Results: The study included 30 patients with SLE. Internal consistency of the MFIS was excellent with Cronbach’s $\alpha = 0.97$ for the complete scale. Excellent test-retest reliability was found with ICC = 0.95 (95% confidence interval: 0.88-0.98, p < 0.05). Construct validity was confirmed by Spearman’s correlation (VT-SF36: $r_s = -0.73$, p < 0.001 (Fig. 1). MH-SF36: $r_s = -0.74$, p < 0.001 (Fig. 2)) and PCA with explained variance from the first two principal components (PC) (VT-SF36: PC1 = 60.2%, PC2 = 8.5%. MH-SF36: PC1 = 58.5%, PC2 = 7.4%). No significant correlation was found between the MFIS and SLEDAI ($r_s = 0.04$, p = 0.84) or SLICC Damage Index ($r_s = 0.32$, p = 0.08).

Conclusion: The present study found the multidimensional assessment of fatigue with MFIS to be a reliable and valid instrument in SLE. The MFIS might provide more detailed information about fatigue in future studies. In agreement with some previous studies we found no association between fatigue and disease components exist.

Disclosure of Interests: None declared

DOI: 10.1136/annrheumdis-2020-eular.1293

FRI0584 SCREENING AND IDENTIFICATION OF BIOMARKERS IN MYOSITIS

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Background: Myositis, including dermatomyositis and polymyositis, is autoimmune disorders that is characterized by muscle degeneration in the proximal extremities, with the complications of weakness of muscles, interstitial lung disease and vascular lesions, even leading to death in an acute progressive process[1,2]. However, the molecular mechanisms of myositis are rarely understood.

Objectives: Identify the candidate genes in myositis.

Methods: Microarray datasets GSE128470, GSE48280 and GSE39454 were extracted from the Gene Expression Omnibus (GEO) database. The differentially expressed genes (DEGs) and function enrichment analyses were conducted. The protein-protein interaction network and the analyses of hub genes were performed with STRING and Cytoscape.

Results: There were 98 DEGs, of which the function and pathways enrichment analyses showed defense response, immune response, response to virus, inflammatory response, response to wounding, cell adhesion, cell proliferation, cell death and macromolecule metabolic process. 20 hub genes were identified, of which 7 including IRF9 TRIM22 MX2 IFITM1 IFI6 IFI44 IFI44L had not been reported in the literature, related to the response to virus, immune response, transcription from RNA polymerase II promoter, cell apoptosis, cell death. The verification analysis about the 7 genes in GSE128314 showed significant differences in myositis.

Conclusion: In conclusion, DEGs and hub genes identified in our study showed the potential molecular mechanisms in myositis, providing the helpful targets for diagnosis and clinical strategy of myositis.

References:
Acknowledgments: The authors acknowledge the efforts of the Gene Expression Omnibus (GEO) database. The interpretation and reporting of these data are the sole responsibility of the authors.

Disclosure of Interests: None declared

DOI: 10.1136/annrheumdis-2020-eular.1831

Figure 1. Two dimensional representation of Set A generated using dimensionality reduction (tSNE) and clustering (DBScan).

Clusters represented similar yet distinct clinical phenotypes; e.g. patients diagnosed with “other headache syndrome” were separated into four distinct clusters characterized by migraines, neurofibromatosis, epilepsy or brain cancer, all resulting in patients presenting with headaches (Fig. 1 & 2). Though EMR databases tend to be noisy, our method was also able to differentiate misclassification from true cases; SLE patients with RA codes clustered separately from true RA cases.

Figure 2. Phenotype Spectrographs (PheSpecs) of four clusters characterized by “Other headache syndromes”, driven by codes relating to migraine, epilepsy, neurofibromatosis or brain cancer.

Conclusion: We have shown that EMR data can be used to identify and visualize latent structure in patient categorizations, using an approach based on dimension reduction and clustering machine learning techniques. Our method can identify misclassified patients as well as separate patients with similar problems into subsets with different associated medical problems. Our approach adds a new and powerful tool to aid in the discovery of novel risk factors in complex, heterogeneous diseases.

References:

Disclosure of Interests: Marc Maurits: None declared, Thomas Huizinga Grant/research support from: Ablynx, Bristol-Myers Squibb, Roche, Sanofi, Consultant of: Ablynx, Bristol-Myers Squibb, Roche, Sanofi, Marcel Reinders: None declared, Soumya Raychaudhuri: None declared, Elizabeth Karlson: None declared, Erik van den Akker: None declared, Rachel Knevel: None declared

DOI: 10.1136/annrheumdis-2020-eular.3489