ASSOCIATION OF MONOCYTE CHEMOATTRACTANT PROTEIN 1 -2518A>G POLYMORPHISM WITH CAROTID ARTERY INTIMA-MEDIA THICKNESS IN PATIENTS WITH DIABETIC NEPHROPATHY

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INTRODUCTION and AIMS

Monocyte chemoattractant protein-1 (MCP-1) is highly expressed in human atherosclerotic plaques and plays an important role in cardiovascular diseases. Single nucleotide polymorphism (SNP) *MCP1 -2518 A>G* results to increased expression of MCP-1 protein in subjects carrying a G allele (AG/GG genotypes) and probably initiation and progression of atherosclerosis. Carotid artery intima-media thickness (cIMT) is a surrogate marker of atherosclerosis. A recent study showed an association of *MCP1 -2518 A>G* polymorphism with cIMT in a cohort of patients with type 2 diabetes mellitus. We sought to determine the association between the *MCP1 -2518 A>G* polymorphism and the severity of carotid atherosclerosis in patients with diabetic nephropathy.

METHODS

The study population comprised 109 unrelated patients (mean age 68.1±8.6 years) who were diagnosed with diabetic nephropathy (Table 1). To evaluate macroangiopathy, cIMT was measured using a high resolution, real-time B-mode ultrasonograph. MCP1 -2518 A>G polymorphism was determined using PCR-RFLP. Cumulative effect of cardiovascular disease (CVD) risk was assessed by calculation of each individual's CVD score: addition of 1 point for each CVD co morbidity present (myocardial infarction, stroke, angina and peripheral arterial disease) and then calculating the sum for all four CVD events. The CVD score of each individual ranged from 0 (absence of any CVD co morbidity) to 4 (presence of all four CVD events).

RESULTS

cIMT significantly increased as the CVD score progressed (CVD score 0: 0.79±0.03 mm, CVD score 1: 0.86±0.03 cm, CVD score 2: 0.96±0.05 cm, CVD score 3: 1.01±0.05 cm, CVD score 4: 0.94±0.18 cm, p<0.001) (Figure 1). The frequencies of *MCP1* genotypes were: AA (n=56) 51.4%, AG (n=47) 43.1%, and GG (n=6) 5.5%. cIMT in patients with the *MCP1* AG or GG genotype was not significantly different than in patients with the AA genotype (0.96±0.26 cm vs. 0.91±0.21 cm, p=0.3) (Table 2).

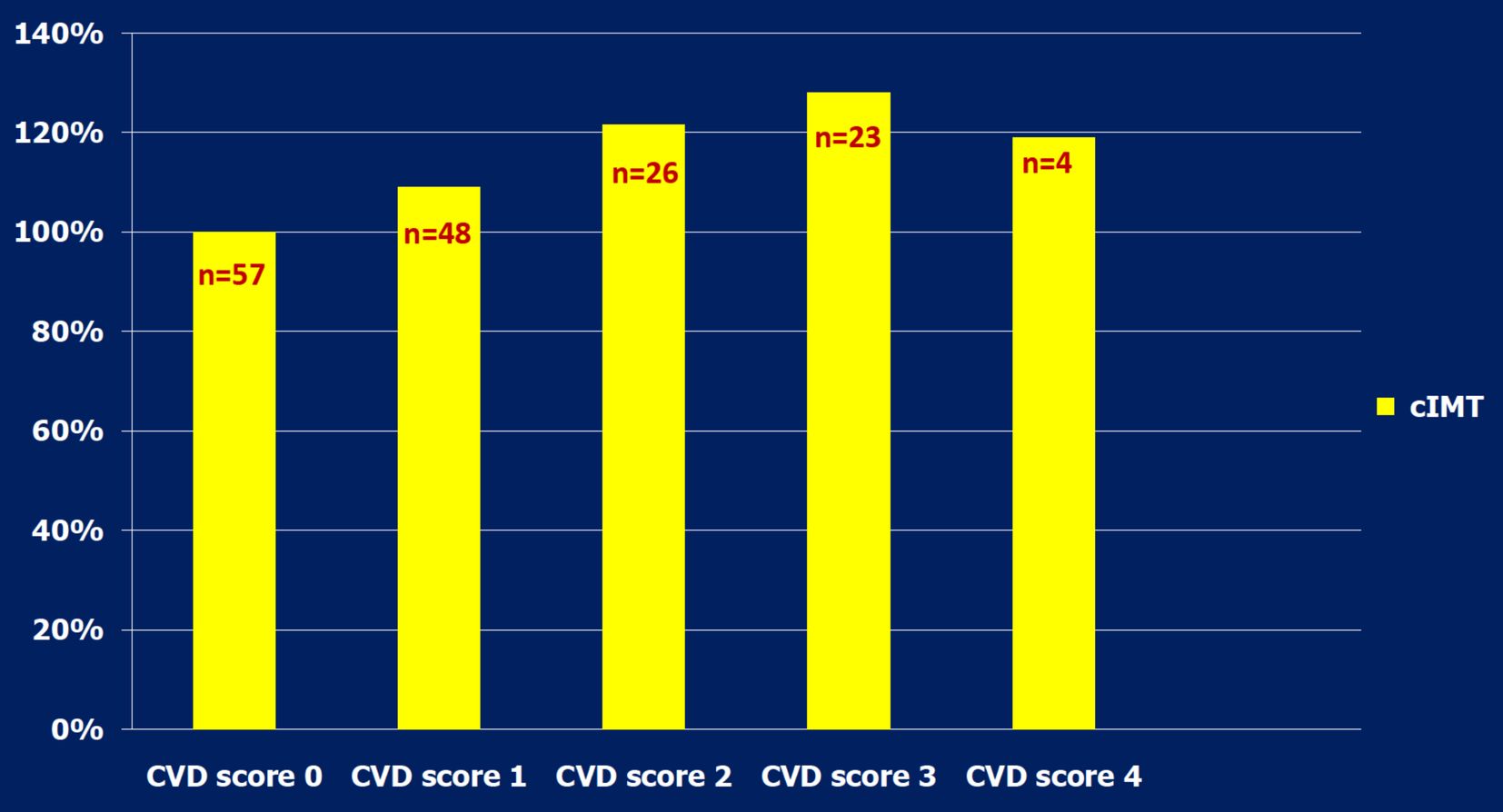
Table 1: Anthropometric, clinical and biochemical parameters

n=109 patients (47 M & 62 F)	*Mean value ± SD*
Age (y)	68.1 (8.6)
SBP (mm Hg)	138.6 (20.5)
DBP (mm Hg)	77.1 (10.7)
BMI (kg/m^2)	30.8 (5.3)
Duration of T2DM (y)	14.6 (7.7)
Fasting glucose (mg/dl)	164.1 (66)
HbA1c (%)	7.4 (1.1)
eGFR (ml/min/1.73m^2)	47.1 (33.2)
Total cholesterol (mg/dl)	174.0 (44.7)
LDL-cholesterol (mg/dl)	96.1 (37.0)
HDL-cholesterol (mg/dl)	46.0 (13.1)
Triglycerides (mg/dl)	163.0 (82.4)
Mean cIMT (cm)	0.94 (0.24)

Table 2: MCP1 -2518 A>G genotypes and cIMT

MCP1 -2518 A>G genotypes	n(%)	cIMT (cm)	p
AG/GG	47/6 (48.6%)	0.96±0.26	0.3
AA	56 (51.4%)	0.91±0.21	

Figure 1: cIMT according to CVD scores



CONCLUSIONS

cIMT increased significantly with cardiovascular disease severity in patients with diabetic nephropathy but was not associated with *MCP1 -2518 A>G* polymorphism.

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