



Assessment and Evaluation of Thyroid Dysfunction in Seropositive Rheumatoid Arthritis A Hospital-Based Prospective Observational Study.

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Abstract

Background: Rheumatoid arthritis (RA) is a chronic systemic autoimmune disorder that frequently coexists with other autoimmune conditions, including thyroid dysfunction. Early identification of thyroid abnormalities in RA patients is clinically important, as untreated thyroid disorders may worsen fatigue, musculoskeletal symptoms, cardiovascular risk, and overall quality of life. **Aim:** To assess and evaluate thyroid dysfunction in patients with seropositive rheumatoid arthritis and to correlate it with disease activity. **Materials and Methods:** This hospital-based observational study was conducted in the Department of General Medicine, Mahatma Gandhi Medical College & Hospital, Jaipur, from April 2024 to September 2025. A total of 100 consecutive patients with confirmed seropositive RA, aged 18–85 years, were enrolled. Serum T3, T4, TSH, and Anti-thyroid peroxidase (Anti-TPO) antibody levels were measured. Disease activity was assessed using the DAS28 score. Data were analysed using SPSS v29.0 with chi-square, Student's t-test, ANOVA, Pearson correlation, and odds ratio; $p < 0.05$ was considered significant. **Results:** The mean age was 44.6 ± 15.15 years, with a marked female predominance (78%). Thyroid dysfunction was present in 36% of patients, with subclinical hypothyroidism the most common pattern (18%), followed by overt hypothyroidism (10%), subclinical hyperthyroidism (5%), and overt hyperthyroidism (3%). Anti-TPO positivity was observed in 28% of patients and was strongly associated with thyroid dysfunction (OR = 8.3; 95% CI 2.9–23.8; $p < 0.001$). Serum T3 and T4 levels showed a significant negative correlation with DAS28 ($r = -0.47$ and $r = -0.72$ respectively; $p < 0.001$), while TSH showed a strong positive correlation ($r = 0.72$; $p < 0.001$). **Conclusion:** Thyroid dysfunction, particularly hypothyroid patterns and Anti-TPO positivity, is common in patients with seropositive rheumatoid arthritis and is significantly associated with greater disease activity. Routine thyroid function and Anti-TPO screening should be considered in RA patients, particularly those with moderate to severe disease, to facilitate early diagnosis and improve clinical outcomes.

Keywords: Rheumatoid arthritis; Thyroid dysfunction; Anti-TPO; DAS28; Subclinical hypothyroidism; Autoimmune thyroid disease.



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INTRODUCTION

Rheumatoid arthritis (RA) is a chronic, systemic autoimmune disorder characterised by persistent synovial inflammation and the production of autoantibodies, particularly rheumatoid factor and anti-citrullinated peptide antibodies.¹ The disease primarily targets synovial joints, where ongoing immune-mediated inflammation leads to synovial hypertrophy, cartilage degradation, and progressive bone erosion. Over time, repeated inflammatory episodes disrupt normal joint architecture, resulting in pain, stiffness, deformity, reduced functional capacity, and significant long-term disability.²

Beyond articular involvement, RA is a multisystem disease, and many patients develop extra-articular manifestations affecting the skin, eyes, lungs, cardiovascular system, and haematologic system, further contributing to disease burden and morbidity.³

The global prevalence of RA has remained approximately 0.46% over the past four decades, underscoring its substantial contribution to chronic inflammatory disease worldwide. In addition to its articular and systemic manifestations, RA has been increasingly recognised to coexist with other autoimmune conditions, including thyroid dysfunction.³

Thyroid dysfunction encompasses a spectrum of disorders, including overt hyperthyroidism, overt hypothyroidism, subclinical hyperthyroidism, and subclinical hypothyroidism. These conditions may present with varying clinical and biochemical profiles, ranging from subtle hormonal imbalances without obvious symptoms to clinically significant metabolic and systemic disturbances.^{4,5} Autoimmune thyroid disease (AITD) represents the most common underlying cause of thyroid dysfunction. The two principal forms of AITD are Hashimoto's thyroiditis and Graves' disease. Hashimoto's thyroiditis is typically associated with progressive thyroid destruction leading to hypothyroidism, whereas Graves' disease results in thyroid hyperactivity and thyrotoxicosis. Both conditions are characterised by immune-mediated mechanisms involving autoantibodies directed against thyroid-specific antigens, including thyroid peroxidase (TPO), thyroglobulin (Tg), and the thyrotropin (TSH) receptor. The shared autoimmune basis of RA and AITD suggests potential common genetic, immunological, and environmental factors contributing to their coexistence.^{6,7}

Laboratory evaluation plays a central role in the diagnosis and classification of thyroid disorders. The most commonly assessed biochemical parameters include thyroid-stimulating hormone (TSH), triiodothyronine (T3), thyroxine (T4), free triiodothyronine (fT3), and free thyroxine (fT4). Although reference ranges may vary slightly depending on laboratory methods, the overall patterns of hormonal changes used to identify thyroid dysfunction are well established.

Both thyroid disease and rheumatoid arthritis are chronic conditions that often coexist and may share overlapping symptoms such as fatigue, weight changes, and musculoskeletal complaints. Early manifestations of thyroid dysfunction are frequently nonspecific and can be masked by features of other chronic diseases, including RA, particularly when biochemical thyroid testing is not performed. Undiagnosed or untreated thyroid dysfunction may lead to significant health consequences, including cardiovascular complications, metabolic disturbances, reduced quality of life, and, in severe cases, life-threatening outcomes. Evidence regarding the risk of thyroid dysfunction among patients with RA remains inconsistent. McCoy et al. reported that individuals with RA did not have a significantly higher risk of hypothyroidism compared with the general population, while Graves' disease has been reported to show a notable comorbidity with RA. Against this background, the present study was conducted to assess and evaluate thyroid dysfunction in patients with seropositive rheumatoid arthritis.

Aim and Objectives

Aim

To assess and evaluate thyroid dysfunction in patients with seropositive rheumatoid arthritis.

Objectives

- To assess the relationship of serum T3, T4, and TSH levels with seropositive rheumatoid arthritis.
- To study the association of Anti-thyroid peroxidase (Anti-TPO) antibody with seropositive rheumatoid arthritis.
- To evaluate the severity of thyroid dysfunction in patients with seropositive rheumatoid arthritis.
- To correlate thyroid dysfunction with disease activity and severity in seropositive rheumatoid arthritis patients.

MATERIALS AND METHODS

Study Design

This was a hospital-based observational study conducted to assess and evaluate thyroid dysfunction in patients with seropositive rheumatoid arthritis.

Place and Duration of Study

The study was conducted in the Department of General Medicine, Mahatma Gandhi Medical College & Hospital, Jaipur, Rajasthan, over a period of one and a half years from April 2024 to September 2025.

Study Population and Sample Size

The study population comprised patients diagnosed with seropositive rheumatoid arthritis attending the Outpatient Department (OPD) or admitted to the Inpatient Department (IPD). A total of 100 consecutive patients fulfilling the inclusion criteria were enrolled.

Ethical Clearance

Prior approval from the Institutional Ethics Committee was obtained before commencement of the study. Written informed consent was obtained from all participants. In patients unable to provide consent, consent was obtained from the legally authorised attendant.

Selection Criteria

Inclusion criteria

- Patients of either sex.
- Age between 18 and 85 years.
- Confirmed cases of seropositive rheumatoid arthritis.
- Patients willing to participate and provide written informed consent.

Exclusion criteria

- Age less than 18 years or more than 85 years.
- Patients unwilling to participate.
- Patients refusing to sign the informed consent form.

Study Methodology

After enrolment, a detailed history was taken from each participant, including duration of illness, presenting complaints, treatment history, and associated comorbidities. General physical and systemic examinations were performed. Vital parameters including pulse rate, blood pressure, respiratory rate, and temperature were recorded.

Investigations

All enrolled patients underwent the following investigations:

- Complete blood count (CBC).
- Blood sugar profile.
- Liver function test (LFT).
- Kidney function test (KFT).
- Routine urine examination.
- Rheumatoid factor (RF).
- Anti-cyclic citrullinated peptide (Anti-CCP) antibody.
- C-reactive protein (CRP).
- Serum triiodothyronine (T3), thyroxine (T4), and thyroid-stimulating hormone (TSH).
- Anti-thyroid peroxidase (Anti-TPO) antibody.

Whenever clinically indicated, a thyroid scan and ultrasonography (USG) of the neck/thyroid were performed.

Assessment of Disease Severity

Disease activity and severity of rheumatoid arthritis were assessed using the Disease Activity Score in 28 joints (DAS28), calculated using the number of tender joints, swollen joints, ESR/CRP values, and patient global health assessment. Based on DAS28, patients were classified as:

- Remission: <2.6
- Low disease activity (mild): 2.6–3.2
- Moderate disease activity: >3.2–5.1
- High disease activity (severe): >5.1

Statistical Analysis

Data were entered into Microsoft Excel and analysed using SPSS v29.0. Quantitative variables were expressed as mean $\pm$ standard deviation (SD); qualitative variables as frequency and percentage. The chi-square test, Student's t-test, ANOVA, Pearson correlation coefficient, and odds ratio were applied wherever appropriate. A p-value < 0.05 was considered statistically significant.

RESULTS

Table.1 Demographic profile and thyroid status distribution among study participants

Variable	Category	No. of patients	Percentage (%)
Mean age $\pm$ SD	44.6 $\pm$ 15.15 years.		
Gender	Female	78	78.00
	Male	22	22.00
Thyroid status	Euthyroid	64	64.00
	Subclinical hypothyroidism	18	18.00
	Overt hypothyroidism	10	10.00
	Subclinical hyperthyroidism	5	5.00
	Overt hyperthyroidism	3	3.00

Majority of patients belonged to the 31–40 years age group (25%), followed by 21–30 years (22%) and 41–50 years (18%). Patients aged 51–60 years and 61–70 years constituted 16% and 15% of the study population, respectively, while only 4% of participants were older than 70 years. A marked female predominance was observed, with females accounting for 78% of the study population, whereas males constituted 22%. Regarding thyroid status, most participants were euthyroid (64%). Among patients with thyroid dysfunction, subclinical hypothyroidism was the most common abnormality, observed in 18% of cases, followed by overt hypothyroidism in 10%. Subclinical hyperthyroidism and overt hyperthyroidism were less frequent, reported in 5% and 3% of patients, respectively.

Table 2. Descriptive statistics of disease duration and thyroid hormone levels

Parameter	Mean $\pm$ SD
Duration of RA (years)	6.01 $\pm$ 1.38
T3 (ng/dL)	2.35 $\pm$ 0.74
T4 (μ g/dL)	2.29 $\pm$ 0.93
TSH (μ IU/mL)	3.47 $\pm$ 1.04

The mean duration of rheumatoid arthritis was 6.01 $\pm$ 1.38 years, indicating a moderately long-standing disease in the study population. The mean values of T3, T4, and TSH suggest variability in thyroid hormone levels, with values tending toward altered thyroid function in a subset of patients.

Table 3. Distribution of disease severity (DAS28) among study participants

Disease severity (DAS28)	No. of patients (%)	Mean $\pm$ SD
Mild (2.6–3.2)	30 (30.00)	2.38 $\pm$ 0.29
Moderate (>3.2–5.1)	38 (38.00)	4.49 $\pm$ 0.31
Severe (>5.1)	32 (32.00)	6.21 $\pm$ 0.75
Total	100 (100.00)	4.41 $\pm$ 1.59

Moderate disease activity was the most common (38%), followed by severe (32%) and mild (30%). The overall mean DAS28 score was 4.41 $\pm$ 1.59, indicating moderate average disease activity.

Table 4. Distribution of Anti-TPO status among study participants

Anti-TPO status	No. of patients	Percentage (%)
Positive	28	28.00
Negative	72	72.00
Total	100	100.00

Anti-TPO positivity was observed in 28% of patients, while 72% were Anti-TPO negative, indicating that a smaller proportion of patients had evidence of autoimmune thyroid involvement.

Table 5. Association between Anti-TPO status and thyroid dysfunction

Anti-TPO status	Dysfunction present, n (%)	Dysfunction absent, n (%)
Positive	22 (50.00)	6 (10.71)
Negative	22 (50.00)	50 (89.29)
Total	44	56
p-value	<0.001	

The association between Anti-TPO status and thyroid dysfunction was statistically significant ($p < 0.001$). Among Anti-TPO positive patients, 50% had thyroid dysfunction, whereas 89.29% of Anti-TPO negative patients did not, indicating a strong association between Anti-TPO positivity and thyroid dysfunction.

Table 6. Association between disease severity (DAS28) and thyroid dysfunction

Disease severity (DAS28)	Dysfunction present, n (%)	Dysfunction absent, n (%)
Mild (2.6–3.2)	8 (18.18)	22 (39.29)
Moderate (>3.2–5.1)	18 (40.91)	20 (35.71)
Severe (>5.1)	18 (40.91)	14 (25.00)
Total	44	56
p-value	0.088	

Although patients with thyroid dysfunction were more frequently in the moderate and severe groups (40.91% each) compared with the mild group (18.18%), the association did not reach statistical significance ($p = 0.088$). The observed clinical trend, however, suggests greater thyroid involvement with higher disease activity.

Table 7. Comparison of mean serum T3, T4, and TSH levels across DAS28 categories.

Disease severity (DAS28)	T3 (ng/dL) Mean $\pm$ SD	T4 (μ g/dL) Mean $\pm$ SD	TSH (μ IU/mL) Mean $\pm$ SD
Mild (2.6–3.2)	2.84 $\pm$ 0.55	3.09 $\pm$ 0.51	2.47 $\pm$ 0.31
Moderate (>3.2–5.1)	2.27 $\pm$ 0.85	2.41 $\pm$ 0.89	3.46 $\pm$ 0.88
Severe (>5.1)	1.98 $\pm$ 0.42	1.40 $\pm$ 0.29	4.41 $\pm$ 0.73
Total	2.35 $\pm$ 0.74	2.29 $\pm$ 0.93	3.47 $\pm$ 1.04
p-value	<0.001	<0.001	<0.001

Mean serum T3 and T4 levels showed a statistically significant decline with increasing disease severity ($p < 0.001$ for both), while mean TSH levels showed a statistically significant rise ($p < 0.001$). These findings demonstrate a hypothyroid pattern that becomes more pronounced with worsening RA disease activity.

Table 8. Correlation of thyroid hormone levels with disease severity (DAS28)

Parameter	Correlation coefficient (r)	p-value
T3 vs DAS28	–0.47	<0.001
T4 vs DAS28	–0.72	<0.001
TSH vs DAS28	+0.72	<0.001

Serum T3 showed a moderate negative correlation with DAS28 ($r = -0.47$), while serum T4 showed a strong negative correlation ($r = -0.72$). In contrast, TSH demonstrated a strong positive correlation with DAS28 ($r = +0.72$). All correlations were statistically significant ($p < 0.001$), indicating that altered thyroid function is closely linked with greater RA disease activity.

Table 9. Predictors of thyroid dysfunction – logistic regression analysis

Variable	Odds ratio (OR)	95% CI	p-value
Anti-TPO positivity	8.3	2.9–23.8	<0.001
High TSH	5.6	2.6–12.1	<0.001

Anti-TPO positivity was a strong predictor of thyroid dysfunction (OR = 8.3; 95% CI: 2.9–23.8; $p < 0.001$), and elevated TSH levels were also significantly associated with thyroid dysfunction (OR = 5.6; 95% CI: 2.6–12.1; $p < 0.001$).

DISCUSSION

This hospital-based observational study was undertaken to assess and evaluate thyroid dysfunction in patients with seropositive rheumatoid arthritis (RA), with special emphasis on the relationship of serum thyroid hormones (T3, T4, and TSH), Anti-TPO antibody status, and their correlation with disease activity. RA is a chronic systemic autoimmune inflammatory disorder that affects not only joints but is also associated with several extra-articular manifestations and autoimmune comorbidities. Thyroid dysfunction, particularly autoimmune thyroid disease, has been increasingly recognised as one of the common endocrine associations of RA. Early identification of thyroid abnormalities is clinically important, as untreated thyroid dysfunction may worsen fatigue, musculoskeletal symptoms, cardiovascular risk, and quality of life.

Demographic Profile

In the present study, the mean age of participants was 44.6 ± 15.15 years, indicating that the majority belonged to the middle-aged group. Most patients were in the 31–40 years age group, followed by 21–30 years and 41–50 years. These findings agree with the known epidemiology of RA, which most commonly affects individuals in the fourth and fifth decades of life. Similar observations have been reported by Nadeem et al.⁹ (mean age 48.2 ± 12.1 years), Kumar et al.¹⁰ (mean age 56.65 ± 9.72 years), and Ferdoush et al.¹¹ (mean age 47.2 ± 11.3 years). The relatively lower proportion of elderly patients in our study may be attributable to reduced healthcare-seeking behaviour, multiple comorbid illnesses, or decreased survival in patients with longstanding severe disease.

A marked female predominance was observed, with females constituting 78% of the study population and males 22%. This is comparable to Nadeem et al.⁹ (86.8% female), Kumar et al.¹⁰ (75.8% female), and Ferdoush et al.¹¹ (76% female; F:M ratio 3.1:1). The higher prevalence of RA among females may be attributed to hormonal influences, particularly oestrogen-mediated immune modulation, and a greater predisposition to autoimmune disease in women.

Pattern of Thyroid Dysfunction

The majority of patients in our study were euthyroid (64%), while thyroid dysfunction was observed in 36%. Subclinical hypothyroidism was the most common pattern (18%), followed by overt hypothyroidism (10%), subclinical hyperthyroidism (5%), and overt hyperthyroidism (3%). These findings indicate that hypothyroid disorders, particularly subclinical hypothyroidism, are more common in patients with seropositive RA. This is clinically important because subclinical hypothyroidism often goes unrecognised due to nonspecific symptoms such as fatigue, weakness, and musculoskeletal discomfort, which may overlap with manifestations of RA.

The higher prevalence of hypothyroid states in RA patients may be explained by common autoimmune mechanisms. Both RA and autoimmune thyroid disease involve dysregulation of T-cell and B-cell-mediated immunity, persistent inflammation, cytokine activation, and shared genetic susceptibility (such as HLA associations); coexistence of these disorders is therefore biologically plausible. Comparable findings have been reported by Ferdoush et al.¹¹ (thyroid dysfunction in 26%), Datta et al.¹² (37% in cases vs 18% in controls), Kumar et al.¹⁰ (subclinical hypothyroidism 33.3%), Maheshwari et al.¹³ (subclinical hypothyroidism 20.0%; overt hypothyroidism 12.3%), and Arain et al.¹⁴ (thyroid dysfunction 41.2%). These findings collectively support the increased frequency of thyroid dysfunction, especially hypothyroid states, among RA patients.

Disease Severity (DAS28)

Most patients in our study had moderate disease activity (38%), followed by severe (32%) and mild (30%), with an overall mean DAS28 of 4.41 ± 1.59 , indicating moderate average disease activity. This suggests that many patients seek medical care after substantial disease progression or continue to have persistent inflammatory activity despite treatment, reflecting the considerable burden of active RA in our population. Fransen et al.¹⁵ similarly reported that the majority of their participants had moderate to high disease activity.

The association between thyroid dysfunction and DAS28 categories did not reach statistical significance ($p = 0.088$), comparable to the findings of Tekaya et al.¹⁶ ($p = 0.37$). However, an important clinical trend was noted: patients with thyroid dysfunction were more frequently distributed in the moderate and severe disease activity groups, while those without dysfunction more commonly fell into the mild category. The absence of statistical significance may be related to limited sample size or subgroup variations, but the observed trend remains clinically relevant and warrants further evaluation in larger studies.

Thyroid Hormone Profile and Disease Activity

A significant decline in serum T3 levels was observed with increasing severity of RA. Mean T3 levels were highest in mild disease and progressively decreased in moderate and severe disease ($p < 0.001$). This inverse relationship suggests that lower T3 levels are linked with greater inflammatory burden. Chronic systemic inflammation may impair the peripheral conversion of T4 to T3 through altered deiodinase enzyme activity, resulting in a low-T3 state or non-thyroidal illness syndrome. Pro-inflammatory cytokines such as TNF- α , IL-1, and IL-6 may also suppress thyroid hormone metabolism. Comparable observations have been reported by Nadeem et al.⁹, Arain et al.¹⁴, and Maheshwari et al.¹³

Serum T4 levels also showed a significant decline with increasing disease severity ($p < 0.001$), suggesting that more severe inflammatory activity may be associated with suppression of thyroid hormone production, altered thyroid hormone metabolism, or progression from subclinical to overt thyroid dysfunction. This is supported by Nadeem et al.⁹ (low T4 in 10.9%), Arain et al. (significant difference in FT4 across groups), and Maheshwari et al.¹³ (low FT4 in 6%).

In contrast, TSH levels rose significantly with increasing disease severity ($p < 0.001$). Patients with severe disease had the highest mean TSH values, while those with mild disease had the lowest. Elevated TSH accompanied by relatively lower T3 and T4 supports a hypothyroid tendency that becomes more prominent with rising RA activity. Chronic inflammation, autoimmune thyroiditis, altered hypothalamic–pituitary–thyroid axis regulation, and treatment-related metabolic effects may all contribute. Similar results have been reported by Nadeem et al.⁹ (raised TSH in 44.7%), Shafique et al.¹⁴, and Maheshwari et al.¹³

Correlation Analysis

Correlation analysis further reinforced the association between thyroid dysfunction and RA disease activity. Serum T3 showed a moderate negative correlation with DAS28 ($r = -0.47$), serum T4 a strong negative correlation ($r = -0.72$), and TSH a strong positive correlation ($r = +0.72$), all statistically significant ($p < 0.001$). Comparable findings have been reported by Talukdar et al.¹⁷ and Kumar et al.¹⁰, supporting the concept that thyroid hormonal imbalance is associated with higher RA disease activity and emphasising the need for routine thyroid function monitoring in patients with active RA.

Anti-TPO Antibody Status

Anti-TPO positivity was observed in 28% of patients, suggesting that a considerable proportion of seropositive RA patients have coexisting thyroid autoimmunity. Anti-TPO antibodies are strongly associated with autoimmune thyroiditis and are recognised predictors of future thyroid dysfunction; their presence in RA further supports the concept of shared autoimmune susceptibility.

A highly significant association was found between Anti-TPO positivity and thyroid dysfunction ($p < 0.001$): 50% of Anti-TPO positive patients had thyroid dysfunction compared to 10.71% of those who were Anti-TPO negative. Logistic regression demonstrated that Anti-TPO positive individuals had 8.3 times higher odds of developing thyroid dysfunction (95% CI 2.9–23.8). This is an important clinical finding, as Anti-TPO testing may help identify RA patients at increased risk for thyroid disease even before overt biochemical abnormalities develop. Comparable findings have been reported by Fransen et al., who observed Anti-TPO positivity in 57.46% of cases versus 36.44% of controls (OR 2.69; 95% CI 1.70–4.13).

Elevated serum TSH was independently associated with thyroid dysfunction (OR 5.6; 95% CI 2.6–12.1; $p < 0.001$), highlighting the diagnostic and predictive value of TSH measurement in RA patients. As TSH is the most sensitive initial screening test for thyroid dysfunction, routine assessment may be particularly beneficial in patients with active disease, unexplained fatigue, persistent symptoms despite treatment, or positive thyroid autoantibodies.

Limitations

Certain limitations should be acknowledged. As this was a hospital-based observational study, the findings may not be fully generalisable to the broader community. The cross-sectional design limits the ability to establish a causal relationship between thyroid dysfunction and RA severity; longitudinal studies with follow-up would help determine whether thyroid abnormalities contribute to worsening RA or develop secondary to chronic inflammation. Although the sample size of 100 was adequate for the primary analysis, subgroup analyses were limited, particularly for less common categories such as hyperthyroidism. In addition, factors such as medication use (DMARDs, corticosteroids), nutritional status, and comorbid illnesses may also influence thyroid function and warrant further evaluation in future studies.

CONCLUSION

Thyroid dysfunction is common in patients with seropositive rheumatoid arthritis, with subclinical hypothyroidism being the most frequent abnormality, followed by overt hypothyroidism. Anti-TPO positivity was strongly associated with thyroid dysfunction, with affected patients having 8.3 times higher odds of dysfunction compared to Anti-TPO negative individuals. Serum T3 and T4 levels declined significantly while TSH increased significantly with rising DAS28 scores, indicating a hypothyroid pattern that becomes more pronounced with greater disease activity.

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