Possible Interaction between renin angiotensin system and Apelin/APJ system in obesity-associated hypertension

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ABSTRACT

Background: WAT is now recognized as the largest endocrine organ of the body. WAT secretes a number of bioactive peptides and proteins, collectively termed "adipokines". Adipokines have different biological effects, including blood pressure control. Dysregulated production and release of specific adipokines (RAS& apelin) from WAT in the setting of obesity may contribute to hypertension. Based on previous concepts, the present study aimed to clarify role of RAS in obesity induced hypertension& to clarify role of apelin in obesity, also to determine possible interaction between RAS& apelin. **Materials &methods**: 63rats used in this study divided into 3main groups, group I given standerd rat chow, group II given high fat sucrose diet for 4 weeks, group II given high fat sucrose diet for 10 weeks. Each group was further divided into 3subgroups (n=7), non- treated (group1,4,7), ACEI(group2,5,8), L-NAME(group3,6,9). At the end of experiment BMI, Systolic blood pressure, blood glucose and serum triglycerides were measured. Visceral adipose tissue (epididymal fat) was collected for gene expression of AT1R, ANG1-7 R, apelin apj and VEGF. **Results**: High fat sucrose diet for 4 weeks leads to significant increase in body weight, BMI, blood glucose and serum triglycerides associated with significant increase in apelin&AT1 expression. High fat sucrose diet for 10 weeks leads to significant increase in blood pressure with significant decrease in Ang1-7expression. Captopril caused significant reduction in body weight, BMI, blood glucose and serum triglycerides associated with significant increase in apelin apj & Ang1-7expression. Correlation was proved between different genes expressed on adipose tissue. **Conclusion:** obesity leads to development of hypertension. ACEI decrease blood pressure significantly in obese hypertensive rats

through increased Ang 1-7&apelin receptors expression. L-NAME raise blood pressure significantly in obese rats through increased AT1 receptor expression. AngII, Ang1-7&apelin all interact to regulate ABP in obese subject.

KEY WORDS :

RAS, Apelin , Obesity induced hypertension.