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Lipidology Apo B is Important? Become Atherogenic? In Brain, Heart? How?

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Introduction

LDL-C is a cause of cardiovascular disease. It is also the factor to attack.

The metabolism of Apo B contains lipoproteins, these are absorbed in intestinal cells, such as chylomicrons; these chylomicrons will be metabolized in the liver as LDL, VLD L1, VLD L2, and LDL 1, 2. How hyperlipidemia is produced? Atherosclerosis Apo B contains lipoproteins that produce atherosclerosis. There is a consensus that low-density lipoproteins cause atherosclerotic cardiovascular disease. There is genetic, epidemiological evidence, and clinical studies, low-density lipoproteins cause cardiovascular, pathophysiological, and genetic disease. Since lipoproteins enter the arterial wall by a mechanism called Transcytosis, it is a passive filtration through the endothelium. Transcytosis involves ALK1, SRB1, and Cavilin. Estrogens inhibit LDL, this explains why women are protected from cardiovascular disease before menopause. Experimental studies indicate that hypercholesterolemia and hyperglycemia may increase LD transcytosis, LDL through the endothelium.

There is one in Apo B 100 that has positive receptors that mediate the union with the arterial wall in Proteoglycans. Chylomicrons and VLDLs cannot enter the endothelium because they are heavy, only LDL particles. They are less than 70 nanometers. Triglycerides are cleansed by lipolysis as well as cholesterol esters. The lipid metabolism postponed dial is governed by Apo B 48 containing particles that are secreted not only by chylomicrons but also by the cell. About half of Apo B 48 contains particles of LDL derived from chylomicrons of lipolysis, and half by direct secretion. The Apo B 100 and the Apo B 48 of both liver and intestine, can raise triglycerides, the remainder, or with the retention of lipoproteins, those that initiate atherosclerosis by modifying the MLL, performing a cellular response, and causing inflammation of the macrophage. Oxidized phospholipids have been found in inflamed tissues, in the form of a non-enzymatic oxidation of lipids, including atherosclerotic lesions.

These become accumulated from atherosclerosis, due to the absence of LDL. The trial testifies that anti-inflammatory therapy with Canakinumab, a monoclonal antibody which neutralizes interleukin, does not lower the incidence of cardiovascular events. Canakinumab was effective in decreasing adverse cardiac events over 3.7 years in another study. Methotrexate failed in any cardiovascular effect. Retained lipoproteins cause early lesion development and accelerated retention of LDL C aggregation. Lipid deposition Macrophages infiltrates the arterial wall. The progression mechanism of a stable cardiovascular injury is apolipoprotein, macrophage inflammation, inflammatory mediators and the fibrous layer. In unstable lesions is seen an accumulation of the apolipoprotein retained; there will be an effect on necrotic cells, inflammatory mediators, therefore fibrous layer will be thinner and subsequently become broken. This mechanism triggers oxidative stress, proteolysis and lipolysis.

The pharmacotherapy will be PCK9 and Inclisiran; these will increase the number of LDL in the liver. Recent studies show that their administration for six months, will reduce by 50% the LDL levels, while PCK9 only reduces them by 50 to 70%. Currently, there is a treatment for homozygous autosomal dominant hyperlipidemia since birth. Intravascular Ultrasound is the gold standard method to observe the progression or regression of the atheroma plaque as shown in the pictures from our laboratory (Figures No.1 and No.2). There are works with Evolocumab with an Intracoronary Ultrasound to observe the progression and regression of the plaque. The best strategy is to reduce them with the drugs LDL, PCK9, Inclisiran, Bempedoic acid. Risk factors are cigarette smoking, hypertension, diabetes, triglyceride elevation, decreased HDL, and elevated lipoprotein. In conclusion, we describe the mechanism of atherogenesis in all vascular beds and the treatment of plaque progression and regression, with the new pharmacotherapy.

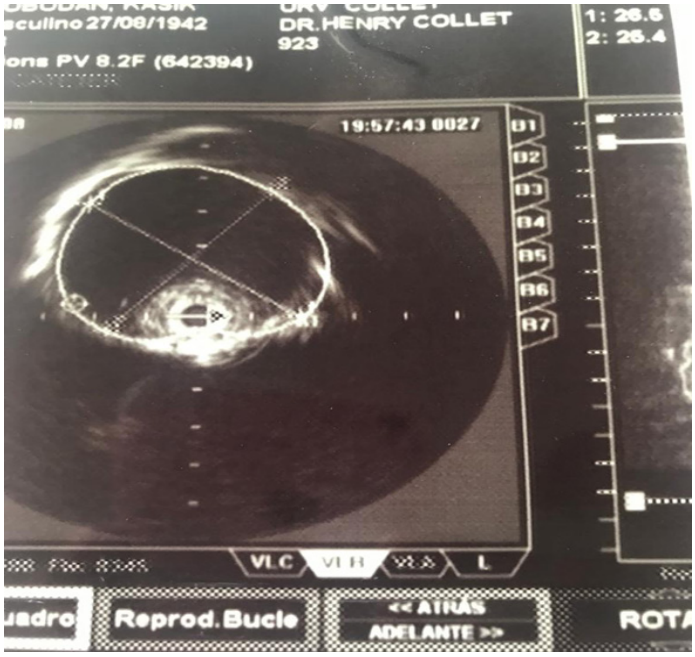

Figure 1

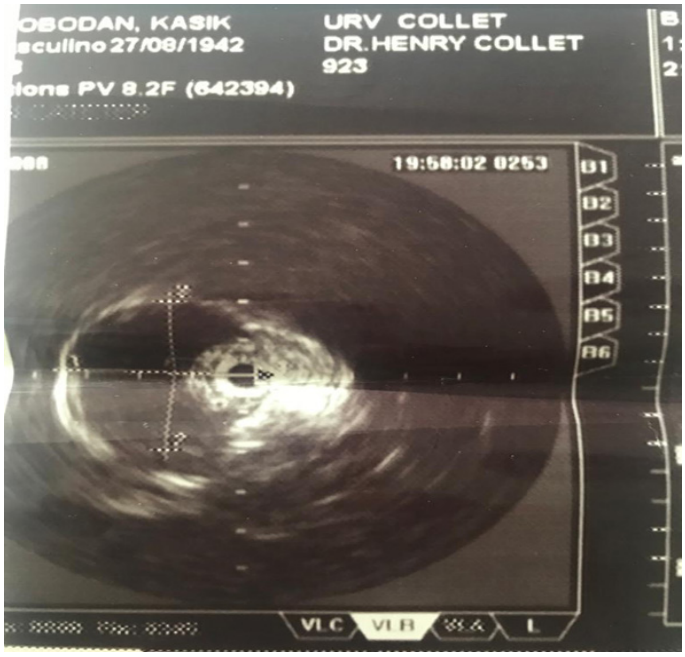

Figure 2

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