In the univariate analysis, surgery was significantly associated with a lower probability of reaching CKD ($p=0.042$; OR 0.2; 95% CI 0.03-0.94). In the multivariate analysis, the tendency to benefit from surgery was maintained but the statistical significance was lost, probably due to the low number of patients.

In the subgroup analysis, the tendency to benefit from surgery was maintained in patients with or without anticoagulation, immunosuppressants, statins, with primary or secondary APS, with uni- or bilateral stenosis, with or without dyslipidemia, but this benefit was lost in smokers independent of the grade of stenosis.

Conclusion: RAS is a treatable cause of hypertension and a poor prognostic marker in APS patients.

In this study, APS patients with RAS who underwent intervention with angioplasty or stenting had a trend to a lower probability of developing CKD in contrast to studies in atherosclerotic RAS. The beneficial effect of surgery was lost in smoking patients. In this relatively young population mortality was high.

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SAT0240 THE PROGNOSIS OF TWO DISTINCT CLINICAL PHENOTYPES OF SLE-PAH

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Background: Based on the characteristics of systemic lupus erythematosus-associated pulmonary arterial hypertension (SLE-PAH), Sun *et al* has put forward a scoring system to distinguish two clinical phenotypes as vasculitic and vasculopathic subtypes[1]. A weighted score ≥ 2 suggested a vasculitic subtype by combining two factors: The time interval between SLE and PAH diagnosis <2 years and ≥ 2 years were 1 and 0 point; SLE Disease Activity Index (SLEDAI) >9 , 5-9 and ≤ 5 were 2, 1, 0 point, respectively. While the vasculitic subtype seemed to have poorer prognosis in Sun's research, other study has shown controversial result[2].

Objectives: To find out the prognosis of two distinct clinical phenotypes of SLE-PAH.

Methods: Between 2008 and 2019, a SLE-PAH cohort confirmed by right heart catheterization (RHC) from Guangdong Provincial People's Hospital was included. Other groups of pulmonary hypertension were excluded. Based on the scoring system, patients were divided into vasculitic (weighted score ≥ 2)

and vasculopathic subtypes (weighted score <2). The endpoint was PAH-related mortality. Survival status were confirmed by clinic follow-up data or phone call.

Results: A total of 53 SLE-PAH patients were enrolled. The cases of vasculitic and vasculopathic subtype were 14 and 39, respectively. Ten endpoint events occurred. Eight attributed to PAH and the cause could not be traced in two which were still included in study. The pooled 1-, 3-, 5-year survival rates were 85.7%, 78.6%, 65.5% in vasculitic subtype, and 93.9%, 87.5%, 87.5% in vasculopathic subtype, respectively. Kaplan-Meier analysis showed vasculitic subtype tended to have a poorer prognosis than vasculopathic subtype ($p=0.16$, HR 2.4, 95%CI 0.5-13.8, figure 1).

Conclusion: The prognosis of the two phenotypes of SLE-PAH was statistically indifferent while the vasculitic subtype showed a trend of worse prognosis. Further studies are needed.

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SAT0241 PLATELET INDICES COULD BE SIMPLE RELIABLE PREDICTORS OF THROMBOTIC EVENTS IN PATIENTS WITH ANTIPHOSPHOLIPID SYNDROME

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Background: Platelet activation is considered as a pivot pathogenic process to be responsible for thromboembolism in antiphospholipid syndrome (APS). several studies shown that platelet indices including platelet distribution width (PDW), mean platelet volume (MPV), large platelet rate (P-LCR) are associated with platelet activation.

Objectives: This study aims to determine the correlation between platelet indices and thrombotic events in patients with APS.

Methods: Platelet activation is considered as a pivot pathogenic process to be responsible for thromboembolism in antiphospholipid syndrome (APS). several studies shown that platelet indices including platelet distribution width (PDW), mean platelet volume (MPV), large platelet rate (P-LCR) are associated with platelet activation.

Results: A total of 207 patients [135(65.2%) female, 72(34.8%) male], median age 35(IQR 10)] was classified into thrombotic group ($n=150, 72.5\%$) and non-thrombotic group ($n=57, 27.5\%$). PDW, MPV, P-LCR were significantly higher in thrombotic group than non-thrombotic group (13.0 ± 3.4 vs. 11.2 ± 7.3) $p < 0.001$, 10.7 ± 1.4 vs. 10.0 ± 3.0 , $p < 0.001$, 30.25 ± 11.6 vs. 25.1 ± 10.4 $p < 0.001$, respectively]. No differences in age, WBC count, hsCRP and C3 were observed between groups. Also, HGB was found to be notably higher in thrombotic group than non-thrombotic group (143 ± 29 vs. 132 ± 15 , $p = 0.001$). Upon univariate logistic analysis, PDW (OR 1.554, 95%CI 1.289-1.873, $p < 0.001$), MPV (OR 1.772, 95%CI 1.268-2.476, $p = 0.001$), P-LCR (OR 1.089, 95%CI 1.040-1.140, $p < 0.001$) were all significantly associated with the occurrence of thrombosis. In multivariate logistic analysis, only PDW and positive LA were identified to be risk factors of thrombotic events (Table 1). The ROC curve showed that PDW combined with positive LA was a reliable indicator of thrombotic events with an AUC of 0.796 (95%CI 0.728-0.864). The optimal cut-off value for PDW was 12.4fl with a sensitivity of 72.0% and specificity of 77.2%.

Conclusion: This study confirmed that PDW, P-LCR and MPV (especially PDW) were significantly associated with thrombotic events in APS patients, which could support the theory of platelet activation being a crucial factor of thrombosis in APS. Caution should be raised when patients with positive LA has relatively high PDW level.

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SAT0242 REGIONAL VARIATION IN CARDIOVASCULAR DISEASE AMONG SLE PATIENTS

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Objectives: To evaluate whether the risk of cardiovascular disease (CVD) including myocardial infarction (MI) and cerebrovascular (CVA) differs across geographic regions among SLE patients.

Figure 1. Survival curves for patients with systemic lupus erythematosus-pulmonary arterial hypertension (SLE-PAH) in two distinct subtypes. RHC, Right Heart Catheterization.

Methods: We identified SLE patients using two ICD codes 60 days apart within two years recorded in Medical Services Plan (MSP) or hospital discharge database (DAD). We defined the second of two diagnosis dates as the index date. We included incident SLE patients (7-year continuous registries in MSP before the first diagnosis date) with an index date between 1997 and 2012 and excluded patients with previous MI or CVA before the index date. We followed each patient from the index date up to 10 years and censored at the date of death date, leaving the province, or March 31, 2015.

We assessed the incident CVD that was defined as the first ever diagnosis of MI or CVA recorded in DAD or as the primary cause of death in Vital Statistics. We also evaluated MI and CVA separately.

The Province's publicly administered and funded health care system is organized into five regional health authorities (HA): Interior (IHA), Fraser (FHA), Vancouver Coastal (VCHA), Vancouver Island (VIHA), and Northern (NHA) [Figure 1(a)]. We assigned each patient the HA she/he was registered at the index date. We extracted baseline covariates using the information during a period of 365 days prior to the index date, including socio-demographic characteristics, health care resource use, comorbidities, and prescription medication use. We calculated the incident rate (IR) of MI, CVA, and CVD (first ever MI or CVA) by HA. Using Cox Proportional Hazard model adjusting for potential confounders at baseline, we estimated the adjusted hazard ratios (aHR) of CVD for each HA compared to FHA or VCHA which have the large proportion of provincial population and SLE patients. We evaluated the regional disparities in MI and CVA separately using the same methods.

Results: We included 3,960 incident SLE patients free of CVD at baseline with a mean (SD) age of 48.5 (15.8), including 726 (18.3%) from IHA, 1634 (42.3%) from FHA, 854 (21.6%) from VCHA, 504 (12.7%) from VIHA, and 242 (6.1%) from

NHA. During 26378 person-year (PY) follow-up, 133 patients developed incident CVD including 91 MI and 43 CVA. [Table 1]

The IR of CVD varied from 35 in FHA to 76 per 10,000 PY in IHA [Figure 1(b)]. IHA had significantly higher risk of CVD than FHA (aHR=1.93, 95%CI=1.17-3.2) and VCHA (aHR=2.05, 95%CI=1.17-3.58).

The IR of MI varied from 24 in FHA to 52 per 10,000 PY in IHA [Figure 2(a)]. IHA had significantly higher risk of MI than FHA (aHR=2.09, 95%CI=1.14-3.83).

The IR of CVA varied from 11 in FHA to 32 per 10,000 PY in VIHA [Figure 2(b)]. VIHA had significantly higher risk of CVA than FHA (aHR=2.79, 95%CI=1.14-6.85) and VCHA (aHR=3.66, 95%CI=1.34-10.01).

Conclusion: Compared with FHA and VCHA, IHA had higher risk of CVD and VIHA had higher risk of CVA. IHA also had higher risk of MI than FHA.

Disclosure of Interests: None declared

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Vasculitis

SAT0243

SUBPHENOTYPES IN POLYARTERITIS NODOSA (PAN): TARGET ORGAN ASSOCIATIONS OF A WORLDWIDE COLLABORATION STUDY

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Background: There is a paucity of information on the current phenotypes, ethnic and geographic differences of PAN. A global PAN study group has been working for clinical subphenotype and GWAS studies.

Objectives: This study is aimed to look for target organ associations in PAN.

Methods: PAN patients fulfilling the EMA vasculitis classification algorithm were recruited. In addition to baseline characteristics, treatment and outcome data, occurrence of any of the clinical manifestations related to PAN during disease course was recorded.

Factor analysis was used to analyse target organ associations of 306 patients. Five factors were identified by factor analysis of variables sex, paediatric-onset, HBV, monogenic disease relationship, cutaneous features, musculoskeletal symptoms, constitutional symptoms and involved areas (abdominal, renal, neurologic, ENT, cardiac, pulmonary).

Results: PAN cohort from 7 countries were used (Italy: n=59, Japan: n=39, Mexico: n=29, Slovenia: n=14, Sweden:11, TUR: n=106, UK: n=48). 306 (M/F: 171/135 and Caucasian 77.1%, Asian 13.4%, and Hispanic 9.5%) patients were included. 8 were HBV-related, and 22 of TUR patients had a monogenic form of disease (FMF n=15, DADA2 n=7). 21.8% of patients were cutaneous-only PAN patients. 48.4% of patients had radiologic, 64% had biopsy-proven PAN. Median age at disease onset was 40 (IQR 27.0-57.5) years. During a median 57 (16-120) months follow-up, 39 (13%) patients died.

Factor analysis revealed 5 factors that explained 54.1% of the original information on the matrix as follows: **Factor 1**, represented the association between gastrointestinal and renal involvement, male gender and negatively associated with cutaneous features; **Factor 2**, the association between monogenic relationship with paediatric onset disease; **Factor 3**, any of musculoskeletal findings with positive constitutional symptoms; **Factor 4** any neurologic involvement was associated with ENT and pulmonary findings; **Factor 5** cardiac involvement in non-HBV patients (Table).

The eigenvalues of the 5 factors were 2.034, 1.470, 1.427, 1.079 and 1.030, in decreasing order, i.e., the highest contribution to the overall variance in the matrix came from the togetherness of the 4 clinical and demographic characteristics that made up Factor 1.

Table 1. Incident Rates and HR

	HA	Incidence		aHR (95%CI)		
		N	PY	IR (95%CI) 10,000 PY	Reference: FHA	Reference: VCHA
CVD	IHA	35	4579	76(54,105)	1.93(1.17,3.2)	2.05(1.17,3.58)
	FHA	39	11197	35(25,47)	ref	1.06(0.64,1.76)
	VCHA	27	5844	46(31,66)	0.94(0.57,1.57)	ref
	VIHA	22	3165	70(44,103)	1.65(0.96,2.83)	1.74(0.98,3.11)
	NHA	10	1592	63(31,110)	1.81(0.86,3.8)	1.92(0.88,4.17)
MI	IHA	24	4579	52(34,76)	2.09(1.14,3.83)	1.81(0.95,3.47)
	FHA	27	11197	24(16,34)	ref	0.87(0.48,1.56)
	VCHA	21	5844	36(23,54)	1.15(0.64,2.07)	ref
	VIHA	12	3165	38(20,64)	1.29(0.64,2.58)	1.12(0.54,2.31)
	NHA	7	1592	44(19,85)	2.11(0.88,5.11)	1.84(0.74,4.56)
CVA	IHA	11	4579	24(12,41)	1.79(0.71,4.55)	2.35(0.8,6.88)
	FHA	12	11197	11(6,18)	ref	1.31(0.49,3.48)
	VCHA	7	5844	12(5,23)	0.76(0.29,2.03)	ref
	VIHA	10	3165	32(16,55)	2.79(1.14,6.85)	3.66(1.34,10.01)
	NHA	<5	1592	19(5,49)	1.45(0.36,5.72)	1.89(0.44,8.22)

Figure 1. HA and CVD Incident Rate

Figure 2. MI and CVA incident Rate by HA