



What are the Anti-Rheumatic Drug Prescribing Patterns in Rheumatoid Arthritis Patients With Chronic Kidney Disease?: A Single-Center Cross-Sectional Study

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Abstract

Aim: This study evaluated the distribution of anti-rheumatic drug treatments in rheumatoid arthritis (RA) patients with chronic kidney disease (CKD) across different renal stages.

Material and Method: A cross-sectional analysis included 72 RA patients with CKD (estimated glomerular filtration rate <60 mL/min/1.73 m² for >3 months). Demographic characteristics, disease duration, laboratory results, current RA medications, and renal replacement therapy status were recorded. Additionally, the presence of extra-articular manifestations, comorbidities (including hypertension, diabetes mellitus, hyperlipidemia, coronary artery disease, cerebrovascular disease, osteoporosis, and malignancy were obtained from the electronic patient files), history of prior infections, underlying etiology of CKD, and availability of renal biopsy reports were retrospectively extracted from hospital electronic medical records. Patients were stratified by CKD renal stage (3, 4, 5).

Results: Mean age was 66.7±11.8 years; 73.6% were female. Hypertension (84.7%) and diabetes (33.3%) were prevalent comorbidities. CKD etiology was undetermined in 65% of patients. Overall, 97.2% received conventional synthetic disease-modifying antirheumatic drugs (csDMARDs), 12.5% biologic disease-modifying antirheumatic drugs (bDMARDs), and 51.4% glucocorticoids. Hydroxychloroquine was the most common csDMARDs (76.4%), while methotrexate use differed significantly by CKD renal stage (stage 3: 27.7%; stage 4: 9.1%; stage 5: 0%; p=0.028). Among bDMARDs, rituximab (stage 3 only), TNF inhibitors (all stages), and tocilizumab (stage 4) were used. Etanercept was preferred in dialysis-dependent patients.

Conclusion: CKD stage significantly influences RA treatment selection. Methotrexate is avoided in stage 5 CKD, while hydroxychloroquine remains the predominant csDMARD. Leflunomide and sulfasalazine use in advanced CKD exceeds prior reports. Individualized therapy, adjusted for renal function and comorbidities, is essential. Larger prospective studies are needed to validate these findings.

Keywords: Antirheumatic agents, chronic renal insufficiency, rheumatoid arthritis

INTRODUCTION

Rheumatoid arthritis (RA) is a chronic, progressive inflammatory disease characterized by articular and extra-articular involvement. RA is one of the most common systemic inflammatory diseases, affecting 0.6–1.0% of the general population (1). Its prevalence in Türkiye is 0.56% (2). The most prevalent comorbidity in RA patients is cardiovascular disease (3). Although determining the precise prevalence of chronic kidney disease (CKD) in RA patients can be challenging, CKD represents a substantial

proportion, affecting approximately 10-30% of patients (4-6). Nephropathy in RA can stem from drug toxicity (e.g., nonsteroidal anti-inflammatory drugs (NSAIDs), methotrexate, cyclosporin), secondary renal amyloidosis, or various glomerulonephritis (GN). Numerous studies have identified RA as a risk factor for reduced estimated glomerular filtration rate (eGFR) (5,7).

RA and CKD are chronic conditions that can mutually influence each other. While drugs used to treat RA can adversely affect the kidneys, the presence of CKD can

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complicate RA management. Since the metabolism and excretion processes of some medications used in RA treatment are renal, dose adjustments or alternative therapies may be necessary in this patient group. Therefore, regular patient monitoring and personalized treatment plans are critical. Controlling disease activity may prevent chronic inflammation from leading to kidney damage. Some studies suggest that treatments reduce the risk of CKD development (8).

Recent academic studies evaluating rheumatologic medications used in RA patients with CKD have particularly focused on the safety and efficacy of biological therapies (9-11). In this study, we aimed to evaluate not only biological therapies but all anti-rheumatic drug treatment preferences and their distribution according to renal stages.

MATERIAL AND METHOD

This cross-sectional study included 72 consecutive RA patients with CKD admitted to the Rheumatology Outpatient Clinic of Ankara Bilkent City Hospital between November 2022 and November 2023. All patients fulfilled the 2010 American College of Rheumatology/European League Against Rheumatism (ACR/EULAR) classification criteria for RA (12). Exclusion criteria were defined as concurrent inflammatory rheumatic diseases, pregnancy, lactation and advanced liver failure that could affect our drug prescribing. Demographic characteristics, disease duration, laboratory results, current RA medications, and renal replacement therapy status were recorded. Additionally, the presence of extra-articular manifestations, comorbidities (including hypertension, diabetes mellitus, hyperlipidemia, coronary artery disease, cerebrovascular disease, osteoporosis, and malignancy were obtained from the electronic patient files), history of prior infections, underlying etiology of CKD, and availability of renal biopsy reports were retrospectively extracted from hospital electronic medical records.

Recorded laboratory values included: Rheumatoid factor (RF), anti-cyclic citrullinated peptide (anti-CCP) antibody, serum creatinine, eGFR, urine protein-to-creatinine ratio (UPCR).

Kidney function was evaluated using the eGFR (with the CKD-EPI formula). Patients with persistently reduced eGFR (<60 mL/min/1.73 m²) for >3 months were classified as having CKD, staged according to KDIGO guidelines as follows (13):

- **Stage 3:** eGFR 30–59 mL/min/1.73m² (moderate reduction)
- **Stage 4:** eGFR 15–29 mL/min/1.73m² (severe reduction)
- **Stage 5:** eGFR <15 mL/min/1.73m² (kidney failure)

Patients infection history was evaluated. Infections requiring hospitalization were classified as major infections, while those managed in outpatient settings were recorded as minor infections.

The data were analysed using the SPSS (version 22.0, IBM Corp., Armonk, NY, USA). The conformity of the numerical data to normal distribution was assessed

by both visual (Histogram and Detrended Q-Q charts) and analytical (Shapiro-Wilk or Kolmogorov-Smirnov tests) methods. Normally distributed numerical data were presented as mean±standard deviation, while non-normally distributed numerical data were presented as median (interquartile range). Categorical variables were presented as number (percentage). Chi-square tests were used to compare nonparametric data. p<0.05 was considered statistically significant.

The study was approved by the Ankara Bilkent City Hospital ethics committee (Approval no: E1-22-2932, Date: October 5, 2022) and was conducted in accordance with the Declaration of Helsinki.

RESULTS

This study included 72 RA patients with CKD. The mean age was 66.74±11.80 years, and 73.6% (n=53) were female. The median disease duration was 13 years. RF and/or anti-CCP antibody positivity was present in 66.7% (n=48) of patients. Extra-articular involvement was observed in 11 patients (15.3%), comprising pulmonary involvement in 8 patients and cutaneous rheumatoid nodules in 3 patients.

The most prevalent comorbidities were hypertension (84.7%), diabetes mellitus (33.3%), hyperlipidemia (30.6%), coronary artery disease (30.6%), and osteoporosis (29.2%). Malignancy was present in one patient, who had dual malignancies (cervical and pancreatic cancer). A history of infection was reported in 36.1% of patients, the majority of which did not require hospitalization (Table 1).

Table 1. Characteristics of RA patients with chronic kidney disease	
Characteristic	
Age, years, mean (±SD)	66.74±11.80
Gender, Female, n (%)	53 (73.6)
BMI, kg/m², mean (±SD)	27.57±5.73
Disease duration (years), median (IQR)	13 (17)
Presence of autoantibodies, n (%)	
RF+ and Anti CCP+	32 (44.4)
RF or Anti CCP +	16 (22.3)
RF- and Anti CCP-	24 (33.3)
Extra-articular involvement, n (%)	11 (15.3)
Comorbidities, n (%)	
Hypertension	61 (84.7)
Diabetes mellitus	24 (33.3)
Hyperlipidemia	22 (30.6)
Coronary artery disease	22 (30.6)
Cerebrovascular disease	2 (2.8)
Osteoporosis	21 (29.2)
Malignancy	1 (1.4)
History of infection, n (%)	
Major	6 (8.3)
Minor	20 (27.8)
RA: rheumatoid arthritis, SD: standard deviation, BMI: body mass index, IQR: interquartile range, RF: rheumatoid factor, Anti CCP: anti-cyclic citrullinated peptide	

Laboratory parameters related to CKD are presented in Table 2. Regarding CKD etiology, the underlying renal cause was undetermined in the majority of patients. Among the known etiologies, diabetes and/or hypertension constituted the largest proportion. Other identified causes included nephrectomy due to calculi, hydatid cyst, NSAIDs associated nephritis, IgA nephropathy, post-infectious GN, membranous GN, minimal change disease, polycystic kidney disease, congenital solitary kidney, and renal amyloidosis secondary to long-term uncontrolled RA activity.

Concerning treatment, 97.2% of patients were receiving conventional synthetic disease-modifying antirheumatic drugs (csDMARDs), 12.5% biologic disease-modifying antirheumatic drugs (bDMARDs), and 51.4% glucocorticoids. Hydroxychloroquine was the most frequently used csDMARD, while methotrexate was the least used. Among bDMARDs, the most commonly used agents, in descending order of frequency, were rituximab, tumor necrosis factor inhibitors (TNFi), and tocilizumab. TNFi agents administered were etanercept (2 patients) and adalimumab (1 patient) (Table 2).

Table 2. Chronic kidney disease characteristics and medication preferences	
eGFR (ml/min/1.73m ²)	41 (29)
Serum creatinine (mg/dL)	1.47 (1.5)
UPCR (mg/g kreatinin)	395 (831)
Renal replacement therapy, n (%)	16 (22.2)
CKD etiology, n (%)	
Unknow	47 (65.3)
Diabetic and/or hypertensive	15 (20.8)
Nephrectomy	2 (2.8)
Amyloidosis	1 (1.4)
Others	7 (9.7)
Renal biopsy, n (%)	13 (18.1)
csDMARDs, n (%)	70 (97.2)
Methotrexate	14 (19.4)
Leflunomide	35 (48.6)
Sulfasalazine	20 (27.8)
Hydroxychloroquine	44 (61.1)
bDMARDs, n (%)	9 (12.5)
TNFi	3 (4.2)
Tocilizumab	2 (2.8)
Rituximab	4 (5.5)
GCs, n (%)	37 (51.4)

The results are presented in the form of median (Interquartile Range), or n (%); eGFR: estimated glomerular filtration rate, UPCR: urine protein-to-creatinine ratio, CKD: chronic kidney disease, csDMARDs: conventional synthetic disease- modifying antirheumatic drugs, bDMARDs: biological disease- modifying antirheumatic drugs, TNFi: tumor necrosis factor inhibitor, GCs: glucocorticoids

When patients were stratified into 3 groups based on CKD stage (stage 3, 4, 5), no significant differences were observed in the overall use of csDMARDs, bDMARDs, or

glucocorticoids between groups. However, a significant difference was found in methotrexate use (stage 3: 27.7%; stage 4: 9.1%; stage 5: 0%; p=0.028). Stage 3 patients most frequently received hydroxychloroquine and glucocorticoids, stage 4 patients received hydroxychloroquine and leflunomide, and stage 5 patients received hydroxychloroquine and leflunomide. methotrexate was not administered to any stage 5 patients. bDMARDs were used in 27.3% (3/11) of Stage 4 patients, compared to 10.6% (5/47) in Stage 3 and 7.1% (1/14) in Stage 5. TNFi use was equally distributed across all three CKD stages. All patients receiving tocilizumab therapy had stage 4 CKD, whereas all patients treated with rituximab had stage 3 CKD. One patient on tocilizumab had RA-associated secondary amyloidosis. Etanercept was the preferred bDMARD for one dialysis-dependent patient (Table 3).

Table 3. Drug preferences according to renal stages in CKD				
Drugs	Stage 3 n=47	Stage 4 n=11	Stage 5 n=14	p value
csDMARDs	47 (100)	10 (90.9)	13 (92.9)	0.122*
Methotrexate, n (%)	13 (27.7)	1 (9.1)	0 (0)	0.028*
Leflunomide, n (%)	22 (46.8)	6 (54.5)	7 (50.0)	0.943*
Sulfasalazine, n (%)	12 (25.5)	4 (36.4)	4 (28.6)	0.798*
Hydroxychloroquine, n (%)	29 (61.7)	7 (63.6)	8 (57.1)	0.936*
GCs, n (%)	28 (59.6)	4 (36.4)	5(35.7)	0.165*
bDMARDs, n (%)	5 (10.6)	3 (27.3)	1 (7.1)	0.296*
TNFi	1 (2.1)	1 (9.1)	1 (7.1)	
Tocilizumab	0 (0)	2 (18.2)	0 (0)	
Rituximab	4 (8.5)	0 (0)	0 (0)	

Due to small sample sizes in Stage 4 and Stage 5, particularly for bDMARD subgroups, statistical comparisons (p-values) for these groups should be interpreted with caution; * Chi-square tests; CKD: chronic kidney disease, csDMARDs: conventional synthetic disease-modifying antirheumatic drugs, GCs: glucocorticoids, bDMARDs: biological disease- modifying antirheumatic drugs, TNFi: tumor necrosis factor inhibitor

DISCUSSION

This study analyzes demographic characteristics, comorbidities, renal etiology, and treatment approaches in RA patients with concomitant CKD. Our findings highlight critical clinical patterns in this specific population, including a high comorbidity burden, uncertainty regarding renal etiology, and CKD stage-specific the real-world prescribing patterns of anti-rheumatic medications in RA patients. Unlike some previous studies primarily focused on bDMARDs, this research provides a valuable assessment of the utilization of all major anti-rheumatic drug classes – csDMARDs, bDMARDs, and glucocorticoids – within this specific patient group.

We observed a csDMARD utilization rate of 97.2% and a bDMARDs utilization rate of 12.5% among our RA patients with CKD. Hydroxychloroquine was the most frequently used csDMARD, while methotrexate was the least used. Our methotrexate usage was approximately 20%, and notably, it was not administered to any stage 5 CKD patients. A

previous study evaluating RA patients with CKD receiving biologic therapy reported methotrexate usage near 50%, although it did not provide a separate assessment by renal stage (14). In our study, the classification of patients according to established CKD stages (3, 4 and 5) is a major strength. This detailed analysis provides important clinical stage-specific information by demonstrating how treatment options, particularly for nephrotoxic or renally cleared drugs such as methotrexate, significantly change with decreasing renal function. Similarly, a study evaluating RA patients with end-stage renal disease (ESRD) also reported no methotrexate use, consistent with our findings in stage 5 patients. In that study, half the patients used glucocorticoids and hydroxychloroquine was the most frequently used csDMARD, while other csDMARDs were rarely used (15). In contrast, within our stage 5 patient group, hydroxychloroquine (57.1%) was also the most common, but leflunomide (50%) and sulfasalazine (28.6%) usage was more prevalent than reported in the aforementioned study. The findings regarding the relatively frequent use of leflunomide and sulfasalazine in Stage 4 and Stage 5 CKD patients, exceeding rates previously reported in the literature, constitute an important contribution. This suggests evolving clinical practices or better recognition of their adjusted dosing feasibility in severe renal impairment. Current ESRD treatment recommendations advise that leflunomide dosing requires no adjustment, sulfasalazine requires a 50% dose reduction, and methotrexate is contraindicated (16). Our findings align with these recommendations.

The most frequently used bDMARDs in our study were rituximab, TNFi agents and tocilizumab, respectively. In contrast to our results, previous studies demonstrated that TNFi were the most frequently preferred bDMARDs, with etanercept being the most commonly selected agent within the TNFi class (14,15). In our study, etanercept was the only TNFi agent administered to patients receiving renal replacement therapy. TNFi agents remain central to RA treatment due to their well-documented efficacy and safety profile. As they are primarily metabolized in lysosomes, their pharmacokinetics are not significantly influenced by renal function (17). The preference for etanercept in our ESRD group may be attributed to its shorter half-life, potentially offering a safety advantage in the context of impaired kidney function (14). Rituximab is known to be usable in CKD patients without requiring dose adjustment (16). All four patients receiving rituximab in our study had stage 3 CKD, and one had a concomitant malignancy diagnosis. A previous Turkish study in RA patients with CKD reported rituximab usage rates similar to ours (18).

When examining bDMARDs use by CKD stage, it was most prevalent in stage 4 CKD patients, among whom tocilizumab usage was common. One patient receiving tocilizumab had a diagnosis of RA-associated secondary amyloidosis. Secondary amyloid A (AA) amyloidosis is a rare and potentially fatal complication observed in patients with long-standing, refractory RA (19). Suppression of serum amyloid A (SAA) protein production is essential for

controlling AA amyloidosis. SAA is synthesized in the liver in response to stimulation by pro-inflammatory cytokines such as interleukin-6 (IL-6), TNF, and interleukin-1. Consequently, reducing the levels of these cytokines is crucial for lowering SAA. While TNF inhibition can decrease SAA levels, normalization of SAA levels has been specifically achieved through IL-6 blockade (20,21). These literature findings support our rationale for prioritizing tocilizumab in patients with amyloidosis.

As the population of RA patients ages, an increase in comorbidities, including CKD, is anticipated. However, the precise frequency of CKD in RA patients and the extent to which common RA comorbidities like hypertension and diabetes contribute to ESRD development remain unclear (15). The most prevalent comorbidities in our cohort—hypertension, diabetes mellitus, hyperlipidemia, and coronary artery disease—are consistent with previous literature (14,18). A prior study conducted at our clinic evaluating comorbidities in inflammatory rheumatic diseases reported a CKD prevalence of 2% in RA patients (22). Other studies, however, have reported higher prevalence rates, ranging from 10% to 30% [4-6]. Conversely, a study examining RA prevalence among ESRD patients found it to be approximately 1% (15).

Extra-articular involvement occurs in approximately 40% of RA patients (23). The subclinical nature of pulmonary disease in many RA patients makes precise epidemiological assessment challenging. A study employing computed tomography (CT) screening in RA patients reported pulmonary involvement in 8% of cases, with interstitial lung disease (ILD) in 52%, pulmonary rheumatoid nodules in 5%, pleural effusion in 3%, and drug-induced lung disease in 3% of those affected (24). Our study did not include systematic CT screening; pulmonary involvement was recorded retrospectively, and ILD was noted in approximately 11% of patients. This finding suggests that pulmonary involvement might be more frequent in RA patients with CKD. This could be explained by several factors in our CKD cohort: older age, longer disease duration, and the potential for uremia and toxin accumulation to exacerbate inflammatory responses, leading to lung tissue damage. Furthermore, medications like methotrexate, used in RA treatment, may contribute to pulmonary fibrosis via drug toxicity in CKD patients.

Regarding CKD etiology, the cause remained unknown in 65% of our patients. This likely stems from the retrospective nature of data extraction from hospital electronic records and potential limitations within the record-keeping system. Among identified causes, diabetes and hypertension were the most common. A study evaluating RA patients with ESRD also identified diabetes and hypertension as the primary etiologies. Miscellaneous causes constituted about 8% in that study, significantly lower than in ours; this discrepancy may be explained by more comprehensive retrospective data in their cohort (15). RA-associated amyloidosis accounted for approximately 1% of cases in our study. While amyloidosis was historically suggested

as one of the most common causes of renal involvement in RA (25,26), more recent studies support our findings, indicating a diminished prevalence of amyloidosis in RA (15,27). CKD associated with NSAIDs was observed in approximately 1% of our patients, which is consistent with the findings from the ESRD study (15).

Our study has several limitations. It is a cross-sectional analysis conducted on a relatively small patient population. This constrains our statistical power and may have impacted the feasibility of robust subgroup analyses. Consequently, caution is warranted when generalizing our findings to the broader CKD population. Certain patient information was collected retrospectively by reviewing medical records. This approach introduces risks of missing data, potential effects of recording errors, and the inherent potential for bias associated with retrospective data collection. Our study lacks a control group comprising RA patients without CKD. This omission limits our ability to make direct comparisons regarding the impact of CKD on RA treatment choices. The study protocol did not systematically capture data on dose reductions or treatment regimen modifications specifically made due to CKD. This gap precluded our ability to assess the practical implications of renal dysfunction on RA treatment management and its potential association with treatment response. The presence of malignancy is an important parameter affecting treatment decisions. The inclusion of these patients is another limitation of the study, but only one patient in our study had malignancy. Future research should involve larger, prospective, controlled, multicenter cohort studies to enhance the generalizability of findings. If feasible, longitudinal follow-up would be valuable to investigate the relationship between CKD progression and RA activity/treatment response.

CONCLUSION

This study demonstrates significant clinical patterns in RA patients with CKD. Treatment approaches were influenced by CKD stage, highlighting its impact on pharmacological selection. While csDMARDs were widely utilized, methotrexate use was notably low and entirely contraindicated in stage 5 CKD patients due to safety concerns. Hydroxychloroquine emerged as the most frequently prescribed csDMARD. Conversely, the use of leflunomide and sulfasalazine in stage 5 CKD patients exceeded rates previously reported in the literature. The management of RA in the context of CKD represents a complex clinical challenge, necessitating individualized treatment strategies attuned to renal function stage and aggressive management of comorbidities. Our findings underscore the need for larger, multicenter, prospective controlled studies to further validate these observations and enhance their generalizability.

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REFERENCES

1. Cross M, Smith E, Hoy D, et al. The global burden of rheumatoid arthritis: estimates from the global burden of disease 2010 study. *Ann Rheum Dis*. 2014;73:1316-22.
2. Tuncer T, Gilgil E, Kaçar C, et al. Prevalence of rheumatoid arthritis and spondyloarthritis in Turkey: a nationwide study. *Arch Rheumatol*. 2017;33:128-36.
3. Yoshida K, Harrold LR, Middaugh N, et al. Time-varying association of rheumatoid arthritis disease activity to subsequent cardiovascular risk. *ACR Open Rheumatol*. 2022;4:587-95.
4. Couderc M, Tatar Z, Pereira B, et al. Prevalence of renal impairment in patients with rheumatoid arthritis: results from a cross-sectional multicenter study. *Arthritis Care Res (Hoboken)*. 2016;68:638-44.
5. Tokoroyama T, Ando M, Setoguchi K, et al. Prevalence, incidence and prognosis of chronic kidney disease classified according to current guidelines: a large retrospective cohort study of rheumatoid arthritis patients. *Nephrol Dial Transplant*. 2017;32:2035-42.
6. Mori S, Yoshitama T, Hirakata N, Ueki Y. Prevalence of and factors associated with renal dysfunction in rheumatoid arthritis patients: a cross-sectional study in community hospitals. *Clin Rheumatol*. 2017;36:2673-82.
7. Fukui S, Winkelmayer WC, Tedeschi SK, et al. Disease activity of rheumatoid arthritis and kidney function decline: a large prospective registry study. *Ann Rheum Dis*. 2025;84:201-9.
8. Sumida K, Molnar MZ, Potukuchi PK, et al. Treatment of rheumatoid arthritis with biologic agents lowers the risk of incident chronic kidney disease. *Kidney Int*. 2018;93:1207-16.
9. Yoshimura Y, Yamanouchi M, Mizuno H, et al. Efficacy and safety of first-line biological DMARDs in rheumatoid arthritis patients with chronic kidney disease. *Ann Rheum Dis*. 2024;83:1278-87.
10. Freitas RV, Godinho F. Rheumatoid arthritis and chronic kidney disease under dialysis - are anti-TNF an option?. *Reumatol Clin (Engl Ed)*. 2022;18:623.
11. Cho SK, Sung YK, Park S, Bae SC. Etanercept treatment in rheumatoid arthritis patients with chronic kidney failure on predialysis. *Rheumatol Int*. 2010;30:1519-22.
12. Kay J, Upchurch KS. ACR/EULAR 2010 rheumatoid arthritis classification criteria. *Rheumatology (Oxford)*. 2012;51:vi5-9.
13. Kidney disease: improving global outcomes (KDIGO) CKD work group. KDIGO 2024 Clinical Practice Guideline for the Evaluation and Management of Chronic Kidney Disease. *Kidney Int*. 2024;105:S117-314.
14. Yağız B, Coşkun BN, Dalkılıç HE, Pehlivan Y. Usage of biological therapy in rheumatoid arthritis patients with chronic renal failure: a retrospective study from a single center. *Uludağ Üniversitesi Tıp Fakültesi Dergisi*. 2022;48:203-8.

15. Paudyal S, Yang FM, Rice C, et al. End-stage renal disease in patients with rheumatoid arthritis. *Semin Arthritis Rheum*. 2017;46:418-22.
16. Weiner SM. Treatment of rheumatic disease with renal insufficiency. *Orthopade*. 2019;48:927-35.
17. Senel S, Kisacik B, Ugan Y, et al. The efficacy and safety of etanercept in patients with rheumatoid arthritis and spondyloarthritis on hemodialysis. *Clin Rheumatol*. 2011;30:1369-72.
18. Sandal Uzun G, Taghiyeva A, Çakır İY, et al. Factors that predict development of chronic kidney disease in patients with rheumatoid arthritis receiving biologic DMARDs and mortality rates. *Int J Rheum Dis*. 2024;27:e14967.
19. Jung JY, Kim YB, Kim JW, et al. Biologic therapy for amyloid A amyloidosis secondary to rheumatoid arthritis treated with interleukin 6 therapy: case report and review of literature. *Medicine (Baltimore)*. 2021;100:e26843.
20. Fukuda M, Sawa N, Hoshino J, et al. Tocilizumab preserves renal function in rheumatoid arthritis with AA amyloidosis and end-stage kidney disease: two case reports. *Clin Nephrol*. 2021;95:54-61.
21. Okuda Y. AA amyloidosis - Benefits and prospects of IL-6 inhibitors. *Mod Rheumatol*. 2019;29:268-74.
22. Koçak Ulucaköy R, Babaoğlu H, Kayacan Erdoğan E, et al. Comorbidity profiles in inflammatory rheumatic diseases: an analytical perspective. *International Journal of Clinical Practice*. 2024;3244597.
23. Guellec D, Cozien S, Ruysse-Witrand A, et al. Prevalence and clinical significance of extra-articular manifestations at diagnosis in the ESPOIR cohort with recent-onset arthritis. *Semin Arthritis Rheum*. 2020;50:409-13.
24. Duarte AC, Porter JC, Leandro MJ. The lung in a cohort of rheumatoid arthritis patients-an overview of different types of involvement and treatment. *Rheumatology (Oxford)*. 2019;58:2031-8.
25. Targońska-Stępnia B, Majdan M. Serum amyloid A as a marker of persistent inflammation and an indicator of cardiovascular and renal involvement in patients with rheumatoid arthritis. *Mediators Inflamm*. 2014;2014:793628.
26. Horak P, Smrzova A, Krejci K, et al. Renal manifestations of rheumatic diseases. a review. *Biomed Pap Med Fac Univ Palacky Olomouc Czech Repub*. 2013;157:98-104.
27. Karstila K, Korpela M, Sihvonen S, Mustonen J. Prognosis of clinical renal disease and incidence of new renal findings in patients with rheumatoid arthritis: follow-up of a population-based study. *Clin Rheumatol*. 2007;26:2089-95.