ORIGINAL ARTICLE

Medical Journal of Western Black Sea
Batı Karadeniz Tıp Dergisi

Med J West Black Sea 2023;7(1): 14-20
DOI: 10.29058/mjwbs.1221562

Can the Prognosis be Predicted in Subacute Thyroiditis?

Subakut Tiroiditte Prognoz Öngörülebilir mi?

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Cite this article as: Saklamaz A and Çiftçi Ö. Can the prognosis be predicted in subacute thyroiditis?. Med J West Black Sea. 2023;7(1):14-20.

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Received
19.12.2022

Revision
13.04.2023

Accepted
14.04.2023

ABSTRACT

Aim: Subacute thyroiditis (SAT) is a thyroid disease that seriously affects the quality of life for patients caused by acute inflammation of the thyroid gland. Apart from classical acute phase reactants, the values and rates obtained from peripheral blood count (mean platelet volume (MPV), neutrophil-to-lymphocyte ratio (NLR), and platelet-to-lymphocyte ratio (PLR)) values are also accepted as practical indicators of systemic inflammation. Our aim in this study is to compare the effects of systemic inflammation markers and the treatments given in the laboratory tests of our patients with a diagnosis of SAT, on the hypothyroid state one year later.

Material and Methods: In this study, which was carried out with a retrospective method, 133 patients were included in the study. The medical data of these patients at the time of SAT diagnosis and one year later were analyzed. 37 patients were in the steroid group and 97 patients were in the nonsteroidal anti-inflammatory drug (NSAID) group.

Results: The male/female ratio was similar in both groups. Female dominance was observed in both groups in patients diagnosed with SAT. The thyroid tests of the groups, which were hyperthyroid at the beginning and euthyroid one year later, were similar between the groups ($p>0.05$). Both groups had an increase in acute phase reactants at baseline (erythrocyte sedimentation rate [ESR] and C-reactive protein (CRP) levels) and normalized after treatment. Neutrophil ($p<0.05$), lymphocyte ($p>0.05$) and platelet ($p<0.05$) counts decreased with the reduction of inflammation. Monocyte count decreased in both groups, but it was significant in the steroid group, but not in the NSAID group. The development of permanent hypothyroidism was 8/37 (21.6%), 24/97 (24.74%) in steroid and NSAID groups respectively ($p>0.05$). There was no statistical difference in inflammation markers (CRP etc.) and follow-up parameters before and after treatment in both groups (steroid vs. NSAID) with and without a diagnosis of permanent hypothyroidism ($p>0.05$).

Conclusion: Inflammation markers and treatments applied in SAT patients did not have a significant effect on the prognosis.

Keywords: Subacute thyroiditis, Glucocorticoid, NSAID, Hypothyroidism

ÖZ

Amaç: Subakut tiroidit (SAT), tiroid bezinin akut inflamasyonu nedeniyle oluşan hastalar için yaşam kalitesini ciddi oranda etkileyen bir tiroid hastalığıdır. Klasik akut faz reaktanları dışında periferik kan sayımından elde edilen değerler ve oranları da sistemik inflamasyonun pratik göstergesi olarak kabul edilmektedir. Bu çalışmada amacımız, SAT tanısı olan hastalarımızın laboratuvar tetkiklerinde sistemik inflamasyon markerlarının ve verilen tedavilerin, bir yıl sonraki hipotiroidik duruma etkisini karşılaştırmaktır.

Gereç ve Yöntemler: Bu retrospektif çalışmada 133 hasta çalışmaya dahil edildi. Bu hastaların SAT tanı anında ve 1 yıl sonraki tıbbi verileri incelendi. 37 hasta steroid grubunda, 97 hasta nonsteroid antiinflamatuar ilaç (NSAİİ) grubunda yer aldı.

Bulgular: Her iki grupta erkek/kadın oranı benzerdi. SAT tanısı alan hastalarda kadın hakimiyeti her iki grupta da görüldü. Grupların başlangıçta hipertiroidi ve bir yıl sonra ötiroid olan tiroid testleri gruplar

arası benzerdi (p>0.05). Her iki grupta da başlangıçta akut faz reaktanlarından artış (eritrosit sedimantasyon hızı [ESR] ve C-reaktif protein (CRP) seviyeleri) ve tedavi sonrası normale gelmiştir. İnflamasyonun azalması ile nötrofil (p<0.05), lenfosit (p>0.05) ve trombosit (p<0.05) sayıları azaldı. Monosit sayısı her iki grupta da azaldı, ancak steroid grubunda anlamlıydı, ancak NSAID grubunda değildi. Steroid ve NSAİİ gruplarında kalıcı hipotiroidi gelişimi sırasıyla 8/37 (%21,6), 24/97 (%24,74) idi (p>0,05). Tedavi öncesi ve sonrasında, her iki grupta da (steroid vs. NSAID) kalıcı hipotiroidizm tanısı alan/almayan arasında inflamasyon belirteçleri (CRP etc.) ve izlem parametrelerinde istatistiksel olarak fark bulunmamıştır (p>0.05).

Sonuç: SAT hastalarında uygulanan inflamasyon belirteçleri ve tedavilerin prognoz üzerine anlamlı bir etkisi olmamıştır.

Anahtar Sözcükler: Subakut tiroidit, Glukokortikoid, NSAİİ, Hipotiroidizm

INTRODUCTION

Subacute thyroiditis (SAT) is a thyroid disease that seriously affects the quality of life for patients caused by acute inflammation of the thyroid gland (1). Most of the time, patients are examined in various branches before the definitive diagnosis is made. Because of the neck pain that hits the ear, antibiotics are first prescribed with pre-diagnoses of throat infection or otitis. For some patients, after the diagnosis is made, the treatment process can be a bit difficult. Patients can be diagnosed easily with pain in the thyroid gland area and increased acute phase responses in blood tests. In most patients, a viral upper respiratory tract infection is detected in the anamnesis before the onset of symptoms. On thyroid ultrasound, areas of inflammation are seen as hypoechoic areas with irregular edges. Sometimes, the image on ultrasound is thought to be malignant, and fine needle aspiration biopsy is mistakenly performed on painful thyroid tissue.

Inflammation markers are high in SAT. Along with classical markers, parameters such as mean platelet volume (MPV), neutrophil / lymphocyte ratio, thrombocyte / lymphocyte ratio, monocyte / lymphocyte ratio obtained from complete blood count are also important markers of inflammation (2-4). These new inflammation markers were also examined in patients with SAT (5-9). These markers were correlated with the inflammation, but there was no correlation with the recurrence of the SAT attack or causing permanent hypothyroidism (7).

When prescribing NSAIDs or glucocorticoids, the patient's clinical condition and the practice of the doctor are important. The degree of inflammation in the thyroid, the level of acute phase reactants in blood tests and the differences in drug selection were investigated (7,10-14). To date, there has been no clear indication of disease recurrence or persistent hypothyroidism.

In this study, we wanted to compare the new inflammation markers of our SAT patients and investigate their effects on the development of permanent hypothyroidism with treatment options.

MATERIAL and METHODS

The records of patients who applied to the endocrinology outpatient clinic between 2018 and 2022 were reviewed. 172 Patients diagnosed with subacute thyroiditis were identified retrospectively. The diagnosis of SAT was made with painful thyroid gland, increased acute phase reactants, thyroiditis area on thyroid ultrasound, and low uptake on thyroid scintigraphy if performed. Of the 172 patients, only those who took steroids and NSAIDs were included in the study. Users of both drugs during their treatment were excluded. For this reason, 37 patients in the steroid group and 96 patients in the NSAID group were enrolled in the study. Patients with acute exacerbation of chronic thyroiditis, bleeding into the thyroid cyst, acute suppurative thyroiditis, and those who used levothyroxine before diagnosis were excluded from the study.

This study was conducted in accordance with the Declaration of Helsinki with the approval of the Izmir University of Economics Faculty of Medicine Ethics Committee.

Laboratory Methods

Thyroid stimulating hormone (TSH) (15), free thyroxine (FT4), free trio-iodothyronine (FT3), anti-thyroglobulin antibodies (Anti-Tg) and anti-thyroid peroxidase antibodies (Anti-TPO) concentrations were measured using chemiluminescent microparticle enzyme immunoassay (CMIA) method. Thyroglobulin (Tg) was measured using an electrochemiluminescence immunoassay (ECLIA); CRP was measured by particle association turbidimetric assay (Cobas Integra 400 plus; Roche Diagnostics, Indianapolis, USA).

Hematological parameters were obtained from standard CBC. The NLR was calculated as the ratio between NEU count and LYM count. The PLR was calculated as the ratio between platelet (PLT) count and LYM count. The MLR was calculated as the ratio between MONO count and LYM count.

Statistical Analysis

Statistical analyzes were performed using Rstudio software (version 0.98.501, Wirtschaftsuniversität Wien Welthandel-

splatz 1 1020 Vienna, Austria). Continuous variables were reported as mean±standard deviation, categorical variables as numbers and percentages. Normality conditions were determined for continuous variables in the groups with the Shapiro Wilk test. The homogeneity of the variances was evaluated with the Levine test. Continuous and categorical variables were compared between groups using one-way ANOVA and Pearson's chi-square test. Paired post-hoc tests were performed on the data, where overall significance was observed in the ANOVA using the LSD test. For homogeneous data, the paired sample t-test was used to compare the pre- and post-treatment data of the groups with the baseline data and the data obtained 1 year later. The independent t-test was used to compare the permanent hypothyroidism (yes vs. no) groups. A p value of <0.05 was considered statistically significant.

Power analysis was calculated using G-Power ver. 3.1.9.7 (Heinrich Heine Universitat Düsseldorf, Germany) software. In the calculation made with the sample numbers in the groups, the effect size value was determined as (d) 0.9 for the steroid group and (d) 0.4 for the NSAID group, and the actual power was calculated as 82.53% and 80.44% for both groups separately. According to Cohen, a scientific study should have at least 80% power and according to this criterion, the study was completed with an appropriate power.

RESULTS

Demographic Data

133 patients were classified in 2 groups. The steroid or NSAIDs given groups had 37 and 96 patients respectively. There was no statistically difference in the mean ages of the groups (p>0.05). The male/female ratios were 10/27 in steroid group and 25/71 in NSAID group (p>0.05). Male/female ratio was similar and female domination was demonstrated in all groups.

Laboratory Data

Thyroid function tests were consistent with hyperthyroidism in the steroid and NSAID groups at the time of admission (Table 1). A significant difference was observed between the thyroid function tests (TSH, FT4, FT3) of the two groups before and 1 year after treatment (p<0.05). There was no significant difference in the values of Anti-Tg and Anti-TPO in both groups, measured at 1-year intervals (p>0.05). While there was no significant difference in thyroglobulin value in the steroid group (p>0.05), a statistical difference was found in the thyroglobulin value in the NSAID group (p<0.05). In

Table 1: Demographic and laboratory parameters of patients (before and one year later)

	STEROID (n=37)			NSAID (n=96)		P
Age (years)	43.5±11.8			43.0±10.1		0.808
Male/Female	10/27			25/71		0.909
	Pretreatment	**1 Year Later**	**p**	**Pretreatment**	**1 Year Later**	**p**
TSH (0.5-4.4 uIU/mL)	0.44±1.02	3.33±3.61	0.018	0.37±0.75	4.18±9.67	<0.001
FT3 (2-4.4 ng/dL)	6.72±3.19	2.86±0.63	<0.001	5.28±3.07	2.73±0.48	<0.001
FT4 (0.93-1.7 ng/dL)	2.77±1.44	1.15±0.35	<0.001	2.02±1.10	1.02±0.30	<0.001
Anti-Tg (<115 uIU/mL)	251.14±343.69	162.82±236.82	0.07	123.84±212.58	87.167±168.59	0.336
Anti-TPO (<34 uIU/mL)	8.01±5.15	7.20±3.98	0.054	31.43±97.63	24.63±77.97	0.112
Thyroglobulin (ng/dL)	461.69±902.45	126.16±244.41	0.052	257.82±233.93	35.131±33.62	0.024
ESR (<20 mm/h)	47.74±19.10	12.27±7.77	<0.001	49.06±25.96	16.05±17.22	<0.001
CRP (<0.5 mg/l)	7.47±6.38	1.12±2.8	0.007	13.64±32.66	0.86±1.75	0.001
NEUTROPHIL	6.10±1.67	4.04±1.55	0.011	5.94±2.13	4.00±1.32	<0.001
LYMPHOCYTE	2.20±0.78	2.04±0.77	0.520	2.32±0.716	2.24±0.52	0.402
MONO	0.651±0.26	0.537±0.15	0.027	0.77±0.24	0.76±0.17	0.653
PLATELETS	349.36±104.16	269.44±58.03	<0.001	309.20±77.40	271.70±67.93	<0.001
NLR (NEU/LYM)	2.91±1.63	1.85±0.76	<0.001	2.776±1.45	1.84±0.77	<0.001
PLR (PLT/LYM)	169.73±82.28	133.39±45.63	0.018	140.67±53.14	124.92±40.79	0.005
MLR (MONO/LYM)	0.32±0.18	0.27±0.12	0.145	0.32±0.16	0.22±0.09	<0.001
MPV	9.30±0.95	9.67±0.70	0.050	9.42±1.27	9.66±1.22	0.271

TSH: Thyrotropin, **FT3:** Free triiodothyronine, **FT4:** Free thyroxine, **Anti-TPO:** Antithyroid peroxidase antibodies, **Anti-Tg:** Anti-thyroglobulin antibodies, **NLR:** Neutrophil-to-lymphocyte ratio, **PLR:** Platelet-to-lymphocyte ratio, **MLR:** Monocyte-to-lymphocyte ratio, **MPV:** Mean platelet volume, **ESR:** Erythrocyte sedimentation rate, **CRP:** C-reactive protein (Data are shown as mean±standart deviation).

two groups the ESR and CRP levels were increased in the pretreatment period and decreased with the treatment (p<0.05). The pretreatment ESR level of the steroid group was lower than the other group (p<0.05). One year after the treatment the ESR level of the steroid group was significantly lower than the NSAID group (p<0.05). In the beginning the CRP levels of the two groups were increased and one year later the levels were decreased. There was no statistically difference between the groups (p>0.05).

There was a significant difference in neutrophil counts in both groups before and after treatment, respectively (p<0.05). There was no significant difference in lymphocyte counts before and after treatment in both groups (p>0.05). While there was a statistical difference in monocyte counts during the acute attack and 1 year later in the steroid group (p<0.05), no difference was found in the NSAID group (p>0.05). Platelet counts were found to be significantly higher in both groups during the attack (p<0.05). While NLR and PLR values were high during the attack in both groups, a significant decrease was observed in the values 1 year later (p<0.05). While the decrease in MLR was not significant in the steroid group after the treatment (p>0.05), the decrease in the value was significant in the NSAID group (p<0.05). No significant difference was observed in MPV values before and after the treatment in both groups (p>0.05).

Permanent Hypothyroidism

Permanent hypothyroidism observed in steroid and NSAID groups were 8/37 (21.6%), 5/97 (24.74%) in steroid and NSAID groups respectively (p>0.05).

Before treatment, there was no statistically significant difference between the CRP measurement levels (Yes=11.3±9.3 vs. No=6.2±4.2) between those with and without a diagnosis of permanent hypothyroidism in the steroid group (p=0.143). Similarly, no statistically significant difference was found between the CRP measurement levels (Yes=9.4±9.7 vs. No=15.2±10.3) in the NSAID group between those with and without a diagnosis of permanent hypothyroidism (p=0.230). After treatment, there was no statistically significant difference between the CRP measurement levels (Yes=1.3±3.2 vs No=0.5±0.3) in the steroid group between those with and without a diagnosis of permanent hypothyroidism (p=0.494). Similarly, no statistically significant difference was found between the CRP measurement levels (Yes=1.9±2.6 vs No=0.68±1.5) in the NSAID group between those with and without a diagnosis of permanent hypothyroidism (p=0.148).

Also, there was no statistical difference in other inflammation markers (CRP etc.) before and after treatment in both groups (steroid vs. NSAID) with and without a diagnosis of permanent hypothyroidism (p>0.05) (Table 2 and Table 3)

DISCUSSION

Subacute thyroiditis is a disease that usually develops after a viral infection and is characterized by inflammation in the thyroid gland.

The age of our patients was between 30-50 years old and female dominant. This situation was consistent with the publications in the literature (5,16).

Table 2: Comparison of pre-treatment inflammation markers in steroid and NSAID groups of patients with and without persistent hypothyroidism.

Variables	STEROID (n=37)		p	NSAID (n=96)		p
	Permanent hypothyroidism			Permanent hypothyroidism		
	No (n=29)	Yes (n=8)		No (n=72)	Yes (n=24)	
ESR (<20 mm/h)	47.0±18.6	52.5±24.3	0.504	49.01±25.6	65.8±41.5	0.271
CRP (<0.5 mg/l)	6.2±4.2	11.3±9.3	0.143	15.2±35.2	9.4±9.7	0.230
NEUTROPHIL	6.6±2.4	5.7±1.8	0.284	6.0±2.3	5.9±2.0	0.821
LYMPHOCYTE	2.2±0.8	2.4±0.7	0.485	2.4±0.83	2.4±0.36	0.905
MONO	0.69±0.21	0.64±0.17	0.556	0.70±0.26	0.76±0.39	0.447
PLATELETS	319.8±100.5	312.4±67.8	0.831	311.6±74.4	297.1±90.64	0.495
NLR (NEU/LYM)	3.6±3.0	2.6±1.2	0.323	2.8±1.6	2.5±0.84	0.436
PLR (PLT/LYM)	167.3±86.7	139.5±48.4	0.354	142.7±53.3	130.6±52.9	0.408
MLR (MONO/LYM)	0.34±0.11	0.29±0.12	0.272	0.32±0.16	0.32±0.17	0.935
MPV	9.2±1.3	8.6±1.5	0.277	8.9±1.5	9.3±1.21	0.304

TSH: Thyrotropin, **FT3:** Free triiodothyronine, **FT4:** Free thyroxine, **Anti-TPO:** Antithyroid peroxidase antibodies, **Anti-Tg:** Anti-thyroglobulin antibodies, **NLR:** Neutrophil-to-lymphocyte ratio, **PLR:** Platelet-to-lymphocyte ratio, **MLR:** Monocyte-to-lymphocyte ratio, **MPV:** Mean platelet volume, **ESR:** Erythrocyte sedimentation rate, **CRP:** C-reactive protein (Data are shown as mean±standart deviation).

Table 3: Comparison of post-treatment inflammation markers in steroid and NSAID groups of patients with and without persistent hypothyroidism.

Variables	STEROID (n=37)		p	NSAID (n=96)		p
	Permanent hypothyroidism			Permanent hypothyroidism		
	No (n=29)	Yes (n=8)		No (n=72)	Yes (n=24)	
ESR (<20 mm/h)	11.9±7.8	13.6±8.1	0.621	14.8±14.1	21.9±30.9	0.286
CRP (<0.5 mg/l)	1.3±3.2	0.5±0.3	0.494	0.68±1.5	1.9±2.6	0.148
NEUTROPHIL	4.2±1.4	3.9±1.5	0.637	3.9±1.3	4.0±1.1	0.738
LYMPHOCYTE	2.1±0.6	2.4±1.2	0.242	2.3±0.5	2.3±0.4	0.518
MONO	0.57±0.17	0.49±0.14	0.174	0.50±0.16	0.56±0.17	0.188
PLATELETS	281.5±81.1	269.1±60.9	0.664	268.7±69.9	283.1±56.9	0.442
NLR (NEU/LYM)	2.1±1.0	2.1±1.8	0.907	1.8±0.8	1.8±0.6	0.748
PLR (PLT/LYM)	143.9±59.2	137.0±80.5	0.777	124.9±42.2	125.1±33.8	0.981
MLR (MONO/LYM)	0.29±0.11	0.24±0.14	0.239	0.23±0.09	0.25±0.10	0.446
MPV	8.8±1.5	9.1±1.4	0.627	9.2±1.5	9.6±0.96	0.360

TSH: Thyrotropin, **FT3:** Free triiodothyronine, **FT4:** Free thyroxine, **Anti-TPO:** Antithyroid peroxidase antibodies, **Anti-Tg:** Anti-thyroglobulin antibodies, **NLR:** Neutrophil-to-lymphocyte ratio, **PLR:** Platelet-to-lymphocyte ratio, **MLR:** Monocyte-to-lymphocyte ratio, **MPV:** Mean platelet volume, **ESR:** Erythrocyte sedimentation rate, **CRP:** C-reactive protein (Data are shown as mean±standart deviation).

ESR and CRP are found to be high in SAT patients when evaluated together with clinical findings at the time of diagnosis. While these two parameters are found to be high in the active period of the disease, they decrease to normal levels with the decrease of inflammation. In the literature, ESR and CRP were found to be higher in SAT patients compared to control groups (5,7,13). There was no difference between the steroid group and the NSAID group during the illness and after 1 year. Since ESR and CRP are nonspecific parameters, it should be evaluated together with clinical findings for diagnosis in patients with SAT.

In inflammation, there are changes in cell distribution in the hemogram due to cytokines. While leukocytosis and neutrophil dominance occur in bacterial infections, leukopenia and lymphocytosis are observed in viral infections. While lymphocytosis is expected to be observed as a viral factor that is generally responsible for the etiology of SAT, neutrophil dominance was found in SAT patients in the study by Ergün and Tuzcu (5). The NLR ratio is a parameter that indicates systemic inflammation (17). In the study of Calapkulu et al., It was shown that NLR was higher in SAT patients compared to the control group and correlated with classical acute phase responses (ESR, CRP) (7). In our study, NLR was found to be significantly higher at the time of first diagnosis compared to the situation after 1 year, in parallel with inflammation. No significant difference was found between steroid users and NSAIDs. In the literature, MLR, PLR and MPV have been shown to be increased in cardiovascular, malignant and inflammatory diseases as markers of inflammation (17). In one study, NLR, PLR, and MLR also reported a significant increase in SAT patients. They found no difference in MPV compared to the control group (6). In another study, PLR and NLR also reported significantly higher values in SAT patients and significantly lower values in MPV (7). In the study of Ergün and Tuzcu, NLR was found to be higher in the SAT group compared to the control group, while MPV values were reported to be significantly lower in the SAT group (5). In our study, PLR values at the time of diagnosis of SAT were found to be significantly higher in both groups compared to the values 1 year later. There was no significant difference between the groups. In both groups, it was observed that MLR values were high during the period of high inflammation, and the values decreased after 1 year. This decrease was significant ($p<0.05$) in the steroid group, but not in the NSAID group ($p>0.05$). In our study, a non-significant increase was observed in MPV values in both groups.

In patients with SAT, thyroid hormone levels increase temporarily in blood tests due to damage to thyroid follicles and TSH levels also decrease. During this period, it is not necessary to give a drug that will suppress thyroid hormone synthesis. Symptomatic treatment is sufficient. The height of FT4 and FT3 has been reported in the literature, and the high ratio of FT4 / FT3 is also highlighted (5-7). Similar results were found in our study.

Thyroid autoantibodies can be detected mildly positive in patients with SAT. In general, Anti Tg was found to be higher positive. In the study of Ergün and Tuzcu, AntiTg was reported to be significantly different from Anti TPO compared to the control group (5). In the study of Taşkaldıran et al., autoantibodies were not found to be significantly higher (6). In our study, Anti Tg levels were found to be higher than Anti TPO and higher in the steroid group than in the NSAID group.

There are a limited number of studies in the literature on the treatment modality in SAT and the development of permanent hypothyroidism (10-12). Theoretically, it has been suggested that the severity of attacks in SAT may be an important factor affecting persistent hypothyroidism. However, the levels of inflammation markers indicating the severity of the attack or the width of the thyroiditis areas on ultrasound were evaluated, but no significant correlation was found. Similarly, no significant effect of the drugs used in the treatment was found on permanent hypothyroidism (11,16,18,19). In our study, in accordance with the clinical situation, inflammation markers were correlated with the parameters showing classical acute phase reaction at the time of diagnosis, but it was observed that they had no effect on determining the prognosis. Also in our patients, treatment drug option did not affect the permanent hypothyroidism one year after the first examination ($p>0.05$). In 2017, a similar publication was published by me as a letter to the editor in Acta Endocrinologica (Buc), using more limited data on inflammation markers and a smaller number of patients (20). 81 patients were examined in 3 groups (steroids, NSAIDs, steroids+NSAIDs) and no correlation was found between the drug and persistent hypothyroidism in that publication (20).

The limitation of our study is that the treatment drugs and doses are not homogeneous for all patients. So these should be taken into consideration because this is a retrospective study.

We think that the level of inflammatory markers and treatment options at the time of first diagnosis of the disease have no effect on the development of permanent hypothyroidism. However, prospective studies in more homogeneous groups are needed.

Acknowledgment

None.

Author Contributions

Concept: **Ali Saklamaz,** Design: **Ali Saklamaz,** Data collection or processing: **Ali Saklamaz, Özcan Çiftçi,** Analysis or Interpretation: **Ali Saklamaz,** Literature search: **Özcan Çiftçi,** Writing: **Ali Saklamaz,** Approval: **Özcan Çiftçi.**

Conflicts of Interest

The authors declare no conflict of interest.

Financial Support

None to declare.

Ethical Approval

The present study was approved by the Ethics Commitee of Izmir University of Economics, Faculty of Medicine Since the study was retrospective, informed consent was not obtained from the patients.

Review Process

Extremely peer-reviewed and accepted.

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