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Coexistence of Systemic Lupus Erythematosus and Papillary Thyroid Carcinoma: A Case Report

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Abstract:

Introduction: Autoimmune diseases, including systemic lupus erythematosus (SLE), are associated with an increased risk of developing malignant tumors. Thyroid cancer is the most common cancer of the endocrine system. It has been shown that SLE increases the risk of thyroid cancer by approximately twofold, especially in young women. Possible causes of this association include the relationship between autoimmunity and carcinogenesis, high disease activity, the impact of immunosuppressive treatment and the appearance of connective tissue disease symptoms during the neoplastic process (paraneoplastic syndrome).

Case report: We present the case of a 40-year-old female SLE patient who has been treated with multiple immunosuppressive drugs, including glucocorticosteroids (gcs), hydroxychloroquine, azathioprine, methotrexate, mycophenolate mofetil (MMF), and epratuzumab (anti-CD22). Complete remission of the disease was not achieved. The clinical presentation was characterised by skin changes indicative of subacute cutaneous lupus erythematosus, as well as vasculitis, arthritis, weight loss and haematological abnormalities.

In 2024, an ultrasound examination of the thyroid gland revealed a focal lesion in the right lobe and a thyroid biopsy raised suspicion of papillary carcinoma. A total thyroidectomy was performed, followed by the administration of an ablative dose of J-131 and suppressive doses of L-thyroxine. Several weeks after surgery, a significant improvement in the skin lesions was observed. Symptoms of SLE remission persist to date.

In summary, any systemic connective tissue disease, including SLE, requires oncological vigilance. The coexistence of SLE and differentiated thyroid cancer is rare. Periodic testing for antithyroid antibodies and ultrasound examinations of the thyroid gland are recommended for all patients with SLE.

Keywords:

papillary thyroid cancer, paraneoplastic syndrome, systemic lupus erythematosus

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Introduction

Systemic lupus erythematosus (SLE) is a chronic, multi-organ autoimmune inflammatory disease. Its pathogenesis is complex and involves genetic factors, environmental influences, and immune system dysfunction. Disease symptoms can affect any organ, most commonly the joints, skin, kidneys, bone marrow, nervous system, and cardiovascular system [1]. Skin involvement occurs in 80-85% of SLE patients. Cutaneous lesions are acute, subacute, or chronic and most often appear on skin exposed to UV radiation. Subacute cutaneous lupus erythematosus (SCLE) manifests as annular or papulosquamous lesions that in many cases mimic psoriasis [2].

Paraneoplastic syndrome refers to a clinical syndrome accompanying or preceding malignancy, resulting from bioactive substances secreted by tumor cells, but not related to the primary or metastatic tumor location. Paraneoplastic syndromes affect various systems and organs, including the gastrointestinal tract, nervous system, musculoskeletal system, and skin. Among paraneoplastic rheumatologic syndromes, dermatomyositis (DM), polymyositis, scleroderma-like syndromes, and vasculitis hold particular significance. Systemic lupus erythematosus is a rare paraneoplastic syndrome [3-7].

In this paper, a case of a patient with SLE and papillary thyroid cancer is presented, and possible causes of this association are analyzed.

Case report

A 40-year-old woman was diagnosed with SLE in 2013 based on clinical and laboratory features like: malar rash, photosensitivity, oral mucosal erosions, alopecia, non-erosive arthritis involving the proximal interphalangeal and metacarpophalangeal joints, leukopenia with lymphopenia, hypocomplementemia, elevated antinuclear antibody titer of 1:640 with a speckled pattern, and positive anti-SSA/Ro (+++), anti-SSB/La (++), anti-Ro-52 (+++), and anti-dsDNA antibodies (Table 1). Initial therapy consisted of chloroquine (250 mg daily) and low-dose prednisone (5 mg daily), yielding partial clinical response. Over the next five years, recurrent flares of polyarthritis and facial rash prompted multiple rheumatology consultations. Maintenance therapy with chloroquine or hydroxychloroquine was continued, supplemented by intermittent high-dose intravenous methylprednisolone pulses. Azathioprine was briefly used but discontinued due to elevated transaminases. The patient was also treated with methotrexate and epratuzumab (anti-CD22). Partial disease remission was achieved, and persisted 2018.

In 2019, an exacerbation of SLE occurred, manifesting as intensified photosensitive rash across the face, neck, and décolletage, accompanied by alopecia, arthralgia and myalgia (Figure 1). High-dose intravenous methylprednisolone was administered, followed by initiation of mycophenolate mofetil (MMF). Only partial remission of skin lesions occurred, with reduction in joint and muscle pain.

In 2023, the patient required rehospitalization for severe SCLE, recurrent mucosal erosions, alopecia, cutaneous vasculitis, and significant weight loss (Figure 2). Thyroid ultrasonography identified a focal lesion in the right lobe (TIRADS 5). Fine-needle aspiration biopsy raised suspicion of papillary thyroid carcinoma (PTC). Total thyroidectomy, performed in 2024, confirmed classic variant PTC (pT1bN0M0). Postoperative management included radioiodine ablation (I-131) and suppressive levothyroxine therapy. Surveillance thyroid scintigraphy and thyroglobulin levels, assessed several months post-ablation, demonstrated an excellent response according to American Thyroid Association criteria.

Notably, within weeks of thyroidectomy, significant improvement in skin lesions and weight gain were observed (Figure 3). Clinical remission of SLE has been maintained to the present. Current

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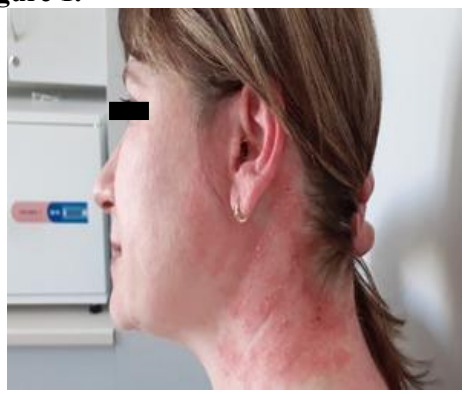
maintenance regimen comprises hydroxychloroquine (200 mg daily) and MMF (1 g daily), without glucocorticosteroids.

Table 1: Immunological test results for the patient presented

Immunological tests	Results
ANA	The titer 1:640, speckled pattern SS-A(+++), SS-B (+++), Ro-52 (+++)
RF-IgM	positive
aCCP	negative
anty-dsDNA (ELISA assay)	negative
cANCA	negative
pANCA	negative
Cryoglobulins	negative
aTPO	negative
aTG	negative
TRAb	negative
C3 complement (mg/dl) (range 82-93)	78
C4 complement (mg/dl) (range 12-16)	10

ANA: antinuclear antibodies; RF-IgM: rheumatoid factor in IgM class; aCCP: anti-cyclic citrullinated peptide antibodies; anti-dsDNA: anti-double stranded DNA antibodies: cANCA: cytoplasmic antineutrophil cytoplasmic antibodies; pANCA: perinuclear antineutrophil cytoplasmic antibodies: aTPO: anti-thyroid peroxidase antibodies: aTG: anti-thyroglobulin antibodies: TRAb: TSH receptor antibodies

Figure 1.

A 

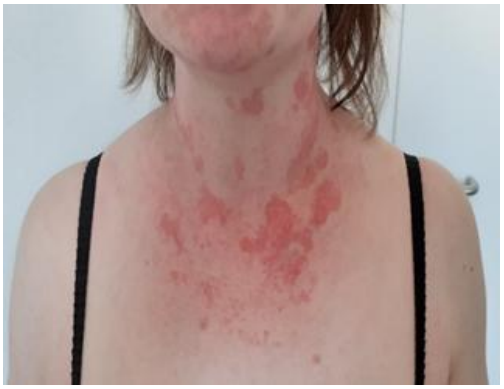
B 

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Figure 2

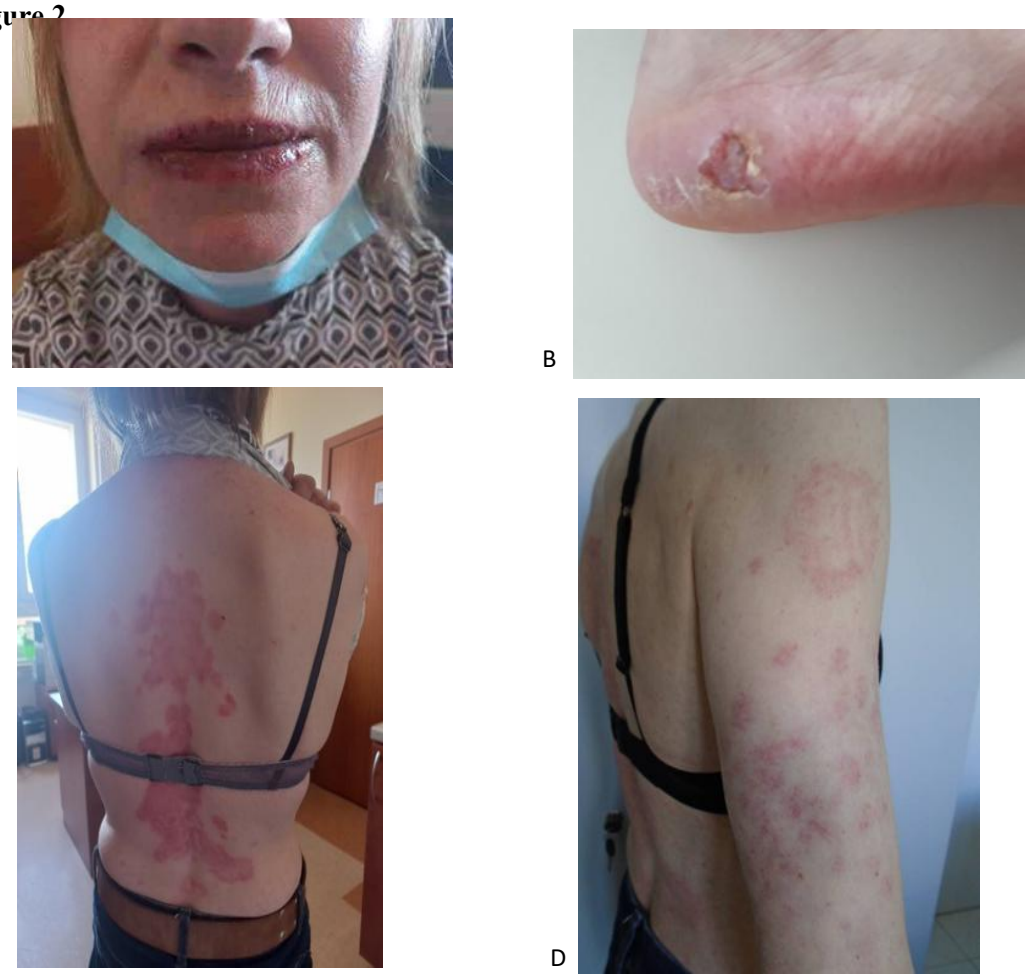
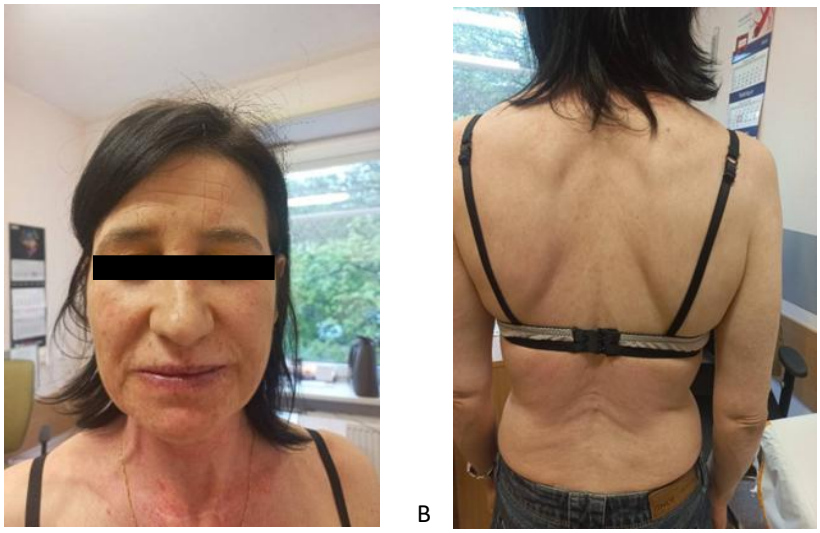


Figure 3.



Discussion

This case report presents a patient with long-standing SLE and predominant involvement of the skin, joints and mucous membranes. Despite treatment with various immunosuppressants (chloroquine/hydroxychloroquine, methotrexate, mycophenolate mofetil, anti-CD22), complete remission of symptoms was not achieved. Diagnosis and surgical resection of papillary thyroid carcinoma were associated with substantial

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improvement in mucocutaneous lesions, resolution of arthralgia, and significant weight gain.

Previous studies have demonstrated a higher incidence of malignancies in SLE patients compared to the general population [8]. A meta-analysis of 48 cohort studies (247575 SLE patients) identified SLE as a risk factor for 17 cancers, including lung, laryngeal, vaginal, cervical, anal, lymphoma, leukemia, and thyroid cancer [9]. These data were consistent with previous studies, which also demonstrated an increased risk of malignancies in SLE patients [10-13].

Thyroid cancers represent the most common endocrine malignancies [14]. Pathogenesis involves genetic factors (MAP kinase pathway activation, PI-3K/AKT signaling mutations, RET proto-oncogene mutations), environmental influences, and autoimmune thyroid diseases [15-18]. Papillary thyroid carcinoma is the most common histopathological subtype of thyroid cancer (85%), characterized by slow growth and favorable prognosis. It can occur at any age, with peak incidence observed between 30 and 50 years. The most frequent clinical manifestation is a painless thyroid nodule and enlargement of regional lymph nodes [15,19].
Most reviewed studies report approximately a twofold increased thyroid cancer risk in SLE patients. Jun et al. showed that the prevalence of thyroid cancer in SLE patients was 1.81%, which was significantly higher than that of the control group (1.30% ; $p< 0.001$) [11,12,17,20].

Thyroid cancer in SLE patients most commonly occurred in young women under 50 years of age with the majority of cases being papillary thyroid carcinoma [10,11,21,22].

Increasing evidence highlights the association between autoimmunity and oncogenesis, with numerous data indicating shared pathogenetic mechanisms and overlapping molecular pathways that promote both processes [23]. A key factor facilitating understanding of the increased incidence of thyroid cancer in patients with connective tissue diseases is that autoimmune thyroid diseases represent risk factors for thyroid cancer [24]. SLE exhibits a strong association with autoimmune thyroid diseases. Antithyroid antibodies, including anti-thyroid peroxidase (anti-TPO) and anti-thyroglobulin (anti-TG), are present in 20-45% of SLE patients [12,25]. Both SLE and autoimmune thyroid diseases are characterized by increased interferon-gamma activity, suggesting shared immunopathogenetic mechanisms [12]. Boi et al. demonstrated an association between increased papillary thyroid carcinoma incidence and the presence of antithyroid antibodies and elevated thyrotropin (TSH) levels [26]. Antonelli et al. found that 80% of SLE patients with papillary thyroid cancer had concomitant autoimmune thyroid disease, whereas only 30% of SLE patients without thyroid malignancy had thyroid autoimmunity [21]. Autoimmune thyroid disease was excluded in the case presented in this report.
High SLE activity may contribute to elevated thyroid cancer risk [24,27]. According to some authors, enhanced immune activation leads to persistent inflammation, chronic oxidative stress, and DNA damage, promoting malignant transformation [28]. This hypothesis could explain malignancy emergence in our patient with long-standing active SLE.

Liu et al. and Sun et al. offered novel insights into SLE and thyroid cancer through Mendelian randomization analysis, demonstrating that genetic variants associated with SLE confer increased thyroid cancer risk. This association was observed in European populations but not in East Asian cohorts. Researchers developed a thyroid cancer prognostic index (SLEscore) based on four SLE-related genes (IFITM1, RAP1GAP, MT1A, ALAS2) [29,30].
Immunosuppressive drugs represent another potential cause of increased malignancy risk in SLE patients. Cyclophosphamide particularly elevates lymphoma and bladder cancer risk [31,32]. A study by Cho et al. found no statistically significant association between immunosuppressive therapy and cancer in SLE patients [33].

Certain malignancies present with paraneoplastic syndromes that mimic rheumatic diseases, particularly SLE features [24]. SCLE-like skin lesions most commonly co-occur with lung, esophageal, and oral cavity cancers [4,5,7]. Abbott et al. describes a case of a patient with small-cell lung carcinoma who developed SCLE-like rash and extensive painful oral and nasal ulcers and esophageal mucositis [5]. Another study describes a case of a patient who achieved complete remission

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of nephrotic syndrome in the course of SLE following surgical resection of colorectal cancer [3].

To date, singular cases of paraneoplastic syndromes have been reported in patients with papillary thyroid carcinoma, including polymyalgia rheumatica [34], polymyositis [35], dermatomyositis [36], Still's disease [37] and vasculitis [6].

In the case of the described patient, SLE symptoms remitted following thyroid cancer resection, suggesting a possible paraneoplastic etiology of SLE. To date, no similar case has been reported in the literature. The influence of SLE activity and administered treatment on malignancy development cannot be definitively excluded.

Conlusions

Coexistence of SLE and thyroid cancer occurs rarely. Autoimmune thyroid diseases increase thyroid cancer risk, therefore, periodic antithyroid antibody testing and thyroid ultrasound are recommended for all SLE patients. Subacute cutaneous lupus erythematosus that is resistant to treatment may be associated with cancer. All autoimmune diseases, including SLE, require oncologic vigilance.

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