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lesions. However, clinical role of adhesion molecules in the pathogenesis of hypertensive vascular lesions is not yet clear, especially in obesity induced hypertension. To assess the role of adhesion molecules in that mechanism, the relationship between carotid intima-media thickness (IMT) as hypertensive vascular lesions and plasma levels of soluble adhesion molecules such as E-selectin, VCAM-1,and ICAM-1 was statistically analyzed. Carotid IMT was measured by B-mode ultrasonography and plasma levels of adhesion molecules were by ELISA method. Four hundred and sixty one subjects who underwent annual health checkups at our center were enrolled in this study. VCAM-1 was significantly correlated with blood pressure (BP)($p<0.05$), and body mass index (BMI)($p<0.01$). There were no correlations between ICAM-1 and BP nor BMI. E-selectin and VCAM-1 were significantly increased with BP ($p<0.001$), and BMI ($p<0.001$). Carotid IMT was significantly correlated with E-selectin. E-selectin and VCAM-1 were significantly increased with BP, and E-selectin was also significantly increased with BMI. There was no cross reaction between BP and BMI. In conclusion, E-selectin and VCAM-1 may play an important role in the pathogenesis of hypertensive vascular lesions, and E-selectin may be a key factor for obesity induced hypertensive vascular lesions.

Key Words: Adhesion molecules, Hypertensive vascular lesion, Obesity

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PIOGLITAZONE BLUNTS THE BLOOD PRESSURE RESPONSE TO ANGIOTENSIN II IN INSULIN-RESISTANT ZUCKER RATS

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Thiazolidinediones are high-affinity agonists of peroxisome proliferator-activated receptors (PPAR)-γ which have been found to lower blood pressure (BP) in animal models of diabetes and reno-vascular hypertension. The mechanisms leading to this decrease in blood pressure are still unclear. Since the renin-angiotensin system is a main regulator of blood pressure, we investigated the effects of pioglitazone on blood pressure, plasma renin activity and on the blood pressure response to exogenous angiotensin II in insulin-resistant Zucker rats.

Pioglitazone 20mg/kg/d or vehicle were administered for 4 weeks to 8-weeks old fa/fa Zucker rats. Pioglitazone-treated rats were heavier than vehicle-treated rats (respectively 481g±8g vs 437±5g, $p=0.0002$, mean±SEM) and ate more (35.6±0.5g/d vs 28.9±0.3, $p<0.0001$). The increase in blood sugar after an intra-venous glucose tolerance test was significantly attenuated in the pioglitazone treated rats at 10,15 and 30 minutes. Systolic (SBP), diastolic (DBP) blood pressure and heart rate (HR) were lower in pioglitazone-treated rats: SBP: 124±3 mmHg vs 144±3, $p<0.001$; DBP: 80±2 vs 94±2, $p<0.001$; HR: 369±6 vs 397±8, $p<0.01$. The BP response to exogenous angiotensin II was significantly attenuated in pioglitazone treated rats. With the 25 ng/kg Ang II dose the increase in BP was 27.8±2.4 mmHg with pioglitazone and 37.5±3.3 with the vehicle ($p=0.04$) and with the Ang II 100 ng/kg dose the increase in BP was respectively 36.1±2.7 mmHg and 49.2±2.3 ($p=0.003$). Plasma renin activity was comparable in both groups.

In conclusion, these results show that pioglitazone blunts the BP response to angiotensin II in insulin-resistant Zucker rats. This effect may partially explain the blood pressure lowering effect of PPAR-γ agonists.

Key Words: glitazone, angiotensin II, Zucker

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EFFECTS OF POSTPRANDIAL BLOOD PRESSURE AND PULSE PRESSURE ON CAROTID REMODELING AND ATHEROSCLEROSIS IN ESSENTIAL HYPERTENSION AND DIABETES MELLITUS

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Postprandial glucose and lipid play an important role in the cardiovascular diseases. Recently, pulse pressure (PP) and microalbuminuria are also thought to be atherosclerotic risk factors. It is unclear whether the changes of postprandial BP and PP are associated with the carotid remodeling and atherosclerosis in essential hypertension (EH) and type 2 diabetes mellitus (DM). To study the effect of postprandial BP and PP on carotid artery. Ambulatory BP were monitored from 30 min before food intake until 120 min after meal. Common carotid artery (CCA), plaque rate and intima-media thickness (IMT) of carotid artery were examined by echocardiography. Urinary microalbuminuria (UMA) were detected. Based on the difference of BP and PP change before and after food intake, 92 EH patients (50M/42F, mean age 52 yrs) and 83 type 2 DM (45M/38F, mean age 55 yrs) were divided into: group 1 (ΔSBP <10,ΔDBP <5,ΔPP <10mmHg,) and group 2 (ΔSBP≥10,ΔDBP≥5, ΔPP≥10mmHg). Compared to BP and PP before food intake, increased postprandial SBP, DBP and PP were observed in 92%, 86% and 82 % EH patients, respectively. Compared to group 1, remarkable damage of carotid artery was found in group 2: CAA, 6.0 +/- 1.1 vs 7.8+/- 1.1 mm, P< 0.01; IMT, 0.65 +/-0.08 vs 0.76 + /- 0.11 mm, P< 0.01; plaque rate, 38% vs 69 %, P < 0.01; UMA, 30 +/- 3 vs 70 +/-4 mg / L, P < 0.01. Compared to BP and PP before meal, increased postprandial SBP, DBP and PP were observed in 24%, 20% and 48 % DM patients. Compared to group 1, abnormal parameters were found in group 2: CAA, 5.6 +/- 1.5 vs 6.6+/- 1.4 mm, P< 0.05; IMT, 0.57 +/-0.06 vs 0.64 + /- 0.05 mm, P< 0.05; plaque rate, 38% vs 61 %, P < 0.01; UMA, 50 +/- 3 vs 73 +/-5 mg / L, P < 0.01. It concluded that EH and type 2 DM had a different status of postprandial BP and PP. Increased UMA and postprandial PP had an effect on carotid remodeling and atherosclerosis in EH and DM (Supported by NCSF grant 39725013).

Key Words: Hypertension, Type 2 Diabetes, Carotid Atherosclerosis