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Effect of Beta-Blockers on Serum Cholesterol in Patients with Cardiac Diseases

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ABSTRACT

Hypertension, hyperlipidemia, and cigarette smoking are major risk factors for coronary heart disease. Since many antihypertensive drugs alter plasma, lipid levels it is a subject of current discussion that these agents may increase associated coronary risk and therefore offset the beneficial effects of lowering blood pressure. The purpose of this paper is to review clinical and experimental data in the literature on the influence of data in the literature on the influence of beta-blockers on lipid metabolism. Atenolol lowered LDL + VLDL cholesterol slightly. Propranolol lowered HDL cholesterol and increased total triglycerides. It is suggested that the metabolic effects of antihypertensive drugs could be of special importance in the long-term treatment of mild hypertension.

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INTRODUCTION

Beta-blockers, also called beta-adrenergic blocking agents, treat a variety of conditions, such as high blood pressure and migraines. Find out more about this class of medication. Beta blockers, also known as beta-adrenergic blocking agents, are medications that reduce your blood pressure. Beta-blockers work by blocking the effects of the hormone epinephrine, also known as adrenaline. Arteriosclerosis occurs when the blood vessels that carry oxygen and nutrients from your heart to the rest of your body (arteries) become thick and stiff — sometimes restricting blood flow to your organs and tissues. Healthy arteries are flexible and elastic, but over time, the walls of your arteries can harden, a condition commonly called hardening of the arteries.

Atherosclerosis is a specific type of arteriosclerosis, but the terms are sometimes used interchangeably. Atherosclerosis refers to the buildup of fats, cholesterol and other substances in and on your artery walls (plaques), which can restrict blood flow. These plaques can burst, trigger a blood clot. Although atherosclerosis is often considered a heart problem, it can affect arteries anywhere in your body. Atherosclerosis may be preventable and is treatable. Several drugs used for antihypertensive therapy may interact with lipoprotein metabolism and increase associated coronary risk factors. Beta-blocker monotherapy with cardioselective or non-cardioselective beta blockers without intrinsic sympathomimetic activity (ISA) usually increases serum triglyceride and decreases the concentration of high-density lipoprotein (HDL), especially HDL2 cholesterol. With the exception of the non-cardio selective beta blocker sotalol, beta-blocker therapy has little influence on the serum total cholesterol or low-density lipoprotein (LDL) cholesterol concentrations. The magnitude of these changes in serum lipids does not significantly differ between cardio selective and non-cardio selective beta blockers.

LITERATURE REVIEW

Leren P, Eide I, Foss OP, Helgeland A, Hjermann I, Holme I, Kjeldsen SE, Lund-Larsen PG.

The effects on blood lipids and uric acid of six different antihypertensive drugs used alone, and of five different combinations of two antihypertensive drugs, are reported here. Prazosin significantly lowered serum low-density lipoprotein and very low-density lipoprotein (LDL + VLDL) cholesterol and total triglycerides while maintaining high-density lipoprotein (HDL) levels. Atenolol lowered LDL + VLDL cholesterol slightly. Both pindolol and

hydrochlorothiazide (HCTZ) were neutral, while oxprenolol increased total triglycerides. Propranolol lowered HDL cholesterol and increased total triglycerides and uric acid. The combination of prazosin plus pindolol has a direct favorable lipid profile, while the combination of propranolol plus HCTZ lowered HDL cholesterol and increased total triglycerides. The combination of propranolol plus prazosin lowered HDL cholesterol, but to a lesser degree than propranolol alone, which suggests that prazosin was not able to completely counteract the negative effects of propranolol on HDL. Methyldopa plus HCTZ, and HCTZ plus amiloride was neutral with regard to effects on blood lipids. It is suggested that the metabolic effects of antihypertensive drugs could be of special importance in the long-term treatment of mild hypertension.

Krone W, Müller-Wieland D, Greten H.

Hypertension, hyperlipidemia, and cigarette smoking are major risk factors for coronary heart disease. Since many antihypertensive drugs alter plasma lipid levels it is a subject of current discussion that these agents may increase associated coronary risk and therefore offset the beneficial effects of lowering blood pressure. The purpose of this paper is to review clinical and experimental data in the literature on the influence of data in the literature on the influence of antihypertensive drugs on lipid metabolism. The thiazides hydrochlorothiazide and chlorthalidone cause an elevation of plasma triglycerides and very low-density lipoprotein (VLDL) but have little effect on total cholesterol, low-density lipoprotein (LDL) and high-density lipoprotein (HDL). The unspecific beta-blockers, e.g. propranolol, do not affect total cholesterol and LDL but increase total triglycerides and VLDL and decrease HDL. The changes in plasma lipids and lipoproteins caused by cardio-selective beta-blockers, e.g. atenolol and metoprolol, and unspecific beta-blockers with intrinsic sympathomimetic activity.

Klein W.

Meta-analysis of several large interventional trials in patients with mild to moderate hypertension has shown that coronary events are reduced to a much lesser extent than expected. One of the possible explanations for this are the metabolic side-effects of diuretics and beta-blockers used in these trials that may counteract their beneficial blood-pressure-lowering effect. Diuretics, especially thiazide, increase total cholesterol (+5%) and LDL-cholesterol (+10%), while beta blockers decrease HDL-cholesterol (-5%) and increase

triglycerides (+20%). Calcium antagonists and ACE-inhibitors do not affect lipids, and alpha-blockers have some beneficial effects. Regarding the carbohydrate metabolism, diuretics and beta blockers decrease insulin sensitivity, increase plasma insulin, LDL-cholesterol, and triglycerides, and reduce HDL-cholesterol. Calcium channel blockers are neutral, while alpha-blockers and ACE-inhibitors improve glucose tolerance and reduce insulin resistance.

MATERIALS AND METHODS

Therapy with 10 to 40 mg once daily of propranolol a new angiotensin converting enzyme inhibitor, was compared with therapy with 50 to 100 mg once daily of atenolol in a double-blind randomized in 100 patients with a diastolic blood pressure (determined with the patient seated) of 95 to 115 mm Hg. A total of 50 patients (25 men and 25 women with a mean age of 49.4 years and a mean blood pressure at entry into the trial received propranolol, and 80 patients (men and women with a mean age of 50.9 years and a mean blood pressure at entry of 156.6 [16.6]/101.2 [5.7] mm Hg) received atenolol. After a placebo run-in period the patients received increasing dosages of medication every 2 weeks until the target diastolic blood pressure of 90 mm Hg or less was achieved on two consecutive visits, the maximum dosage was reached.

RESULT

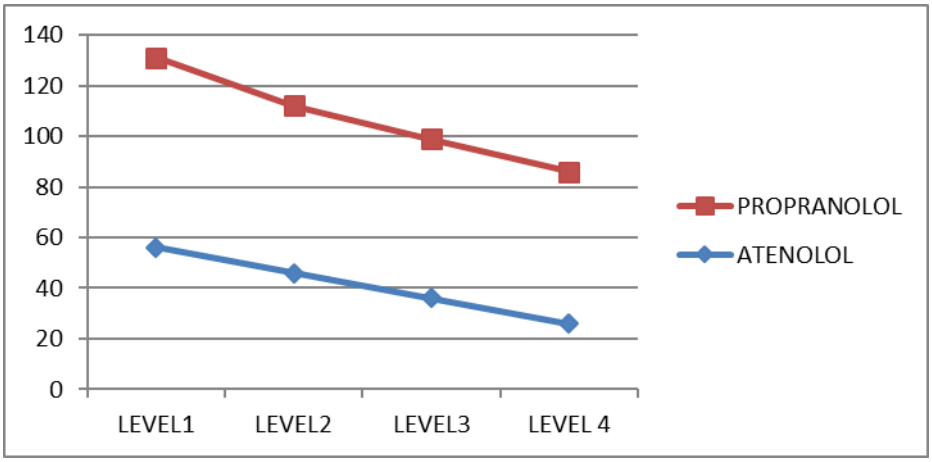

Figure No 1: Figure showing the effect on lipids

CONCLUSION

Hypertension, hyperlipidemia, and cigarette smoking are major risk factors for coronary heart disease. Since many antihypertensive drugs alter plasma lipid levels it is a subject of current

discussion that these agents may increase associated coronary risk and therefore offset the beneficial effects of lowering blood pressure. The purpose of this paper is to review clinical and experimental data in the literature on the influence of data in the literature on the influence of antihypertensive drugs on lipid metabolism. The thiazides hydrochlorothiazide and chlorthalidone cause an elevation of plasma triglycerides and very low-density lipoprotein (VLDL) but have little effect on total cholesterol, low-density lipoprotein (LDL) and high-density lipoprotein (HDL). The unspecific beta-blockers, e.g. propranolol, do not affect total cholesterol and LDL but increase total triglycerides and VLDL and decrease HDL.

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