SLE clusters had similar levels of nucleic acid-specific AABs and Ro52 AABs, but were distinguished by significant differences in histone 2A (H2A) AAB levels. Renal manifestations were significantly enriched in the NA Ro52 cluster and the AA nucleolin/histone cluster compared to other SLE patient clusters of the same ethnicity.

Abstract THU0263 – Figure 1

Conclusion: Expanded AAb profiles can be used to identify SLE subsets that are more likely to have renal manifestations of disease.

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THU0264

ANALYSIS OF SURVIVAL ASSOCIATED WITH PULMONARY ARTERIAL HYPERTENSION IN PATIENTS WITH SYSTEMIC LUPUS ERYTHEMATOSUS

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Background: Identifying clinical variables that predict outcome of Systemic lupus erythematosus (SLE)-pulmonary arterial hypertension (PAH) is important.[1] Data focusing on SLE-PAH is lacking as previous studies were based on connective tissue disease (CTD)-PAH patients.

Objectives: To examine clinical, serological and echocardiographic features that are 1) associated with SLE-PAH at baseline and 2) predictors of mortality in SLE-PAH.

Methods: Data on SLE-PAH patients (SLE-PAH group, n=67) attending the Prince of Wales Hospital, from 2008 to 2018 was retrieved. Diagnosis of PAH was based on echocardiogram showing pulmonary artery systolic pressure (PASP) > 30mmHg. Age-, sex- and disease duration-matched SLE patients with PASP < 30mm Hg were selected from the same database as control (SLE-non PAH group, n=154). Clinical, serological, echocardiographic features and medication at baseline (PAH diagnosis) were collected. Censor date was death or last visit on or before 31st December 2018. PAH targeted therapy and immunosuppressant used for PAH were recorded after diagnosis of PAH. Hazard ratio were analysed by cox regression model, and adjusted by age-, sex-, disease-duration, comorbidities and baseline medication.

Results: The cohort (n=221) mean age was 42.7 +/- 12.6 with 96.3% female, and a disease duration of 8.26 +/- 7.61 years. At baseline, the SLE-PAH group had higher anti-ds DNA titre; lower haemoglobin level; higher prevalence of renal impairment; but a lower prevalence of arthritis and thrombocytopenia/neutropenia and anti-phospholipid antibodies compared to the control (Table 1). SLE-PAH group's echocardiogram had a higher prevalence of reduced left ventricular ejection fractions, moderate-

severe tricuspid regurgitation and pericardial effusion compared to the control (Table 1). Amongst the SLE-PAH group, 11/67 had a right heart catheterization done and all were confirmed to have PAH. 8/11 patients received PAH specific treatment and 23/67 received escalated immunosuppressive therapy for presumed PAH, within 1 years of PAH diagnosis. The mortality rate of the SLE-PAH group was 3.82 (95% confident interval 1.50-9.83; p=0.005) compared to the control after adjusting for baseline parameters and medication used after the diagnosis of PAH. Poor prognostic factors at baseline are summarized in table 2.

Table 1. Comparison between SLE-PAH and control at baseline

	SLE non-PAH (N=154)	SLE-PAH (N=67)	p-value
Female	149 (96.8%)	64 (95.5%)	0.701
Age, Mean (SD)	41.84 (11.76)	44.78 (14.18)	0.111
Follow up time (year) [Median (IQR)]	9.78 (4.39-12.42)	8.24 (4.54-12.07)	0.835
Disease duration (year), Median (IQR)	6.85 (1.29-11.77)	7.33 (0.94-15.06)	0.681
Death at end of follow up (%)	8/154 (0.05%)	14/67 (20.1%)	0.001
Renal impairment	25/154 [16.3%]	19/67 [29.7%]	0.026
Arthritis	91/154 [59.9%]	29/67 [43.3%]	0.023
Thrombocytopenia/neutropenia	78/154 [51.3%]	24/67 [35.8%]	0.047
Anti-ds DNA titre (median [IQR])	52 IU/ml [IQR 0-292.5]	160 IU/ml [IQR 0-789.5]	0.036
Haemoglobin (median [IQR])	11.7 g/dL [IQR 10.5-12.9]	10.8 g/dL [IQR 10-10.2]	0.009
Anti-phospholipid antibodies	38/154 [25.5%]	8/67 [12.1%]	0.027
Reduced left ventricular ejection fractions	1/154 [0.6%]	6/67 [9%]	0.003
Moderate-severe tricuspid regurgitation	2/154 [1.3%]	20/67 [29.9%]	<0.001
Pericardial effusion	22/154 [14.3%]	17/67 [25.4%]	0.047

Table 2. Predictors of mortality in SLE-PAH at baseline

	Alive (N=53)	Death (N=14)	p-value
Hyponatremia	7/49 [14%]	4/12 [30.8%]	p=0.011*
Renal impairment	11/51 [21.6%]	8/13 [61.5%]	p=0.007*
Severe tricuspid regurgitation	2/53 [3.8%]	2/14 [14.3%]	p=0.071

Conclusion: SLE-PAH had 3.8-fold increase in mortality despite treatment with PAH specific therapy and immunosuppressants. Hyponatremia and renal disease were associated with poor survival outcome in SLE-PAH patients.

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THU0265

IDENTIFYING COMORBID FIBROMYALGIA IN SYSTEMIC LUPUS ERYTHEMATOSUS USING PATIENT-REPORTED OUTCOMES

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Background: Fibromyalgia (FM) is disproportionately common in patients with systemic lupus erythematosus (SLE) and difficult to diagnose on a background of classical SLE symptoms [1]. The Multi-Dimensional Health Assessment Questionnaire (MDHAQ) has been shown to be useful to recognise improvement over 2 months in a variety of rheumatic conditions including SLE [2] but has not previously been shown to be useful to alert the clinician to comorbid FM in the latter condition. Conversely, the 2011 FM self-report questionnaire is disease-specific and available for use in clinical and epidemiological studies. Administration of multiple forms may be difficult in a busy clinical setting.

Objectives: To identify comorbid FM in patients with SLE using patient-reported outcomes (PROs) from the routinely distributed MDHAQ, in comparison to the 2016 revision of the 2010/2011 FM criteria.

Methods: Patients with SLE completed an MDHAQ and the 2011 FM Criteria questionnaire. FM status was assigned using the 2016 revision of the 2010/2011 FM criteria as the gold standard. The MDHAQ features