and found to have excellent correlation with disease activity index and rSLEDAI scores.

P35 MEASURING DSDNA ANTIBODIES IN THE ELDERLY: POSITIVE RESULTS RARELY INDICATE LUPUS

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Background Antibodies to dsDNA are reported to be specific for lupus and are included in the main classification criteria, although pitfalls when used as a 'screening' test in patients with multiple medical symptoms have been described. Lupus is typically diagnosed in the first 50 years of life and clinically active disease is unusual in the elderly. We evaluated dsDNA requests made in elderly patients (>80 years) in our centre.

Methods Review of 23,287 consecutive dsDNA antibody requests made through the University of Birmingham Immunology Lab between 2009 and 2019. Review of available clinical records for patients with positive results.

Results 993 dsDNA antibody requests were made for patients >80 years old (mean 84.2 years), representing 0.04% of total requests. 578 were female, 411 were male and 5 unknown. 138 (13.9%) requests were made from primary care, 80 (8.0%) from the critical care/emergency department, 623 (62.5%) from medical wards and outpatients and 97 (4.4%) from rheumatology. The remainder were from neighbouring hospitals. 67 patients (6.7%) tested positive for dsDNA by ELISA (lab normal range) and 31 (3.1%) by Crithidia.

Clinical records were available for 58 patients with positive dsDNA antibodies. Three were known to have longstanding SLE. No patients were diagnosed as having SLE at or after the time of dsDNA antibody testing. Sixteen patients had other autoimmune diseases including rheumatoid arthritis, Sjogrens Syndrome, myasthenia gravis, microscopic polyangiitis, polymyalgia rheumatica, bullous pemphigoid and immune thrombocytopenia. Four patients had underlying malignancy (one each of lung, prostate, bladder and colon). The remainder had a mix of infections and general medical presentations.

One hundred randomly selected patients with negative dsDNA antibody were reviewed and no cases of lupus were identified, leading to a calculated specificity of dsDNA antibodies for lupus of only 64.5%.

Conclusions Antibodies to dsDNA are not uncommon in unwell elderly patients presenting to hospital or primary care. They are rarely indicative of SLE. The reported high specificity of dsDNA antibodies for lupus are not applicable in this age group.

P36 USING MACHINE LEARNING TO IDENTIFY AND STRATIFY PATIENTS WITH JUVENILE-ONSET SLE

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Background Juvenile-onset Systemic Lupus Erythematosus (JSLE) is a complex disease characterised by diagnosis and treatment delays. We applied a machine learning (ML) approach to explore new diagnostic signatures for JSLE based on immune-phenotyping data.

Methods Immune-phenotyping of 28 T-cell, B-cell and myeloid-cell subsets in 67 age and sex-matched JSLE patients and 39 healthy controls (HCs) was performed by flow cytometry. A balanced random forest ML predictive model was developed (10,000 decision trees). 75% of sample data was randomly selected as a training set, the remaining 25% out-of-bag data was used for validation. Receiver operator characteristic, 10-fold cross validation, Sparse Partial Least Squares-Discriminant Analysis (sPLS-DA) and linear regression was used to validate the model.

Results In JSLE, a global change in immunological architecture was established compared to HCs: many of the immune cell relationships identified in HCs using correlation comparison analysis were inverted or exacerbated in JSLE, including significantly inverted correlations between intermediate monocytes and memory B-cell populations and CD4/CD8 memory T-cells and B-cell memory in SLE versus HCs. Using immune-phenotyping data a ML model was developed and validated (accuracy=87.80%) showing that JSLE patients could be distinguished from HCs with high confidence using immunological parameters. The top variables contributing to the model included CD19⁺ unswitched memory B-cells, naïve B-cells, CD14⁺ monocytes and memory T-cell subsets. The 'JSLE immune signature' was also successfully validated using sPLS-DA and linear regression. To assess whether the validated signature could be used to further stratify JSLE patients, K-mean clustering was applied. Four JSLE groups each with a distinct immune and clinical profile were identified. Finally, network analysis identified specific clinical features associated with each of the top JSLE immune-signature variables.

Conclusion Using a combined ML approach, a distinct immune signature was identified that discriminated between JSLE patients and HCs and further stratified patients. This signature could have diagnostic and therapeutic implications.

P37 NON-INVASIVE BIOMARKERS OF DISEASE ACTIVITY IN SYSTEMIC LUPUS ERYTHEMATOSUS

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Purpose The possibility of exploring other body fluids to identify potential biomarkers of systemic inflammation has emerged as a non-invasive alternative to blood samples for both diagnosis and control of disease activity. We investigated the levels of innate-immunity related biomarkers in saliva, serum, and urine from Systemic Lupus Erythematosus (SLE) patients and their correlation to each other and to disease activity.

Methods We included 84 SLE patients and 20 controls from the general population, all participants underwent a thorough clinical examination. Disease activity as measured with the Systemic Lupus Activity Measure (SLAM) and SLE Disease

Activity Index-2K(SLEDAI-2K) was recorded. Matched saliva, serum, and urine were collected, and the levels of colony-stimulating factor (CSF)-1, tumor necrosis factor (TNF)- α , interferon- γ -induced protein (IP)-10, monocyte chemoattractant protein (MCP)-1 and calprotectin were analysed by enzyme-linked immunosorbent assays (ELISA) and Bead-based Multiplex immunoassay.

Results TNF- α , IP-10 and MCP-1 in saliva, serum and urine, and CSF-1 in saliva and serum provided diagnostic ability to distinguish SLE from controls (area under the curve >0.659 , $p<0.05$ for all). CSF-1, TNF- α , IP-10 and MCP-1 in saliva, serum and urine as well as calprotectin in saliva and urine were increased in SLE patients. Levels of CSF-1 in saliva, serum and urine, IP-10 in serum and urine, and calprotectin in saliva and urine, as well as TNF- α , IP-10 and MCP-1 in urine correlated positively with measures of disease activity. TNF- α , IP-10 and MCP-1 in urine and CSF-1 and IP-10 in serum were elevated in patients with active renal disease ($p<0.05$ all comparisons).

Conclusion The proposed biomarkers provide diagnostic ability to distinguish SLE from controls and they also reflect disease activity. This is a promising result suggesting that in some situations either saliva or urine samples might be capable to be an alternative to serum when diagnosing and monitoring patients with SLE.

P38

DESCRIPTIVE ANALYSIS OF PATIENTS POSITIVE FOR ANTI-PCNA ANTIBODIES

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Background Proliferating cell nuclear antigen (PCNA) antibodies are a rare type of antinuclear antibodies (ANA), historically considered highly specific for systemic lupus erythematosus (SLE). Recent studies showed they can be present in other autoimmune diseases, viral infections or tumors. Our aim was to clinically describe patients positive for anti-PCNA antibodies.

Methods We retrospectively identified patients with a PCNA pattern by indirect immunofluorescence (IIF), subsequently confirmed by immunoblot, selected among patients tested for ANA, between January 2006 and August 2019. Clinical and analytical data were obtained from medical records.

Results 11014 samples (excluding follow-up tests) were tested for ANA, 2642 (23.99%) were positive (titer $>1/160$), 26 (0.98%) had a PCNA pattern by IIF but only 6 (0.23%) were anti-PCNA positive. Mean patients' age was 51.5 ± 17.93 years-old and the majority were female (83%). Half had SLE (including one Overlap Syndrome). Other autoimmune diseases included Antiphospholipid syndrome [$n=2$, 33%], Systemic Sclerosis [$n=1$, 17%] and Behçet Disease [$n=1$, 17%]. One patient developed metastasis from an occult neoplasia and none had viral disease. Almost all patients had cutaneous manifestations [$n=5$, 83%]. Half had articular [$n=3$, 50%] and neurological manifestations [$n=3$, 50%]. Median ESR and RCP were slightly elevated. None

had complement consumption. Mean GFR was normal, with one patient showing low level of proteinuria. Anti-dsDNA, anti-SSA, anti-M2 and anti-cardiolipin were also found in the sera. Two patients were under hydroxychloroquine [$n=2$, 33%] and one under prednisolone 10 mg [$n=1$, 17%] (table 1).

Abstract P38 Table 1 Descriptive analysis of the clinical and analytical data of the 6 patients positive for anti-PCNA

Disease duration (years), median [min, max]	1 [1,17]
<i>Autoimmune disorders:</i>	
Systemic lupus erythematosus (SLE), n (%)	2 (33)
Secondary Antiphospholipid syndrome, n (%)	2 (33)
Systemic Sclerosis (SyS), n (%)	1 (17)
Overlap syndrome (SLE + SyS), n (%)	1 (17)
Behçet disease, n (%)	1 (17)
No evidence of autoimmune disorder, n (%)	1 (17)
<i>Other diseases:</i>	
Malignancies, n (%)	1 (17)
Viral infections, n (%)	0 (0)
<i>Clinical manifestations:</i>	
Skin, n (%)	5 (83)
Joints, n (%)	3 (50)
Neurological, n (%)	3 (50)
Constitutional, n (%)	2 (33)
Renal, n (%)	2 (33)
Muscle, n (%)	1 (17)
Sicca, n (%)	1 (17)
<i>Analytical data (at time of PCNA positive test)</i>	
RCP (mg/dL), median [min, max]	0.86 [0.04, 22.89]
ESR (mm/h), median [min, max]	38.5 [6, 113]
Number of patients with complement consumption (%)	0 (0)
GFR (MDRD Equation, mL/min/1.73 m ²), mean $\pm$ SD	92.9 $\pm$ 44.8
Number of patients with proteinuria, n (%)	1 (17)
<i>Serological data</i>	
Positive anti-dsDNA antibodies, n (%)	1 (17)
Positive anti-SSA antibodies, n (%)	1 (17)
Positive anti-Mitochondrial M2 antibodies, n (%)	1 (17)
Positive anti-Cardiolipin antibodies, n (%)	1 (17)

Conclusions Our study confirmed that anti-PCNA antibodies can be present in other autoimmune diseases and are not SLE specific. Given its rare prevalence, evidence about the clinical relevance of anti-PCNA antibodies is lacking. Larger studies are needed to further evaluate its role.

Skin: Malar rash and/or photosensitivity and/or oral/genital ulcers and/or Raynaud phenomenon and/or telangiectasias and/or microstomy and/or digital ulcers and/or skin sclerosis;

Joints: Arthralgias and/or arthritis;

Neurological: Focal neurological symptoms and/or dementia and/or cerebrovascular disease;

Constitutional: Fatigue and/or weight loss; **Renal:** serum creatinine elevation and/or proteinuria and/or hematuria; **Muscle:** Myalgias and/or myositis;

Sicca: Dry mouth/eyes;

RCP: Reactive C protein;

ESR: Erythrocyte sedimentation rate;

GFR: Glomerular filtration rate.