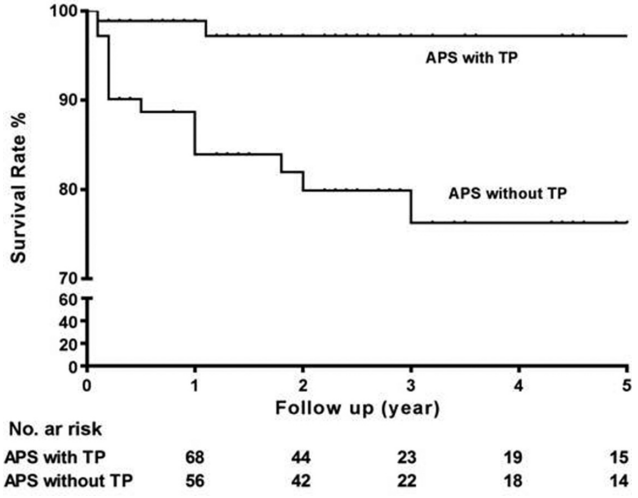
Abstract 69 Table 1 Baseline characteristics of APS patients

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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