Dyslipidemia in hemodialysis patients: Can it be a factor associated with ventricular hypertrophy?

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ABSTRACT

Lipid abnormalities are common in hemodialysis patients, they are potentially atherogenic. The aim of our work is to study the quantitative changes in lipid parameters and find a correlation between dyslipidemia and the occurrence of left ventricular hypertrophy (LVH) in chronic hemodialysis patients followed in our training. We conducted a prospective study in 36 chronic hemodialysis patients for at least one year, non-diabetic, non-smoking. The dialysis membrane used was helixone. Dyslipidemia was defined by a TG levels > 1.5 g / l and / or a rate of CT> 2g / l. The mean age was 45.53 ± 13.42 years, 30.5% were overweight. Causal nephropathy was undetermined in 55.6% of cases. The quality of the dialysis was characterized by a KT / V Average 1.12 ± 0.21.

Results

Sixty-seven percent of patients had LVH. An analytical study was carried out between the group of patients with LVH (group I) and those who had not (group II). We found a significant increase of TG (p = 0.001) (2.14 ± 0.76 vs 1.19 ± 0.21), the rate of CT (p = 0.001) (2.31 ± 0.66 vs 1.20 ± 0.32), the rate of LDL-C (p = 0.001) (1.83 ± 0.38 vs 0.89 ± 0.23) and CLDL / CHLD ratio (p = 0.001) (5.41 ± 1.98 vs 2.09 ± 0.83) and a significant decrease in the rate of HDL-C (0.36 ± 0.11 vs 0.47 ± 0.16) in group I by compared to group II. Nineteen percent of patients were on statin. No complication of rhabdomyolysis was observed. Assessment of lipid profile should be integrated into the strategy for the management of chronic hemodialysis since cardiovascular risks they are exposed.

Introduction

The cardiovascular morbidity and mortality are increased in patients on dialysis. Dyslipidemia is a major atherogenic factor. It is more frequent and severe renal function is impaired. The aim of our work is to study the quantitative changes in lipid parameters and find a correlation between dyslipidemia and the occurrence of left ventricular hypertrophy (LVH) in hemodialysis patients on dialysis. Dyslipidemia was present in 66.5% of our patients. Sixty-seven percent of patients had LVH. An analytical study was carried out between the group of patients with LVH (group I) and those who had not (group II). We found a significant increase of TG (p = 0.001) (2.14 ± 0.76 vs 1.19 ± 0.21), the rate of CT (p = 0.001) (2.31 ± 0.66 vs 1.20 ± 0.32), the rate of LDL-C (p = 0.001) (1.83 ± 0.38 vs 0.89 ± 0.23) and CLDL / CHLD ratio (p = 0.001) (5.41 ± 1.98 vs 2.09 ± 0.83) and a significant decrease in the rate of HDL-C (0.36 ± 0.11 vs 0.47 ± 0.16) in group I by compared to group II. Nineteen percent of patients were on statin. No complication of rhabdomyolysis was observed.

Discussion

Renal failure is an independent cardiovascular risk factor [1]. Chronic renal failure have a secondary dyslipidemia involves quantitative and qualitative abnormalities of lipoproteins. These disturbances are present from the very early stage of renal failure development with the progression of the disease, and are not corrected by maintenance dialysis (1,2).

The mechanism of dyslipidemia is not probably involving a clear increase in the synthesis of triglycerides decreased lipolysis due to an alteration in lipoprotein lipase activity etiology controversial : decreased synthesis of LPL in particular in relation to insulin resistance whose appearance coincides with the IRC with decrease LPL activity, stocks depletion LPL hepamin by repeated administration in the hemodialysis, existence of a plasma inhibitor of LPL or increased apo CIII recognized quite early in the evolution of the chronic renal failure? (2,3)

The most common lipid abnormality in chronic uremic patients is hypertriglycerideremia. It appears that its prevalence increases with the decrease in glomerular filtration rate above 50 ml / min. It affects nearly 80% of chronic uremic. The plasma concentrations of triglycerides are in the range from 2 mmol / L; those of total cholesterol, however, little changed during the progression of I'IRC. The plasma concentration of LDL-Chol is usually normal (90%), whereas that of the cholesterol in high density lipoprotein (HDL) is decreased.

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Cholesterol is redistributed from HDL to very low density lipoproteins (VLDL). There is an accumulation of VLDL and especially lipoproteins intermediate density (IDL), and an enrichment of LDL and HDL triglyceride (3,4,5).

In our work, lipid disorders consist essentially hypertriglyceridaemia accompanied by an increase in total cholesterol and LDL cholesterol and a decrease in HDL cholesterol.

Cardiovascular accidents are a major cause of morbidity and mortality during chronic renal failure (6, 7). Chronic uremic patients have the classic risk factors of atherosgenèse such as age, diabetes, hypertension, smoking, but also more specific factors such as uremia lipid disorders characteristics, hyperfibrinogenaemia, hyperhomocysteinemia (11). Uremic dyslipidemia is a major cardiovascular risk factors as it is strongly associated with stroke in patients with CKD (6,8,9). Has been shown recently that lipid disorders, especially lowering plasma concentrations of HDL-Chol, were predictive markers of stroke in these patients (6).

In chronic hemodialysis patients the prevalence of hypertension is much greater. Thus, in the Framingham cohort, CKD patient had a higher prevalence (71% versus 43%) (1). Its pathogenesis is multifactorial and is readily associated with left ventricular hypertrophy. In our study, we found a clear correlation between the presence of dyslipidemia and the occurence of left ventricular hypertrophy in our hemodialysis patients.

**Conclusion:**

The present study shows the pro atherogenic lipid over our renal insufficient patients. The risk of cardiovascular mortality is the most important especially in the presence of left ventricular hypertrophy (5,12). The evaluation of lipid profile should be integrated into the strategy for the management of chronic hemodialysis and perhaps lipid-lowering therapy or lipid-lowering dietary requirements should be included in treatment protocols.

**Bibliography**


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