LIPOPROTEIN (a): A CONCEALED PRECURSOR OF INCREASED CARDIOVASCULAR RISK? A REAL-WORLD REGIONAL LIPID CLINIC EXPERIENCE.

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Abstract

Objective: Lipoprotein (a) [Lp(a)] is a potent genetically predisposed, independent cardiovascular risk factor. Elevated Lp(a) levels ≥30 mg/dL may alter risk stratification. We present real-life clinical characteristics of patients with increased Lp(a) levels attending a University Lipid Clinic.

Methods: We included patients attending the University of Ioannina Hospital Lipid Clinic with Lp(a) levels ≥30 mg/dL. Patient medical history, concomitant medications and laboratory results were reported during scheduled periodic visits over a median 22-month follow-up.

Results: One hundred seven patients (median age 59 years, 50.5% females) were included with median Lp(a) levels of 67 mg/dL (range: 30-175). Of patients, 24.4% had established cardiovascular disease (CVD): 10.3% and 5.7% positive personal history of myocardial infarction (MI) and stroke, respectively, 6.5% carotid artery disease and 1.9% lower extremities arterial disease (LEAD). In addition, 34.6% of participants had heterozygous familial hypercholesterolemia (heFH), 37.4% positive family history of premature CVD, 29.9% hypertension and 12.1% diabetes. Regarding hypolipidemic treatment, 67.3% were receiving statin therapy and 17% additional ezetimibe at baseline visit. In total, 80% and 36% were receiving statin treatment and additional ezetimibe, respectively, during follow-up. Low-density cholesterol (LDL-C) levels and LDL-C corrected for Lp(a) levels were significantly reduced in statin-naive patients by 37% and 40%, in statin up-titrated patients by 30% and 41%,

and in patients on stable statin treatment by 24% and 31%, respectively, over a median 22-month follow-up. In contrast, Lp(a) levels were overall increased by 9%.

Conclusion: Our data confirm the high prevalence of established CVD (24.4%), FH (34.6%) and positive familial history of premature CVD (37.4%) in patients with elevated Lp(a) levels. Noteworthy, Lp(a) levels were marginally increased during treatment follow-up.

Key-words: Lipoprotein (a), LDL-C, Cardiovascular Disease, Familial Hypercholesterolemia, statins