prescribed at the onset of the disease were more often TNFα blockers in gr C and rituximab in gr S.
Conclusion: According to our observations, the combination of RA with SjS leads to a milder course of the RA. Early diagnosis of SjS in RA pts is important for understanding the course of the disease and determining the prognosis.
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POS0629 PREVALENCE AND FACTORS ASSOCIATED WITH SARCOPENIA IN PATIENTS WITH RHEUMATOID ARTHRITIS

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Background: Sarcopenia is a progressive and generalized skeletal muscle loss associated with falls, fractures, physical disability, and mortality, described as age-related or secondary. Systemic inflammatory diseases, such as rheumatoid arthritis (RA), are well-known causes of secondary sarcopenia. However, the exact prevalence of sarcopenia in patients with RA is still unknown, partly due to the heterogeneous definitions of sarcopenia adopted in different studies.
Objectives: To assess the prevalence of sarcopenia in a cohort of patients affected by RA, and to evaluate the influence of age, sex, comorbidity, disease duration and activity, antibody status and therapies on sarcopenia.
Methods: We conducted a retrospective observational study on adult patients affected by RA undergoing evaluation at our outpatient clinic from January 2009 and July 2021. All patients underwent dual-energy X-ray absorptiometry (DXA) for assessment of body composition; diagnosis of sarcopenia was defined using Skeletal Muscle Mass Index (SMI), as proposed by consensus EWGSOP2 (1). We collected relevant demographic, clinical, therapeutic, and laboratory data at the time of DXA. We excluded patients affected by neoplastic disorders and/or malnutrition. Binary logistic regression analysis was employed to define predictors and protective factors of developing sarcopenia.
Results: A total of 266 patients (82.7% women) with a median age of 58.4 (IQR 14.4) years were included in the study. The prevalence of sarcopenia was 27.44%. From the binary logistic regression analysis, we found that the use of oral glucocorticoids (GCs) at a daily dose > 3.25 mg of prednisone-equivalent was significantly associated with sarcopenia (β 0.68, p = 0.047, aOR 1.98, 95% CI 1.009 – 3.881) (Figure 1). We found a significant inverse correlation between conventional disease-modifying antirheumatic drug (c-DMARDs) and sarcopenia (β -0.71, p = 0.027) as well. Age, sex, disease duration, mean disease activity - expressed as disease activity score based on 28 joints (DAS-28), erosive and seropositive disease - and biologic disease-modifying antirheumatic drug (b-DMARDs) therapy were not predictors of sarcopenia, albeit seropositive status showed a correlation trend with increased prevalence of sarcopenia (p = 0.092).

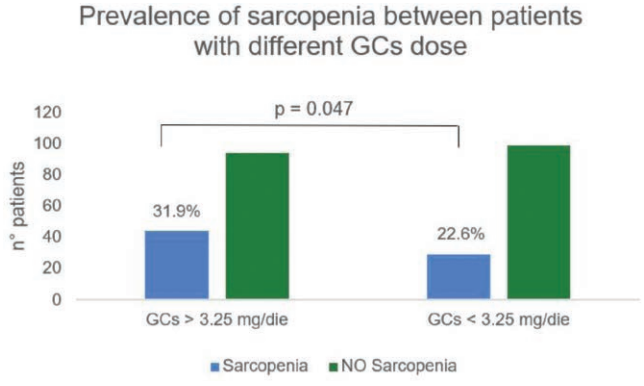


Figure 1.

Conclusion: Our study showed that sarcopenia is a common complication in RA. Glucocorticoid therapy was associated with an increased prevalence of sarcopenia, while c-DMARDs acted as protective factors, possibly decreasing chronic inflammation. No correlation was found with b-DMARDs, possibly due to association with longer and more aggressive rheumatic disease.

REFERENCES:
[1] Cruz-Jentoft AJ, Bahat G, Bauer J, Boirie Y, Bruyère O, Cederholm T, et al. Sarcopenia: revised European consensus on definition and diagnosis. Age Ageing. 2019;48:16–31
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POS0630 INCIDENCE AND ASSOCIATED FACTORS OF CANCER IN PATIENTS WITH RHEUMATOID ARTHRITIS

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Background: Rheumatoid arthritis (RA) is an autoimmune disease primarily characterized by joint inflammation along with systemic manifestations and increased risk of cardiovascular disease. Furthermore, it has been suggested that RA itself and medications administered might increase the risk of cancer.
Objectives: To assess cancer incidence in RA patients and factors associated with cancer development in RA patients.
Methods: In this single centre retrospective cohort study, the data of 2000 patients (76.3% female, mean age at the last follow-up: 59.5 ± 13.4 years) who were followed up between 2005 and 2020 and fulfilled 2010 ACR/EULAR classification criteria were scrutinised. Patients' demographic, clinical and serological characteristics, and smoking history were recorded. Multivariable logistic regression analysis was performed to determine the associated factors with cancer development. The standardized incidence rate (SIR) was calculated by dividing the number of cancer cases observed in a given age and gender group by the expected number.
Results: After a median follow-up of 9 years, 71 (3.6%) patients were diagnosed with cancer and the number of patients with more than one malignancy was 5 (0.5%). Cancer types in each sex were demonstrated in Table 1. Seropositivity rate of the cohort was 86.0% and 376 out of 1102 (34.1%) patients were smokers. Glucocorticoids were the most common used agent in the treatment of RA (75.2%), followed by methotrexate (68%), hydroxychloroquine (65.1%), sulfasalazine (37.7%), leflunomide (35.5%), biological disease-modifying antirheumatic drugs (11.4%) and tofacitinib 4%. In multivariable logistic regression analysis performed of 1102 patients without missing data of smoking history, only smoking and age were independently associated with malignancy (Odds Ratios were 2.11 (95% Confidence Interval (CI) 1.07-4.14 and 1.05 (95% CI 1.02-1.07), respectively). Treatment modalities and RA-related clinical factors were not related to cancer development (data not shown). When compared with the 2017 Cancer Statistics of Turkey, the incidence of malignancy in our RA cohort was similar to the corresponding age and gender groups. The SIRs in male and female patients were calculated as 0.93 (95% CI: 0.82-1.05 p= 0.25) and 0.92 (95% CI: 0.77-1.09 p= 0.34), respectively.

Table 1. Number of malignancy distribution by gender of patients

	Female	Male
	n=47	n= 24
Breast, n (%)*	15 (100.0)	0 (0.0)
Urogenital system, n (%)*	8 (53.4)	7 (46.6)
Prostate, n (%)*	0 (0.0)	5 (100.0)
Lung, n (%)*	3 (30.0)	7 (70.0)
Lymphoma, n (%)*	4 (57.1)	3 (42.9)
Thyroid, n (%)*	6 (100.0)	0 (0.0)
Skin, n (%)*	5 (83.3)	1 (16.7)
Colorectal, n (%)*	2 (50.0)	2 (50.0)
Head and Neck, n (%)*	0 (0.0)	3 (100.0)
Multipl Myeloma, n (%)*	1 (50.0)	1(50.0)
Others, n (%)*	7 (87.5)	1 (12.5)

*Row Percent

Conclusion: Our results show that patients with RA do not require additional screening beyond the nationally recommended guidelines. Medications used, including tumor necrosis factor inhibitors, did not increase the risk of cancer. In conclusion, all patients with RA should receive standard age- and sex-appropriate cancer screening.
REFERENCES:
[1] McInnes IB, G. Schett, The pathogenesis of rheumatoid arthritis. N Engl J Med 2011; 365: 2205-19.

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[2] De Cock D, Hyrich K. Malignancy and rheumatoid arthritis: Epidemiology, risk factors and management. Best Practice & Research Clinical Rheumatology. 2018;32(6):869-86.

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POS0631 EVEN LOW-DOSE GLUCOCORTICOID USE IS A RISK FACTOR FOR CLINICAL FRACTURES IN PATIENTS WITH RHEUMATOID ARTHRITIS: TEN-YEAR FINDINGS OF THE TOMORROW STUDY

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Background: Patients with rheumatoid arthritis (RA) who have sarcopenia and stiff or painful joints might be at increased risk of falls and fractures.
Objectives: The present study aimed to prospectively identify the incidence of clinical fractures and associated risk factors in patients with RA in a cohort study named the TOMORROW (UMIN000003876) that started in 2010.
Methods: We evaluated anthropometric parameters, bone mineral density (BMD), disease activity, RA medication at entry and observed the incidence of clinical fractures during ten years in 202 patients with RA (mean age, 58.6 y; medication with biological agents, 54.9%) and 202 age- and sex-matched non-RA volunteers (Vo) (mean age, 57.4 y). We compared the incidence of clinical fractures between patients with RA and Vo for ten years, and analyzed the risk factors for clinical fractures using Cox proportional hazard model.
Results: The incidences of clinical fractures were 0.036 and 0.024/person-year in patients with RA and Vo, respectively. Cox proportional hazard model revealed that low BMD at the thoracic vertebrae (< 0.7 g/cm^2) and history of fractures at entry were significantly associated with the incidence of clinical fractures (Hazard ratio [HR]1.737, p=0.020 and HR1.514, p=0.047, respectively) in all participants. RA morbidity, however, was not (HR1.398, p=0.112). In patients with RA, medication with GC at entry was a significant risk factor for clinical fractures (HR1.898, p=0.017). Additionally, a mean GC dose (≥ 2 mg/day) at entry and during the ten-year period increased risk for fractures (HR 2.189, p=0.004, 1.866, p=0.022, respectively).
Conclusion: RA per se was not a risk factor for clinical fractures in this cohort. Low BMD at the thoracic vertebrae at entry and the use of GC with even low dose at entry and during ten years were significantly associated with an increased frequency of clinical fractures among patients with RA.
Disclosure of Interests: None declared
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POS0632 EXPRESSION OF PD-1 AND PD-L1 IN DIFFUSE LARGE B-CELL LYMPHOMA PATIENTS WITH PRE-EXISTING RHEUMATOID ARTHRITIS – A POSSIBLE ASSOCIATION WITH HIGH RHEUMATOID ARTHRITIS DISEASE ACTIVITY

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Background: Patients with rheumatoid arthritis (RA) are at increased risk of developing diffuse large B-cell lymphoma (DLBCL). Immunotherapy blocking programmed cell death protein 1 (PD-1) and its ligand PD-L1 has substantially improved outcome in a number of malignant diseases but has so far been disappointing in DLBCL. However, current research seeks to identify DLBCL subgroups responsive to PD-1 blocking agents. Whether patients with pre-existing rheumatoid arthritis (RA) might constitute such a subgroup is unknown. Expression of PD-1 and PD-L1 has not previously been studied in a cohort of RA-DLBCL patients.
Objectives: To determine intratumoural expression of PD-1 and PD-L1 in DLBCL patients with pre-existing RA and relate the expression to clinical characteristics and survival outcome.
Methods: We included 103 RA-DLBCL patients and 74 DLBCL controls without rheumatic disease. PD-1 and PD-L1 immunohistochemical markers were applied on tumour tissue microarrays. The number of PD-1+ tumour infiltrating leukocytes (TILs) and proportions of PD-L1+ tumour cells and TILs were calculated and correlated to clinical data. For RA patients, disease activity had been individually scored repeatedly over time based on swollen and tender joint counts, erythrocyte sedimentation rate (ESR), and the treating physician's global assessment of disease activity from onset of RA until lymphoma diagnosis(1). Values of cumulative disease activity for each patient were calculated as area under the curve (AUC). To investigate a possible influence of RA disease severity on the expression of PD-1 and PD-L1, RA patients with DLBCL were divided in two groups using the upper quartile of the AUC values as cut off for the highest disease activity group.
Results: Expression of PD-1 in TILs and PD-L1 in tumour cells was similar in RA-DLBCL and DLBCL controls. In RA-DLBCL, high expression of PD-L1 in tumour cells was significantly more common in patients with the most severe RA disease (45% vs 20%, p=0.04) (Table 1) and was associated with inferior overall survival in multivariable analysis (HR 4.62, 95% CI, 1.55-13.71).

Table 1. Clinical and lymphoma related parameters and their distribution in the RA-DLBCL patients according to high and low expression of PD-L1 in tumour cells

	Entire cohort	High PD-L1 in tumour cells[1]	Low PD-L1 in tumour cells	P-value*
All patients, n (%)	**103 (100)**	**11 (100)**	**89 (100)**	
Women, n (%)	**61 (59)**	**6 (55)**	**54 (61)**	**0.70**
Age ≥60, n (%)	**85 (83)**	**10 (91)**	**72(81)**	**0.68**
DLBCL subtype, n (%)				**0.35**
GCB	**30 (29)**	**2 (18)**	**27 (30)**	
Non-GCB	**69 (67)**	**9 (82)**	**57 (64)**	
Missing	**4 (4)**	**0 (0)**	**5 (6)**	
Ann Arbor stage III-IV, n (%)	**65 (63)**	**5 (45)**	**57 (64)**	**0.31**
Missing	**4 (4)**	**1 (10)**	**3 (3)**	
EBV status, n (%)				**<0.001**
Positive	**9 (9)**	**4 (36)**	**5 (6)**	
Missing	**2 (2)**	**0 (0)**	**2 (2)**	
RA highest disease severity group, n (%)				**0.04**
Yes	**25 (25)**	**5 (45)**	**18 (20)**	
Missing	**4 (4)**	**1 (10)**	**3 (3)**	
Active RA treatment, n (%)†				**0.96**
Yes	**39 (38)**	**4 (36)**	**34 (38)**	
Missing	**3 (3)**	**1 (10)**	**2 (2)**	

RA, rheumatoid arthritis; DLBCL, diffuse large B-cell lymphoma; GCB, germinal centre B-cell like;EBV, Epstein-Barr virus; DMARD, disease modifying anti-rheumatic drug; TIL, tumour-infiltrating leukocytes; [1] High expression ≥17% PD-L1+ tumour cells of all tumour cells; *According to chi-square or Fischer's exact test; †Active RA treatment= DMARD and/or corticosteroids;

Conclusion: Our results indicate an association between a more severe RA disease and increased expression of PD-L1 in DLBCL tumour cells. This might have implications for the prognosis of the lymphoma as well as the chance of response to immunotherapy and warrants further studies also in other chronic inflammatory condidions.
REFERENCES:
[1] Baecklund E, Iliadou A, Askling J, Ekbom A, Backlin C, Granath F, et al. Association of chronic inflammation, not its treatment, with increased lymphoma risk in rheumatoid arthritis. Arthritis Rheum. 2006;54(3):692-701.
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POS0633 HIGH SENSITIVITY TROPONIN I IS ASSOCIATED WITH FOCAL FIBROSIS AS DETECTED BY CARDIAC MR IN EARLY, TREATMENT-NAIVE RA

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Background: RA individuals are known to have excess CVD risk, equivalent to that of type 2 diabetes mellitus[1]. We recently reported vascular and

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