Conclusions It was established the relationship of antiphospholipid syndrome with the process of atherosclerosis.

The presence of atherosclerotic plaques is not associated with traditional risk factors.

Not establish a connexion between antiphospholipid antibodies and IMT.

Proven connexion between aCL and carotid plaques.

Not establish correlation between aPL and Ca score.

Persons with APS have a higher incidence of Calcium score versus healthy controls.

67 SOME ANTI-CALDIOLIPIN-BETA2-GPI ANTIBODIES BRING THROMBOPHILIC DIATHESIS BY THE DUAL REACTIVITY TO DNA AND INTERNALISATION TO LIVE CELLS ACCOMPANYING DNA

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Background and aims In antiphospholipid syndrome (APS), antibodies reactive to CL-beta2-GPI are known to be the important pathogenic factor, but the mechanism of the interaction between the antibodies and cells, and the reason why APS is highly associated with SLE are not fully elucidated.

Methods Since we obtained a monoclonal antibody WB-6 which shows reactivity to CL-beta2-GPI and induces a prothrombotic state in normal mice by tissue factor expression upon circulating monocytes, we tried to clarify how this antibody interacts with live cells.

Results In the current study, we found unexpectedly that WB-6 reacted with DNA by direct-binding ELISA which was confirmed by inhibition ELISA. The result of epitope mapping on the domain 1 of beta2-GPI suggested that WB-6 binds to the arginine- and lysine-rich peptides close to the N-terminal of beta2-GPI, not directly but indirectly via DNA. Incubation of endothelial cell lines or monocytic THP-1 cells with WB-6 revealed that WB-6 enter into the live cells. Because pre-treatment of the cells with DNase 1 significantly reduced the internalisation, and addition of extracellular DNA into the culture significantly increased the internalisation, this phenomenon is likely to be resulted from interaction of WB-6 and cell surface DNA.

Conclusions These results suggest that some anti-DNA antibodies show dual reactivity with CL-beta2-GPI via DNA, and this may contribute to the high percentage of association with SLE in APS. Such an antibody can enter live cells with DNA, and activate intracellular DNA sensors to induce tissue factor expression, without participation of the cell surface bata2-GPI and its still controversial receptors.

68 IDENTIFYING CLINICAL AND EPIDEMIOLOGICAL RISK FACTORS ASSOCIATED WITH THROMBOSIS AND PREGNANCY MORBIDITY IN A LARGE COHORT OF CHINESE APS PATIENTS

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Background and aims The evaluation of thrombotic and pregnancy risks associated with antiphospholipid antibodies (aPLs) in individual patients is challenging. Our objective was to identify potential clinical and epidemiological predictors of thrombosis and pregnancy morbidities in a large Chinese antiphospholipid syndrome (APS) cohort.

Methods This cohort included 177 consecutive APS patients and 146 asymptomatic aPLs control patients who attended the rheumatology clinic at People's Hospital of Beijing University Health Science Centre. All APS patients fulfilled the 2006 revised criteria APS. All control patients had at least one persistent positive aPLs without any other criteria APS manifestations. When assessing risk factors associated with pregnancy morbidities, only reproductive age (age <45) female controls were used. Chi-squared or Fisher's exact test univariate analysis and multivariable logistic regression analyses were used to assess association between different clinical and epidemiological risk factors and clinical manifestations.

Results Of the 177 APS patients, 134 (75.7%) were women with a mean age of 43. 5 (S.D. 16). When comparing to controls, risk factors associated with thromboembolic events included: Raynaud's phenomenon (odds ratio (OR)=2.371, 95% Confidence interval (CI) 1.039–5.637, p=0.0462), hypertension (OR=1.829, 95% CI 1.114–3.05, p=0.022), and smoking (OR=3.941, 95% CI 1.816–8.799, p=0.0004). Age, hyperlipidemia, diabetes, hypocomplementemia, and thrombocytopenia did not demonstrate significant association with thrombosis. None of the analysed clinical characteristics showed significant association with pregnancy morbidities. A high frequency of thrombocytopenia and hypocomplementemia were observed in both APS patients and control patients with persistent +aPLs.

Conclusions Smoking, Raynaud's phenomenon, and hypertension are potential predictors of thromboembolic events in +aPLs Chinese patients.

69 LONG-TERM PROGNOSIS AND PREDICTING FACTORS OF CHINESE PATIENTS WITH ANTIPHOSPHOLIPID SYNDROME

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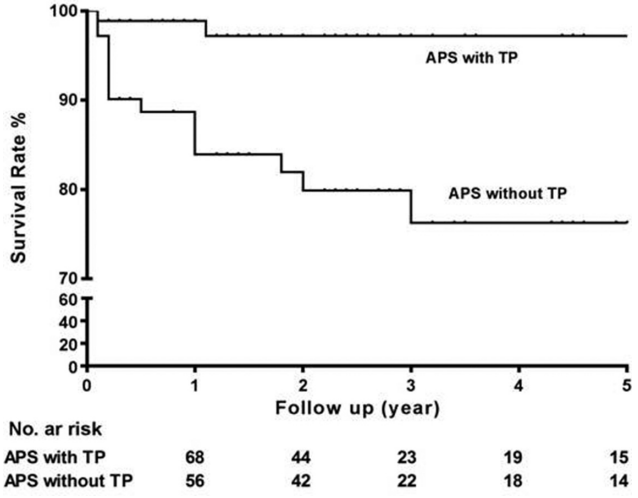
Background and aims The aims of the present study were to assess and identify the prognostic factors of the long-term outcomes and mortality of antiphospholipid syndrome (APS) in Chinese patients.

Methods Records of 160 patients with APS admitted to Peking Union Medical College Hospital in Beijing between 2005 and 2015 were investigated. Demographic characteristics, cumulative clinical and laboratory features, autoantibody profiles were retrieved from the database. Survival rates were studied by Kaplan-Meier method, and COX proportional hazard model was adopted to perform the analysis of predicting factors for mortality.

Results The entire cohort consisted of 110 (68.8%) female and 50 (31.3%) male patients. Mean (SD) age was 36.5±14.9 years. In total, 50.6% of the patients had primary APS, 45.9% had APS associated with SLE. The most prevalent immunological features at baseline were LA (71.3%), aCL (55.0%), and β2GPI(49.4%). No significant statistical

Abstract 69 Table 1 Baseline characteristics of APS patients

Clinical characteristics	Prevalence	Thrombotic event	
		No (N=51)	Yes (N=109)
Female, n/%	110(68.8%)	42(82.4%)	68(62.4%)
Age, year, mean+SD	36.5±14.9	34.2±14.9	37.4±14.9
Thrombotic events			
Arterial thrombosis	59(36.9%)	-	59(54.1%)
Venous thrombosis	72(45.0%)	-	72(66.1%)
Coexist of arterial and venous thrombosis	22(13.8)	-	22(20.2%)
Systemic autoimmune diseases	79(49.4%)	33(64.7%)	46(42.2%)
Thrombophylic risk factors			
Smoking	8(5.0%)	2(3.9%)	5(5.5%)
Dyslipidemia	20(12.5%)	6(11.8%)	14(12.8%)
HTN (systolic>140)	24(15.0%)	7(13.7%)	17(15.6%)
ACL	88(55.0%)	32(62.7%)	56(51.4%)
β2GP1	79(49.4%)	31(60.8%)	48(44.0%)
Lupus anticoagulants	114(71.3%)	39(76.5%)	75(68.8%)
Tri-positive	41(25.6%)	21(41.2%)	20(18.3%)
Thrombocytopenia	71(44.4%)	23(45.1%)	48(44.0%)
Hypocomplementaemia	59(36.9%)	25(49.0%)	34(31.2%)



Abstract 69 Figure 1

differences were found in the clinical presentation of the APS according to the presence or absence of any of these antibodies. During the 10 year period, 16 (10.0%) patients (8 female and 8 male) died. The overall 1, 3, and 5 year survival rate was 92.6%, 89.1% and 87.1%, respectively. The most common causes of death were severe thrombotic events, including pulmonary embolism, strokes and myocardial infarction (43.8% of total deaths), infections (18.8%). COX proportional hazard model show thrombocytopenia is the independent prognostic factor of mortality (HR 8.228, 95% CI 1.866–36.282).

Conclusions Patients with APS develop significant morbidity and mortality despite current treatment. More attention should be devoted to APS patients with thrombocytopenia.

New therapies and therapeutic targets – other autoimmune diseases

70 5-AMINOLEVULINIC ACID COMBINED WITH FERROUS IRON AMELIORATE GRAFT-VERSUS-HOST-INDUCED SYSTEMIC SCLEROSIS IN THE MOUSE

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Background and aims Scleroderma or systemic sclerosis (SSc) is a clinically heterogeneous rheumatologic autoimmune disease characterised by skin, internal organs and blood vessels, and there is no effective therapy. The purposes of current study are to develop a model of GvHD-induced scleroderma that more fully represents human condition, and to investigate the effects of 5-aminolevulinic acid (5-ALA), an intermediate of heme synthesis, enhance HO-1 activity to cleave heme to form biliverdin, CO, and iron on this model.
Methods Scl-GvHD was induced by injection of lymphocytes from B10.D2 mice into BALB/c mice deficient in mature T and B cells (recombination activating gene 2 null mice).
Results We successfully established an scl-GvHD model, which is similar to the human disease particularly in the skin, progressive inflammation and fibrosis of internal organs including lung, kidney, and liver. We found that treatment with 5-ALA and iron (Fe^{2+}) significantly reduced progressive inflammation and fibrosis in the skin and ear. Furthermore, by quantitative real-time PCR analysis, 5-ALA and Fe^{2+} suppressed the inflammatory cytokines and TGF-β, type I collagen mRNAs expression. These results indicate that combination treatment with 5-ALA and Fe^{2+} exhibited a protective effect on tissue fibrosis and inflammation of scl-GvHD model mice.
Conclusions The model of GvHD-induced SSc has shown most of symptoms of human disease and is likely to contribute to better understanding of the disease mechanism. Furthermore, efficacy of the 5-ALA has important implication for clarifying the mechanism of HO-1 activity in autoimmune diseases, and may provide a favourable opportunity for clinical therapy.

71 ACTIVATION OF MGLUR7 ATTENUATES THE DEVELOPMENT OF ALLERGY-INDUCED ANAPHYLAXIS

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