

[P60] PLASMA LEVELS OF ASYMMETRIC DI METHYL ARGININE AND ENDOTHELIAL DYSFUNCTION IN DIABETIC SUBJECTS WITH NEUROPATHIC FOOT ULCER

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Aim: Evaluate the relationship between plasma Asymmetric–Di-Methyl-Arginine (p-ADMA) level and endothelial dysfunction (ED) in diabetic subjects with neuropathic foot ulcer (NFU), and study the possible predictors of p-ADMA level.

Method: 80 diabetic subjects of matched age, sex and BMI were included; 40 with NFU(G1), 20 with peripheral nerve dysfunction (PND) (G2) and 20 without PND (G3), plus 20 matched healthy subjects (G4). Subjects with renal or hepatic impairment, ischemic heart disease, smoking or using statins were excluded. Flow-mediated-dilatation (FMD) of brachial artery and Carotid-intima-media-thickness (CIMT) were measured using high-resolution ultrasound to evaluate ED and subclinical atherosclerosis, respectively. p-ADMA levels were assayed by ELISA kits supplied by EAGLE-BIOSCIENCES, INC (Germany).

Results/Discussion: G1&2 had a significantly lower FMD than G3&4 [-5.09(-22.5-22.92), 4.67(-15-23.91) vs. 15.74(8.33-36.59) and 20.1(10.0-46.15) %, respectively] ($p < 0.001$), and higher CIMT [0.9(0.6-1.5), 0.9(0.6-1.3) vs. 0.6(0.5-0.8) and 0.7(0.5-0.9) cm, respectively] ($p < 0.001$). However, there was no significant change in p-ADMA between the study groups [704.5(508-3611), 687(286-2863), 678(506-874), 642(383-797) ng/L, respectively] ($p = 0.126$). p-ADMA was positively correlated with diabetes duration, systolic blood-pressure, serum total cholesterol, triglycerides and CIMT ($r = 0.299$, $p = 0.007$, $r = 0.298$, $p = 0.007$, $r = 0.390$, $p < 0.001$, $r = 0.237$, $p = 0.034$, $r = 0.330$, $p = 0.003$, respectively), with no significant correlation with FMD ($r = -0.176$, $p = 0.118$). FMD was inversely and strongly related to CIMT ($r = -0.520$, $p < 0.001$). p-ADMA levels were significantly higher in uncontrolled hypertensive patients in comparison to controlled and normotensive subjects ($p = 0.026$). Metformin users and hypertensive subjects on ACEIs or ARBs had the lowest p-ADMA levels than the non users ($p < 0.001$, $p = 0.007$, respectively).

Conclusion: The remarkable ED in diabetic subjects with NFU is unlikely to be due to alteration in p-ADMA. Further studies are needed in order to conclude a causal association between p-ADMA and ED in this group of patients.