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| Document Title | : ASYMMETRIC DIMETHYL-L-ARGININE IN DIABETES MELLITUS AND |
| | HYPERLIPIDEMIA |

ثنائي ميثيل -ال- أرجينين غير المتماثل في مرضى السكري وزيادة معدل الدهون

: Arabic

Document Language Abstract

: Asymmetric dimethyl-L-arginine (ADMA) is an endogenous inhibitor of nitric oxide synthase (NOS). Endothelium-derived nitric oxide is the most potent endogenous vasodilator. NO also powerfully inhibits key processes in atherogenesis and restenosis, and therefore vasoprotective. The elaboration of NO is also required for angiogenesis. By inhibiting the synthesis of NO, ADMA increase vascular resistance, inhibits angiogenesis, and promotes vascular disease. Accumulative evidence indicates that ADMA is a critical mediator of the adverse effects on NOS of all cardiovascular risk factors. ADMA also appears to be an independent risk factor for future cardiovascular events and total mortality. ADMA may have diagnostic relevance as a novel cardiovascular risk marker. In the present investigation, it is aimed to determine serum ADMA concentrations in male and female healthy normal subjects as well as in diabetic and hyperlipidemic patients in order to find out any possible relationship. A total of 300 male and female individuals ranging from 20-65 years, were randomly selected and divided as follows: A 100 of normal healthy subjects (52 males, 48 females); 100 of diabetic patients (51 males, 49 females); 100 of hyperlipidemics patients (50 males, 50 females). It was found that the concentration of serum ADMA was significantly increased in diabetic and hyperlipidemic patients of both sexes when compared to their matched controlled subjects. ADMA concentration in control group was less than that measured in the diabetic and hyperlipidemic male group (0.82 ± 0.04) μ mol/l, 1.09 \pm 0.07 and 1.31 \pm 0.64, respectively; p <0.001). However, ADMA concentration in control female group was less than that measured in the diabetic and hyperlipidemic female group $(0.88\pm0.06 \mu mol/l, 1.22\pm0.08, 1.45\pm0.09,$ respectively; p < 0.001). The present study also showed that serum concentration of ADMA was significantly correlated with diabetes and that serum levels of ADMA were positively correlated with glucose in diabetic males (r =0.41, p=0.01) and females (r =0.51, p= 0.01). Also, there was a positive correlation between ADMA serum concentration and lipid profile (except HDL-cholesterol) in hyperlipidemic males (TC: r =0.71, p=0.01; TAG: r =0.70, p=0.01; and LDL: r =0.34, p=0.05) and females (TC: r =0.77, p=0.01; TAG: r =0.51, p=0.01; and LDL: r =0.46, p=0.01). This finding suggests that ADMA can be used as a biological marker to suggest an increase risk of atherosclerosis, because ADMA is accumulated to factors that themselves produce other changes including in increasing ADMA.

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