after AMI remodeling. Importantly, a selective PPARγ inhibitor T0070907 (at a dose of 1 mg/kg/g for 21 days) prevented the protective effects of QL against AMI remodeling. Cardiac function were decreased after the combination of PPARγ inhibitor with QL. Myocardial infarct size and apoptosis-related factors were all increased, confiming the inhibition effect of PPARγ inhibitor on QL. Thses results indicated that QL attenuates cardiac remodeling after AMI through PPARγ. To further claify whether QL takes effetcs in acute stage or remodeling stage, mice was either treated for 3 days or from 3 days-21days. It is found that QL performed functional effect mainly during chronic stage, and had no evident effect on acute stage as determined by measuring the percentage of myocardial ischemic infarct size/area-at-risk (AAR).
Conclusions: QL attenuates cardiac remodeling after AMI. The effects of QL in attenuating cardiac remodeling after AMI was at least partially via targeting PPARγ.

GW25-e5193

The remodeling of gallbladder artery and altered expression of calcium handling genes in hypertensive patients

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Objectives: The remodeling of gallbladder artery in hypertensive patients and its underlying mechanisms are poorly understood. The present study observed the morphological and histological changes of gallbladder arteries and investigated the mechanisms of calcium handling genes involved in remodeling.
Methods: A total of 44 patients with biliary calculus underwent cholecystectomy at department of hepatobiliary surgery in Affiliated Hospital of Zunyi Medical College from Jun, 2011 to Mar, 2012. Among them, 21 patients without risk factors were selected on age and sex-matched method and divided into control group (n=11, normal blood pressure) and hypertensive group (n=10). HE staining and Masson staining were used to observe the morphology changes of arteries. The intima-media thickness (IMT), intimal cross-sectional area (ICSA), medial cross-sectional area (MCSA), collagen volume fraction (CVF) of intima and media was analyzed by computer image analysis system. The protein expressions of α-smooth muscle actin (α-SMA) and proliferating cell nuclear antigen (PCNA) were detected by immunohistochemical technique. The mRNA expression levels of embryonic smooth muscle myosin heavy chain (SMemb) and calcium handling genes were detected by Realtime PCR.
Results: Compared with control group, IMT (79.5±4.7 vs 51.2±4.3 μm), intima-media thickness to internal diameter ratio (IMT/ID) (0.25±0.02 vs 0.17±0.01), ICSA to internal diameter ratio (ICSA/ID) (67.7±9.2 vs 39.6±8.7) and MCSA to ID ratio (MCSA/ID) (242.4±20.7 vs 153.3±19.7) were increased in hypertensive group (all $P<0.05$); Compared with control group, in intima or media of artery, CVF (0.36±0.03 vs 0.17±0.03; 0.36±0.02 vs 0.28 ±0.02, all $P<0.05$) and cell proliferation index (0.61±0.05 vs 0.36±0.05; 0.73±0.05 vs 0.54±0.05, all $P<0.01$) were increased in hypertension subjects; In media of artery, the gene expression of SMemb, sodium pump α_1 subunit and transient receptor potential canonical channel type 1 (TRPC1), TRPC3 were increased, while sodium pump α_3 subunit and plasma membrane calcium-transporting ATPase 4 were decreased in hypertensive group (all $P<0.05$).
Conclusions: This study provides evidences that hypertension is associated with the remodeling of the gallbladder artery. The phenotypic change of vascular smooth muscle cell and the abnormal expression of the calcium handling genes may play an important role in the arterial remodeling.

GW25-e1736

The role of neutrophil-lymphocyte ratio in Takayasu arteritis disease monitoring

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Objectives: Blood neutrophil-lymphocyte ratio (NLR) is an indicator of the overall inflammatory status of the body. Takayasu arteritis (TA) is a chronic non-specific inflammatory disease. The objective of the present study was to assess whether the NLR would be useful in TA disease monitoring. Additionally, a possible relationship between NLR and other inflammatory markers in patients with TA was also investigated.
Methods: Eighty-seven patients and 59 healthy controls were enrolled in the study. The neutrophil and lymphocyte counts were recorded, and the NLR was calculated from these parameters. Disease activity and severity in patients with TA were defined according to the National Institutes of Health and Ishikawa's criteria, respectively.
Results: NLR values were higher in patients with TA compared with healthy controls [2.02 (1.68-3.00) vs. 1.70 (1.37-2.16), $P<0.001$]. There were also significant differences in the NLR values between patients with active disease and patients in remission [2.63 (1.95-4.13) vs. 1.81 (1.50-2.38), $P<0.001$]. Patients with severe TA showed significantly higher NLR values than those with mild-moderate TA [2.09 (1.74-4.20) vs. 1.94 (1.64-2.68), $P=0.033$]. In patients with available longitudinal data, NLR values at the active phase were significantly higher than those at the stable phase [2.86 (2.28-4.84) vs. 2.13 (1.51-3.14), $P=0.001$]. Moreover, NLR values were found to be correlated with inflammatory markers, including C-reactive protein ($\rho= 0.262$, $P=0.014$), erythrocyte sedimentation rate ($\rho = 0.255$, $P=0.017$), and white blood cell count ($\rho = 0.429$, $P<0.001$).
Conclusions: NLR values were significantly increased in patients with active TA exhibiting severe complications. These results indicated that NLR may be a useful marker to assess disease activity, severity, and progression of TA.

GW25-e0290

Pharmacologic approach to defective protein trafficking in the E637K-hERG mutant with PD-118057 and thapsigargin

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Objectives: Treatment of LQT2 is inadequate. Many drugs which can pharmacologically rescue defective protein trafficking in LQT2 also result in potent blockade of HERG current, negating their therapeutic benefit. It is reported that PD-118057 and thapsigargin can rescue LQT2 without hERG channel blockade, but the precise mechanism of action is unknown. Furthermore, the effect of PD-118057 and thapsigargin on the dominant negative E637K-hERG mutant has not been previously investigated.
Methods: The whole-cell Patch-clamp technique was used to assess the effect of PD-118057 and thapsigargin on the electrophysiological characteristics of the rapidly activating delayed rectifier K^+ current (I_{kr}) of the hERG protein channel.Western blot was done to investigate pharmacological rescue on hERG protein channel function.
Results: In our study, PD-118057 was shown to significantly enhance both the maximum current amplitude and tail current amplitude, but did not alter the gating and kinetic properties of the WT-hERG channel, with the exception of accelerating steady-state inactivation. Additionally, thapsigargin shows a similar result as PD-118057 for the WT-hERG channel, but with the exception of attenuating steady-state inactivation. However, for the WT/E637K-hERG channel, PD-118057 had no effect on either the current or on the gating and kinetic properties. Furthermore, thapsigargin treatment did not alter the current or the gating and kinetic properties of the WT/E637K-hERG channel, with the exception of opening at more positive voltages.
Conclusions: Our findings illustrate that neither PD-118057 nor thapsigargin play a role in correcting the dominant-negative effect of the E637K-hERG mutant.

GW25-e4298

Frequency of Hyponatremia and Short Term Clinical Outcomes in Patients Hospitalized for Heart Failure

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Objectives: The objective of this study was to determine the frequency of hyponatremia and short term clinical outcomes in patients hospitalized for heart failure.
Methods: This study was carried out from August 9, 2011, to July 29, 2012. Both male and female patients aged 14 years and above admitted for heart failure fulfilling the inclusion criteria, were included in the study. Admission Serum sodium was measured in all patients. Those having serum sodium of ≤135mmol/L were defined as hyponatremic. All the patients were managed according to guidelines. All patients were followed during their hospital stay. Patients who survived were discharged on standard HF medications and followed till the end of six month for 6-month mortality and re-admissions for heart failure.
Results: A total of 241 patients were included in the study. Mean age was 59.2±14.9 (18-100) years. The number of female patients was 123 (51%) while male were 118 (49%). Based on age patients were divided into two groups, Group I included patients less than 60 years (41.9%, n-101)and Group II included 60 years and above (58.1% (n-140). Mean serum sodium was 136±5.1mmol/L (116-151). Hyponatremia (serum sodium ≤135mmol/L) was found in 35.3% (85) patients. The overall in-hospital mortality rate was 5.4%. Lower admission serum sodium was associated with higher in-hospital mortality, 8.2% for the lower sodium group compared with 3.8% for those patients with normal serum sodium ($P=0.23$). Mean length of hospital stay (LOS) for overall CHF patients was 3.8±2.4 days. Lower admission serum sodium was associated with longer mean hospital LOS, 4.1±2.3 for lower sodium group compared with 3.7±2.4 for normonatremic group. Overall 6-month follow up mortality was 19.6%, while it was higher in hyponatremic group 28.0% compared to normonatremic patients 15.3% ($P=0.03$). Six month follow up readmission rate was 30.7%. Hyponatremic group had readmission rate of 34.7% compared with 28.7% in normonatremic patients ($P=0.36$).
Conclusions: Hyponatremia in hospitalized patients with heart failure is common and is associated with longer hospital stay, higher in-hospital and early post-discharge mortality.

GW25-e4585

Effects of CD147/MMP-2 Pathway on Early Left Ventricular Remodeling in Spontaneously Hypertensive Rats

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