MHz transducer a statistically significantly higher median DT was showed in all skin areas, included thighs ($p<0.01$). Finally, a positive statistically significant correlation was observed between the two transducers in the evaluation of DT ($p<0.0001$), as well as between both probes and mRSS ($p<0.0001$ for both).
**Conclusions:** This study suggests that subclinical dermal involvement may be detectable by skin high frequency US already in patients with limited cutaneous SSc. This study confirms that DT can be better assessed in SSc patients by using a 22 MHz US probe, and suggests that DT might be underestimated by using US probes of lower frequency (18 MHz). However, the DT values obtained using both probes resulted significantly correlated together and with the mRSS.
**References:**
[1] Clements PJ, et al. Arthritis Rheum 2000;43:2445–54.
[2] Moore TL, et al. Rheumatology 2003;42:1559–63.
[3] Sulli A, et al. Ann Rheum Dis. 2014;73:247–51.
[4] Czirják L, et al. Ann Rheum Dis. 2007;66:966–9.
[5] Hesselstrand R, et al. Rheumatology 2008;47:84–7.
[6] Kaloudi O, et al. AnnRheum Dis.2010;69:1140–3.
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## OP0038 MYOSITIS AUTOANTIBODIES OUTPERFORM CLINICAL SUBGROUP CLASSIFICATION IN PREDICTING MUSCLE WEAKNESS IN MYOSITIS PATIENTS

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**Background:** Myositis patients may be classified as belonging to one of four clinical groups: dermatomyositis (DM), polymyositis (PM), clinically amyopathic dermatomyositis (CADM) or necrotizing myositis (NM). Alternatively, myositis patients may be classified according to myositis autoantibody status.
**Objectives:** The aim of this study was to determine whether clinical groups or myositis autoantibodies provide better prognostic categories with regard to muscle involvement in these patients.
**Methods:** All Johns Hopkins Myositis Center patients from 2002 to 2015 with a myositis specific autoantibody confirmed by two different immunologic techniques were included. Autoantibody groups accounting for less than 2% of the final sample size were excluded. Strength (analyzed as the average of deltoid and hip flexor strength using Kendall's scale) and log transformed CK levels were compared between the different autoantibody groups using multilevel regression models adjusted for age, time from disease onset, sex, race and treatments. Models with different combinations of key variables were compared using the likelihood ratio test to ascertain if autoantibody groups and clinical subgroups provided the same amount of information regarding muscle weakness and CK levels over time.
**Results:** 483 patients with 4181 visits were included and 10 different autoantibody groups were identified. Muscle weakness and CK levels followed a gradient among both antibody and clinical groups. Anti-SRP patients had the greatest weakness, followed by anti-HMGCR, anti-Mi2 and anti-NXP2, and then anti-Jo1. CK levels were highest in anti-HMGCR patients, followed by anti-SRP, anti-PL7, anti-Jo1 and anti-Mi2. Interestingly, strength and CK levels were dissociated in two groups: anti-NXP2 patients had significant weakness with low CK levels and anti-PL7 patients were relatively strong despite high CK levels. Multilevel regression models showed autoantibody groups explained the strength and the CK variability better than the clinical groups (AIC difference>20). Indeed, adding clinical groups to a model using only autoantibodies did not improve the model's ability to predict strength (p=0.2) and only mildly improved its ability to predict CK (p=0.01). In comparison, adding the autoantibodies to a model using the clinical groups resulted in a marked improvement in predicting both CK and strength (both $p<0.001$).
**Conclusions:** In patients with myositis, autoantibody status predicts strength and CK levels better than clinical grouping.
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# SLE, Sjögren's and APS - clinical aspects

## OP0039 A POPULATION-BASED STUDY ON MORTALITY AND THE INFLUENCE OF MEDICATION USE IN 4356 PATIENTS WITH SYSTEMIC LUPUS ERYTHEMATOSUS AND 21845 MATCHED CONTROLS FROM THE UNITED KINGDOM

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**Background:** Systemic lupus erythematosus (SLE) has been associated with an increased mortality rate. However, population-based data on all-cause, age-specific and sex-specific mortality risk are limited and data on the influence of medication exposure on mortality risk in SLE are scarce.
**Objectives:** To estimate the magnitude of the risk from all-cause, age-specific, and sex-specific mortality in patients with SLE and relative risks compared with matched controls, and to evaluate the influence of medication exposure on mortality risk in SLE.
**Methods:** We conducted a population-based cohort study using the Clinical Practice Research Datalink (from 1987 to 2012). Each SLE patient (n=4356) was matched with up to 6 controls (n=21845) by age and sex. Multivariate Cox regression analysis estimated adjusted relative rates (RR) of mortality, and time interaction terms to evaluate mortality timing patterns. Time-dependent Cox models were used to evaluate the association of glucocorticoid use and hydroxychloroquine use on mortality and were adjusted for age, sex, lifestyle parameters, comorbidities and comedication.
**Results:** A total of 442 out of 4356 SLE patients died during the study period. Patients with SLE had an increased mortality rate for all-cause mortality compared with age- and sex-matched subjects, after adjustment for confounders (adjusted RR 1.80, 95% CI 1.57–2.08). Glucocorticoid use in the previous six months raised the mortality rate while the adjusted RR was 45% decreased with low dose hydroxychloroquine use. The RR was highest in patients aged 18–39 years (adjusted RR 4.87, 95% CI 1.93–12.3) and slightly higher in females (adjusted RR 1.82, 95% CI 1.56–2.13) compared to male patients (adjusted RR 1.68, 95% CI 1.19–2.39). The mortality rate was significantly increased for patients with a history of dementia, seizures, diabetes, cancer, and renal disease (Table 1).

Table 1. Risk of all-cause mortality within SLE patients (n=4356), stratified according to organ damage (reference = no risk factor)

| | Person years (x1000) | Deaths | IR (/1000) | Adjusted RR* (95% CI) |
|---|---|---|---|---|
| Dementia | 0.1 | 14 | 140.0 | 2.99 (1.74–5.14) |
| Seizures | 1.4 | 37 | 26.4 | 2.33 (1.66–3.28) |
| Cerebrovascular event | 1.9 | 73 | 38.4 | 1.28 (0.99–1.65 |
| Renal disease | 2.0 | 86 | 43.0 | 1.40 (1.09–1.78) |
| Osteoporotic fracture | 5.1 | 110 | 21.6 | 1.06 (0.85–1.32) |
| Diabetes mellitus | 0.9 | 45 | 50.0 | 1.90 (1.39–2.59) |
| Malignancy | 2.0 | 95 | 47.5 | 1.90 (1.50–2.40) |

*Adjusted for: recent use of corticosteroids, recent use of antimalarials, recent use of benzodiazepines.

**Conclusions:** Patients with SLE have a 1.8-fold increased mortality rate compared with the general population. Glucocorticoid use, female sex and young age are associated with an increased mortality risk while low dose hydroxychloroquine use significantly reduces the mortality rate. In addition, special attention should be paid to lupus patients with neuropsychiatric complications, diabetes, malignancy or renal disease since these subgroups of patients are at high risk of death.
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## OP0040 INTEGRATION OF SALIVARY-GLAND ULTRASONOGRAPHY IN CLASSIFICATION CRITERIA FOR PRIMARY SJÖGREN'S SYNDROME: AN INTERNATIONAL VIGNETTE-BASED STUDY

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**Background:** The recent classification criteria sets for primary Sjögren's syn-