ORIGINAL ARTICLE

Presence of organ-specific antibodies in patients with systemic sclerosis

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KEY WORDS

antithyroid antibodies, autoimmune thyroid disease, systemic sclerosis

ABSTRACT

INTRODUCTION According to the literature, organ-specific antibodies may be present in the course of systemic sclerosis (SSc).

OBJECTIVE The aim of this study was to assess the prevalence of antithyroid antibodies (antithyroid peroxidase antibodies [anti-TPO] and antithyroglobulin antibodies) and of antimitochondrial antibodies (AMAs), as well as to evaluate their clinical significance in patients with SSc.

PATIENTS AND METHODS The study involved 86 consecutive in-hospital patients with SSc (32 patients with diffuse cutaneous SSc [dcSSc] and 54 with limited cutaneous SSc [lcSSc]). Patients were observed for autoimmune thyroid diseases (ATDs) and primary biliary cirrhosis (PBC). Serum samples were obtained from each patient.

RESULTS Positive antithyroid antibody titers were observed in 27 patients (31%) and positive AMA titers—in 11 patients (13%). ATD was diagnosed in 26 patients (30%) and PBC—in 10 patients (12%) with SSc. No significant differences in the prevalence of antithyroid antibodies were found between patients with dcSSc and those with lcSSc, but the prevalence of AMAs was significantly higher in patients with lcSSc compared with those with dcSSc. The prevalence of anti-Ro-52 antibodies was significantly higher in the SSc group with positive anti-TPO antibody titers compared with the SSc group with negative anti-TPO antibody titers. The prevalence of anticentromere antibodies (ACAs) was significantly higher in the SSc group with positive AMA titers compared with the SSc group with negative AMA titers.

CONCLUSIONS The prevalence of organ-specific antibodies in SSc patients is relatively high. The prevalence of AMAs is higher in patients with lcSSc than in those with dcSSc and is strongly associated with the presence of ACAs. Patients with SSc should be evaluated for coexisting ATDs and PBC.

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INTRODUCTION Systemic connective tissue diseases are characterized by the presence of non-organ-specific autoantibodies, directed against all tissues, and organ-specific autoantibodies, directed against individual organs. Polyautoimmunity is essential for the clinical picture as well as the course of connective tissue diseases and is still being investigated.[1,2] Numerous studies have described various clinical consequences resulting from the presence of organ-specific antibodies in patients with rheumatoid arthritis and Sjögren syndrome. The presence of these antibodies can significantly alter the course of the underlying diseases and enhance the symptoms of the musculoskeletal system, generalized joint or muscle pain, and fibromyalgia. Several studies have described the effects of various organ-specific antibodies on the course of rheumatic diseases.[1-4]

Systemic sclerosis (SSc) is a connective tissue disease characterized by vascular abnormalities, multiorgan fibrosis, and immune system alterations with overproduction of non-organ-specific antibodies.[5] SSc is known to be frequently accompanied by other non-organ- and organ-specific autoimmune disorders, as they share genetic and possibly environmental factors.[6,7] There are numerous studies describing the coexistence of autoimmune thyroid diseases (ATDs), autoimmune pancreatic, intestinal, and hepatic diseases, or primary biliary cirrhosis (PBC) with SSc.[8-13] ATD

TABLE 1 Characteristics of the study groups

Characteristic		SSc (n = 86)	dcSSc (n = 32)	lcSSc (n = 54)	*P* value
sex	female	69	23	46	NS
	male	17	9	8	
age, y		53.0 ±13.07 (19–81)	54.2 ±11.34 (22–81)	52.3 ±14.14 (19–77)	0.02
duration of diseases, y		6.8 ±6.30 (0.5–30)	5.43 ±4.94 (0.5–21)	7.61 ±7.01 (0.5–30)	NS

Data are presented as number of patients or mean ± SD (range).

Abbreviations: dcSSc, diffuse cutaneous systemic sclerosis; lcSSc, limited cutaneous systemic sclerosis; NS, nonsignificant; SSc, systemic sclerosis

is the most common organ-specific autoimmune disease, which affects about 1.5% of the population, mainly women, whereas PBC is a chronic and usually progressive liver disease with the presence of antimitochondrial antibodies (AMAs).[8,14,15] In some cases, organ-specific antibodies can be detected in SSc patients without or with minor signs and symptoms of thyroid, gut, pancreatic, or liver diseases.[13,15,16]

The antibodies that play the most important role in the pathogenesis of ATD are antithyroid peroxidase antibodies (anti-TPO), antithyroglobulin antibodies (anti-TG), and anti-thyroid-stimulating hormone (TSH) receptor antibodies (anti-TSHR).[8,16] Studies have demonstrated that antithyroid antibodies can affect the development of various manifestations of the locomotor system, such as arthralgia, fibromyalgia, or myalgia. Tagoe et al,[17] who studied 46 patients with chronic lymphocytic thyroiditis, reported polyarthralgia in 98%, fibromyalgia in 59%, Raynaud phenomenon in 28%, and dryness syndrome in 26% of the patients. Moreover, AMAs have been detected in some patients with SSc and were strongly associated with anticentromere antibodies (ACAs).[18] Several studies have reported an increase in antithyroid antibody or AMA titers in patients with SSc, but little is known about their connection with the clinical course and different manifestations of SSc.[8,10,15,16,19] The aim of this study was to assess the prevalence of antithyroid antibodies (anti-TPO and anti-TG) and AMAs in SSc patients. Moreover, since polyautoimmunity can modify the course of the underlying disease, we compared the presence of different manifestations of SSc in the group of patients with and without organ-specific antibodies.

PATIENTS AND METHODS The study included 86 consecutive patients with SSc (69 women and 17 men) hospitalized in the Department of Rheumatology and Connective Tissue Diseases, Medical University of Lublin, Lublin, Poland. Patients fulfilled the American College of Rheumatology classification criteria of SSc.[20] Seventeen patients had SSc overlaping on other connective tissue diseases (CTDs). Le Roy et al[21] classified patients as having limited cutaneous SSc (lcSSc) or diffuse cutaneous SSc (dcSSc) (TABLE 1). The prevalence of antithyroid antibodies (anti-TPO and/or anti-TG) and AMAs was determined in both groups of patients. Moreover, the different clinical and serological manifestations were assessed in the group of SSc patients with and without antithyroid antibodies or AMAs. The prevalence of ATD and PBC was determined in both groups. The data regarding the coexistence of ATD or PBC were obtained retrospectively based on available medical records and anamneses from previous hospitalizations. The available medical records were based on laboratory markers, including the levels of TSH, thyroid hormones, antithyroid antibodies, AMAs, and alkaline phosphatase, as well as on ultrasound examination and/or biopsy of the thyroid or liver.

All patients provided written informed consent to participate in the study according to the Declaration of Helsinki, and the ethical committee approval was obtained.

Organ involvement was assessed according to the clinical symptoms and results of diagnostic tests. Interstitial lung disease was defined as "a ground glass" pattern or bibasilar pulmonary fibrosis revealed on a high-resolution computed tomography scan. To interpret the pulmonary function, the DLCO test (% predicted diffusing capacity for carbon monoxide) was performed.[22] Heart involvement was established as arrhythmia, conduction disturbances, or heart failure. Pulmonary arterial hypertension was defined as systolic pulmonary arterial pressure exceeding 35 mmHg on Doppler echocardiography.[23] Myalgia or myositis was assessed as pain or weakness in muscles or increased serum creatine phosphokinase levels. Joint involvement was considered as joint tenderness and swelling. Gastrointestinal tract involvement was defined according to the clinical symptoms such as dysphagia, heartburn, diarrhea, or bloating and was examined by a barium swallow. Renal involvement was defined as the development of scleroderma renal crisis or presence of proteinuria and elevated creatinine levels. Calcinosis and digital erosions were also assessed. The clinical data of the study groups are presented in TABLE 2. Serum samples were obtained from each patient. Anti-TPO and anti-TG antibodies were detected using direct chemiluminescence methods on Advia Centaur XP Systems (Siemens Healthcare, Warszawa, Poland). The samples were classified as positive when exceeding 60 IU/ml for anti-TPO and anti-TG antibodies. AMAs were assessed using the immunoblotting method.

TABLE 2 Prevalence of clinical and serological manifestations in patients with systemic sclerosis (n = 86)

calcinosis		21 (24)
digital ulcers		15 (17)
gastrointestinal tract involvement		55 (64)
interstitial lung disease		42 (49)
primary heart involvement		31 (36)
PAH on echocardiography		17 (20)
kidney involvement		23 (27)
SRC		2 (2)
joint involvement	arthralgia	71 (83)
	arthritis	31 (36)
myalgia or myositis		17 (20)
overlap syndrome		17 (20)
anti-centromere antibodies		19 (22)
anti-Scl-70 antibodies		29 (34)

Data are presented as number (percentage) of patients.

Abbreviations: PAH, pulmonary arterial hypertension; SRC, scleroderma renal crisis

TABLE 3 Prevalence of antithyroid antibodies and autoimmune thyroid diseases in the study groups

Parameter	SSc (n = 86)	dcSSc (n = 32)	lcSSc (n = 54)	*P* value
anti-TPO	22 (26)	8 (25)	14 (26)	NS
anti-TG	22 (26)	9 (28)	13 (24)	NS
anti-TPO or anti-TG	27 (31)	11 (34)	16 (30)	NS
anti-TPO and anti-TG	17 (20)	6 (19)	11 (20)	NS
ATD	26s (30)	10 (31)	16 (30)	NS

Data are presented as number (percentage) of patients.

Abbreviations: anti-TG, antithyroglobulin antibodies; anti-TPO, antithyroid peroxidase antibodies; ATD, autoimmune thyroid disease; others, see TABLE 1

Moreover, the study groups were tested according to the presence of antibodies applying the commercial test, EUROLINE Systemic Sclerosis Profile (EUROIMMUN, Lübeck, Germany), which is used to determine antibodies against SSc-specific antigens, such as antitopoisomerase I (anti-Scl-70) and ACAs, anti-RNA polymerase III (anti-RNA pol III), rarer anti-PM/Scl, anti-Ku, anti-Th/To anti-Ro52, and autoantibodies against nucleolus-organizing region-90 (anti-NOR90). Detection and interpretation of the results were performed electronically using the Euroimmun–EUROLINE Scan. All calculations were performed with Statistica 10.0 PL (StatSoft, Kraków, Poland). Data were analyzed using the χ^2 test for comparisons between groups. A *P* value of less than 0.05 was considered statistically significant.

RESULTS Our study showed that 27 patients (31%) had positive antithyroid antibody (aTPO or anti-TG or both) titers in the SSc group. There were no significant differences in the prevalence of antithyroid antibodies between the dcSSc and lcSSc groups. ATD was diagnosed in 26 patients (30%) with SSc. Moreover, we did not observe any significant differences in the presence of ATD between the dcSSc and lcSSc groups (TABLE 3). Furthermore, in the group of 26 patients with ATD, 2 patients had hypothyroidism, 7 patients had subclinical hypothyroidism, 1 patient had hyperthyroidism, and 4 patients had subclinical hyperthyroidism. As for the clinical manifestations of SSc, no significant differences in the prevalence of lung, heart, gastrointestinal tract, kidney, or muscle and joint involvement were observed between the SSc group with positive antithyroid antibody titers and the SSc group with negative antithyroid antibody titers. The results are presented in TABLE 4.

Interestingly, the prevalence of anti-Ro-52 antibodies was demonstrated to be significantly higher in the SSc group with positive anti-TPO antibody titers, compared with the SSc group with negative anti-TPO antibody titers (52.9% vs 22.0%, $\chi^2 = 5.49$; $P = 0.02$). No significant differences in the prevalence of anti-Scl-70, ACAs, anti-Pm/Scl, anti-RNA pol III, anti Th1/Th0, anti-Ku, and anti-NOR 90 antibodies were found between the study groups. Moreover, 11 patients (13%) with SSc had positive AMA titers. Of note, the prevalence of AMAs was demonstrated to be significantly higher in the lcSSc group compared with the dcSSc group ($P = 0.004$). Additionally, PBC was diagnosed in 10 patients with SSc (12%). The presence of PBC was found to be more common in the lcSSc group compared with the dcSSc group ($P = 0.05$) (TABLE 5). Furthermore, of 10 patients with SSc and PBC, 8 patients had clinically silent course of PBC (apart from elevated cholestatic enzyme levels) and 2 patients developed cirrhosis and chronic liver failure.

No significant differences in the prevalence of lung, heart, gastrointestinal tract, kidney, or muscle and joint involvement were observed between the SSc group with positive AMA titers and the SSc group with negative AMA titers. Data are presented in TABLE 6. Interestingly, the prevalence of ACAs was demonstrated to be significantly higher in the SSc group with positive AMA titers compared with the SSc group with negative AMA titers (64% vs 16%, $\chi^2 = 7.3$; $P = 0.007$). No significant differences in the prevalence of anti-Scl-70, Ro-52, anti-Pm/Scl, anti-RNA pol III, anti-Th1/Th0, anti-Ku, and anti-NOR 90 antibodies were found between the study groups. The SSc patients with positive AMAs were found to have an overlap of more than one CTD significantly more frequently, as compared with the SSc group without AMAs (55% vs 15%, $\chi^2 = 4.2$; $P = 0.04$). Additionally, the prevalence of PBC was observed to be significantly higher in SSc patients with positive anti-TPO antibody titers, as compared with SSc patients with negative anti-TPO antibody titers (22.7% vs 6.8%, $\chi^2 = 4.12$, $P = 0.04$).

TABLE 4 Clinical and serological differences between patients with systemic sclerosis with positive and negative antithyroid antibody titers

Parameter		SSc group		*P* value
		anti-TPO/anti-TG positive (n = 27)	anti-TPO/anti-TG negative (n = 59)	
calcinosis		8 (30)	13 (22)	NS
digital ulcers		4 (15)	11 (19)	NS
gastrointestinal tract involvement		18 (67)	37 (63)	NS
interstitial lung disease		16 (53)	26 (44)	NS
primary heart involvement		9 (33)	22(37)	NS
PAH (echocardiography)		6 (22)	11 (19)	NS
kidney involvement		7 (25)	16 (27.1)	NS
SRC		0	2 (3)	NS
joint involvement	arthralgia	23 (85)	48 (81)	NS
	arthritis	10 (37)	21 (36)	NS
overlap syndromes		7 (25)	10 (16)	NS
myalgia or myositis		5 (19)	12 (20)	NS
anti-Scl-70 antibodies		11 (41)	18 (33)	NS
anticentromere antibodies		6 (22)	13 (16)	NS

Data are presented as number (percentage) of patients.

Abbreviations: see TABLES 1 and 2

TABLE 5 Prevalence of antimitochondrial antibodies and primary biliary cirrhosis in the study groups

Parameter	SSc (n = 86)	dcSSc (n = 32)	lcSSc (n = 54)	*P* value
AMA	11 (13)	1 (3)	10 (18)	0.04 (dcSSc vs lcSSc)
PBC	10 (12)	1 pts (3)	9 (17)	0.05 (dcSSc vs lcSSc)

Data are presented as number (percentage) of patients.

Abbreviations: AMA, antimitochondrial antibodies; PBC, primary biliary cirrhosis; others, see TABLE 1

DISCUSSION The presence of organ-specific antibodies, such as antithyroid antibodies or AMAs, in the course of CTDs, has been documented in numerous studies.[10,14,16,24-26] Moreover, one autoimmune disease was often associated with one or more diseases, suggesting common immunological dysfunction in the pathogenesis of the diseases. The same applies to ATDs and their association with other CTDs. Although ATDs, such as Hashimoto disease or Graves disease, have been reported with different autoimmune diseases, Hashimoto thyroiditis has been observed more frequently. Hashimoto thyroiditis or the presence of antithyroid antibodies (or both) occurs in rheumatoid arthritis, Sjögren syndrome, systemic lupus erythematosus, mixed connective tissue disease, or SSc.[7,9,14,16,27,28] According to different reports, the prevalence of antithyroid antibodies ranged from 30% to 38%, yet the diagnosis of ATD was established in 15% to 20% of SSc patients.[6,9,16,28-31] In addition, Danielides et al[16] have found a significant increase in anti-TPO antibody titers only in patients with lcSSc, as compared with healthy controls (32.6% vs 14%, $P = 0.003$). Moreover, SSc patients with positive anti-TPO titers had a significantly higher incidence of the HLA DR15 haplotype than those who were anti-TPO-negative. Therefore, HLA DR15 may represent the immunogenic marker of anti-TPO production in patients with SSc.[6,32] Moreover, genetic variation in the gene for cytotoxic T-lymphocyte antigen-4 (CTLA-4) has been found to be associated with ATD.[1,33]

There is little information about the association of anti-TPO or anti-TG antibodies with the clinical course and various manifestations of SSc. There is some evidence for the incidence of generalized locomotor pain, fibromyalgia, muscle and spinal pain, or even polyneuropathy is higher in patients with antithyroid antibodies. Other accompanying symptoms include fatigue, alopecia, Raynaud phenomenon, or dryness.[1,2] Toki et al,[8] who studied 210 patients with SSc, identified 30 patients with ATD (14.3%), including 29 with Hashimoto disease (13.8%) and 1 patient with Graves disease (0.5%). Moreover, they found that all patients with ATD were female, and AMA positivity, complications of Sjögren syndrome, severe facial skin sclerosis, and atrophy of the thyroid gland were significantly more prevalent in SSc patients with ATD. Furthermore, Ugurlu et al[31] revealed that anti-TPO and anti-TG antibodies

TABLE 6 The clinical and serological differences between SSc patients with positive and negative antimitochondrial antibody titers

Parameter		SSc group		*P* value
		AMA positive (n = 11)	AMA negative (n = 75)	
calcinosis		3 (27)	18 (24)	NS
digital ulcers		2 (18)	13 (17)	NS
gastrointestinal tract involvement		6 (55)	49 (65)	NS
interstitial lung disease		5 (45)	37 (49)	NS
primary heart involvement		4 (36)	27 (36)	NS
PAH (echocardiography)		2 (18)	15 (27)	NS
kidney involvement		1 (9)	22 (29)	NS
SRC		0	2 (3)	NS
joint involvement	arthralgia	8 (73)	63 (84)	NS
	arthritis	4 (36)	27 (36)	NS
overlap syndromes		6 (55)	11 (15)	0.04
myalgia or myositis		3 (27)	14 (19)	NS
anti-Scl-70 antibodies		4 (36)	25 (33)	NS
anticentromere antibodies		7 (64)	12 (16)	0.007

Data were presented as number (percentage) of patients.

Abbreviations: see **TABLE 5**

significantly correlated with a-Scl-70 antibodies ($P = 0.003$; $P < 0.001$).

Our results are comparable with the literature data regarding the prevalence of antithyroid antibodies in SSc; 31% of SSc patients in our study had positive antithyroid antibody titers, and ATD was diagnosed in 30% of SSc patients. In contrast to the literature data, we did not find differences in the prevalence of antithyroid antibodies or ATD between the dcSSc and lcSSc groups. Moreover, we did not observe differences in the clinical course of SSc between the SSc group with positive antithyroid antibodies and the SSc group with negative antithyroid antibodies.

It has been reported that the symptoms from musculoskeletal system are more common in SSc patients with antithyroid antibodies.[1-3] Our findings did not reveal such differences. As for serological markers, it was interesting that the presence of anti-Ro 52 antibodies was higher in the group with positive anti-TPO antibody titers. No such data are available in the literature and there are only few reports referring to the presence of anti-Ro 52 antibodies in patients with positive ACA titers.[34,35] It cannot be excluded that the presence of a-Ro antibodies could be associated with Sjögren syndrome, which has been found to be more common in patients with anti-TPO antibodies.[1-4]

Dry eye syndrome is a frequent manifestation in SSc patients. Waszczykowska et al[36] described different ocular manifestations in SSc and concluded that dry eye syndrome, cataract, increased intraocular pressure, and vascular abnormalities are more common in SSc patients, as compared with the control group.

The association between autoimmune liver diseases and SSc has been described in many studies. PBC is the most frequent autoimmune liver disease in SSc patients.[18,19] Various studies have reported that PBC occurred in about 10% to 15% of SSc cases, most commonly in the lcSSc subtype.[10,15,29] Furthermore, SSc predicted the diagnosis of PBC in 59% of patients.[29,34] PBC mostly accompanied lcSSc and generally was clinically silent, despite elevated cholestatic enzyme levels and the presence of AMAs.[29] AMAs are detected in 10% to 20% of patients with SSc and are strongly associated with ACAs and the lcSSc subtype.[12,15,19] Additionally, the incidence of ACAs is higher in SSc coexisting with PBC than in lcSSc alone. Positive ACA titers in patients with PBC indicate the risk of future lcSSc.[15,29] Our results are similar to the literature data. In our study, PBC was diagnosed in 12% and AMAs in 13% of patients with SSc. We also found that PBC and AMAs were more common in the lcSSc group compared with the dcSSc group. Moreover, the prevalence of ACAs was demonstrated to be higher in the SSc group with positive AMA titers, compared with the SSc group with negative AMA titers. Imura-Kumada et al,[10] who studied 225 SSc patients, demonstrated that 37 of them (16.4%) had AMAs and 22 (9.8%) had PBC. They also revealed that AMAs and ACAs were independently associated with PBC in SSc patients and that AMAs and ACAs are likely to indicate an increasing risk of PBC in SSc patients.[10] Liberal et al[18] found ACAs in up to 30% of patients with PBC and 80% of patients with PBC/SSc.

The literature reports comparing the clinical course of SSc in the groups of SSc patients with and without AMAs are sparse. Cavazzana et al[12] reported that teleangiectasia and calcinosis were less frequent in the group of SSc patients with AMAs, as compared with SSc patients without

AMAs. On the other hand, another study indicated that PBC had a slower progression and better prognosis in SSc/PBC compared with PBC alone.[29] Our findings did not demonstrate the differences in the clinical course of SSc between the SSc groups with positive and negative AMAs. Interestingly, SSc patients with positive AMA titers frequently presented more than one CTD, as compared with SSc patients with negative AMA titers. It is well known that the coexistence of SSc with organ-specific autoimmune disease could predispose to other CTDs.[7,29] In some cases, 3 or more CTDs overlap or some other nonrheumatic autoimmune diseases occur.[30,37] Moreover, it has been reported that ACAs indicate a subset of more severe primary Sjögren syndrome with a unique phenotype, including the features of lcSSc and a lower frequency of anti-Sjögren syndrome-related antigen A/antigen B antibodies.[38]

In conclusion, the prevalence of organ-specific antibodies, such as antithyroid antibodies and AMAs in SSc, is relatively high. The prevalence of AMA is higher in the lcSSc group, as compared with the dcSSc group. Moreover, AMAs are strongly associated with ACAs. Considering the above, patients with SSc should be evaluated for coexisting ATDs and PBC. Polyautoimmunity can markedly alter the course of the disease, affect the development of various noncharacteristic locomotor symptoms, and modify the disease activity, thus complicating the therapeutic process. Therefore, extended diagnostic screening for accumulating autoimmune diseases with organ-specific antibodies seems reasonable in patients with SSc. Moreover, it is particularly important to search for organ-specific antibodies in cases with rare symptoms, which are not characteristic of the underlying disease and markedly alter its course. Atypical symptoms are likely to be associated with the presence of organ-specific antibodies. Our findings require further observation and assessment of the effects of antithyroid antibodies and AMAs on the course of disease and long-term prognosis.

Contribution statement EW and MM conceived the idea for the study. MM and EW contributed to the design of the research. All authors were involved in data collection. EW, MD, AK, and JT analyzed the data. JT was involved in statistical analysis. EW, AK, and MM were involved in data interpretation. EW and MM were involved in preparing the final revision of the manuscript. MM coordinated funding for the project. All authors edited and approved the final version of the manuscript.

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Obecność przeciwciał swoistych narządowo u chorych na twardzinę układową

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SŁOWA KLUCZOWE

autoimmunologiczne choroby tarczycy, przeciwciała przeciwtarczycowe, twardzina układowa

STRESZCZENIE

WPROWADZENIE Według danych z piśmiennictwa w przebiegu twardziny układowej (TU) mogą występować przeciwciała swoiste narządowo.

CELE Celem pracy było określenie częstości występowania przeciwciał przeciwtarczycowych (przeciwko tyreoperoksydazie [*antithyroid peroxidase antibodies* – anty-TPO] oraz przeciwko tyreoglobulinie) i przeciwmitochondrialnych, a także ich znaczenia klinicznego u chorych na TU.

PACJENCI I METODY Obserwacją objęto 86 kolejnych hospitalizowanych pacjentów z TU (32 pacjentów z uogólnioną i 54 z ograniczoną postacią TU). Pacjentów obserwowano pod kątem występowania autoimmunologicznych chorób tarczycy (ACHT) i pierwotnej marskości żółciowej (PMŻ). Próbki krwi pobierano od każdego pacjenta.

WYNIKI U 27 pacjentów (31%) stwierdzono dodatnie przeciwciała przeciwtarczycowe, a u 11 (13%) – przeciwciała przeciwmitochondrialne. ACHT zdiagnozowano u 26 pacjentów (30%), a PMŻ – u 10 pacjentów (12%) z TU. Nie stwierdzono różnic pod względem częstości występowania przeciwciał przeciwtarczycowych między pacjentami z uogólnioną i ograniczoną postacią choroby, jednak częstość występowania przeciwciał przeciwmitochondrialnych była istotnie wyższa u chorych na ograniczoną TU w porównaniu z chorymi z uogólnioną postacią choroby. Częstość występowania przeciwciał anty-Ro-52 była wyższa u chorych na TU z obecnością przeciwciał anty-TPO w porównaniu z grupą bez obecności anty-TPO. Częstość występowania przeciwciał antycentromerowych była istotnie wyższa u chorych na TU z obecnością przeciwciał przeciwmitochondrialnych.

WNIOSKI Częstość występowania przeciwciał przeciwtarczycowych u chorych na TU jest relatywnie wysoka. Częstość występowania przeciwciał przeciwmitochondrialnych jest wyższa u chorych z ograniczoną postacią TU niż u chorych z uogólnioną postacią TU i ściśle wiąże się z obecnością przeciwciał przeciwcentromerowych. Pacjentów z TU powinno się diagnozować pod względem współistnienia ACHT i PMŻ.

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