SHORT COMMUNICATION AND COMMENTARIES

Atherogenic Dyslipidemia: Q and A for Patients

Anita L R Saldanha[1], Ana Paula Pantoja Margeotto[1], Andre Luiz Valera Gasparoto[2], Tania Leme da Rocha Martinez[1]

[1]Department of Nephrology, BP - A Beneficência Portuguesa de São Paulo, São Paulo, Brazil, [2]Intensive Care Unit, BP - A Beneficência Portuguesa de São Paulo, São Paulo, Brazil

ABSTRACT

Much has been done in terms of prevention of cardiovascular diseases, being one of them the achievement of ideal levels of lipoproteins according to each one of the patients goal, calculated by risk stratification. Even so there remains a residual risk and it may be caused by many less as yet defined risk factors, being one of the most prevalent, the atherogenic dyslipidemia. Atherogenic Dyslipidemia is defined by a triad of alterations: high triglycerides, low high density lipoprotein cholesterol and normal to elevated low-density lipoprotein of the most deleterious kind: small and dense low-density lipoprotein particles. This circumstance occurs most frequently in patients with insulin resistance or diabetes. The mechanism responsible for this metabolic profile stems from increased catabolism of high density lipoprotein particles rich in triglycerides. In individuals with hypertriglyceridemia, including postprandial, esterified cholesterol transfer protein facilitates the transfer of triglycerides from very low density lipoprotein to high density lipoprotein, resulting in the formation of high density lipoprotein particles enriched with triglycerides and depletated cholesterol. These particles are known to have higher atherogenic potential. There are several clinical and epidemiological evidences that support the concept that individuals with type 2 diabetes mellitus have an increased cardiovascular risk. The lipid phenotype often found in this population consists of hypertriglyceridemia and high density lipoprotein cholesterol. The average concentration of low density lipoprotein cholesterol does not present quantitative differences, being distinguished, however, by the high atherogenicity profile by the presence of small and dense particles.

Key words: Atherogenesis, dense low-density lipoprotein, diabetes, dyslipidemia, lipoproteins, metabolic syndrome

Among the changes in lipid profile, the condition that presents high triglycerides, low high-density lipoprotein-c, and relatively normal low-density lipoprotein (LDL)-c, but with small and dense molecules, is a condition of atherogenic dyslipidemia, a disorder commonly associated with altered glucose tolerance test, insulin resistance, obesity, type II diabetes mellitus, and systemic arterial hypertension. Dyslipidemia can cause the formation of the atherosclerotic plaque when the vascular endothelium becomes vulnerable due to the oxidation of lipoproteins, facilitating its entry into macrophages, where excess cholesterol is housed, triggering the process of atherogenesis.

IN THE LATEST STUDIES ON DYSLIPIDEMIAS, WE NOTICED A HIGHER PERCENTAGE OF WOMEN AFFECTED BY THE DISEASE. WHAT ARE THE MAIN REASONS FOR THIS STATISTIC?

Such data depend on the universe in which these surveys were conducted. In studies, I participated in, published in the Brazilian Archives of Cardiology, in more than 90,000 adults participating in the workforce, there was no significant

Address for correspondence:
Tania Leme da Rocha Martinez, BP - A Beneficência Portuguesa de São Paulo, Rua Comandante Ismael Guilherme, 358 - Jardim Lusitânia, CEP 04031-120 - São Paulo – SP, Brazil.

differentiation. What is there is a higher incidence of overweight and obesity in women, many of these cases with dyslipidemia. Furthermore, when they pass the menopause range, they lose the protection of estrogen so that dyslipidemias appear that did not occur by the action of this hormone. The problem of dyslipidemia is as important in men as in women.[1]

ARE SMALL AND DENSE LDL MOLECULES A MAJOR RISK FACTOR FOR THE ATHEROSCLEROSIS PROCESS AND, CONSEQUENTLY, FOR CARDIOVASCULAR DISEASES?

This lipoprotein is more recurrent in diabetic, pre-diabetic, or insulin resistance patients. The risk condition occurs when LDL is atherogenic (modified LDL), being able to enter the cell called macrophage – which originates the foamy cell and creates the fatty stria, which may turn out to be an atheroma plaque, responsible for arterial obstruction, such as the coronary artery that irrigates the heart. It is common for a diabetic patient to present a risk linked to LDL, and sometimes, on laboratory tests, this LDL will be little or nothing increased; in this patient, LDL is high atherogenic risk because he is in a heavier lipoprotein. In lipoproteins, there is a layer of protein involving lipids, and this characteristic of LDL – being small and dense – is due to the fact that there is a higher proportion of apoprotein B (apo B) that coats LDL.[2]

WHAT ARE THE MOST EFFECTIVE TREATMENT ACTIONS IN REDUCING TRIGLYCERIDE LEVELS?

For normalization and achievement of goals in triglyceride values, the most important is healthy lifestyle: Practicing physical activities, maintaining a balanced diet, not smoking, not being sedentary, and having normal sugar levels. High triglyceride levels are often corrected by controlling lifestyle, but if the patient is diabetic, pre-diabetic or has insulin resistance, in addition to lifestyle changes, it is also necessary to perform glycoglycemia and glycated hemoglobin controls. When, after all, triglyceride levels remain high, there is a need for medication use and, according to the guidelines, statin treatment is started to normalize these levels. If it does not normalize, fenofibrate is associated – although it is not official consensus, it is common among many professionals to start drug treatment by fibrates.[3]

PATIENTS WITH DIABETES HAVE A HIGHER PREVALENCE OF DYSLIPIDEMIAS. WHAT CARE SHOULD THE DOCTOR TAKE WHEN EVALUATING A PATIENT WITH CHANGES IN HIS LIPID PROFILE?

As a rule, patients with diabetes have dyslipidemias. This has to do with various mechanisms associated with metabolic disorders in diabetes. Carbohydrate metabolism, in particular glucose metabolism, is step by step with the metabolism of triglycerides, which are part of our energy storage. Glucose is our fast energy supply. When glucose falls into the blood, we have as energy reserve the triglycerides, which then divide into three molecules of monoglycerides, and in this division, there is the detachment of energy. Cholesterol in diabetic patients is also located in the denser LDLs. In dyslipidemia, it may present LDL at the highest dosage than normal, but may also not be as altered as a number, but altered as LDL conformation, because it is small and dense.[4,5]

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