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The Incidence of Atorvastatin-induced Abnormal Liver Function in Chinese Patients: A Systematic Review and Meta-analysis
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OBJECTIVES
To study the incidence of atorvastatin-induced abnormal liver function in Chinese patients.

METHODS
Literatures were searched in PubMed, EMBASE, CENTRAL, SinoMed, CMCI, CNKI, VIP and Wanfang database (from their inception to Feb 8, 2015). Randomized controlled trial (RCT), Cohort Study, case series and quasi-experiment studies were included, and generic drugs were excluded. Data extraction and verification were conducted by two reviewers independently. Open Meta-Analyst software with a binary random effect model was applied to compute the incidence of atorvastatin-induced abnormal liver function in different doses and periods, and the results were described by rates and 95% confidence intervals (95% CI).

RESULTS
According to our searching strategy and including criteria, 2386 literatures (2298 Chinese literatures and 88 English ones) published between 2001 and 2015 were included, among which there were 1984 RCT studies, 45 cohort studies, 258 case series studies and 99 quasi-experiment studies. 1140 literatures reported the adverse drug reactions (ADRs) in the process of follow-up, but 1246 literatures did not explicitly mentioned any ADRs. 182430 patients with few cases of abnormal liver function in each groups (2/566, 9/99 quasi-experiment studies. 1140 literatures reported the adverse drug reactions (ADRs) in the process of follow-up, but 1246 literatures did not explicitly mentioned any ADRs. When based on hepatic transaminases (ALT and AST) the ones with no ADRs. When based on ALT, the ratio to achieve therapeutic INR at day 3, day 5, day 7 were 81 patients using oral warfarin were randomly assigned to two groups according to different kinds of dosage regimen. The control group included 42 cases started with 3 mg while research group included 39 cases, started with the initial warfarin dosage by the guide of body mass index (BMI) (BMI<25mg;25-30,6mg). Follow up one month, to compare the time that INR first stabilized at target range(2.0~3.0), the ratio within stabilized target range at different time, the time that INR stabilized at target range, the times to exceed therapeutic INR (INR>3.0), maintenance dose, and the incidence of bleeding or thrombosis episodes.

RESULTS
There was no statistical significance between the two groups in baseline characteristics included age, sex, height, weight, BMI, comorbid conditions, drug combination, Baseline INR, left atrial thrombus and pulmonary embolism. Comparing the safety between two groups: there were two cases bleeding slightly in control group and one case in research group, Both groups had no thrombotic events. There was no statistical significance between the two groups (P>0.05). Compared with control group, the ratio to achieve therapeutic INR at day 3, day 5, day 7 were significantly higher in research group (38.9% vs. 11.9% at day 3, 51.3% vs. 23.8% at day 5, 71.8% vs. 45.2% at day 7). There was statistical significance between the two groups (P<0.05). Compared with control group, the times to exceed therapeutic INR was significantly shorter time to achieve therapeutic INR at the first time (5 days vs. 7 days). There was no statistical significance between the two groups (P>0.05). Compared with control group, research group required a significantly shorter time to achieve therapeutic INR at the first time (5 days vs. 7 days). There was no statistical significance between the two groups (P<0.05).

CONCLUSIONS
Our study shows that phosphocreatine plays a crucial role in the inhibition of myocardial fibrosis induced by AngII through partially suppressing the CF proliferation and collagen synthesis, which is possibly associated with the inhibition of excessively activated ERK2/1.

GW26-e1296
A research on initial dosage of warfarin and target ratio
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OBJECTIVES
To further acknowledge the optimal initial dosage and offer more reasonable evidence for the application of warfarin in clinical work, the research is adopted by comparing the efficacy and safety of two dosage regimens to the patients with atrial fibrillation (AF) or pulmonary embolism (PE) by using different initial dosages of warfarin according to the warfarin 3mg and to the BMI.

METHODS
81 patients using oral warfarin were randomly assigned to two groups according to different kinds of dosage regimen. The control group included 42 cases started with 3 mg while research group included 39 cases, started with the initial warfarin dosage by the guide of body mass index (BMI) (BMI<25mg;25-30,6mg). Follow up one month, to compare the time that INR first stabilized at target range(2.0~3.0), the ratio within stabilized target range at different time, the time that INR stabilized at target range, the times to exceed therapeutic INR (INR>3.0), maintenance dose, and the incidence of bleeding or thrombosis episodes.

RESULTS
There was no statistical significance between the two groups in baseline characteristics included age, sex, height, weight, BMI, comorbid conditions, drug combination, Baseline INR, left atrial thrombus and pulmonary embolism. Comparing the safety between two groups: there were two cases bleeding slightly in control group and one case in research group, Both groups had no thrombotic events. There was no statistical significance between the two groups (P>0.05). Compared with control group, the ratio to achieve therapeutic INR at day 3, day 5, day 7 were significantly higher in research group (38.9% vs. 11.9% at day 3, 51.3% vs. 23.8% at day 5, 71.8% vs. 45.2% at day 7). There was statistical significance between the two groups (P<0.05). Compared with control group, the times to exceed therapeutic INR was significantly shorter time to achieve therapeutic INR at the first time (5 days vs. 7 days). There was no statistical significance between the two groups (P>0.05). Compared with control group, research group required a significantly shorter time to achieve therapeutic INR at the first time (5 days vs. 7 days). There was no statistical significance between the two groups (P>0.05). Compared with control group, the ratio to achieve therapeutic INR at day 3, day 5, day 7 were significantly higher in research group (38.9% vs. 11.9% at day 3, 51.3% vs. 23.8% at day 5, 71.8% vs. 45.2% at day 7). There was statistical significance between the two groups (P<0.05). Compared with control group, research group required a significantly shorter time to achieve therapeutic INR at the first time (5 days vs. 7 days). There was no statistical significance between the two groups (P>0.05). Compared with control group, the times to exceed therapeutic INR (INR>3.0) was significantly shorter time to achieve therapeutic INR at the first time (5 days vs. 7 days). There was no statistical significance between the two groups (P>0.05).

CONCLUSIONS
Our study shows that phosphocreatine plays a crucial role in the inhibition of myocardial fibrosis induced by AngII through partially suppressing the CF proliferation and collagen synthesis, which is possibly associated with the inhibition of excessively activated ERK2/1.

GW26-e0750
Effect of phosphocreatine on angiotensinIIinduced proliferation and collagen synthesis in neonatal rat cardiac fibroblasts
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OBJECTIVES
The aim of this study is to investigate the effect of phosphocreatine (PC) on angiotensinII(ANGII)-induced cardiac fibroblasts in neonatal rats in vitro and to clarify its mechanism of action.

METHODS
The model of myocardial fibrosis induced by AngII was established, the effect of phosphocreatine on cultured cardiac fibroblasts(CF) proliferation in the presence or absence of excessive angiotensinII(ANGII) was assessed by flow cytometry assay, the area of myocardial collagen was observed by VG staining and the expression of the phosphorylated extracellular signal-regulated kinase (pERK1/2) was detected by immunohistochemistry in each group. The cardiac fibroblasts were randomly divided into four groups: control group, the AngII group (1·10-5mol/L) the angiotensinII treated group (1·10-5mol/L) and the AngII-phosphocreatine treated group (1·10-5mol/L +1mmol/L).

RESULTS
The model of myocardial fibrosis was successfully established. Compared to the control group, distribution of CFs in G0/G1 and G2/M phase in AngII group was decreased (P<0.01) with conversely, the increase of the proportion in S phase, the collagen synthesis and the expression of pERK1/2 protein of CFs(P<0.01). However, no significant difference was observed in cell cycle distribution, collagen synthesis of CFs and the expression of pERK1/2 protein between the control and the phosphocreatine treated group(P>0.05). Compared to the AngII group, the percentage of CFs in the G0/G1 phase and G2/M phase was increased with simultaneously, the reduction of S phase and the collagen synthesis of CFs(P<0.01). Noteworthy, phospho-status of ERK1/2 in the AngII-phosphocreatine treated group demonstrated a higher expression than that in the control group as well as lower than that in the AngII group(P<0.01).

CONCLUSIONS
Our study shows that phosphocreatine plays a crucial role in the inhibition of myocardial fibrosis induced by AngII through partially suppressing the CF proliferation and collagen synthesis, which is possibly associated with the inhibition of excessively activated ERK2/1.

GW26-e4473
Cholesterol in Remnant-Lipoprotein as Measured by Different Methods
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