Objectives: To assess the accuracy of US and MRI and to define possible cutoff values for the diagnosis of pSS.
Methods: Twenty-three patients with pSS according to AECG criteria and typical histology of the SG biopsy and 12 patients with sicca-syndrome and normal SG histology were included in the study. Two experienced ultrasound experts (C.D. 6y, T.D.Z. 5y) did ultrasound of the SG using a B-mode score (0–48 points [1]) and real-time sonoelastography (RTS; 0–16 pts [2]). Morphology of the parotid glands was also assessed by MRI (0–12 pts). We obtained clinical data (C-reactive protein (CRP), antinucleary-antibodies (ANA), Ro-/La-antibodies, Gamma globulins, patient questionaires ESSDAI and SSDI). The statistically analysis was carried out using Kolmogorov-Smirnov-test, student's t-test, or Man-Whitney-U-test. Correlations were performed using Spearman-rang-correlations.
Results: Patients with pSS had significantly higher B-mode- (average =25 [2–44] vs. 9 [1–20], $p<0.001$) and RTS-scores (6.5 [2–13] vs. 4 [1–9], $p<0.001$) than sicca-patients. The same was also found for MRI-assessment (6,96 vs. 2,33, p=0.001). In a Spearman rank correlation, clinical parameters were linked to the imaging techniques The B-mode showed significant inverse correlations with the Saxon-test (r=-0.505, p=0.002) and a positive correlation with MRI (r=0.792, $p<0.0001$). No correlation was found for the activity scores ESSDAI (p=0.221) or SSDI (p=0.219). The MRI score had an inverse correlation with the Saxon-test (r=-0.523, p=0.001). Both imaging techniques showed no relationship with ESR or CRP. We also generated ROC curves of both imaging methods to define possible cutoff values for the diagnosis. For B-Mode we would recommend a value of 12 points (sensitivity 82,6% and specificity 91,7%) and for MRI 3.5 points (78,3% and 91,7%).
Conclusions: Sonography and MRI detected typical morphological changes in the SG of pSS with high sensitivity and specificity. Both methods could become valuable tools for the diagnosis of pSS.
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FRI0269 "IF IT'S NOT MULTIPLE SCLEROSIS, LOOK FOR A CONNECTIVE TISSUE DISEASE": ATYPICAL DEMYELINATING DISORDERS REFERRED TO A TERTIARY RHEUMATOLOGY CENTRE

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Background: Central nervous system (CNS) demyelination is the hallmark of multiple sclerosis (MS), but may rarely occur in connective tissue diseases (CTD), mainly systemic lupus erythematosus (SLE) and antiphospholipid syndrome (APS).
Objectives: To examine whether patients with demyelinating syndrome and atypical features for MS may exhibit an underlying connective tissue disease.
Methods: Patients referred to the Rheumatology and Clinical Immunology Unit of "Attikon" University Hospital for a demyelinating disorder characterized as "atypical for MS" following neurologic evaluation, were included in the study. All cases were discussed with a neurologist with expertise in MS and CNS magnetic resonance imaging were assessed by an experienced neuroimagist. Cases included either i) a clinical syndrome suggestive of a demyelinating process (demyelinating optic neuropathy, myelopathy, internuclear opthalmoplegia etc.) and/or ii) abnormal imaging of the central nervous system (CNS) with features suggestive of demyelination (ie. presence of supra- and infratentorial lesions, periventricular location, gadolinium enhancement, Dawson's fingers, hypointense T1-lesions ("black holes"), "dirty" white matter). Patients with brain lesions in MRI more compatible with non-specific white matter hyperintensities (WMHI, ie. of possible ischemic/microvascular etiology) were excluded from the study.
Results: 21 patients were included in the study [all women, mean (SD) age at first neurologic manifestation 37.7 (10.6) years]. 17 patients had MRI findings of a demyelinating process in the CNS (brain lesions only in 7, spinal cord lesions only in 4 and both brain and spinal cord lesions in 5 patients); WMHI or normal findings were found in 4 patients with optic neuritis. In this selected group of patients, detailed rheumatologic evaluation revealed clinical and laboratory findings suggestive of a CTD in all patients. The most common findings were: arthritis (80.5%), Raynaud's phenomenon (42.8%), photosensitivity (38.1%), malar rash/erythema (33.3%), livedo reticularis (23.8%) and leukopenia (23.8%). Antinuclear antibodies were positive in two-thirds of patients (66.7%), anti-Ro/La in 19.0% and aPL only in one patient (4.8%). Final diagnoses were undifferentiated CTD, in 10 patients, frank SLE in 9 patients, primary obstetric APS and RA, in one patient each. After a median (range) follow-up of 3 (1–14) years, three patients fulfilled the criteria for MS and received MS-specific therapy, thus were subsequently classified as having an overlap of two diseases.
Conclusions: CNS lesions suggestive of demyelination on MRI must be distinguished from non-specific lesions of microvascular etiology. In cases of demyelinating syndromes not fulfilling criteria for MS, features of an underlying CTD, suggestive of SLE, are frequently found. A small percentage of patients may go on to develop frank MS during follow-up, thus longitudinal monitoring is necessary.
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FRI0270 URINARY AND SERUM NEUTROPHIL GELATINASE-ASSOCIATED LIPOCALIN AS A BIOMARKER IN INDIAN CHILDREN WITH SYSTEMIC LUPUS ERYTHEMATOSUS: RELATION TO RENAL INVOLVEMENT AND OVERALL DISEASE ACTIVITY

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Background: Renal involvement often results in long-term morbidity in childhood Systemic Lupus Erythematosus (cSLE). Neutrophil gelatinase associated lipocalin (NGAL) has been shown to be a reliable structural biomarker for the early diagnosis of kidney injury in many clinical scenarios.
Objectives: This study aimed to detect levels of urinary and serum NGAL and changes in flare and improvement on longitudinal follow up in cSLE in a real world clinical scenario.
Methods: Children <14 years of age attending the Pediatric Rheumatology clinic, fulfilling the 1997 SLE criteria were recruited. Urine and serum samples were collected during routine clinical review and SLE Disease Activity Index (SLEDAI) assessed. Children were divided into 3 categories, active renal, active non-renal and inactive lupus. Active lupus was defined as SLEDAI >4. Urinary and serum levels of NGAL (uNGAL; sNGAL) were assessed by ELISA. Urinary values were normalized for urinary spot creatinine. In addition, some patients were longitudinally followed up and resampled when their disease activity changed.
Results: The study included 122 (F:M =91:31) children, 54 had active renal,14 had active non-renal and 54 had inactive disease. Median (IQR) age was 8.8 (6.5–10.7) years and disease duration was 10 (3–24) months.26 children with nephrotic syndrome and 49 age and gender matched healthy controls were also recruited. Children with active renal lupus had significantly higher uNGAL as compared to other categories.Although sNGAL was significantly higher in active renal as compared to inactive lupus,there was no difference between renal and non-renal active lupus (Table 1). On longitudinal follow up, uNGAL levels increased markedly prior to a flare, significantly higher in renal compared to a non-renal flare ($p<0.05$). On the other hand, active lupus children had a significant fall in their uNGAL on follow up. Their was good correlation between change in SLEDAI and change in absolute uNGAL levels (r =0.84, $p<0.01$). Overall, on ROC analysis, uNGAL classified active renal versus active non-renal and inactive combined with an AUC of 0.986 (95% CI 0.972–1.0). The sensitivity and specificity of a uNGAL cutoff off value of 25750 ng/ml was 96.3 and 91.2% respectively.
Conclusions: Urinary NGAL is a sensitive marker of renal involvement in SLE disease activity and can also be a reliable tool for monitoring renal disease activity changes.

Abstract FRI0270 – Table 1

	Inactive cSLE (n=54)	Active renal cSLE (n=54)	Active non-renal cSLE (n=14)	Healthy controls (n=49)	Nephrotic controls (n=26)
Demographics					
Age (years) (mean±SD)	10.6±3.2	10.9±2.5	10.7±1.9	–	11.0±2.0
Age at onset (years) (mean±SD)	*8.4*± 3.1	*9.4*±2.3	*8.9*±2.4	-	–
Female: male ratio	39:15	41:13	11:3	–	18:8
Disease duration (months) (mean±SD)	23.7±32.4	16.6±23.1	14.5±19.2	–	–
Disease activity					
SLEDAI (mean±SD)	2.1±1.0	20.9±6.6	7.1±2.0	–	–
Renal SLEDAI (mean±SD)	0	12.5±2.7	0	–	–
Serum NGAL (pg/ml) (Median (IQR)*	4400 (2332.5–5400)	18245 (16137.5–21062.5)	18375 (14375.0–26142.5)	–	–
Urine NGAL (pg/ml) Median (IQR)**	14100 (11250–19650)	41875.0 (36243.75–46575)	18475 (15287.5–22500)	1170 (658.5–1714.5)	25040 (19637.5–31762.5)
Urine_NGAL_pg/mg_creatinine Median (IQR)**	14743.7 (10487.7–21051.8)	33288.65 (24610.7–44243.9)	14541.2 (11939.3–22043.08)	1008 (597.7–2042.79)	21874.5 (16328.5–31143.8)

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FRI0271 PREVALENCE AND ASSOCIATED FACTORS OF DEPRESSIVE DISORDERS IN CHINESE PATIENTS WITH SYSTEMIC LUPUS ERYTHEMATOSUS (SLE)

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Background: Psychiatric symptoms are common in patients with SLE. Most studies utilized self-rated scales of psychiatric symptoms for evaluation. Formal diagnosis of depression was not established by psychiatric interviews.
Objectives: To determine the prevalence of depressive disorders, severity of depressive symptoms and the associated clinical and socioeconomic factors in Chinese patients with SLE.
Methods: Patients who fulfilled ≥4 ACR criteria for SLE were randomly recruited from rheumatology out-patient clinics and hospital admission in a 9-month period. Psychiatric disorders were diagnosed by a direct interview with the psychiatrist using the Chinese-bilingual Structural Clinical Interview for DSM-IV Axis I disorders, patient research version (CB-SCID-I/P). The severity of depressive symptoms was assessed by the validated Chinese Hamilton Depressive Rating Scale (HAM-D). Patients were also asked to complete the Beck Depression Inventory (BDI), Medical Outcomes Study Social Support Survey (MOS-SSS-C) and the WHO Quality of Life Measure-Abbreviated Version (WHOQOL-BREF, BREF (HK)). SLE disease activity (SLEDAI), organ damage (SLICC/SDI) and socio-demographic were collected and correlated with the presence of psychiatric disorders. Logistic regression models were used to study the independent factors associated with depressive disorders and the severity of depressive symptoms.
Results: 175 SLE patients were studied (95%women, age 39.2±12.4 years, SLE duration 10.3±6.7 years). 27 (15%) and 37 (21%) patients were diagnosed with a current depressive (52%major depressive disorder, 22%dysthymia) or anxiety (35%generalized anxiety, 14%panic, 14%phobia, 8%adjustment) disorders, respectively. Patients with depressive disorders, as compared to those without psychiatric disorders, had more active SLE (p=0.03) and were more likely to have a history of psychiatric diagnosis (p<0.001) and financial assistance from Government (p=0.04). Independent factors associated with a depressive disorder were SLEDAI score (1.13 [1.02–1.24];p=0.02), perceived poor social support (p=0.03) and a past history of psychiatric disorders (p=0.003). Age, disease duration and other socio-economic variables such as educational level and marriage status were not correlated with the presence of a depressive disorder. Being separated/divorced (β=0.19;p=0.02), having a higher SLEDAI score (β=0.16;p=0.02), SLE duration (β=-0.18;p=0.02) and a past history of psychiatric disorders (β=0.18;p=0.01) were independently associated with higher HAM-D scores, which reflect more severe depressive symptoms. Depressive disorders and severity of depressive symptoms were significantly associated with poorer quality of life. ROC analysis showed that a cut-off of 14 points of the self-rated BDI had a sensitivity of 89% and a specificity of 83% for differentiating a current depressive disorder from those without.
Conclusions: A diagnosis of depressive disorders is prevalent in Chinese patients with SLE. Independent risk factors are more active disease, perceived poor social support and a past history of psychiatric disorders. Patients with more active SLE, shorter disease duration, a past history of psychiatric disorders and being separated were associated with more serious depressive symptoms. The self-rated BDI provides a good screening tool for identifying depressive disorders in SLE patients.
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FRI0272 EARLY DAMAGE ASSESSMENT AND PREDICTION OF DAMAGE ACCRUAL IN PRIMARY SJÖGREN'S SYNDROME USING SALIVARY GLAND ULTRASONOGRAPHY DURING 2 YEARS OF FOLLOW-UP

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Background: Early detection and prediction of glandular damage in primary Sjögren's syndrome (pSS) is of pivotal importance in patients' stratification and treatment. Recently, ultrasonography (SGUS) has appeared as a promising tool for the assessment of salivary gland involvement in pSS. However, few studies have specifically explored its role in the early identification of salivary gland damage and in monitoring disease progression over the follow-up.
Objectives: a) to explore the contribution of SGUS in the early assessment of pSS-induced glandular damage and in monitoring damage accrual during the follow-up b) to identify predictive factors associated with the development and progression of sonographic changes in salivary glands.
Methods: An inception cohort of 54 pSS (AECG 2002) patients was included in this study at the diagnosis and prospectively followed after 6, 12 and 24 months. Demographic, clinical data and the ESSDAI of the patients were collected at each study visit as well as SGUS score that was calculated every time by the same radiologist. Sonographic assessment of glandular damage was performed by evaluating the size of the parotid and submandibular glands (normal/reduced) and the presence/absence of hyperechoic bands in more than 50% of the glands (i.e fibrosis). Descriptive statistics and logistic regression were used for the data analysis.
Results: We included 54 patients (51F:3 M) with a median duration of symptoms before diagnosis of 11 months (range 36–5 mo) and a median (IQR) ESSDAI at baseline of 5 (1–8). We found that at baseline 13 patients out of 54 (24.1%) already presented one element of damage in their SGUS evaluation, 14/54 (25.9%) more than one element of damage, whereas only 27/54 (50%) of patients did not present any sonographic sign suggestive for damage. In particular, hyperechoic bands were detected in the parotid glands of 12/54 (22%) patients and in the submandibular glands of 21/54 (38.9%); reduced parotid and submandibular gland size were described in 5/54 (9.3%) and in 10/54 (18.5%) of the cases, respectively. Predictive factors associated with sonographic salivary gland damage at the diagnosis were the ESSDAI at baseline (OR =1.13 [95% CI 1.0 to 1.27], p=0.04) and the positivity for Rheumatoid factor (RF) (OR =4.47 [95% CI 1.29 to 15], p=0.02). During the follow-up, 15/54 (27.8%) patients presented a progression of their salivary gland damage. We specifically observed a significant increase in the frequency of reduced parotid (9.3% vs 25.9%, p=0.004) and submandibular gland size (18.5% vs 33.3%, p=0.008) at the end of follow-up. Predictive factors for damage accrual during follow-up were: minor salivary gland focus score (OR =3.5 [95% CI 1.1 to 10.6], p=0.03) and the ESSDAI at baseline (OR =1.64, [95% CI 1.0 to 2.6]p=0.03).
Conclusions: Sonographic assessment of glandular damage apparently revealed a relative high frequency of already established signs of glandular damage in pSS patients at baseline, especially in patients with positive RF and higher ESSDAI. When routinely used, SGUS may accurately allowed to monitor damage accrual over the follow-up, ultimately contributing to a better clinical management of patients.
Disclosure of Interest: None declared
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FRI0273 ROLE OF PROCALCITONIN AND C-REACTIVE PROTEIN IN SCREENING OF INFECTION IN PATIENTS WITH SYSTEMIC LUPUS ERYTHEMATOSUS

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Background: Previous studies revealed conflicting results regarding the role of procalcitonin in predicting infections in patients with systemic lupus erythematosus (SLE).
Objectives: This retrospective study aimed to analyse the role of procalcitonin (PCT) and C-reactive protein (CRP) in predicting infections in SLE patients, and to determine an optimal cut-off value for PCT and CRP for defining sepsis.
Methods: This study was carried out in one single tertiary centre. Adult patients (>18-year-old) with underlying SLE who were admitted to hospital between 1st Jan 2007 and 31st Dec 2015 were included. Demographic data, PCT and CRP upon admission, and other clinical parameters were obtained. Infection was defined by positive culture, or based on clinical and radiological evidence with subsequent response to antimicrobial treatments. Active SLE disease activity was defined by SLEDAI, and also by SLE-related manifestations not included in SLEDAI. Mann-Whitney test was used to test the difference between numerical parameters between patients with and without infection. Spearman's correlation was used to analyse the correlation between PCT and CRP. Receiver operating characteristic (ROC) curves were plotted to define an optimal cut-off values for PCT and CRP in infection.
Results: 33 (27 female & 6 male) SLE patients were included. Both mean and median age were 42-year-old. Among the 21 septic patients, 9 had active lupus and 12 had inactive disease. All but one of the 12 non-septic patients had active lupus. All 4 patients with underlying renal failure belonged to the infection group. There were 13 and 3 patients on immunosuppressive treatments in infection and non-infection groups respectively.
In patients with infection, mean PCT was 5.74ng/ml and mean CRP was 77.22mg/L. In those without, these were 0.29ng/ml and 20.04mg/L respectively. Both PCT (p=0.014) and CRP (p=0.016) levels were significantly higher in patients with infection than those without. There was no significant difference between the PCT and CRP levels in both septic patients (PCT p=0.862, CRP p=0.247) and non-septic patients (PCT p=1.000, CRP p=0.500) regardless of their SLE disease activity.
PCT level correlated positively with CRP level (r=0.456, p=0.008), but it had no correlation with age (p=0.978), gender (p=0.424), underlying renal failure (p=0.304), steroid (p=0.053) or other immunosuppressants (p=0.132) use.
ROC curves of PCT and CRP showed a similar area under curve (AUC) of 0.756 and 0.752 respectively. The cut-off for PCT was 0.78ng/ml (sensitivity 61.9%, specificity 91.7%), giving a positive predictive value (PPV) of 92.9%. The cut-off for CRP was 9.35mg/l (sensitivity 85.7%, specificity 66.7%), giving a PPV of 81.8%. Combining both PCT and CRP above their cut-offs, the specificity of predicting infection improved to 100% but the sensitivity worsened to 52%.
Conclusions: Both PCT and CRP were useful in predicting infection SLE patients regardless of their disease activity. The cut-off of PCT at 0.78ng/ml and CRP at 9.35mg/l gave satisfactory positive predictive value for infection.