

assessed while the plasma level of VPO1 in patients and the expression of VPO1 in arterial tissues was measured. Cultured human aorta vascular smooth muscle cells were treated with ANGII, and the proliferation activity, VPO1 expression, H₂O₂ and HOCL level were examined. The effect of VPO1 RNA interference, apocynin, catalase and PD98059 on VPO1 expression and the proliferation activity of cells were observed.

Results The VPO1 level/expression was significantly increased in patients with essential hypertension and in spontaneously hypertensive rats concomitant with definite vascular remodeling by evaluating the intima-media thickness, pressure-strain elastic modulus and stiffness index of carotid artery in patients, as well as the media thickness, lumen diameter, media thickness/lumen diameter ratio and mean nuclear area in artery media in spontaneously hypertensive rats. The angiotension II-stimulated cell proliferation of human aorta smooth muscle cells was inhibited by knockdown of VPO1 using small hairpin RNA. Moreover, the NADPH oxidase inhibitor, apocynin, the hydrogen peroxide scavenger, catalase, but not the ERK1/2 inhibitor, PD98059 attenuated Ang II-mediated upregulation of VPO1 and generation of hypochlorous acid.

Conclusions VPO1 is a novel regulator of vascular smooth muscle cell proliferation via NADPH oxidase/H₂O₂/VPO1/ERK1/2 pathways and plays an important role in vascular remodelling during hypertension.

e0595 ANGIOTENSINII MODULATES ION PUMPS OF SMOOTH MUSCLE CELLS DERIVED FROM UMBILICAL ARTERY OF HUMAN NEONATES WITH HYPERTENSIVE FAMILY HISTORY

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Objective To investigate sodium pump and calcium pump activities and mRNA expression level and the changes after AngiotensinII (AngII) treatment in human umbilical artery smooth muscle cells (HUASMCs) isolated from neonates with positive hypertensive family history (FH⁺) or with negative hypertensive family history (FH⁻).

Methods Ion pump activities in cultured HUASMCs were detected by spectrophotography. The mRNA expression of sodium pump α_1 -subunit and plasma membrane Ca²⁺-ATPase isoform 1 (PMCA1) in FH⁺ and FH⁻ HUASMCs was measured by RT-PCR.

Results Sodium pump, calcium pump activities in FH⁺ HUASMCs were higher than those in FH⁻ group (p<0.05), but the mRNA expression of sodium pump α_1 subunit and PMCA1 showed no difference between two groups. In FH⁻ group, after 24-h treatment, AngII (1×10⁻⁷ mol/L) elevated the activities of sodium pump (4.62±0.26 vs 3.52±0.33) and calcium pump (4.00±0.31 vs 3.01±0.32), and up-regulated sodium pump α_1 -subunit mRNA expression (0.946±0.099 vs 0.697±0.050, n=5, p<0.01), however higher concentration AngII (1×10⁻⁶ mol/L) suppressed the activities of sodium (2.47±0.27) and calcium pump (1.79±0.27), and down-regulated sodium pump mRNA expression (0.445±0.065). Whereas, in FH⁺ groups, both concentration (10⁻⁶ and 10⁻⁷ mol/l) of AngII suppressed the activities of sodium pump (3.49±0.34, 2.21±0.23 vs 4.70±0.44) and calcium pump (2.85±0.31, 1.87±0.16 vs 4.27±0.48), but only AngII (10⁻⁷ mol/l) down-regulated their mRNA expression (α_1 -subunit: 0.515±0.133 vs 0.885±0.097, PMCA₁: 0.165±0.049 vs 0.397±0.046, n=5, p<0.01).

Conclusions The activity of sodium pump and calcium pump is increased in FH⁺ HUASMCs. AngII inhibits both Na⁺ and Ca²⁺ ion pumps activities and mRNA expression in FH⁺ HUASMCs, and

may have biphasic effects on ion pump activities and mRNA expression in FH⁺ HUASMCs.

e0596 ROLE OF MONOCYTE CHEMOATTRACTANT PROTEIN-1 ON LARGE ARTERIAL STRUCTURAL AND FUNCTIONAL CHANGE IN PREHYPERTENSIVE SUBJECTS

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Background and Objects Elevated blood pressure causes a change in vascular remodelling and arterial stiffness. Dynamic development of the inflammatory reaction may play a role in the early increase of blood pressure. Monocyte chemoattractant protein-1 (MCP-1) which has a chemotactic effect on monocytes/macrophages, is an initial factor of inflammation. However, whether monocyte chemoattractant protein-1 (MCP-1) is altered in the change of large arterial structure and function in prehypertensive subjects has been incompletely investigated.

Method According to the criteria of JNC7, 160 subjects were divided into three groups: (1) normotensive group (n=57), (2) prehypertensive group (n=50) and (3) hypertensive group (n=53). Brachium-ankle pulse wave velocity (BaPWV) was measured by an automatic wave-form analyser (Form PWV/ABI) and carotid artery intima-media thickness (IMT) was determined ultrasonographically. MCP-1 mRNA level were obtained by real time RT-PCR.

Result In prehypertensives, MCP-1, baPWV and IMT levels are higher than that in normotensives (p<0.01) and lower than that in hypertensives (p<0.01). MCP-1 mRNA level correlated linearly and significantly with baPWV and IMT (p<0.01), even after adjustments for confounding variables.

Conclusions Large artery remodelling has been found in prehypertensive subjects. PWV and IMT were closely related to the level of blood pressure. MCP-1 may play a role structural and functional vascular changes in prehypertensive subjects.

e0597 SERUM HDL-C LEVELS CORRELATED WITH THE HAEMODYNAMIC AND SEVERITY IN PATIENTS WITH IDIOPATHIC PULMONARY ARTERIAL HYPERTENSION

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Aims Although dyslipidemia was an established risk factor for cardiovascular disease, its role in the pathogenesis of pulmonary arterial hypertension (PAH) is still unclear. The aims of the current study were to elucidate the clinical significance of the serum dyslipidemia levels in patients with idiopathic PAH (IPAH).

Methods Serum dyslipidemia levels (total-cholesterol (TC), triglyceride (TG), high-density lipoprotein cholesterol (HDL-C), low-density lipoprotein cholesterol (LDL-C)) and other clinical datas collected from 90 consecutive adult patients with IPAH from April 2008 to Dec 2009 were retrospectively evaluated in our center. Right heart catheterisation was performed in all patients. Forty-five age and sex-matched healthy volunteers served as control subjects.

Results The levels of serum TC and HDL-C was significantly decreased in patients with IPAH compared with control subjects (3.77±0.86 vs 4.23±0.73 mmol/l, p<0.05; 1.04±0.31 vs 1.46±0.31 mmol/l, p<0.001 respectively). Serum HDL-C levels decreased in proportion to the severity of WHO function. Compared with the high HDL-C group, the low HDL-C group demonstrated significantly lower in 6 min walk distance (6MWD), cardiac output

(CO), mixed venous saturation (SvO₂) and PaCO₂; whereas significantly higher in pulmonary vascular resistance (PVR) and serum uric acid (UA) levels. Serum HDL-C levels positively correlated with 6MWD ($r=0.34$, $p<0.001$), CO ($r=0.35$, $p<0.001$), SvO₂ ($r=0.40$, $p<0.001$) and PaCO₂ ($r=0.289$, $p<0.05$); negatively correlated with UA levels ($r=-0.43$, $p<0.001$) and PVR ($r=-0.30$, $p<0.05$).

Conclusion Serum HDL-C levels correlated with the clinical severity of IPAHA and maybe serve as a novel risk factor for the malignant disease.

e0598 THE RESEARCH OF THE RELATION BETWEEN THE REFRACTORY HYPERTENSION AND THE HOMOCYSTEINE

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Objective To explore the relation between the refractory hypertension and the homocysteine (HCY).

Methods 164 patients with hypertension were randomly divided into refractory hypertension group ($n=76$) and non-refractory hypertension group ($n=88$), and meanwhile 60 healthy persons were regarded as the control group. we checked their homocysteine valuation and had the statistics analysis.

Results The homocysteine valuation of refractory hypertension group was significantly higher than those of the control group ($p<0.01$) and was also higher than those of non-refractory hypertension group ($p<0.05$). On the other hand. The homocysteine valuation of non-refractory hypertension group was higher than those of the control group ($p<0.05$).

Conclusion The blood homocysteine may be considered a relationship factor with refractory hypertension.

e0599 THE IMPACT OF VALSARTAN ON ARTERIAL STIFFNESS IN PATIENTS WITH CORONARY HEART DISEASE AND HYPERTENSION

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Objective To examine whether the angiotensin II receptor blockers valsartan would improve arterial stiffness to a greater extent than an equivalent antihypertensive medication, the calcium channel blocker amlodipine in patients with coronary heart disease combined with hypertension.

Methods 75 patients with coronary heart disease combined with hypertension in all accepted brachial-ankle pulse wave velocity (ba-PWV) and central aortic blood pressure measurement. They were administered to take valsartan in a dose of 160mg per day (valsartan group, $n=35$) or amlodipine 5–10 mg per day (amlodipine group, $n=40$) respectively soon after to ensure equivalent BP control. Measurements of ba-PWV and central aortic BP were carried out again after 24 weeks.

Results After 24 weeks there were no statistical differences in coronary artery Gensini score, left ventricular ejection fraction, mean heart rate and types of therapy medication between valsartan group and amlodipine group ($p>0.05$). Systolic blood pressure, diastolic blood pressure and pulse blood pressure of brachial artery as well as central artery between baseline level and end of the study had no statistical differences between two groups ($p>0.05$), and had significant decrease in both groups ($p<0.01$). The levels of central artery BP and brachial BP controlled by valsartan were similar to amlodipine. There were no significant differences in ba-PWV

between two groups at baseline (12.6 ± 2.6 vs 2.1 ± 2.6 , $p>0.05$), however a significant decrease were observed in valsartan group after 24 weeks (12.6 ± 2.6 vs 10.8 ± 1.9 , $p<0.01$), while no significant changes appeared in amlodipine group (12.1 ± 2.6 vs 11.4 ± 2.7 , $p>0.05$).

Conclusion Valsartan may improve arterial stiffness to a significantly greater extent than amlodipine despite similar central artery and brachial BP control.

e0600 IMPACTS OF OBSTRUCTIVE SLEEP APNOEA ON THE BLOOD PRESSURE IN HYPERTENSIVE PATIENTS UNDER THE OPTIMAL MEDICATION

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Background and Objective Obstructive sleep apnoea (OSA) is a prevalent disease, however only 10% OSA patients receive regular treatment. OSA is an independent risk factor for hypertension and cardiovascular diseases. We aim to investigate the impacts of obstructive sleep apnoea on the blood pressure in hypertensive patients under the optimal medication though office blood pressure and 24 h ambulatory blood pressure monitoring (24 h ABPM), respectively.

Methods 52 patients with hypertension were enrolled consecutively and all received the optimal medication for hypertension. An overnight polysomnography and a 24 h ABPM were performed to each patient. According to the apnoea-hypopnoea index, the patients were divided into four groups: no OSA group ($AHI<5$, $n=13$), mild OSA group ($5\leq AHI<15$, $n=19$), moderate OSA group ($15\leq AHI<30$, $n=11$), severe OSA group ($AHI\geq 30$, $n=9$). The results of 24 h ABPM and office pressure were compared respectively.

Results As to the 24 h ABPM results, 24 h systolic and diastolic pressures were significantly higher in severe OSA group than no OSA group (p value is 0.036 and 0.022), and night-time systolic and diastolic pressures were significantly higher too (p value is 0.046 and 0.024) in severe OSA group. Whereas no significant differences were found when compare day-time systolic and diastolic pressures between groups. Moreover, night-time diastolic pressure was significantly higher in severe OSA group than mild OSA group (p value is 0.039). After adjusting the confounders including age, sex, BMI, smoking and drinking history, and cardiovascular diseases, the statistic differences still remained. However, Office blood pressure including systolic and diastolic blood pressure had no significant differences between each two groups.

Conclusion Severe OSA significantly increases blood pressures, especially night-time blood pressures, of hypertensive patients who receive the optimal medication for hypertension. 24 h ABPM is more accurate than office pressure to evaluate the blood pressure of hypertensive patients with OSA.

e0601 EFFECTS OF BMI ON ISCHAEMIC STROKE IN HOSPITAL PATIENTS WITH ESSENTIAL HYPERTENSION

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Objective To investigate the association between BMI and the incidence of ischaemic stroke in essential hypertensive (EH) patients.