TABLE 1. Body composition parameter at baseline and after 6 months of treatment with a bDMARDs in patients with AS (n=77)

	Mean±SD	Difference, mean±SD	95% CI		P*
			Lower	Upper	
Weight at baseline, kg	77.19±15.71	0.75±3.80	0.04	1.47	0.04
BMI at baseline, kg/m ²	27.94±16.25	0.06	0.02	0.04	0.04
FMI at baseline, kg/m ²	0.30±1.29	0.06	0.02	0.04	0.04
FFMI at baseline, kg/m ²	0.54	0.01	0.02	0.04	0.04
SMM at baseline, kg	18.42±2.27	0.05	0.02	0.04	0.04
VAT at baseline, liters	-0.27	0.04	0.02	0.04	0.04
Weight at 6 months, kg	77.94±16.25	0.75±3.80	0.04	1.47	0.04
BMI at 6 months, kg/m ²	27.94±16.25	0.06	0.02	0.04	0.04
FMI at 6 months, kg/m ²	0.30±1.29	0.06	0.02	0.04	0.04
FFMI at 6 months, kg/m ²	0.54	0.01	0.02	0.04	0.04
SMM at 6 months, kg	18.42±2.27	0.05	0.02	0.04	0.04
VAT at 6 months, liters	-0.27	0.04	0.02	0.04	0.04

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FRI0293

EFFECTIVENESS OF SWITCHING BETWEEN TNF INHIBITORS IN PATIENTS WITH AXIAL SPONDYLOARTHRITIS: IS THE REASON TO SWITCH RELEVANT?

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Background: It has been common practice to start a second TNF inhibitor (TNFi) in patients with axial spondyloarthritis (axSpA) who discontinue their first TNFi. It remains unclear if the reason for discontinuation of the first TNFi influences the response to the second.

Objectives: To assess if the reason of discontinuation of the first TNFi influences the response to the second TNFi.

Methods: Patients with axSpA from the ReumaPt registry, who discontinued their first TNFi and started a second TNFi and who had complete data on Ankylosing Spondylitis Disease Activity Score (ASDAS) and Bath Ankylosing

Spondylitis Disease Activity Index (BASDAI) at baseline, 3 and 6 months for their first TNFi were included. Then, they were followed every 6 months up to 12 years. The main outcome was the ASDAS clinically important improvement (ASDAS CII). Secondary outcomes were ASDAS major important improvement (ASDAS MI); ASDAS low disease activity (ASDAS LDA); ASDAS inactive disease (ASDAS ID) and BASDAI 50. The reason for discontinuation of the first TNFi was defined as: i) Primary failure, ASDAS CII was not achieved at 3 or 6 months; ii) Secondary failure, ASDAS CII achieved at 3 or 6 months but lost in ≥1 follow-up visit; iii) Adverse events; iv) Other (e.g. pregnancy, surgery). The response to the first TNFi at 3 and 6 months was compared to the response to the second TNFi at the same visits, adjusting for age, gender and C-reactive protein (CRP). The association between the reason of discontinuation of the first TNFi and response to the second TNFi over time was tested in generalized estimating equations (GEE) models, adjusted for age, gender and CRP.

Results: In total, 193 patients (53% male, mean age 45 (SD:11) years) were included, with a median follow-up time on the second TNFi of 1.5 years. Patients had a lower response to the second TNFi compared to the first TNFi according to the main outcome (ASDAS CII) at 3 months (41% vs 51%) and 6 months (35% vs 56%). There was an association between the reason to discontinue the first TNFi and response to the second TNFi as defined by the most stringent outcomes (ASDAS MI and ASDAS ID), but not for ASDAS CII (Table). Compared to patients who discontinued their first TNFi due to primary failure, patients were more likely to achieve ASDAS ID with the second TNFi when they discontinued their first TNFi due to secondary failure (OR: 7.3 [(95%CI: 1.9; 27.7)], adverse events (OR: 9.1 [2.5; 33.3]), or other reasons (OR: 7.7 [1.6; 37.9]).

Conclusion: In axSpA, response to the second TNFi is worse compared to the first TNFi. Patients with a secondary failure to the first TNFi have a better response to the second TNFi compared to those discontinuing the first TNFi due to primary failure, particularly for most stringent outcomes.

Table. Association between the reason for discontinuation of the first TNFi and response to the second TNFi

Reason to discontinue first TNFi*	Outcome for the second TNFi				
	OR (95% CI)				
	ASDAS-CII (N=135)	ASDAS-MII (N=135)	ASDAS-LDA (N=166)	ASDAS-ID (N=166)	BASDAI50 (N=147)
(ref Primary failure)					
-Secondary failure	1.9 (0.7;4.8)	4.8 (1.3;18.2)	1.2 (0.6;2.4)	7.3 (1.9;27.7)	1.4 (0.6;3.0)
-Adverse events	1.5 (0.6;3.5)	2.4 (0.6;9.6)	0.9 (0.5;1.7)	9.1 (2.5;33.3)	1.1 (0.5;2.3)
-Other	1.0 (0.3;3.8)	1.7 (0.1;19.4)	1.0 (0.4;2.4)	7.7 (1.6;37.9)	0.5 (0.1;1.7)

*GEE models with the reason of discontinuation of the first TNFi as predictor (reference category: primary failure); all models adjusted for age, gender and C-reactive protein. OR in bold are statistically significant (p<0.05).

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FRI0294

MUSCULOSKELETAL MANIFESTATIONS IN PATIENTS WITH INFLAMMATORY BOWEL DISEASE TREATED WITH VEDOLIZUMAB

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Background: Musculoskeletal manifestations (MEM) are frequent extraintestinal symptoms in patients suffering from inflammatory bowel disease (IBD), affecting up to 40% of them. Tumor necrosis factor inhibitors (TNFi) are effective in both IBD and IBD-related spondylarthritis (SpA). Additionally, vedolizumab (VDZ), an $\alpha 4\beta 7$ integrin inhibitor with selective action on intestinal tissue, has been recently proposed as 1st line treatment on TNFi refractory IBD. The effectiveness of VDZ in MEM has not been properly evaluated but even exacerbation of previously diagnosed SpA has been described.

Objectives: The main objective is to analyse the occurrence of articular exacerbations in patients with IBD-related SpA treated with VDZ. The secondary objective is to analyse the new-onset MEM in IBD patients treated with VDZ.

Methods: Descriptive study of a retrospective cohort of every adult with IBD (Crohn's disease -CD- and ulcerative colitis -UC-) patients starting treatment with VDZ in a tertiary hospital. All data were collected as a collaboration between the Rheumatology and Gastroenterology Departments, through revision of the clinical history and databases from both departments. In patients previously diagnosed of SpA exacerbation was assessed, defined as a clinical worsening causing a treatment modification. The patients with new-onset MEM were classified as: i) nonspecific arthralgia (NSA), not suggestive of SpA; and ii) SpA according to ASAS criteria. A statistical analysis was performed using frequency charts

Results: A total of 61 patients were included, 55.7% women and with an mean (SD) age of 50 (17) years. The proportion of UC and CD was similar (49% and 51%, respectively). Among the patients studied, 12 (19.7%) had a diagnosis of IBD-related SpA and 3 (25%) of them suffered articular exacerbation of SpA within 3,5 and 6 months of treatment. On the other hand, 9 (14.7%) patients showed new-onset MEM, 3 (33%) of them showed symptoms and clinical and/or radiological findings compatible with axial SpA. In 2 of the cases a treatment with a cDMARD was used and the other one required a combination therapy between iTNF and VDZ. The remaining 6 (67%) patients were classified as NSA and inflammatory arthritis was discarded. Table 1 shows the demographic and clinical characteristics of patients included in the analysis.

Table 1. Demographic, clinical characteristics and symptoms onset in patients included in the study.

	Total (n=61)	Diagnosed SpA (n=12)		New-Onset MEM (n=9)	
		Stable (n=9)	Exacerbation (n=3)	New-onset SpA (n=3)	NSA (n=6)
Age (years), mean $\pm$ SD	50 $\pm$ 17	55 $\pm$ 19	50 $\pm$ 7.5	38 $\pm$ 13	50 $\pm$ 14
Gender (female), n (%)	34 (55.7%)	6 (66.7%)	3 (100%)	1 (33.3%)	3 (50%)
BMI (Kg/m ²), mean $\pm$ SD	24.7 $\pm$ 4.3	27.5 $\pm$ 5.5	28.3 $\pm$ 3.9	30.2 $\pm$ 0.5	24.3 $\pm$ 4.6
Smoking habit (smokers), n (%)	11 (18%)	1 (11.1%)	0 (0%)	2 (66.7%)	0 (0%)
CD diagnosis, n (%)	31 (50.8%)	2 (22.2%)	1 (33.3%)	3 (100%)	2 (33.3%)
UC diagnosis, n (%)	30 (49.2%)	7 (77.8%)	2 (66.7%)	0 (0%)	4 (66.7%)
IBD follow-up, (years) mean $\pm$ SD	11 $\pm$ 9.6	10.6 $\pm$ 10	2 (1-29) *	12 $\pm$ 6.8	9.8 $\pm$ 4.6
bDMARD naïve, n (%)	7 (11.5%)	2 (22.2%)	0 (0%)	0 (0%)	0 (0%)

*shown as median (range).

Conclusion: Switching TNFi treatment to VDZ in patients with IBD-related SpA was found to be associated with articular exacerbation of SpA in 1 out of 4 patients within the first 5 months. New-onset MEM is also observed in up to 15% of patients with IBD treated with VDZ. A multidisciplinary assessment of these patients is necessary in order to achieve a proper management of their diseases.

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FRI0295

ADALIMUMAB LEVELS ARE ASSOCIATED WITH DRUG RETENTION RATES IN AXIAL SPONDYLOARTHRITIS PATIENTS

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Background: Production of anti-drug antibodies (ADA) could cause low serum anti-TNF levels and low drug retention rates.

Objectives: To assess the relationship between serum adalimumab (ADL) levels and drug retention rates in patients with axial SpA.

Methods: Single-center prospective study in patients with axial SpA, according to ASAS criteria, being treated with ADL. In the first part of the study, from December 2010 to June 2016, data was collected and serum samples were taken. In a second part of the study, clinical records were reviewed to find out the dates and reasons of treatment discontinuation. Information was collected on age, gender, body mass index (BMI), date of diagnosis of SpA, laboratory data, including HLAB27, erythrocyte sedimentation rate (ESR) and C-reactive protein (CRP), prior biological treatment for SpA, concomitant conventional synthetic disease modifying anti-rheumatic drugs (csDMARDs). SpA disease activity was assessed by BASDAI, ASDAS and PGA scores. A serum sample was taken from all patients just before the next adalimumab dose. ADL and ADA levels were analysed with ELISA Promonitor®, (Proteomika, Progénica, Grifols) and correlated with SpA activity using BASDAI and ASDAS scores. Cox proportional hazards model was performed in order to assess the relation of variables with drug interruption.

Results: Up to January 2016, 51 consecutive patients were included. The mean (range) age was 46,9 (18–68) and 47.1% were women. HLAB27 was positive in 82,4% of patients. Mean disease duration was 122,9 months (2-408) and mean duration of treatment with adalimumab was 17,8 months (1-69). ADL was the first biological agent received in 36 patients (70.6%). Eleven patients (21.6%) were on concomitant treatment with receiving concomitant csDMARDs, mainly methotrexate (MTX) (15.7%) and sulfasalazine (5.9%). The mean (SD) activity scores were BASDAI 4,0 $\pm$ 2,3; ASDAS-PCR 2,1 $\pm$ 1,1 and ASDAS-VSG 2,1 $\pm$ 1,0.

ADA prevalence was 27.5%, with none detected in the 21.6% taking csDMARDs (p=0.021). ADL level was normal (>3mg/l) in 36 patients (70.6%), all without ADA. Fifteen patients (29.4%) had subtherapeutic ADL levels (<3mg/l), with ADA in 14 (93%).

Total ADL treatment time was 241,16 patient/years, and mean ADL treatment time was 4,73 years. Cox model results were resumed in table 1. Multivariate study show that ADL level > 3mg/L was a protective factor for ADL interruption (HR 0.01 (0.00-0.59, p=0.026), while previous etanercept treatment was a risk factor for ADL interruption 9.54 (1.23-74.08, p=0.031).

Table 1. Cox model.

		Continue	Discon= tinue	Univariate HR	Multivariate HR
ADL level	<3mg/L	13 (86,7%)	2 (13,3%)	0.05 (0.00-0.65,	0.01 (0.00-0.59,
	$\geq$ 3mg/L	28 (77,8%)	8 (22,2%)	p=0.022)	p=0.026)
Concomitant	Yes	8 (72,7%)	3 (27,3%)	0.56 (0.14-2.26,	0.28 (0.05-1.65,
DMARD	No	33 (82,5%)	7 (17,5%)	p=0.419)	p=0.159)
BMI	Normal	13 (86,7%)	2 (13,3%)	2.56 (0.54-12.23,	0.48 (0.02-9.75,
	Overweight/ obese	27 (77,1%)	8 (22,9%)	p=0.239)	p=0.634)
Age	Mean (SD)	46,4 (12,2)	48,7 (11,6)	0.93 (0.24-3.60,	0.60 (0.10-3.84,
				p=0.912)	p=0.594)
Gender	Men	19 (70,4%)	8 (29,6%)	0.43 (0.09-2.03,	0.58 (0.03-10.06,
	Women	22 (91,7%)	2 (8,3%)	p=0.283)	p=0.705)
Infliximab	Yes	4 (40,0%)	6 (60,0%)	3.31 (0.92-11.87,	1.37 (0.23-8.24,
previous	No	37(90,2%)	4 (9,8%)	p=0.066)	p=0.731)
Etanercept	Yes	2 (22,2%)	7 (77,7%)	8.17 (2.09-31.90,	9.54 (1.23-74.08,
	No	39 (92,9%)	3 (7,1%)	p=0.003)	p=0.031)

ADL: adalimumab; BMI: body mass index; DMARD: disease modifying antirheumatic drug; SD: standard deviation.