AB0217 NON MEDICAL SWITCH FROM ETANERCEPT ORIGINATOR TO BIOSIMILAR GP2015 IN PATIENTS WITH CHRONIC INFLAMMATORY ARTHROPATHIES

M. Colaci[1], M. L. Aprile[1], A. La Rosa[2], A. DI Maggio[2], L. Malatino[1].
[1]*Rheumatology Centre, Ospedale Cannizzaro, Clinical and Experimental Medicine, University of Catania, Catania, Italy*; [2]*Servizio di Farmacia Ospedaliera, Ospedale Cannizzaro, Catania, Italy*

Background: In the last decades, new biologic drugs were introduced for the treatment of chronic inflammatory arthropathies, progressively leading to a relevant increase of medical costs. However, the introduction of biosimilars (biologic molecules similar to branded drugs with expired patent) permitted to optimize the financial resources. The non-medical switch (NMS) is the switch from a biologic originator to a biosimilar agent for economic reasons only, on the basis of the substantial equivalence as regards efficacy and safety between originator and biosimilar drugs.
In literature, several evidences from clinical trials and registry studies showed that the switch from etanercept originator to biosimilar SB4 was safe [1]. Instead no sufficient data may be found regarding the biosimilar GP2015.
Objectives: We aimed to evaluate efficacy, safety, and retention rate in a series of patients with chronic arthritis treated with etanercept originator who underwent to NMS towards the ETN biosimilar GP2015.
Methods: From March to June 2020, all patient referred in our Centre affected by rheumatoid arthritis (RA), psoriatic arthritis (PA) and axial spondyloarthritis (axSpA) treated with etanercept originator and in remission/low disease activity for at least 6 months underwent to NMS. Data on disease activity (DAS28-PCR/CDAI/SDAI; DAPSA; BASDAI), eventual adverse events and causes of withdrawal of therapy were collected at 2, 4 and 6 months after the switch.
Results: We recruited 71 consecutive patients (M/F: 24/47; mean age 55,8± 11,1 years; 39 RA; 15 PA; 17 axSpA; mean duration therapy 7.3±3.8 years). Disease activity was unchanged for almost all patients after 6 months from the switch (median ΔDAS28-PCR/CDAI/SDAI: 0,1/0/0,5; median ΔDAPSA: 0; median ΔBASDAI: 0) Moreover, the 6-month retention rate was 97.2%. Only 2 patients (2.8%) switched back to the originator due to loss of efficacy in one case and adverse events in the second case (paraesthesia, headache, dizziness and worsening of arthralgia).
Conclusion: Our study confirmed that the NMS from ETN originator to GP2015 represents a safe practice that maintains the efficacy of the current treatment.
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AB0218 NON MEDICAL SWITCH FROM ADALIMUMAB ORIGINATOR TO THE BIOSIMILAR GP2017 IN PATIENTS AFFECTED BY CHRONIC ARTHRITIS

M. Colaci[1], M. L. Aprile[1], A. La Rosa[2], A. DI Maggio[2], L. Malatino[1].
[1]*Rheumatology Centre, Ospedale Cannizzaro, Clinical and Experimental Medicine, University of Catania, Catania, Italy*; [2]*Servizio di Farmacia Ospedaliera, Ospedale Cannizzaro, Catania, Italy*

Background: The non-medical switch (NMS) from originator drugs to biosimilars is a possible strategy adopted by rheumatologists in order to control the expansion of pharmaceutic costs for the treatment fo patients affected by chronic arthritis. To date, few real life data on the switch from adalimumab (ADA) originator to its biosimilars are available.
Objectives: We aimed to evaluate efficacy and safety in a single-Centre cohort of patients affected by chronic arthritis who switched from ADA originator to the biosimilar GP2017.
Methods: Patients affected by rheumatoid arthritis (RA), psoriatic arthritis (PA) and axial spondyloarthritis (axSpA) already on therapy with ADA originator in remission or low disease activity for at least 6 months were switched to GP2017 (March- June 2020). Data on disease activity (DAS28-PCR/CDAI/SDAI; DAPSA; BASDAI), eventual adverse events and causes of withdrawal of therapy were collected at 2, 4 and 6 months after the switch.
Results: 88 patients were enrolled (M/F 36/52; mean age 55.8 ± 12 years; 25 RA, 32 PA, 31 axSpA, mean duration of therapy 6.3 ± 3.8 years). No statistically significant difference was observed in median DAS28-PCR/CDAI/SDAI values at the baseline and after 6 months [1.03 (0.96-3.43) vs. 1.21 (0-3.7) / 0 (0-15) vs. 0 (0-17) / 0 (0-15) vs. 0.5 (0-17.5)], DAPSA [0 (0-12.2) vs. 0 (0-15)] and BASDAI [0 (0-4.3) vs. 0 (0-6.4)]. The retention rate was 93.2%. 6/88 patients (6.8%) switched back to the originator. The causes of discontinuation were: disease reactivation in a single case (1.1%), subjective reasons/ nocebo effect in 5/88 cases (5.7%), including: general malaise and transient increased of blood pressure (n.1), dizziness, paraesthesia, arthralgia, headache (n.1), itch *sine materia* (n.1) and subjective worsening without objective disease flare (n.2).
Conclusion: This is the first real life study showing that the NMS from ADA originator to GP2017 represents a safe practice that maintains the efficacy of the current treatment. However, a few cases of switchback were described, mainly attributed to nocebo effect [1].
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AB0219 EXPLORING LONG-TERM DRUG EFFECTIVENESS AND DRUG SURVIVAL FOR RITUXIMAB REFERENCE DRUG IN TREATMENT OF RHEUMATOID ARTHRITIS PATIENTS IN ORDINARY OUTPATIENT CLINIC

K. Łosińska[1], M. Wilk[1], A. H. Pripp[2], M. Korkosz[1,3], G. Haugeberg[4,5].
[1]*University Hospital, Division of Rheumatology and Immunology, Cracow, Poland*; [2]*Oslo University Hospital, Oslo Centre of Biostatistics and Epidemiology, Oslo, Norway*; [3]*Jagiellonian University Medical College, Department of Rheumatology, Cracow, Poland*; [4]*Sorlandet Hospital, Division of Rheumatology, Department of Medicine, Kristiansand, Norway*; [5]*NTNU, Norwegian University of Science and Technology, Department of Neuromedicine and Movement Science, Faculty of Medicine and Health Sciences, Trondheim, Norway*

Background: In randomized controlled trials (RCTs) rituximab (RTX) has been shown to effectively suppress inflammation and reduce structural joint damage in rheumatoid arthritis (RA) patients [1]. There is a lack of RA data on RTX effectiveness and drug survival when used in real life practice.
Objectives: To explore long-term drug effectiveness and drug survival for RTX used to treat RA patients in ordinary clinical practice.
Methods: The study population included RA patients treated between 2006 and 2020 with RTX at an outpatient clinic in Norway. Patients were monitored using recommended measures for disease activity and patient reported outcomes (PROs). Drug effectiveness was assessed with random intercept linear mixed models. Drug survival was described using Kaplan-Meier survival analysis. Baseline predictors of drug survival were assessed with multivariable Cox proportional hazard models.
Results: In database a total of 246 RA patients (females 74.8%) were identified to have been treated with RTX. At baseline mean (SD) age was 59.1 (13.5) years, disease duration 13.0 (10.2) years, RF positive 88.8%, ACPA positive 92.1%. Majority of patients had first cycle RTX dosage of 2000 mg (82.9%). At baseline patients currently using conventional synthetic disease modifying anti-rheumatic drugs (csDMARDs) were 51.5% (methotrexate 39.4%), prednisolone 73.6% and a total of 17.1% were biologic DMARDs (bDMARDs) naïve. In table 1 mean (SE) values for disease activity and PROs are shown for baseline and subsequent years after baseline for 5 years follow up.

Table 1. Changes in disease activity and PROs at baseline and subsequent 1-year periods

	Baseline N=246	1 year N=246	2 year N=204	3 year N=163	4 year N=130	5 year N=111
CRP (mg/L)	23.1 (2.1)	21.3 (1.6)	11.7 (0.9)	8.6 (1.4)	6.2 (0.7)	6.7 (0.9)
ESR (mm/hr)	32.1 (1.4)	31.1 (1.3)	22.3 (1.1)	17.6 (1.2)	14.3 (1.0)	13.6 (1.2)
SJC28 (0-28)	6.3 (0.4)	5.4 (0.3)	3.2 (0.3)	2.2 (0.2)	1.6 (0.2)	1.5 (0.2)
TJC28 (0-28)	7.1 (0.4)	6.6 (0.3)	3.6 (0.3)	2.6 (0.3)	2.2 (0.3)	1.8 (0.3)
DAS28	4.9 (0.1)	4.7 (0.1)	3.6 (0.1)	3.1 (0.1)	2.8 (0.1)	2.7 (0.1)
CDAI	22.9 (0.9)	20.7 (0.7)	12.3 (0.7)	9.4 (0.7)	8.5 (0.6)	7.7 (0.7)
PGA (0-100mm)	57.2 (1.7)	53.7 (1.4)	38.1 (1.6)	33.7 (1.9)	35.0 (2.1)	32.8 (2.2)
MHAQ (0-3)	1.0 (0.0)	0.9 (0.0)	0.7 (0.0)	0.6 (0.0)	0.5 (0.0)	0.5 (0.0)

During follow up all disease activity and PRO measures improved significantly ($p < 0.001$). Least improvement was seen in first year and a more substantial improvement progressed since second year. Percentage of patients with no, moderate and good treatment response defined according to EULAR response criteria [2] is shown in figure 1.