classify IIM in comparison with 1975 Bohan and Peter criteria in Chinese patients.

Methods: Two hundred and twenty-one in-patients with suspected IIM (including 40 children) were retrospectively included in this study. The performance of 2017 EULAR/ACR criteria was evaluated by sensitivity, specificity, positive predictive value, negative predictive value and classification rate, in comparison to the 1975 criteria, with clinical diagnosis as the gold standard.

Results: The sensitivity, specificity, positive predictive value, negative predictive value of the 2017 EULAR/ACR criteria in IIM classification were 92.7%, 87.0%, 90.1% and 90.4% respectively, in contrast to the 1975 Bohan and Peter criteria of 84.0%, 52.2%, 61.8%, and 77.9% (Fig 1). The classification rate of 2017 criteria was also much better than that of 1975 criteria (90.2% vs. 67.4%). The performance of the new criteria in general, as well as the new criteria with muscle biopsy was better. Most IIM patients were correctly further subclassified by the classification tree. The positive rate of myogenic lesion in electromyography (EMG), muscular inflammatory edema in magnetic resonance imaging (MRI) and specific antibodies of myositis were significantly higher in IIM group than those in control group ($p < 0.001$, all).

Conclusion: The 2017 EULAR/ACR criteria exhibited high sensitivity, specificity, classification rate in the Chinese IIM patients, which was superior to the 1975 criteria. The new criteria showed potentials as clinical classification criteria in the future.

Figure 1. Performance of 2017 EULAR/ACR criteria and 1975 Bohan and Peter criteria.

* referred to the overall assessment, including patients *score of ≥ 5.5 without biopsies and score of ≥ 6.7 with biopsies

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AB0697C

THE ASSOCIATION OF ANTI-MELANOMA DIFFERENTIATION-ASSOCIATED PROTEIN 5 AND SEASONAL PATTERNS IN ONSET OF IDIOPATHIC INFLAMMATORY MYOPATHIES IN KOREA

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Background: There was some reports of seasonal association with myositis onset. With the discovery of new myositis-specific autoantibodies

(MSA), detailed grouping of idiopathic inflammatory myopathies was possible. Therefore, we evaluated the seasonal patterns in the onset of idiopathic inflammatory myositis (IIM) with MSA in Korea.

Objectives: To evaluate the correlation between MSA and seasonal patterns of IIM in Korea.

Methods: A total of 90 patients who met the criteria for probable or definite PM or DM and for whom data were collected from 7 referral centers in Korea. 16 myositis-specific autoantibodies were detected by immunoblot with patient's serum. Statistical analyses were performed using a Poisson model that assessed associations of sex, MSA, and month of onset of symptoms or month of diagnosis.

Results: There were no significant seasonal patterns of disease onset in total IIM patients. Among MSAs, anti-synthetase ($n=18$), anti-transcriptional intermediary factor 1 γ (TIF1 γ) ($n=13$), anti-melanoma differentiation-associated protein 5 (anti-MDA5) ($n=12$), and anti-signal recognition particle (SRP) ($n=12$) were analyzed. Among 55 patients with dermatomyositis, patients with anti-MDA5 showed a significant peak in winter ($n=12$, $P=0.05$). This seasonal association was significant in women ($n=10$; $P=0.045$). Patients with anti-synthetase, anti-TIF1 γ or anti-SRP antibodies did not have a significant seasonal onset patterns. There were no significant seasonal patterns in patients without myositis-specific autoantibodies.

Conclusion: Patients with anti-MDA5 showed a seasonality of myositis onset, in winter.

Disclosure of Interests: None declared

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AB0697D

CARDIOVASCULAR RISK FACTORS ARE HIGHER IN MALNOURISHED PATIENTS WITH SYSTEMIC SCLEROSIS

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Background: Cardiovascular disease (CVD) is a well known complication in rheumatoid arthritis and systemic lupus erythematosus. Atherosclerosis (ATS) and its impact on CVD in patients with systemic sclerosis (SSc) still remains unclear. Most of studies are suggesting higher frequency of CVD in SSc in comparison to healthy controls. CVD is a leading cause of death in most developed countries, whereas in SSc population it ranges from 20 to 30%. The etiology of ATS in SSc is unknown. Traditional risk factors, endothelial dysfunction and inflammation can contribute to ATS in SSc population. However, there is lack of data about the impact of the nutritional status on CVD factors in SSc patients.

Objectives: The aim of the study was to determine CVD factors among SSc patients depending on the nutritional status.

Methods: In 55 patients with SSc (72.7% well-nourished, 18.1% malnourished, 9.1% pre-cachexia) and 49 healthy controls we measured markers of endothelial dysfunction (asymmetric dimethylarginine - ADMA), inflammation (C-reactive protein - CRP, high sensitivity C-reactive protein - hsCRP, interleukin 6 - IL-6) and dyslipoproteinemia (oxidized low-density lipoprotein - ox-LDL, high-density lipoprotein- HDL, low-density lipoprotein - LDL and total cholesterol). Nutritional status was determined with subjective global assessment (SGA), body mass index (BMI), bioelectrical impedance analysis (BIA) and anthropometric measurements.

Results: Well-nourished SSc patients had significantly higher level of IL-6 (6.4 ± 10.1 vs. 2.8 ± 3.6 pg/ml; $p=0.002$) and lower HDL cholesterol (49.9 ± 11.6 vs. 57 ± 13.6 mg/dl; $p=0.011$) in comparison to healthy control. In malnourished SSc patients there was higher concentration of ADMA (1.68 ± 0.53 vs. 1.24 ± 0.34 μ mol/l; $p=0.003$), CRP (18 ± 28.8 vs. 2.4 ± 2.5 mg/l; $p=0.04$), IL-6 (21.6 ± 34.9 vs. 2.8 ± 3.6 pg/ml; $p=0.004$) and lower HDL cholesterol (42.5 ± 18.3 vs. 57 ± 13.6 mg/dl; $p=0.01$) in comparison to healthy control. Pre-cachexia SSc group had significantly lower total cholesterol (166.8 ± 28.5 vs. 219.3 ± 40.9 mg/dl; $p=0.008$) and LDL cholesterol (96.8 ± 21.6 vs. 139.6 ± 36 mg/dl; $p=0.007$). There were no differences in concentration of oxLDL and hsCRP in those groups.

Conclusion: Nutritional status may play role in risk of CVD in SSc patients. Although its contribution to morbidity and mortality rates in CVD in SSc is yet to be established.

Disclosure of Interests: None declared

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Spondyloarthritis – treatment

AB0698

EFFECTIVENESS AND SAFETY OF CERTOLIZUMAB PEGOL FOR THE TREATMENT OF AXIAL SPONDYLOARTHRITIS IN REAL-WORLD CLINICAL PRACTICE IN EUROPE: RESULTS FROM A PROSPECTIVE NON-INTERVENTIONAL 12-MONTH COHORT STUDY

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Background: Certolizumab pegol (CZP) is an Fc-free, PEGylated anti-TNF with an established efficacy and safety profile in axial spondyloarthritis (axSpA) in clinical trial settings.¹

Objectives: To report CZP effectiveness and safety in patients (pts) with axSpA, including ankylosing spondylitis (AS; radiographic axSpA) and non-radiographic (nr-) axSpA subpopulations, in routine clinical practice in Europe.

Methods: CIMAX (NCT02354105) was a non-interventional multicentre prospective cohort study observing CZP treatment response and safety over 12 months in a real-world clinical cohort of axSpA pts newly prescribed CZP. The primary outcome was change from baseline (CFB) in Bath Ankylosing Spondylitis Disease Activity Index (BASDAI) to Week (Wk) 52 in pts with available data, with additional outcomes pertaining to effectiveness and safety. Outcomes were evaluated for AS and nr-axSpA subpopulations (diagnosed according to local practice). Pts who received ≥ 1 dose CZP were followed up for adverse events (AEs) (Safety Set [SS]); those with baseline and ≥ 1 post-baseline BASDAI assessment were included in the effectiveness analyses (Full Analysis Set [FAS]). Outcomes are reported using observed case data with no imputation.

Results: 682 axSpA pts were enrolled from 101 European sites, of whom 490 (71.8%) completed the study. Of those enrolled, 672 formed the SS (AS: 469; nr-axSpA: 201) and 564 the FAS (AS: 384; nr-axSpA: 179); 2 (SS)/1(FAS) pts with unconfirmed AS/nr-axSpA were included in the overall axSpA population. 27.5% (185/672[SS]) axSpA pts had previous anti-TNF exposure (AS: 31.1% [146/469]; nr-axSpA: 18.4% [37/201]). BASDAI data were available for 77.8% (439/564) pts at Wk52. In pts with available data, all clinical outcomes were improved at Wk52 in both subpopulations (Table). At baseline, the mean BASDAI was 6.1. At Wk52, the mean BASDAI CFB in pts with available data (n=439) was -2.9 (AS: -2.9 [n=301]; nr-axSpA: -2.8 [n=137]). For pts with and without prior anti-TNF exposure, BASDAI at baseline was 6.1 in both groups (n=165 vs n=399, respectively), and at Wk52, the mean BASDAI CFB was -2.6 (n=127) vs -3.0 (n=312) (AS: -2.7 vs -3.0; nr-axSpA: -2.2 vs -3.0). In the SS, 37.9% (255/672) pts experienced AEs; 20.7% (139/672) experienced drug-related AEs and 6.3% (42/672) serious AEs (1.8% [12/672] reported serious infections); these data were comparable between AS and nr-axSpA (Table).

Conclusion: This is the first multicentre European study to evaluate CZP effectiveness and safety in both axSpA subpopulations in routine practice. Improvements were observed in all signs and symptoms in pts who remained on treatment to Wk52; >70% of those enrolled completed the study. No new safety signals were identified following application of CZP to real-world rheumatological practice.

Table: Clinical and safety outcomes at Week 52 for patients with axSpA in CIMAX

Clinical outcomes (FAS)	AS (n=384)		nr-axSpA (n=179)		Overall axSpA (n=564)	
	BL	Wk52	BL	Wk52	BL	Wk52
BASDAI	6.1 (1.8)	3.2 (2.1)	6.1 (1.7)	3.2 (2.2)	6.1 (1.8)	3.2 (2.2)
Mean (SD)	n=384	n=301	n=179	n=137	n=564	n=439
BASDAI CFB	-2.9 (2.2)	-	-2.8 (2.4)	-	-2.9 (2.3)	-
Mean (SD)	n=384	n=301	n=179	n=137	n=564	n=439
ASAS20	72.8 (209/289)	64.3 (181/236)	69.7 (200/240)	65.3 (74.1)	71.2 (209/289)	64.8 (181/236)
% (n/N) [95% CI]	(67.2, 77.5)	(55.9, 72.7)	(55.9, 72.7)	(65.3, 74.1)	(67.2, 77.5)	(55.9, 72.7)
ASAS40	51.5 (150/291)	48.4 (62/138)	50.3 (112/220)	45.7 (55.3)	50.9 (150/291)	48.4 (62/138)
% (n/N) [95% CI]	(46.8, 57.3)	(39.6, 57.1)	(45.7, 55.3)	(45.7, 55.3)	(46.8, 57.3)	(39.6, 57.1)
ASDAS (CRP) (n)	3.7 (0.9)	2.1 (1.0)	3.5 (0.8)	2.0 (1.0)	3.6 (0.9)	2.1 (1.0)
Mean (SD)	n=345	n=267	n=158	n=115	n=504	n=383
ASDAS-SD	0.9	24.1	0.6	25.6	0.8	24.5
% (n/N)	(1/351)	(65/270)	(1/160)	(10/117)	(4/512)	(95/388)
BASFI	5.4 (2.4)	3.1 (2.4)	5.1 (2.3)	2.9 (2.5)	5.3 (2.4)	3.1 (2.4)
Mean (SD)	n=383	n=301	n=179	n=137	n=563	n=439
Total back pain (n)	6.8 (2.2)	3.3 (2.3)	6.6 (2.4)	3.3 (2.6)	6.7 (2.3)	3.3 (2.4)
Mean (SD)	n=382	n=297	n=177	n=137	n=560	n=435
PRISDA	6.9 (2.3)	3.2 (2.4)	6.9 (1.8)	3.3 (2.6)	6.9 (2.1)	3.2 (2.3)
Mean (SD)	n=384	n=295	n=177	n=135	n=562	n=431
PRISDA	6.3 (1.7)	2.4 (1.9)	6.2 (1.7)	2.1 (1.9)	6.4 (1.7)	2.3 (1.9)
Mean (SD)	n=378	n=295	n=177	n=135	n=556	n=431
Safety outcomes (SS), % (n)	AS (n=469)		nr-axSpA (n=201)		Overall axSpA (n=672)	
	BL	Wk52	BL	Wk52	BL	Wk52
Any AE	16.1 (17)	79.8 (101)	79.8 (101)	77.7 (121)	77.7 (121)	77.7 (121)
Serious AEs	6.2 (29)	6.3 (13)	6.3 (13)	6.3 (12)	6.3 (12)	6.3 (12)
Serious infections	1.5 (7)	2.5 (5)	2.5 (5)	1.8 (12)	2.5 (5)	1.8 (12)
Drug-related AEs	18.6 (87)	24.9 (32)	24.9 (32)	20.7 (139)	20.7 (139)	20.7 (139)

Observed data are presented. [a] includes 1 patient with unconfirmed AS/nr-axSpA classification; [b] CRP values ≥ 2 were reduced with 1 to complete ASDAS; [c] Measured using a numeric rating scale where 0 = 'no pain' and 10 = 'worst possible pain'; [d] adverse events; [e] including spondyloarthritis; [f] ASAS20/ASDAS20/ASDAS40 assessment of spondyloarthritis International Society 2004/ASIS response; [g] ASDAS; [h] Ankylosing Spondylitis Disease Activity Index; [i] ASDAS-SD; [j] ASDAS-SD; [k] ASDAS-SD; [l] ASDAS-SD; [m] ASDAS-SD; [n] ASDAS-SD; [o] ASDAS-SD; [p] ASDAS-SD; [q] ASDAS-SD; [r] ASDAS-SD; [s] ASDAS-SD; [t] ASDAS-SD; [u] ASDAS-SD; [v] ASDAS-SD; [w] ASDAS-SD; [x] ASDAS-SD; [y] ASDAS-SD; [z] ASDAS-SD; [aa] ASDAS-SD; [ab] ASDAS-SD; [ac] ASDAS-SD; [ad] ASDAS-SD; [ae] ASDAS-SD; [af] ASDAS-SD; [ag] ASDAS-SD; [ah] ASDAS-SD; [ai] ASDAS-SD; [aj] ASDAS-SD; [ak] ASDAS-SD; [al] ASDAS-SD; [am] ASDAS-SD; [an] ASDAS-SD; [ao] ASDAS-SD; [ap] ASDAS-SD; [aq] ASDAS-SD; [ar] ASDAS-SD; [as] ASDAS-SD; [at] ASDAS-SD; [au] ASDAS-SD; 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