Table 1.

Patient	Age/Sex	HLA DRB1*11	rs4149056 genotype	MRC at the weakest muscle group*	Dysphagia	CK (IU/L) at diagnosis	Anti-HMGCR titer (CU)	Induction therapy*	Maintenance therapy	Clinical improvement**	CK (IU/L) at last follow-up visit
1	56/M	Yes	TT	2	No	8963	277.8	GC. IVIG.MTX	GC. IVIG. MTX. RTX	Marked	134
2	69/F	Yes	TT	0	Yes	9271	235.9	GC iv bolus. IVIG.	GC. MTX. RTX.	Marked	890
3	64/F	Yes	TT	3	No	4000	242.6	IVIG.	IVIG.RTX	Marked	1284
4	79/M	Yes	TT	4	No	4977	145.6	GC. IVIG.	GC. IVIG.	Complete	92
5	62/F	No	TT	3	No	2116	210.0	GC	GC.MTX.	Marked	236
6	57/F	Yes	TC	4	No	2294	259.3	IGIV	IGIV	Complete	235
7	68/F	Yes	TT	3	No	3273	236.0	GC. IGIV. AZA.	GC. AZA	Complete	249
8	64/M	Yes	TC	4	Yes	11000	179.0	GC iv bolus. AZA.	GC. AZA	Complete	161

*AZA: azathioprine; CK: creatinine kinase; CU: chemiluminescence units; F: female; GC: glucocorticoids; IVIG: intravenous immunoglobulins; M: male; MRC: medical research council scale; MTX: methotrexate; RTX: rituximab; ** Induction therapy initiated within 3 months of diagnosis. **Clinical improvement: no improvement (no improvement in MRC grade), mild improvement (improvement of MRC grade but still requiring assistance for activities of daily living), marked improvement (persistence of mild weakness without functional limitation), and complete improvement (return to baseline with no symptoms or signs of weakness).*

AB1432 THE ASSOCIATION OF PAINFUL AND NON-PAINFUL COMORBIDITIES WITH CENTRAL MECHANISMS OF KNEE PAIN

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Background: Central mechanisms of knee pain occur in the central nervous system and may intensify and prolong pain. Central pain mechanisms might be facilitated by ongoing nociceptive input. A link between multimorbidity and central mechanisms of knee pain is proposed; ongoing sensory inputs due to comorbidities may trigger changes in pain processing by the CNS. This might be particularly expected with painful comorbidities.

Objectives: To investigate potential relationships of painful and non-painful multimorbidity with central mechanisms of knee pain.

Methods: Cross-sectional analysis of self-report data from participants of the Investigating Musculoskeletal Health and Wellbeing cohort, who reported knee as their most bothersome site of joint pain over the previous month, with pain rated ≥1/10, and who had completed FRAIL and CAP-Knee (1) questionnaires. Two indirect measures suggesting central mechanisms involvement in knee pain were used as dependent variables; pain intensity (0-10 numerical rating scale) and CAP-Knee score (0-16 scale). Comorbidities were assigned either "painful comorbidity" or "non-painful comorbidity" status based on IASP classification of chronic pain criteria (2). Multivariable linear regression models, adjusted for age and sex, were employed to explore associations of comorbidity counts with pain intensity and CAP-Knee score.

Results: 736 participants satisfied inclusion criteria. 55% were female, mean age 71 (range 40 to 95). Painful comorbidity count and non-painful comorbidity count each had positive associations with pain intensity (β=0.42, 95% CI=0.29 to 0.54, p<0.001; and β=0.31, 95%CI=0.16 to 0.45, p<0.001, respectively). Painful and non-painful comorbidity counts each also were associated with CAP-Knee score (β=0.80, 95% CI=0.59 to 1.01, p<0.001; and β=0.52, 95% CI=0.27 to 0.77, p<0.001, respectively). Painful and non-painful comorbidity counts each remained significantly associated both with pain intensity and with CAP-Knee scores when both types of comorbidity count were included in the same multivariable model.

Conclusion: Both painful and non-painful comorbidities were positively associated with central mechanisms of knee pain, providing further insight into the interconnectedness of pain processing systems and the rest of the body. The explanation behind these relationships may depend on more than just ongoing nociceptive input. Future work should address possible contributions from genetic, pathophysiological, psychological, and pharmacological factors associated with comorbid diagnosis.

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AB1433 ASSESSING THE CARDIOVASCULAR RISK IN PATIENTS WITH SYSTEMIC LUPUS ERYTHEMATOSUS: QRISK AND GAPSS SCORES HEAD TO HEAD

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Background: Cardiovascular diseases (CVDs) represent one of the most life-threatening conditions that can affect SLE patients. Assessing the potential CVD risk of these patients is still a challenge and an important aspect in the clinical practice. Recently the QRISK3 score has attempted to encompass for SLE augmented thrombotic risk by adding items (such as corticosteroid use) that are missing in traditional CVD risk scores.

Objectives: To apply and compare the QRISK3 and the adjusted Global AntiPhospholipid Syndrome Score (aGAPSS), a validated score to assess CVD and overall thrombotic risk in aPL positive patients, in a cohort of SLE patients with and without a concomitant diagnosis of APS.

Methods: 25-85 years old patients attending San Giovanni Bosco Hospital (Turin) during a period of 6 months (Sep 2019 – Feb 2020) with a confirmed diagnosis of SLE (2019 ACR/EULAR classification criteria) and/or a diagnosis of SAPS (Sidney criteria) were included in the study. QRISK3 has been calculated using the official online calculator (https://qrisk.org/). aGAPSS has been calculated using the validated point values based on aPL profile and independent risk factors: aCL=5, aβ2GPI=4, LA=4, aPS/PT=3, hyperlipidemia=3, hypertension=1.

Results: The analysis included a cohort of 142 SLE patients: 34 SAPS (23.9%) and 108 SLE patients without APS (76.1%), with a mean age of 48±12.9 (SAPS=51.6±12.8/SLE without APS=46.9±12.8). Table 1 summarizes patients characteristics. When focusing on cerebrovascular/coronary events, we found a statistical significance with respect to aGAPSS (pt with event =10.1±6.2 vs pt without event=5.8±6.1; p=0.007), but not QRISK3. Also, a significant association was observed between the occurrence of these events and high risk aGAPSS: p=0.03 for aGAPSS≥8, p=0.01 for aGAPSS ≥9, p=0.008 for aGAPSS ≥10. Moreover, the aGAPSS but not the QRISK3 resulted to strongly correlate with the occurrence of any thrombotic event, both at the uni- and multivariate analysis (univariate: pt with event =8.17±7.1 vs pt without event= 5.41±5.6; p=0.012 / multivariate: p=0.009). The same was observed for gender: male gender resulted to correlate with the occurrence of any thrombotic event at both uni- and multivariate analysis (p=0.017 and p=0.03, respectively). Finally, when focusing on aPL profile, regardless