Methods: Rats were daily exposed to cigarette smoke in the absence and in the presence of losartan. After 6 months, lung morphology and tissue biochemical changes were examined by Immunohistochemistry, Western blotting, RT-PCR and radioimmunoassay. Cell proliferation and ACE2 levels were measured in cigarette smoke extract-challenged rat primary pulmonary artery smooth muscle cells (PASMCs).

Results: Thickened wall of pulmonary arteries with apparent medial hypertrophy along with increased angiotensin II and decreased ACE2 levels were observed in smoke-exposed-only rats. Losartan administration ameliorated pulmonary vascular remodeling, inhibited Ang II elevation and partially reversed the ACE2 decrease in rat lungs. In cultured PASMCs from 3- and 6-month smoke-exposed rats, ACE2 levels were significantly lower than in those from the control rats. Moreover, PASMCs from 6-month exposed rats proliferated more rapidly than those from 3-month exposed or control rats, and cells grew even more rapidly in the presence of DX600, an ACE2 inhibitor, consistent with the in vivo study, in vitro losartan pretreatment also inhibited cigarette smoke extract (CSE)-induced cell proliferation and ACE2 reduction in rat PASMCs.

Conclusions: The results suggest that angiotensin II receptor antagonist may be therapeutically useful in the chronic smoking-induced pulmonary vascular remodeling and ACE2 may be involved as part of its mechanism. Our study might provide insight into the development of new therapeutic interventions for pulmonary vascular remodeling smokers.

GW25-e0504
Effect of Valsartan on expression of miR-145 in rats with Pulmonary Hypertension
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Objectives: Experiments show that miR-145 is a must to keep the vascular smooth muscle contractile phenotype, in pathological conditions, the expression of miR-145 in synthetic-typed vascular smooth muscle is reduced, but change of expression in pathological hypertension condition and the regulatory mechanisms have been rarely reported. This experiment will observe expression changes of miR-145 in pulmonary artery, and apply RAS antagonists-Valsartan to intervene these rats, observing effects on expression of miR-145.

Methods: 36 SD rats were randomly assigned into three groups (Normal group, Control group and Valsartan group) with 12 rats in each group. Rats in Normal group and Valsartan group were respectively given disposable intraperitoneal injection with 2%/MCT (60 mg/kg). Rats in Valsartan group were given intragastric administration and Valsartan group were respectively given disposable intraperitoneal injection with Continuous ambulatory peritoneal dialysis was 0.850 in GLPSS. The cut-off value for GLPSS of -15.6% had 90.4% sensitivity and 84.2% specificity for detection of patients with normal LV ejection fraction. Strain dispersion index, a measure of regional contractile heterogeneity, was higher in patients compared with controls.

Conclusions: 2DS (AFI) allows rapid characterization of regional and global systolic function and may have the potential to detect early abnormal regional and global systolic function in patients with Continuous ambulatory peritoneal dialysis from the normal ejection fraction of left ventricular.

Peripheral Vascular Disease

GW25-e4379
Crossover chimney technique to preserve the internal iliac artery in a ruptured aortic dissection aneurysm with bilateral iliac entry tears 10 years after TEVAR
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Objectives: The natural history of the aortic growth after thoracic endovascular aortic repair (TEVAR) in Stanford B aortic dissection remains unclear. We reported a rare case developing a huge aortic dissection aneurysm 10 years after the primary TEVAR which ruptured before admission.

Methods: By the aortography and multiple distal intimal entry tears locating above and at the origin of the celiac artery, the origin of the right internal iliac artery (IIA) and the end of the left common iliac artery (CIA). The maximum size of the ruptured dissection aneurysm was 137mm×97mm below the renal arteries. The true lumen was compressed and the size of which at the aortic bifurcation was only 8.15mm, Cerebrospinal fluid (CSF) drainage was placed before the procedure to prevent paraplegia. After the exposure of the bilateral common femoral arteries (CFAs), a thoracic stent-graft was deployed above the superior mesenteric artery. The origin of the left IIA was embolized with coils. A crossover sheath was inserted from the right CFA to the left IIA. A Viabahn stent-graft was positioned 2 cm inside the IIA. A unibody bifurcated abdominal stent-graft was deployed below the renal arteries.
Limb extensions were positioned to bilateral external iliac arteries (EIA). After the Viabahn deployment, a self-expandable nitinol stent was deployed overlapping inside the Viabahn, landing in the right EIA and 2 mm distal to the right limb extension. Complete angiography showed there was no endoleak from the IIA or the chimney graft, the false lumen was totally excluded, and the chimney graft to the left IIA was patent. The CSF drainage was removed 48 hours after the procedure, and the patient recovered uneventfully. He was discharged with aspirin antiplalet.

**Results:** The computed tomographic angiography 6 months after the procedure demonstrated a total thrombosis of the false lumen, the patency of all grafts and a good accommodation of the crossover chimney graft with the right iliac limb extension.

**Conclusions:** Our experience showed that type B dissection may develop ruptured aortic dissection aneurysm long time after TEVAR. The multiple entry tears may be troublesome during the reintervention, however the crossover chimney technique may help preserve the IIA flow in case with bilateral iliac entry tears.

**GW25-e5284**

**Peridontal Pathogen exacerbate the development of experimental Abdominal Aortic Aneurysms and stimulate IL-6 and MMP-9 expression**

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**Objectives:** Abdominal aortic aneurysm (AAA) represents a common degenerative vascular condition with life-threatening risks. Periodontal pathogens are detected at a higher frequency from the aortic walls of patients with AAA. However, the role of the periodontal infection remains unclear. The purpose of this study was to analyze the influence of periodontal pathogen on AAA dilation and plausible mechanism.

**Methods:** AAA was produced by peri-ortic application of 0.25 mol/L CaCl2, with NaCl used as a control. The mice were inoculated with live Porphyromonas gingivalis or vehicle once weekly.

**Results:** Eight weeks later, the prior application of CaCl2, the dilatation rates of the the aortic diameter is significantly higher in P. gingivalis infected mice than those without P. gingivalis infection. Immunohistological analysis found significantly higher levels of MMP-9 in the aneurysmal samples of P. gingivalis-challenged mice compared with control mice. Serum IL-6 and MMP-9 levels also significantly elevated in the CaCl2 treated mice with P. gingivalis infection than those without P. gingivalis infection.

**Conclusions:** Chronic P. gingivalis infection stimulates IL-6 secretion favoring MMP-9 expression and ultimately accelerating AAA development. These findings suggest that periodontal pathogen may influence AAA development via MMP-9 induction.

**GW25-e1664**

**Using ultrasound Doppler to optimization of the table speed of lower extremity CT angiography protocols**

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**Objectives:** Scanning with 64-slice multidetector CT (MDCT) is usually faster than blood flow in peripheral arteries of the lower extremities, and the distal arteries of lower extremities are difficult to visualize, particularly in the case of patients with diffuse atherosclerosis and asymmetric arterial disease. Thus, predetermination of the flow velocity in the arterial circulation and aligning the flow velocity and table speed to achieve optimal synchronization of the acquisition with the propagation of the contrast bolus is important. We evaluated the relative efficacy of different table speeds predetermined by using the flow velocity measured by ultrasound Doppler in patients with patients with suspected peripheral arterial occlusive disease (PAOD) undergoing CTA of lower extremity arteries.

**Methods:** This prospectively study enrolled 40 patients with suspected PAOD. The average aorta-popliteal artery flow velocity (VAO-POP) was measured by ultrasound Doppler in each group. Having received corresponding treatment, the mice were inferior Vena Cava ligated. Two days later, the mice were sacrificed and the tissues were harvested for relative tests. At the same time, in the in vitro study, endothelial cells and monocytes were incubated. We treated the cells with different concentrations of bradykinin, followed by lipopolysaccharide induction for 4 hours, and then the protein was extracted and TF expression was measured, after which we searched for the underlying mechanisms.

**Results:** Ligation of inferior Vena Cava induced thrombosis, and this trend was largely attenuated by the intraarterial injection of bradykinin, both in thrombus size and generation rate. Expression of tissue factor in thrombus and the fibrin in the liver of the bradykinin team were also greatly reduced, with inflammation cytokines simultaneously decreased. Correspondently, by up-regulating PI3K/AKT and down-regulating MAPK signaling pathways, bradykinin dose dependently inhibited the expression of tissue factor in the endothelial cells and monocytes.

**Conclusions:** By regulating PI3K/AKT and MAPK signaling pathways bradykinin can reduce the expression of tissue factor and suppress the stasis induced thrombus in intact vessels.

**GW25-e5359**

**Association of dyslipoproteinemia with the disease activity of Takayasu arteritis**

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**Objectives:** Our study aimed to determine whether proatherogenic lipid profiles exist in patients with active TA and to assess the relationship between different lipid profiles and disease activity in TA.

**Methods:** A total of 120 premenopausal female patients with TA and 100 sex-, age-, and body mass index-matched healthy controls were included in our study. The clinical data were collected in detail from all participants.

**Results:** Patients with active TA significantly have higher ratios of apolipoproteinB/apolipoproteinA1 (apoB/apoA1) (0.70 ± 0.27 vs. 0.48 ± 0.14, P < 0.001), and lower levels of apolipoproteinA1 (apoA1) (1.48 ± 0.30 vs. 1.99 ± 0.33 mmol/L, P < 0.001) and high density lipoprotein cholesterol (HDL-C) (1.22 ± 0.33 vs. 1.68 ± 0.38 mmol/ L, P < 0.001) compared with patients with inactive TA. Multiple linear regression analysis found that the apoB/apoA1 ratio was independently associated with TA activity (β = 0.45, P < 0.001). In addition, multivariate stepwise forward regression analysis found that the apoB/apoA1 ratio was the major determinant for bChAP.

**Conclusions:** Our findings indicate that patients with active TA have proatherogenic lipid and lipoprotein profiles. In addition, the ratio of apoB to apoA1 could be a marker to be monitored and a target that needs to be treated in patients with active TA.

**GW25-e4381**

**Popliteal artery embolization in superficial femoral artery interventions: incidence, risk factors, treatment and prognosis**

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**Objectives:** Endovascular therapy has gained acceptance as a primary treatment modality for superficial femoral artery (SFA) disease. Popliteal artery embolization (PTE) is considered a severe complication in SFA interventions. The purpose of this study was to evaluate the incidence, risk factors, treatment and prognosis of PAE in primary SFA percutaneous transluminal angioplasty and stenting (PTA+S).

**Methods:** Chronic SFA arteriosclerosis cases that underwent primary PTA+S were reviewed from a prospectively maintained database. Runoff vessels were evaluated at the time of revascularization for detection of PAE. The primary patency, secondary patency and limb salvage rates were calculated using Kaplan-Meier analysis and compared using log-rank analysis.

**Results:** There were 436 lesions treated in 388 patients with 10 PAE events (2.3%) in total. PAE rate was significantly higher in Transatlantic Inter-Society Consensus (TASC) C/D group compared with TASC A/B group (OR: 8.91, P = 0.002), in chronic total occlusion (CTO) lesions compared with stenotic lesions (P < 0.001), and in group with history of cerebral ischemic stroke (OR: 6.11, P = 0.007). PAE rates were significantly affected by age, sex, smoking, hypertension, diabetes, hyperlipidemia and runoff status. The 12-month and 24-month primary patency, secondary patency and limb salvage rates in PAE group showed no significant differences comparing with non-PAE group.

**Conclusions:** PAE is a rare event in primary SFA PTA+S. TASC C/D lesion, CTO and cerebral ischemic stroke history are risk factors for PAE. PAE can be treated by