CONCLUSIONS Changes in serum concentrations of sex hormones are extensive in adult patients with IPAH. The influence of these changes on the development of PAH and on the outcome of this condition deserves further study.

GW26-e2948
The Application of Intravascular Ultrasound to Evaluate Pulmonary Vascular Properties and Predict Mortality in Pulmonary Arterial Hypertension
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OBJECTIVES We aimed to explore the application of intravascular ultrasound (IVUS) to evaluate pulmonary vascular properties (PVMPs) and predict mortality in pulmonary arterial hypertension (PAH).

METHODS Patients (n = 51) with a systolic pulmonary arterial pressure (SPAP) ≥ 40 mmHg based on echocardiography were prospectively enrolled. After underwent right heart catheterization (RHC) and IVUS, they were divided into 3 groups: PAH associated with connective tissue diseases (PAH-CTD) group (group1, n = 25), PAH due to other causes group (group2, n = 15), and CTDs patients without PH (group3, n = 11). PAH group (groups 1 and 2) was divided into distal and proximal remodeling subtype based on IVUS results. All patients were followed-up to compare the differences among clinical variables, PVMPs and survival rates.

RESULTS A total of 408 segments of pulmonary vessels were studied. PAH group demonstrated a greater mean wall thickness (MWT) (0.30 ± 0.02 vs. 0.21 ± 0.02 mm, P < 0.01) and a higher percentage of MWT (WTP) (13.62 ± 5.99 vs. 9.57 ± 5.97%, P < 0.01) than group 3. Additionally, the pulmonary vascular mechanical properties (PVMPs) in PAH group were found to be worse than those in group 3, with reduced compliance (8.85 ± 4.38 vs. 14.39 ± 6.28%/mmHg, P < 0.01) and stiffness index (0.83 ± 0.09 vs. 0.55 ± 0.06 vs. 2.36 ± 0.28%/mmHg, P < 0.01). However, no difference among the groups was reported.

CONCLUSIONS IVUS may be useful in assessment of PH by evaluating PVMPs and predicting mortality. PAH group demonstrated worse PVMPs than CTDs patients without PH. PAH-CTD patients had better PVMPs than PAH due to other causes. There was no difference between PAH groups in survival curves. However, the distal remodeling subtype had a higher mortality (22.73%) than the proximal remodeling subtype with a hazard ratio of 10.14 (95% confidence interval 2.61–61.00, P < 0.001).

GW26-e4462
Prognostic value of cardiac troponin T and plasma lactate levels among patients with acute pulmonary embolism
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OBJECTIVES To investigate the clinical value of detecting cardiac troponin T (cTnT) and plasma lactate (lac) for risk stratification and prognosis evaluation in patients with acute pulmonary embolism (APE).

METHODS From January 2013 to December 2014, a total of 89 patients were diagnosed with APE. All patients with a diagnosis of APE established by lung scan or spiral computed tomography (CT) and confirmed by pulmonary angiography if necessary. Plasma tropinin T and lactate levels were tested at presentation. We considered lactate values greater than or equal to 2 mmol/L and troponin T values greater than or equal to 0.10 ng/mL to be abnormal. 89 patients with confirmed APE were divided into three groups according to the levels of tropinin T and plasma lactate: Group1 (n = 16): cTnT < 0.1 ng/mL and lac < 2.0 mmol/L; Group2 (n = 35): cTnT > 0.1 ng/mL or lac > 2.0 mmol/L; Group3 (n = 38): cTnT > 0.1 ng/mL and lac > 2.0 mmol/L. Analysis of troponin T and plasma lactate elevated risk stratification in patients with APE’s relationship with clinical prognosis.

RESULTS Of the 89 patients included in the study, the mean age was 67 years (SD 10.2 years) and 45 (50.6%) were women. Clinical harmful events were death caused by pulmonary embolism and clinical deterioration defined as progression to thrombolytic therapy, vasoactive drugs, mechanical ventilation, shock, or cardiopulmonary resuscitation. Patients with clinical harmful events in group 1, 2 and group 3 were 0 (0%) case, 7 (20.0%) cases, 21 (53.3%) cases respectively and a significant difference was observed (P < 0.01). In the group of proceeding clinical harmful events there were significant differences in troponin T and plasma lactate levels (p < 0.01). 5 patients (6%) died in group 3 and 0 (0%) case died in group 1 and group 2. All patients underwent thrombolysis treatment in group 3, 10 (25.6%) patients underwent thrombolysis treatment in group 2, and all patients underwent routine anticoagulation.

CONCLUSIONS Combinational detection of troponin T and plasma lactate has important value for early risk stratification and prognosis evaluation in patients with APE. Patients with pulmonary embolism elevated troponin T and plasma lactate level are at high risk of death and adverse outcome.

GW26-e4721
Circulating Progesterone and Hemodynamic Parameters in Men with Idiopathic Pulmonary Arterial Hypertension
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OBJECTIVES Pulmonary arterial hypertension (PAH) is a female predominant disease. However, women have preserved right ventricle function and better survival compared with men suffering from PAH. This “sex paradox” indicates sex hormones may contribute significantly to the pathogenesis of PAH. We sought to assess the relationship between serum concentration of sex hormone and hemodynamic parameters in men with idiopathic PAH (IPAH).

METHODS In the observational study, we recruited 98 male IPAH patients (mean ± SD age, 41 ± 18 years), at Cardio-Pulmonary Circulation center (Shanghai Pulmonary Hospital), from June 2008 to October 2014. The study included 85 age-matched healthy male control subjects. Serum concentrations of estradiol (E2), testosterone (TT) and progesterone (P) were measured using immunoassays, and the clinical, functional, and hemodynamic compromises were collected at enrollment.

RESULTS Compared with the healthy controls, serum E2 concentrations were increased, and serum TT, P concentrations were decreased in IPAH patients [E2 (153 (99-215) vs 121 (97-140) pmol/l, P = 0.002; TT: 8.8 (4.6-21.0) vs 15.8 (12.0-19.5) pmol/l, P = 0.007; P: 0.32 (0.22-0.43) vs 0.48 (0.30-0.63) ng/ml, P < 0.001]. There were correlations between P and HR, WHO functional class (FC), 6MWD, serum brain natriuretic peptide (BNP), myocardial injury, pulmonary vascular resistance and cardiac output (all P < 0.05). There were no correlations between E2 or TT and the above parameters. At linear regression analysis, P was the predictor most associated to WHO FC, 6MWD, PVR elevation and CO reduction. Furthermore, P was only the independent predictor most associated with PVR elevation after adjustment for age, BSA and WHO FC by multivariate analysis (R² = 0.346, 95% IC: 0.153 ~ 0.589, P = 0.027).

CONCLUSIONS This work demonstrates serum P concentrations were decreased in male IPAH patients. It’s an independent predictor for PVR elevation and could estimate the severity of men with IPAH.

GW26-e5353
Galectin-3: a Potential Biomarker in Pulmonary Arterial Hypertension
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OBJECTIVES Pulmonary arterial hypertension (PAH) is a syndrome resulting from a restricted flow through the pulmonary arterial circulation, giving rise to increased pulmonary vascular resistance and
ultimately right heart failure. Previous reports have suggested that Galactin-3 (Gal-3) induced endothelial cell morphogenesis and regulated myofibroblast activation. The aim of this study was to determine the diagnostic utility of circulating Gal-3 as a potential biomarker of disease severity in PAH.

**METHODS** Gal-3 was measured in plasma from 31 patients with PAH, diagnosed from right heart catheterization, as well as 18 healthy controls by ELISA. Besides, chronic hypoxia induced pulmonary hypertension models were established in Sprague-Dawley rats. Lung tissues were collected for histological analysis including Gal-3 lung qualitative localization by immunohistochemistry. Total mRNA was extracted from pulmonary arteries in rats, then quantitative PCR was performed with total cellular mRNA to measure Gal-3 expression.

**RESULTS** Plasma level of Gal-3 was significantly decreased in PAH patients compared with healthy controls (p < 0.001). Within the subgroups the correlation was given only by idiopathic PAH (IPAH) patients statistically expressed the lower level of Gal-3 (n = 16, p < 0.001). Gal-3 levels inversely correlated with mean pulmonary arterial pressure (mPAP) (r = -0.57, p = 0.021) and pulmonary vascular resistance (PVR) (r = -0.55, p = 0.027), and correlated with cardiac output (r = -0.530, p = 0.035) in IPAH patients. A Gal-3 cut off value less than 1.765 ng/ml yielded 93% sensitivity and 88% specificity for IPAH patients. Immunohistochemistry method identified Gal-3 was distributed throughout the adventitia of the pulmonary arterioles. The expression of Gal-3 mRNA was significantly down-regulated in the pulmonary arteries from lung tissue samples in pulmonary hypertension rats.

**CONCLUSIONS** Gal-3 might be involved in the pathogenesis of PAH, and plasma Gal-3 could serve as a promising new biomarker of diagnosis and disease severity in IPAH.

**GW26-e03831** Effects of adipose tissue-derived stem cells transplanted with adiponectin gene in monocrotaline-induced pulmonary arterial hypertensive rats

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**OBJECTIVES** To observe the effects of adipose tissue-derived stem cells (ADSC) and adiponectin (APN) gene-modified ADSC transplantation in monocrotaline (MCT) induced pulmonary arterial hypertensive (PAH) rats.

**METHODS** Eighty SD rats were randomly divided into normal control group (NC group), PAH group, ADSC group (ADSC group), empty lentiviral vector infected ADSC group (ADSC-V group) and APN gene modified ADSC (ADSC-APN) group. PAH group, ADSC group, ADSC-V group and ADSC-APN group were given 40mg/kg MCT by intraperitoneal injection. Fourteen days after MCT injection, 1.0 x 10^6 cells/ml suspension, including with ADSC, empty lentiviral vector infected ADSC or APN gene modified ADSC, were injected into ADSC group, ADSC-V group or ADSC-APN group through left jugular vein, respectively. Three weeks after cells transplantation, mean pulmonary arterial pressure (mPAP), right ventricular hypertrophy index (RVHI), vasoconstriction and diastolic function, wall to lumen thickness (WT%) and area (WA%) of pulmonary artery were detected. Serum APN levels were assayed by ELISA. The expression of eNOS in pulmonary artery was detected by immunohistochemical staining. Right heart function was evaluated by echocardiography. Plasma metabolites were detected by NMR-based metabolomics technology. The expressions of bone morphogenetic protein-2 (BMP2), p-Smad1/5/8, Smad1/5/8/9, p-AMPK and AMPK were detected by western blot after endothelium-dependent and endothelium-independent relaxation of pulmonary arteries. Meanwhile, eNOS expression in the intima of pulmonary artery was significantly upregulated. Echocardiography showed that right ventricular structure and function were restored. Compared with ADSC alone transplantation, APN gene-modified ADSC transplantation therapy is better than ADSC transplantation alone. Transplantation of APN gene-modified ADSC could significantly decrease the levels of serum glucose and serum lactate and increase the levels of serum alanine. APN upregulated the BMP2 expression of PASMCs in a dose-dependent manner. BMP inhibitor could reduce the expression of BMP2 and p-Smad1/5/8 induced by APN, and promote cells proliferation. AMPK inhibitor could reduce the expression of p-AMPK and BMP2 induced by APN.

**CONCLUSIONS** ADSC and APN gene-modified ADSC could increase the expression of eNOS, improve vasodilation, reduce pulmonary artery remodeling, decreased mPAP, and improve right ventricular function. The transplantation of APN gene-modified ADSC was better than ADSC transplantation alone. APN gene-modified ADSC could improve Warburg effect and alanine metabolism in MCT-induced PAH rats. The mechanism that APN inhibits proliferation of PASMCs may be related to the activation of AMPK and BMP/Samd pathway.