SAT0095

A PERCEIVED BIOLOGICAL CAUSE OF RHEUMATOID ARTHRITIS ONSET IS ASSOCIATED WITH LOWER LEVELS OF DEPRESSED AND ANXIOUS MOOD OVER ONE YEAR COMPARED TO PATIENTS WHO ATTRIBUTED ONSET TO OTHER CAUSES

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Background: Patients with rheumatoid arthritis (RA) attribute onset of their condition to a range of causes including biological, psychological, work-related causes or stressful life events. Causal attributions are associated with a range of short- and long-term outcomes.

Objectives: To compare the 1 year outcomes of early RA patients who attribute a biological cause of onset of RA with patients who perceive non-biological causal attributions.

Methods: The Rheumatoid Arthritis Medication Study (RAMS) is a prospective cohort of patients with RA starting methotrexate for the first time in the United Kingdom. Demographic and clinical data (pain and fatigue visual analogue scales [VAS-pain/VAS-fatigue], disease activity [DAS28], the Hospital Anxiety and Depression Scale [HADS-A, HADS-D] and the Health Assessment Questionnaire [HAQ]) were collected at baseline and 1 year later. At baseline, patients completed the Brief Illness Perception Questionnaire (Brief IPQ), which assesses key beliefs about the impact, controllability, and chronicity of RA. The final item of the Brief IPQ asks patients to report the most important factor they believe caused their disease. These causal attributions were coded as either biological (e.g. age, genetics) or other causes (e.g. work related, stressful life event). Brief IPQ item response scores were compared between these groups using linear regression, adjusted for age and gender. HAQ, DAS28, VAS-pain, VAS-fatigue, HAD-A and HADS-D over follow-up were compared between the two groups using random effects models.

Results: Of 1171 patients, 483 (41.2%) reported a biological cause to be the most important factor in causing their arthritis and 688 (58.8%) reported a non-biological cause. Patients who believed in biological causes were younger (median 58 vs. 61 years; $p=0.003$) and more likely to be women (72.1% vs. 59.2%; $p<0.001$). Other baseline characteristics were similar (Table). Patients who perceived biological causes reported significantly lower concerns about their illness (median: 8 vs. 9; $p=0.004$) and significantly lower emotional impact (median: 5 vs. 6; $p<0.001$). Over follow-up, patients who attributed biological causes reported lower depression and anxiety HADS scores than those holding non-biological attributions (mean difference [95% CI]: HADS-D = -0.76 [-1.19, -0.33]; HADS-A = -0.94 [-1.41, -0.47]). Other outcomes over follow-up were similar.

Abstract SAT0095 – Table 1. Baseline and one year characteristics, stratified by perceived cause of patients' illness

Characteristic	Biological cause		Other cause		p†
	N	median(IQR)	N	median(IQR)	
Age, years	483	58 (48, 68)	688	61 (52, 69)	0.003
Female	348 (72.1)		407 (59.2)		<0.001
Disease duration at baseline, months	483	7 (4, 11)	688	6 (3, 11)	0.094
Swollen joint count					
Baseline	469	4 (2, 9)	650	4 (2, 9)	0.822
One year	301	1 (0, 3)	409	1 (0, 3)	0.527
Tender joint count					
Baseline	470	5 (2, 11)	651	5 (2, 11)	0.959
One year	301	2 (0, 5)	408	2 (0, 6)	0.7823
DAS28					
Baseline	457	4.2 (3.3, 5.1)	630	4.2 (3.3, 5.2)	0.690
One year	271	3.0 (2.3, 4.0)	386	2.8 (2.1, 4.0)	0.283
HAQ Score					
Baseline	481	1.00 (0.38, 1.50)	685	1.00 (0.50, 1.63)	0.602
One year	287	0.75 (0.13, 1.25)	376	0.63 (0.13, 1.25)	0.996
HADS-D					
Baseline	479	4 (2, 8)	682	6 (2, 9)	0.001
One year	285	3 (1, 7)	374	4 (1, 7)	0.071
HADS-A					
Baseline	479	5 (3, 5)	682	6 (3, 10)	0.010
One year	285	4 (2, 8)	372	5 (2, 8)	0.130

† Univariable analysis (Mann-Whitney U test)

DAS28 = Disease Activity Score (28), HADS-A = Hospital Anxiety and Depression Scale [anxiety], HADS-D = Hospital Anxiety and Depression Scale [depression], HAQ = Health Assessment Questionnaire

Conclusions: Different causal attributions of RA are associated with different outcomes and may suggest that education about RA causality could improve patient-centred outcomes; however, that would require further evaluation.

Disclosure of Interest: None declared

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SAT0096

WINDOW OR NO WINDOW: EARLIER IS BETTER WHEN TREATING RHEUMATOID ARTHRITIS

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Background: Previous reports on a window of opportunity in rheumatoid arthritis (RA) may be related to the use of slow acting c(onventional) s(ynthetic) DMARDs. We hypothesised that onset of action of therapy might influence whether there is a window of opportunity or whether 'earlier is just better'.

Objectives: To investigate the association between symptom duration at treatment onset and the achievement of sustained drug free remission (sDFR) in early RA patients initiating therapy including fast acting prednisone or infliximab, compared to patients initiating csDMARD monotherapy.

Methods: We analysed the shape (hyperbolic or linear) of the association between symptom duration and achievement of sDFR (DAS<1.6 and no DMARDs for ≥ 1 year) for patients initiating combination therapy in 3 cohorts: BeSt, IMPROVED study and METEOR. All patients had arthritis symptoms <2 years. In the BeSt study, early RA patients (1987 criteria) were randomised to 4 targeted treatment strategies: arm 1 and 2 started with csDMARD monotherapy, arm 3 with csDMARDs and tapered high dose prednisone and arm 4 with csDMARD and infliximab. Subsequent treatment adjustments aimed at DAS ≤ 2.4 . In the IMPROVED study early RA patients (2010 criteria) were treated with csDMARD and tapered high dose prednisone. Subsequent treatment adjustments aimed at DFR. METEOR is an international observational cohort including daily practice data from patients with a diagnosis and treatment of RA according to the rheumatologist. We selected patients who started on csDMARD monotherapy or a combination of csDMARD with prednisone or anti-TNF and at least 1.5 year follow-up.

Missing DAS were imputed using last observation carried forward. We performed Cox regression analyses with as outcome sDFR and as predictor symptom duration and compared the likelihood ratio tests of a linear model and a model with inclusion of natural cubic spline functions (resulting in a hyperbola), to investigate which model was a better fit for the data.

Results: In BeSt (n=469), IMPROVED (n=421) and METEOR (n=1653) 54, 110 and 10 patients who started with csDMARD and prednisone or anti-TNF combination therapy, and 53 in BeSt and 15 in METEOR who started with csDMARD monotherapy (no monotherapy in IMPROVED) achieved sDFR. A hyperbolic model did not show a better fit for the data than a linear model (for combination therapy in BeSt $p=0.743$, IMPROVED $p=0.337$, METEOR $p=0.608$, for csDMARD monotherapy in BeSt $p=0.609$, in METEOR $p=0.758$). Figure 1 shows the linear association between symptom duration and sDFR per study. These results indicate that the earlier treatment is started, the higher the likelihood of achieving sDFR. However, the data do not suggest that a hyperbolic relationship between treatment onset and outcome sDFR fit the data better.

Conclusions: Our data suggest that there is no evidence for a window of opportunity in early RA in 3 cohorts. This was not related to use of fast acting combination therapy including prednisone or anti-TNF instead of slow acting csDMARD monotherapy nor was it dependent on strict treatment to target in a clinical trial. It remains that earlier treatment initiation is better when aiming to achieve sDFR.