Conclusion: Real life experience from this nationwide TURKBIO registry show higher long-term retention rate of CZP in ax-SpA. Higher baseline disease activity and functional limitation predict discontinuation of CZP. Adding NSAIDs and csDMARDs to the treatment of the patient with poor prognosis cannot increase retention rates.

Figure 1 Drug survival of CZP in patients with Ax-SpA

Table 1. Baseline characteristics of ax-SpA patients who continue and discontinue CZP

	All patients (n=410)	Continue to CZP (n=318)	Discontinue to CZP* (n=54)	p
Females, n (%)	185 (49,7)	157 (49,4)	28 (51,9)	0,736
Age, years	42 (34-49)	41 (34-49)	45 (34-54)	0,064
Symptom duration, years	11 (7-17)	11 (6-16)	12 (8,5-20)	0,054
HLA-B27, n (%)	150 (63,8)	129 (64,5)	21 (60)	0,609
Previous bDMARDs, n (%)				
Adalimumab	54 (14,5)	42 (13,2)	12 (22,2)	0,082
Etanercept	53 (14,2)	40 (12,6)	13 (24,1)	0,025
Golimumab	11 (3)	7 (2,2)	4 (7,4)	0,060
Infliximab	39 (10,5)	35 (11)	4 (7,4)	0,425
Co-treated drugs, NSAID n (%)	206 (55,4)	169 (53,1)	37 (68,5)	0,036
Methotrexate	35 (9,4)	22 (6,9)	13 (24,1)	<0,001
Sulphasalazine	61 (16,4)	40 (12,6)	21 (38,9)	<0,001
Leflunomide	5 (1,3)	2 (0,6)	3 (5,6)	0,023
ESH, mm/h	21,5 (10-37)	21 (10-37)	23 (10-34)	0,999
CRP, mg/dl	7 (3-20)	7 (3-20)	7 (3-22)	0,727
HAQ	0,63 (0,25-0,94)	0,5 (0,25-0,88)	0,75 (0,38-1,25)	0,009
BASFI	21 (7-45)	20,5 (6-41)	31 (13-58)	0,011
BASDAI	30,5 (13-52)	30 (12-50)	43 (23-61,5)	0,002
ASDAS	2,7 (1,8-3,7)	2,7 (1,8-3,6)	2,9 (2,3-4)	0,062

*Discontinue due to ineffectivity.

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Disclosure of Interests: None declared.

DOI: 10.1136/annrheumdis-2021-eular.3140

AB0480

INFLUENCE OF ANTI TNF THERAPIES AND HYPERHOMOCYSTEINEMIA ON BONE MINERAL DENSITY IN ANKILOSING SPONDYLITIS PATIENTS

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Background: Ankylosing spondylitis (AS) is a chronic inflammatory disease associated with an important risk factor of osteoporosis. A high serum level of Homocysteine (Hcy) has been recently recognized as a risk factor for osteoporosis and osteoporotic fractures. Treatment with TNF- α blockers might influence bone metabolism and prevent structural bone damage in AS.

Objectives: The aim of the study was to evaluate the influence of anti-TNF therapies and hyperhomocysteinemia on bone mineral density in AS patients.

Methods: In our retrospective study were enrolled 73 AS patients with anti TNF- α therapies (29 Adalimumab 40mg/2weeks, 24 Etanerceptum50mg/week, 20 Infliximabum5mg/kg/8 weeks) and 45 AS patients non anti TNF- α therapies. All patients included in this study met the modified New York criteria. The current medications used by the non anti TNF- α therapies patients were nonsteroidal anti-inflammatory drugs.

We used Dual energy x ray absorptiometry (DXA) method for measuring bone mineral density (BMD). Osteoporosis is defined as a T score of ≤ -2.5 SD, according to the WHO criteria. BMD of the hip and lumbar spine (L1-L4) was determined at baseline and after 6 months. Hcy concentrations were measured by using a chemiluminescence immunoassay method. The normal serum Hcy level is considered to be below 15mmol/L. In both group we followed the activities disease (C reactive protein-CRP, erythrocytes sedimentation rate-ESR, Bath ankylosing spondylitis disease activity index-BASDAI score) and determined vitamin D3 level.

All parameters were determined at baseline and after 6 months.

Results: The mean age of study group1 was 47.4 ± 11.1 years and 42 ± 9.8 year in group2. The mean disease duration was 11.9 ± 6.1 years. 83,5% of patients were HLA-B27 positive in group1 and 71,1% in group2.

BMD measurements revealed more severe osteoporosis in patients with positive HLA-B27 in both groups at baseline. After 6 months in group1 BMD increased by 0.4% at lumbar spine and 0.1% in the hip and in group2 decreased by 0.9% and 0.6% in the same sites. No significant differences between type of anti-TNF therapies and BMD. In group 1, HLA-B27 positive status was positive correlated with decrease BMD ($p=0.001$) after 6 months. Hcy levels were found significantly increased (14.8 ± 3.8 mg/mL vs. 12.4 ± 4.2 mg/mL; $P < 0.001$) at baseline in both groups. Serum levels of Hcy were inversely correlated to lumbar spine BMD ($p < 0.005$) and femur neck BMD ($p=0.001$) after 6 months in group1. At baseline the mean level of 25-hydroxyvitamin D₃ was 22.8 ± 14.1 ng/mL in group1 and 26.6 ± 12.5 ng/mL in group2. In anti TNF patients group, we found a positive correlation between decrease BMD, low level of 25-hydroxyvitamin D₃ and high level of Hcy ($p=0.001$) after six months. No correlation was found between BMD and BASDAI score and inflammatory parameters (ESR, CRP).

Conclusion: Bone mineral density measurements revealed more severe osteoporosis in positive HLA-B27 AS patients and hyperhomocysteinemia. Association of low level of 25-hydroxyvitamin D₃ and hiperhomocysteinemia is correlated with high-risk fracture in positive HLA-B27 patients. Anti TNF therapies reduce this risk.

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Disclosure of Interests: None declared.

DOI: 10.1136/annrheumdis-2021-eular.3523