Table 1. MCII and PASS of pain and function instruments in hand OA patients in two settings.

Instrument	MCII (95% CI) in RCT†			PASS (95% CI)	
	Absolute units	Percentage	[n]	RCT (n=68)	Cohort (n=126)
MHQ					
Overall function, 0-100*	3.4 (-2.7;9.5)	3.6 (-9.1;16.3)	[23]	55.6 (52.6;58.5)	48.1 (45.7;50.4)
ADL, 0-100*	1.6 (-4.4;7.6)	2.8 (-9.6;15.3)	[23]	71.7 (68.2;75.1)	62.9 (59.8;66.0)
Pain, 0-100	-12.2 (-17.2;-7.1)	-23.1 (-35.4;-10.8)	[16]	47.0 (40.5;53.5)	55.7 (52.0;59.5)
AUSCAN					
Function, 0-36	-3.3 (-5.7;-0.9)	-9.8 (-23.9;4.2)	[23]	17.1 (15.3;19.0)	20.9 (19.4;22.3)
Pain, 0-20	-1.1 (-2.6;0.4)	-1.8 (-18.5;14.9)	[27]	9.0 (8.2;9.9)	11.1 (10.3;11.8)
FIHOA, 0-30	0.1 (-1.6;1.7)	22.7 (-0.7;46.0)	[23]	12.4 (11.1;13.7)	13.9 (12.8;15.0)
VAS pain, 0-100	-11.5 (-18.2;-4.7)	-24.4 (-36.1;-12.8)	[27]	47.7 (42.2;53.3)	48.8 (44.6;53.0)

Direction of effect of all instruments is higher is worse, except those with *.†For all MCII direction of effect was unified, so positive values indicate worse symptoms and negative values values indicate improved symptoms.

Conclusion: The only function instrument with an acceptable threshold for MCII was AUSCAN function, while for pain MHQ and VAS performed better than AUSCAN. PASS values show a relatively high level of tolerance of 50% of the maximum of the scale.

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POS0183 SIGLEC1 AS A TYPE I INTERFERON BIOMARKER IN IDIOPATHIC INFLAMMATORY MYOPATHIES

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Background: Idiopathic inflammatory myopathies (IIM) are autoimmune diseases that mainly affect skeletal muscle, lung, skin and joints. IIM can be separated into dermatomyositis (DM), inclusion body myositis (IBM), antisynthetase syndrome (AS) and immune-mediated necrotizing myopathy (IMNM). Type I interferons (IFN) are known to play a crucial role in the etiopathogenesis of some of these entities such as DM.[1] Sialic acid binding Ig-like lectin 1 (SIGLEC1, CD169) is part of the type I IFN signature found in SLE and DM and is expressed on the cell surface of monocytes. Thus, analysis of SIGLEC1 expression by flow cytometry enables a straightforward assessment of the type I IFN signature. Its utility has been shown for juvenile and adult SLE and other rheumatic diseases but not in IIM.[2,3] The assessment of the type I IFN system in clinical practice is an unmet need and, in this context, SIGLEC1 might be useful.

Objectives: To assess SIGLEC1 expression on monocytes by flow cytometry as a type I IFN biomarker in IIM

Methods: Pediatric and adult patients with a clinical diagnosis of DM, AS, IMNM and IBM and at least one measurement of SIGLEC1 who have been treated at the Department of Rheumatology, Charité - Universitätsmedizin Berlin between 2015 and 2020 were included in this retrospective study. Control groups of healthy individuals (n=19) and SLE patients (n=30) were included. Disease activity was assessed by Physician Global Assessment (PGA) and Childhood Myositis Assessment Scale (CMAS). SIGLEC1 expression on monocytes was analyzed by flow cytometry. Cross-sectional analyses (n=74) were performed using Mann Whitney-U test (MWU) and two-level mixed-effects linear regression model was used for longitudinal analyses (n=26, 110 visits). This study was approved by the local ethics committee of the Charité - Universitätsmedizin Berlin.

Results: 74 patients (adult/juvenile DM: n=21/n=17; AS: n=19; IMNM: n=8; IBM: n=9) were included. In cross-sectional analysis, SIGLEC1 expression was significantly upregulated in adult and juvenile DM patients with moderate to severe disease activity (PGA≥5) compared with adult/juvenile DM patients with no to moderate disease activity (PGA<5) (both p<0.001). In longitudinal analyses, SIGLEC1 correlated with disease activity in juvenile DM (SIGLEC1 vs. CMAS: betaST=-0.65; p<0.001) and adult DM (SIGLEC1 vs. PGA: betaST=0.52; p<0.001), better than Creatine Kinase (CK) (juvenile DM, CK vs. CMAS: betaST=-0.50; p<0.001; adult DM, CK vs. PGA: betaST=0.17; p=0.149). In AS 42.1% of the patients showed elevated SIGLEC1 expression, while it was not upregulated in IMNM and only in two patients with IBM, who were concurrently positive for autoantibodies that affect the type I IFN system (see Figure 1).

Conclusion: SIGLEC1 is a useful biomarker to identify an activated type I IFN system in IIM. Flow cytometry is used widely in laboratory medicine, which could facilitate the implementation of SIGLEC1 into clinical routine.

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Figure 1. SIGLEC1 expression on monocytes in IIM subgroups and control groups; in IIM subgroups, patients with low disease activity (PGA<5) are marked in blue, patients with high disease activity (PGA≥5) are marked in red; mAb/cell, monoclonal antibodies bound per cell

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POS0184 URINE-GALECTINE 3 BINDING PROTEIN (U-GAL3BP) IS A SENSITIVE MARKER OF KIDNEY INFLAMMATION AND RESPONSE TO TREATMENT IN LUPUS

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