for 28 days, then the systolic, diastolic, mean pulmonary artery pressure (PASP, PADP, mPAP) and right ventricle hypertrophy index (RVHI) were measured; the pathological changes of small pulmonary arteries were observed, the thickness index (TI) and area index (AI) of the small pulmonary arteries in three groups were evaluated with microscopy for morphological analysis; echocardiography measuring the internal diameters of the pulmonary trunk assessed pulmonary hypertension in three groups.
Results: ①The PAP(PASP, PADP, mPAP) and RVHI in air embolism group were all significantly higher than these in control group, respectively ($P<0.05$), and these in nicorandil treated group were significantly lower than these in air embolism group, respectively (20.47±3.91 mmHg VS 32.37±3.00 mmHg, 9.35±1.48 mmHg VS 13.49±1.62 mmHg, 13.74±1.86 mmHg VS 21.93±1.10 mmHg and 0.31±0.03 VS 0.47±0.13 $P<0.05$). ②The thickness of wall increased and the cavity became narrow in the small pulmonary arteries of air embolic group, and these were significantly ameliorated in the small pulmonary arteries of nicorandil treated group. The TI and AI in the small pulmonary arteries of air embolic group were significantly higher than these in control group, respectively ($P<0.05$), and these in the small pulmonary arteries of nicorandil treated group were significantly lower than these in air embolic group, respectively (0.38±0.12 VS 0.75±0.13, 0.60±0.16 VS 0.93±0.09 $P<0.05$). ③The internal diameters of the pulmonary trunk was higher in air embolic group than that in control group($P<0.05$); That was redused in nicorandil treated group(0.78±0.06cm VS 0.93±0.14cm, $P<0.05$).
Conclusions: Nicorandil can significantly attenuate the chronic air embolism-induced pulmonary hypertension, improve pulmonary vascular remodeling of the small pulmonary artery and right ventricular hypertrophy and reduse the internal diameters of the pulmonary trunk.

GW25-e5398

Serum High-Density Lipoprotein Cholesterol Levels as a Prognostic Indicator in Patients With Idiopathic Pulmonary Arterial Hypertension

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Objectives: High-density lipoprotein (HDL) cholesterol levels are a strong, independent inverse predictor of cardiovascular disease. The present study aimed to determine whether serum HDL cholesterol levels correlated with disease severity and clinical outcomes in patients with idiopathic pulmonary arterial hypertension (IPAH). The present study aimed to determine whether serum HDL cholesterol levels correlated with disease severity and clinical outcomes in patients with idiopathic pulmonary arterial hypertension (IPAH).
Methods: The serum HDL cholesterol levels were measured in 76 Chinese patients with IPAH and 45 healthy controls, together with other clinical variables. Univariate and multivariate Cox proportional hazards analysis was performed to assess the prognostic value of HDL cholesterol and event-free survival. Event-free survival was estimated using the Kaplan-Meier method.
Results: Serum HDL cholesterol levels were significantly decreased in patients with IPAH compared with controls (1.0±0.3 vs 1.5±0.3 mmol/L; P<0.001). The serum HDL cholesterol levels decreased in proportion to the severity of World Health Organization functional class. Compared to the high HDL cholesterol group, the low HDL cholesterol group demonstrated a significantly lower 6-minute walking distance, cardiac index, mixed venous saturation, and arterial carbon dioxide pressure but significantly greater pulmonary vascular resistance and serum uric acid levels. The serum HDL cholesterol levels correlated positively with the cardiac index (r=0.42; P=0.002) and negatively with the pulmonary vascular resistance (r=-0.25; P=0.04). Serum HDL cholesterol was independently related to event-free survival on multivariate Cox proportional hazards analysis. Kaplan-Meier survival curves according to the median HDL cholesterol value showed that lower HDL cholesterol levels were associated with lower event-free survival.
Conclusions: Serum HDL cholesterol levels might serve as an indicator of disease severity and prognosis in patients with IPAH.

GW25-e0285

The Diagnostic Probability of Suspected Acute Pulmonary Thromboembolism Based on the Objective Clinical Tests

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Objectives: For the diagnostic awareness of acute pulmonary thromboembolism (APTE) was increased in recent years, it became a relatively common cardiovascular emergency. Because the pulmonary arterial bed is occluded by the thrombolus, usually origenated from the deep vein of lower extrimities, an acute life-threatening right ventricular failure might occur which could lead to severe shock and even death. It is a difficult diagnostic task for a clinician because of non-specific clinical presentation of APTE. If early diagnosis were missed, the patients would die of acute right heart failure, potentially fatal early recurrences or chronic right failure due to chronic thromboembolic pulmonary hypertension. The clinical evaluation is important to classify patients into probability categories corresponding to an increasing prevalence of APTE.A prediction rule makes it possible to discriminate suspected APTE patients for clinicians, that has become a key step in all diagnostic algorithms for APTE. The most frequently used clinical prediction rule is Wells score that is based on early clincal manifestations.But the reproducibility was variable due to one subjective item in the rule (alternative diagnosis less likely than APTE). Thus the usefullness of Wells score was limited. For the different medical system and policy of medical insurance between western countries and China, it is much easier to collect ECG and echocardiography information for Chinese physicians. In this study, the diagnositic probablity of suspected APTE based on the objective clinical tests was researched.
Methods: 425 consecutive cases of suspected APTE were enrolled, which included 161 confirmed cases and 264 excluded cases according to the result of CT pulmonary angiography. The items in Wells score including risk factors, symptoms and signs, and two objective variables: electrocardiography (ECG) and echocardiography were studied by Logistic multiple regression analysis.
Results: The odd ratio and 95% CI of the parameters above were: history of APTE or deep venous thrombosis (DVT) 3.22 (1.13-5.20); Recent surgery or immobilization 2.66 (1.28-4.12); cancer1.34 (0.58-2.50); hemoptysis2.85 (1.14-4.97); heart rate >100 beats/min 1.02(0.93-1.40); clinical signs of DVT 2.28 (1.24-3.01); abnormal ECG including negative T wave of lead V_1-V_4 or $S_IQ_{III}T_{III}$ 2.21 (1.55-3.81); abnormal echocardiography including dilation of right ventricle or increased estimated systolic pulmonary arterial pressure 1.95 (1.30-3.18), P<0.05 respectively.
Conclusions: The abnormal ECG and echocardiography were risk factors for the diagnosis of APTE, could be objective evidence for item 'alternative diagnosis less likely than APTE' in Wells score for APTE. It might make Wells score operatable and reproducible.

GW25-e2403

Valsartan attenuates pulmonary arterial hypertension via mitogen-activated protein kinases (MAPKs) signal pathway and Matrix metalloproteinases (MMPs) in rodents

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Objectives: Studies have demonstrated that rennin angiotension system is involved in the pathogenesis and development of pulmonary arterial hypertension (PAH). However, it is controversial whether angiotensin II type I receptor blockers attenuate PAH. Here we examined the prevention effects of valsartan on experimental rodents of PAH.
Methods: Male Sprague-Dawley rats with monocrotaline-induced PAH and male C57 mice with hypoxia-induced PAH were administered oral valsartan for 3 weeks. Heamodynamic and anatomic data were determined in rats and mice. Histopathology, immunohistochemistry, assessments of cell proliferation and stabilization of extracellular matrix were performed in lung tissue of rats.
Results: In comparison with control rodents (rats and mice), valsartan markedly improved the survival and decreased the RV systolic pressure and RV hypertrophy index. Masson's trichrome staining and immunohistochemical analyses revealed that valsartan suppressed cell proliferation, attenuating the pulmonary vascular remodeling. Further, western blot showed that valsartan inhibited the enhanced p38, JNK1/2 and ERK1/2 phosporylation in PAH rats. Similarly, active matrix metalloproteinase-2 (MMP-2) and MMP-9 were significantly attenuated by valsartan. Furthermore, valsartan significantly reduced the increase of TGF-β1 in rat lung tissue.
Conclusions: These results suggest that valsartan attenuates the progression of PAH via inhibition of cell proliferation and MMPs expression and activity in experimental rodents. Thus, valsartan might be a valuable prevention therapeutic approach for PAH.

GW25-e0262

Correlation of gene expressions between cytokines and platelet αIIbβ3 in patients with symptomatic pulmonary embolism

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Objectives: The American Collage of Chest Physicians indicated that infection is a risk factor for VTE. And Inegrin αIIbβ3, a kind of platelet membrane glycoprotein, is the key factor affecting platelet activation. The aim of this study was to investigate the gene expression correlation of cytokines and platelet activation-related mRNAs in peripheral blood mononuclear cells between patients with symptomatic PE.
Methods: Twenty cases of PE patients and twenty sex and age matched controls were recruited into the study. Human cDNA microarray analysis, random variance model corrected T test and multiple linear regression analysis were used to analyze the statistical data.
Results: (1) Fourteen platelet membrane glycoprotein mRNAs were detected. In PE patients, 7 of them includingITGA2B and ITGB3 were up-regulated significantly in the PE group (P<0.01). 2). One hundred and twelve mRNAs of chemokines, interleukins, colony-stimulating factors, interferons and tumor necrosis factors were