Clinical and Biochemical Correlations Obesity - HDL Cholesterol - Ischemic Coronary Heart Disease

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Central obesity is defined by increasing abdominal circumference ranging from certain limits to the characteristics of the ethnic groups of the rated person (> 94 cm for European males and > 80 cm for Euclid women). The metabolic syndrome, commonly associated with central obesity, must meet at least two of the following four criteria: increased triglyceride levels > 159 mg/dL or specific treatment for this type of dyslipidemia; Low HDL-cholesterol < 40 mg/dL or specific treatment for this type of dyslipidemia; increased blood pressure: Systolic BP ≥ 130 mm Hg or Diastolic BP ≥ 85 mm Hg, or HTA treatment previously diagnosed; High blood glucose level ≥ 100 mg/dL or type 2 diabetes diagnosed previously. HDL-cholesterol (HDL-C) is a group of lipoprotein synthesized and secreted by hepatocytes. HDL plays an important role in the metabolism of cholesterol, participating in its transport from extrahepatic tissues to the liver for catabolism and excretion. HDL and LDL participate in maintaining cell cholesterol levels. HDL-cholesterol and apolipoprotein-A values are positive atherosclerosis risk factors. The high level of HDL-C protects the patient, with a low risk of atherosclerosis. HDL dosing is done for assessing the risk of coronary artery disease; diagnosis of hyperlipoproteinemia. Increased body weight is associated with increased total mortality and morbidity due to cardiovascular disease, partly mediated by increased blood pressure and cholesterol, lowering HDL-cholesterol and increasing the likelihood of diabetes.

Keywords HDL-cholesterol, obesity, coronary ischemic disease.

Epidemiological studies have confirmed in recent years that patients with low HDL-cholesterol levels are at high risk of developing coronary artery disease and myocardial infarction [1, 2].

The prolongation of circulating persistence of TG-rich lipoproteins leads to an increase in TG exchange from these particles with HDL2 and LDL-cholesterol esters (type A).

These lipid shifts are mediated by the esterified cholesterol transfer protein (CETP) and cause the HDL2 and LDL content to decrease in cholesterol and increase the TG in cholesterol.

TG from the HDL2 and LDL particles are lysed under the action of hepatic lipase and, after this intervention, result in smaller and more dense HDL3 and LDL particles.

Low HDL3 and small and dense LDL particles are rapidly removed from the circulation, which contributes to lowering plasma HDL [3, 4]. In the Framingham Heart Study and Quebec Cardiovascular Study, the total / HDL cholesterol ratio was the highest predictive value lipid index.

Experimental part

This study evaluated patients diagnosed with obesity in which we quantified the degree of abdominal obesity (CMA), we compared gender, age, evaluated cardiovascular function (presence of hypertension, ischemic coronary artery disease), we studied the presence of disorders endocrine associated by evaluating thyreotropic and corticotropic axes by TSH, FT4, ACTH and cortisol dosing (table 1) and followed the HDL-cholesterol plasma dynamics.

To assess cardiovascular risk we determined HDL-cholesterol, knowing that many epidemiological studies have shown that patients with low plasma levels are at increased risk for coronary heart disease and myocardial infarction [5, 6]. Patient preparation requires that the patient observe a diet unchanged for 3 weeks before harvesting; - have a constant weight; [7].

- not to eat 12-14 h before harvesting (can drink water and black coffee without sugar);

Abstinence from alcohol is recommended 72 h before harvesting;

Dosing technique for HDL cholesterol. Specimen harvested - venous blood [8, 14].

Vacuum container without anticoagulant with / without separator gel.

Processing required after harvest - separate the serum by centrifugation; work the same day or store at 4°C times -20°C.

Volume sample - minimum 0.5 mL serum.

Causes of sample rejection - hemolyzed intensive specimen.

Stability of the test sample is stable for 5-7 days at 2-8 °C; 3 months at -20 °C; a few years at -70 ° C.

Method - spectrophotometric (enzymatic).

<table>
<thead>
<tr>
<th>Interpretation</th>
<th>Value (mg / dL)</th>
</tr>
</thead>
<tbody>
<tr>
<td>Normal</td>
<td>&gt; 40</td>
</tr>
<tr>
<td>Protect factor</td>
<td>&gt; 60</td>
</tr>
</tbody>
</table>

Table 1 REFERENCE VALUES

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All authors contributed equally
Total cholesterol / HDL ratio:
- Low risk: 3.3-4.4
- Average risk: 4.4-7.1
- Moderate risk: 7.1-11
- Increased risk: > 11
Conversion factor: mg / dL x 0.026 = mmol / L

Detection limit - 0.08 mmol / (3 mg / dL) [8,9].
Clinical alert values - <40 mg / dL: are associated with increased risk of coronary artery disease [10-12]
The results are presented in table 2.

**Methods of statistical and mathematical processing used**
SPSS, specialized in statistical statistical calculations, produced by SPSS and the Data Analysis module of MICROSOFT EXCEL, together with the XLSTAT suite for MS Excel, were used for data processing.

The Cramer test verifies the association power between two nominal factors and is used for multi-row tables and columns (for tables 2x2 the phi coefficient is preferred), more precisely measures if each category of one of the factors is preferentially associated with one of categories of the other factors.
The result of this test is recorded with V.
where r and c are the number of rows and columns in the studied incidence table.

The Student t test of comparing the averages for two lots proposes two statistical hypotheses:
- H0 hypothesis (or null hypothesis): the difference between environments is incidental;
- H1 hypothesis: the difference between averages is statistically significant
The p result of the test is probability of making an error if the hypothesis H0 of the test is rejected, a result provided as a number between 0 and 1.
If p is less than 0.05, we reject the hypothesis H0, the null, and admit that the hypothesis H1 is true.
In the comparing test of the media (Student test), we used the following interpretation of the p values, provided directly by the program with which the statistical data processing is performed, by applying the above test:
- p <0.05, the difference between the two media is significant (S);
- p <0.01, the difference between the two media is highly significant (HS);
- p <0.001, the difference between the two media is very high (VHS);
- p > 0.05, the difference between the two media is insignificant (NS).

**Results and discussions**
In the study, we had 118 women and 54 men, the gender distribution of HDL was the following (table 3, fig. 1)
- 48.26% of women and 5.23% of men showed normal HDL (greater than or equal to 50 mg / dL for women and greater than or equal to 40 mg / dL for men);
- The remaining 20.35% of women and 26.16% of men had low HDL (below 50 mg / dL in women, respectively below 40 mg / dL in men).
- From this study, it results that a higher percentage of hipo-HDL cholesterol is compared with that of women (and therefore increased risk for atherosclerotic disease.)

Depending on the association of obesity with endocrine changes we have divided the patients into 3 lots
- group I, patients with obesity not associated with endocrine disorders
- group II, patients with obesity associated with Cushing's disease
- group III, patients with obesity and hypothyroidism.

The batch distribution of HDL was as follows (table 4, table 5):
- In group 1 there were 46 patients (51.69%) with normal HDL and 43 patients (48.31%) with low HDL
- In group 2 there were 37 patients (60.66%) with normal HDL and 24 (39.34%) low HDL patients;
- 9 patients (40.91%) with normal HDL and 13 patients (59.09%) with low HDL were recorded in group 3.

The HDL-grade obesity correlation is as follows (fig. 2).

<table>
<thead>
<tr>
<th>HDL</th>
<th>Lot 1</th>
<th>Lot 2</th>
<th>Lot 3</th>
<th>Total</th>
</tr>
</thead>
<tbody>
<tr>
<td>Minimum</td>
<td>32</td>
<td>32</td>
<td>32.8</td>
<td>32</td>
</tr>
<tr>
<td>Maximum</td>
<td>65</td>
<td>55</td>
<td>51</td>
<td>65</td>
</tr>
<tr>
<td>Average</td>
<td>44.31</td>
<td>43.13</td>
<td>39.59</td>
<td>43.29</td>
</tr>
<tr>
<td>Dev.std.</td>
<td>7.69</td>
<td>5.29</td>
<td>5.31</td>
<td>6.75</td>
</tr>
<tr>
<td>CV</td>
<td>17.15%</td>
<td>12.27%</td>
<td>13.40%</td>
<td>15.55%</td>
</tr>
</tbody>
</table>

Table 4

DISTRIBUTION OF MINIMUM, MEAN AND MAXIMUM VALUES OF HDLC BY LOTS

Of the 59 patients with obesity, grade I: 1 patient (1.69%) had a normal HDL and 58 patients (98.31%), low HDL values;

Of the 76 patients with grade II obesity: 1 patient (1.32%) had a normal HDL and 75 (98.68%) low HDL.
- All 37 patients with grade III obesity had low HDL.

In conclusion, the degree of obesity and abdominal obesity is a risk factor for hipo-HDL cholesterol. This is explained by the association of the hepatic AGL influx, which stimulates the hepatic synthesis of large TG-rich VLDLs which, through changes after release into the bloodstream, will lead to increased plasma concentrations of small and dense LDL and decreased plasma HDLc. Hypertriglyceridemia causes decreased plasma concentrations of HDL2 particles and increased HDL3.

This change in HDL increases cardiovascular risk, as HDL2 particles have antatherogenic effect. HDL and LDL are independent predictors of cardiovascular risk.

Incidence of painful coronary artery disease
The distribution of patients with painful BCI by batch is as follows (table 6):
- In group 1: 28 patients (31.46%) were diagnosed with Painful BCI based on typical angina pain and ischemic changes on the resting ECG;
cholesterol and one patient had normal, in those with obesity grade 75 they had low HDL cholesterol and one patient had normal, and in those with obesity Grade 3 37 patients representing the whole lot had low HDL cholesterol.

In the study, we identified 34.88% of patients with painful ischemic heart disease and 23.84% of patients with painless ischemic heart disease diagnosed on the basis of clinical picture and resting ECG, and after the exercise ECG test, another 4.65 % of patients were diagnosed with BCI. There were no significant differences in the distribution of BCI per lot (p> 0.05)

References
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Table 6
DISTRIBUTION OF PATIENTS WITH PAINFUL BCI BY LOT

<table>
<thead>
<tr>
<th>BCI painful</th>
<th>Yes</th>
<th>No</th>
<th>Total</th>
</tr>
</thead>
<tbody>
<tr>
<td>Lot 1</td>
<td>28</td>
<td>61</td>
<td>89</td>
</tr>
<tr>
<td>Lot 2</td>
<td>24</td>
<td>37</td>
<td>61</td>
</tr>
<tr>
<td>Lot 3</td>
<td>8</td>
<td>14</td>
<td>22</td>
</tr>
<tr>
<td>Total</td>
<td>60</td>
<td>112</td>
<td>172</td>
</tr>
</tbody>
</table>

Table 7
DISTRIBUTION OF PATIENTS WITH BCI PAINLESS ON BATCHES

<table>
<thead>
<tr>
<th>BCI painless</th>
<th>Yes</th>
<th>No</th>
<th>Total</th>
</tr>
</thead>
<tbody>
<tr>
<td>Lot 1</td>
<td>18</td>
<td>71</td>
<td>89</td>
</tr>
<tr>
<td>Lot 2</td>
<td>15</td>
<td>46</td>
<td>61</td>
</tr>
<tr>
<td>Lot 3</td>
<td>8</td>
<td>14</td>
<td>22</td>
</tr>
<tr>
<td>Total</td>
<td>41</td>
<td>131</td>
<td>172</td>
</tr>
</tbody>
</table>

-In group 2: 24 patients (39.34%) were diagnosed with painful BCI, with suggestive clinical symptoms accompanied by electrocardiographic ischaemia;
-In group 3: 8 patients (36.36%) had painful BCI, the remaining 14 patients (63.64%) not experiencing typical angina pain.

There was no significant difference (p> 0.05) in the distribution of painful coronary artery disease in the three groups.

Incidence of painless ischemic coronary heart disease
The distribution of patients with BCI painless by lot is the following (table 7):
-In group 1, 18 patients (20.22%) presented the BCI painless, diagnosed on the basis of ischemic changes on the resting ECG in the absence of angina.
-In group 2, 15 patients (24.59%) presented BCI painless and 46 patients (75.41%) had no ischemic changes on the resting ECG.
-In group 3, 8 patients (36.36%) presented painless BCI and 14 patients (63.64%) had no ischemic changes on the resting ECG.

Conclusions
HDL-cholesterol and apolipoprotein-A values are positive atherosclerosis risk factors.
The high level of HDL-C protects the patient, with a low risk of atherosclerosis. In this study, 54 men and 118 women were diagnosed with obesity grade 1 82 women and 8 men had HDL slightly below normal (less than or equal to 50 mg / dl for women and less than or equal to 40 mg / dl for men), the remaining 35 women and 45 men had a very low HDL.

The degree of obesity and abdominal obesity is a risk factor for hipo-HDL cholesterol.
This is explained by the association of the hepatic AGL influx, which stimulates the hepatic synthesis of large TG-rich VLDLs which, through changes after release into the bloodstream, will lead to increased plasma concentrations of small and dense LDL and decreased plasma HDLC.

In patients with grade 1 obesity, 58 had low HDL cholesterol and one patient had normal, in those with obesity grade 75 they had low HDL cholesterol and one patient had normal, and in those with obesity Grade 3 37 patients representing the whole lot had low HDL cholesterol.