Dyslipidemias and Obesity

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Abstract
The studies that initially suggested the association between obesity, dyslipidemia and coronary heart disease were population-based studies, in which the predictive factors for coronary heart disease were investigated. In a later study of this same Framingham group, it was suggested that obesity was an independent risk marker for coronary artery disease. Other reports showed that also in obese individuals the elevation of the fraction of low-density lipoprotein-LDL and the reduction of the fraction of high-density lipoprotein-HDL had a positive correlation with the risk of coronary heart disease. It was also observed that triglyceridemia would have a positive correlation with the risk of coronary heart disease. Hypertriglyceridemia in the obese results from greater synthesis and less removal of triglyceride-rich lipoproteins. In the obese individual, the greater supply of free fatty acids supplied to the liver promotes the greater production of very low-density lipoprotein-VLDL cholesterol rich in triglycerides. The hyperinsulinemia observed in metabolic syndrome contributes to the increased formation of these particles in the hepatocyte. For the treatment of dyslipidemia associated with obesity, the patient should receive guidance to lose weight, through an adequate diet and physical exercises. Regarding the diet, there is a controversy about which carbohydrate content it should contain, since, once the fat content is decreased, an increase in the carbohydrate content may occur, favoring hyperinsulinism and postprandial hyperglycemia. In individuals undergoing a strict weight losing diet, a transient phase of increased triglyceride and total cholesterol levels and decreased HDL-cholesterol levels may occur. In some cases, it is necessary to use lipid-lowering medications, the choice of which will depend on the type of lipid alteration found and the patient's response to treatment.

Keywords: Obesity, Hypertriglyceridemia, Hypercholesterolemia, Lipoproteins, Exercise, Diet

Abbreviations: HDL: High Density Lipoprotein; LDL: Low Density Lipoprotein; VLDL-c: Very Low Density Lipoprotein Cholesterol.

Introduction
Several epidemiological studies have demonstrated the association between dyslipidemia and the increased risk of coronary heart disease and the beneficial effect of the correction of lipid disorders on the frequency of coronary heart disease. Several researchers [1-5] have suggested that obesity is often associated with these lipid changes and also contributes to the increased risk of coronary heart disease.

The studies that initially suggested the association between obesity, dyslipidemia and coronary heart disease were population-based studies, in which the predictive factors for coronary heart disease were investigated. Thus, it was demonstrated that descendants of Japanese living in Japan had lower mortality from coronary heart disease than those living in Hawaii, who were more obese and had higher triglyceride levels [6,7]. The prospective study of Los Angeles veterans [8], in which patients were followed for 15 years, and the prospective Framingham study [9], in a 12-year follow-up, also demonstrated that obesity and hypercholesterolemia were related to increased risk of coronary heart disease. In a later study of this same Framingham group [10], it was suggested that obesity was an independent risk marker for coronary artery disease.

Other reports showed that also in obese individuals the elevation of the low-density lipoprotein (LDL) fraction and the reduction of the high-density lipoprotein (HDL) fraction had a positive correlation with the risk of coronary heart disease [11-14]. It was also observed that triglyceridemia would have a positive correlation with the risk of coronary heart disease [13-16]. In the prospective multicenter study PROCAM, it was found that, in individuals aged between 20 and 59 years, the prevalence of hypercholesterolemia (>200 mg/dL) ranged from 26 to 76% in men and from 28 to 86% in women, and a positive correlation was observed between cholesterol levels and the degree of obesity in the groups of younger individuals (20 to 29
the omental tissue have a higher rate of fatty acid synthesis than the lipolytic response to catecholamines. Thus, adipocytes from the subcutaneous tissue, as well as a greater lipolytic response to catecholamines [35]. This fact is important in android obesity, where there is a greater supply of free fatty acids to the liver, with consequent greater synthesis of VLDL-c particles rich in triglycerides, altering the lipid profile more unfavorably than in the gynecoid form [1].

For the treatment of dyslipidemia associated with obesity, the patient should receive guidance to lose weight, through an adequate diet and physical exercises. Regarding the diet, there is a controversy about which carbohydrate content it should contain, since once the fat content is reduced, an increase in the carbohydrate content may occur, favoring hyperinsulinism and postprandial hyperglycemia [36,37]. In individuals submitted to a strict weight-losing diet, a transitory phase of increased triglyceride and total cholesterol levels and decreased HDL-c levels may occur [38]. A meta-analysis study of 70 reports in the literature [39]. demonstrated that weight loss is associated with decreased triglyceride levels by 32%, total cholesterol by 13%, LDL-c by 11% and increased HDL-c by 12%.

In relation to physical exercise, it is known that it promotes the improvement of the lipid profile, regardless of weight loss. In a meta-analysis study of 95 reports in the literature [40], a decrease in triglycerides from 11 to 16%, cholesterol from 3 to 10% and an increase in HDL-c by 3%.

In some cases, it is necessary to use lipid-lowering medications, the choice of which will depend on the type of lipid alteration found and the patient's response to treatment.

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Conflict of Interest
None.

References


