipids familial syndromes