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Study of possible relation between maternal serum resistin and insulin resistance in patients with pre-eclampsia

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Introduction: In humans resistin antagonizes the effects of insulin on glucose metabolism in liver and skeletal muscle, interacts with and reinforces inflammatory pathways and may promote endothelial cell activation. Increased resistin levels have been associated with obesity, insulin resistance, metabolic syndrome, type 2 diabetes and increased cardiovascular risk.

Aim: Our study aimed to investigate the utility of maternal serum resistin in women with preeclampsia compared to normal pregnant women and its relation to insulin resistance.

Methods: The study was conducted on 90 females, divided into two groups: Group one: pre-eclampsia (n=60) and group two: healthy pregnant control (n=30). All individuals were subjected to the following after an informed oral and written consent: Full history taking, clinical examination with special emphasis on edema, blood pressure measurement and maternal body mass index (BMI); index [weight (kg)/height^2 (m^2)], determination of gestational age according to the date of the last menstrual period and confirmed by first trimester ultrasound. Laboratory investigations included CBC, AST, ALT, BUN, creatinine, homeostatic model assessment- insulin resistance (HOMA-IR) and serum resistin.

Results: Statistical comparison between pre-eclamptic patients (group one), and the healthy control group (group two) regarding the different studied parameters revealed a highly statistically significant increase in the patients group than the control group regarding SBP, DBP, BMI, CRE, AST, ALT, 50g oral glucose challenge test (GCT), FBG, fasting insulin, HOMA-IR and resistin. On the contrary, there was a highly statistically significant decrease in the patients group than the control group regarding HB.

Conclusion: In this study, it was found that elevated serum resistin levels could be associated with exaggerated insulin resistance in patients with preeclampsia. Further studies are needed to clarify the role of resistin in the patho-physiology of preeclampsia and insulin resistance.

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