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Paraneoplastic Systemic Lupus Erythematosus with Anti-ribosomal P Antibodies in Association with Colon Cancer: Case Report

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Abstract:

Systemic lupus erythematosus (SLE) is a chronic, multi-organ inflammatory disease of autoimmune origin. In some cases, SLE may be the first symptom of cancer. In rheumatology, paraneoplastic syndromes manifest as symptoms of inflammation of joints, muscles, and blood vessels caused by cancer, but not directly related to tumor invasion or the presence of metastases. SLE as a paraneoplastic syndrome has been observed in patients with lymphoma, breast cancer, lung cancer, and rarely gastrointestinal cancer. Here, we present the case of a 69-year-old man with an inflammatory rash on his face, neck, upper limbs and back, as well as oral ulcers, arthritis, and weight loss. Laboratory tests revealed pancytopenia, low complements C3 and C4, and positive antinuclear and anti-ribosomal protein P antibodies. Thoracic, abdominal and pelvic CT scans showed pleural effusion, a single enlarged mediastinal lymph node, irregular thickening of the splenic flexure of the colon and splenomegaly. Colonoscopy revealed a polypoid lesion in the distal transverse colon and descending colon; histopathological examination showed adenocarcinoma NOS low grade. Treatment consisted of high - dose glucocorticoids and hydroxychloroquine. As a result of the treatment, the skin and mucosal lesions, as well as the arthritis, significantly decreased. In December 2025, a partial colon resection was performed. Since the operation, there have been no signs of SLE activity. Any systemic connective tissue disease, including SLE, can be a paraneoplastic syndrome. To date, only one case of paraneoplastic SLE has been reported in patients with colon cancer. Particular attention should be given to the possibility of cancer in SLE cases involving severe skin lesions, general symptoms and an older age at the onset. Anti-ribosomal P antibodies are mainly known as highly specific markers for SLE, but research has indicated they can also be elicited in cancers, potentially resulting from tumor-induced immunogenicity. Many studies indicate that ribosomal P protein or antibodies to P protein may become a new potential diagnostic, prognostic biomarker or promising therapeutic target in colon cancer.

Keywords:
anti-ribosomal P antibodies, colon cancer, paraneoplastic syndrome, ribosomal P protein, systemic lupus erythematosus

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Introduction

Paraneoplastic syndromes (PNS) are clinical syndromes resulting from the immune responses to the presence of cancer. The symptoms of PNS are not caused by tumor infiltration of tissues, but rather by the action of autoantibodies, cytokines, hormones, or peptides produced by cancer cells [1]. Rheumatological PNS are rare, most commonly occurring within two years prior to a cancer diagnosis. These include inflammatory myopathies, polyarthritis, polymyalgia rheumatica, lupus-like syndromes and vasculitis [2]. 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. Symptoms can affect any organ, most commonly the joints, skin, kidneys, nervous system and cardiovascular system. To date, only a few cases of paraneoplastic SLE have been reported in patients with lymphomas, cancers of the lungs, breast, thyroid, reproductive system, and digestive system [3-9]. Paraneoplastic SLE most commonly manifests as skin involvement, arthritis and hematological abnormalities, and rarely as lupus nephritis [3-6,10,11]. Anti-ribosomal P antibodies (anti-Rib P) have been identified for the first time in SLE where they can induce damage of the central nervous system, hepatitis and nephritis. It has been reported that ribosomal proteins have been implicated in various pathological conditions, including autoimmune diseases and malignancies. Their dysregulated expression is significantly associated with tumorigenesis, highlighting their potential as diagnostic biomarkers [12,13].

This paper presents a case study of an SLE patient with anti-Rib P and colon cancer, and explores the potential causes of this association.

Case Report

A 69-year-old man suspected of having SLE was admitted to the Rheumatology Clinic in September 2025. His first health problems occurred in June 2025: an inflammatory rash on the skin of his face, neck, and chest following sun exposure. The skin lesions resolved after treatment with glucocorticoid creams. In September 2025, massive erythematous-scaly lesions reappeared on the face, neck, upper limbs, and back; additionally, erosions of the oral mucosa and vermilion border of the lips made eating difficult, along with wrist arthritis (Photos 1-4), low-grade fever, and weight loss. Immunological tests showed the presence of antinuclear antibodies, as well as anti-Rib P (+++), nucleosomes (++), p-ANCA and decreased complement components C3 and C4 (see Tables 1 and 2). A chest CT scan revealed fluid in both pleural cavities, as well as isolated enlarged mediastinal and hilar lymph nodes (Photo 5). An abdominal and pelvic CT scan revealed thickening of the wall of the splenic flexure of the colon and splenomegaly (Photo 6). A trepanobiopsy showed no significant changes.

A bronchofibroscopy with EBUS was conducted and tests for tuberculosis and other bacterial infections were negative. Histopathological examination of the lymph node did not confirm neoplastic changes. A colonoscopy revealed a polypoid lesion in the distal transverse colon and descending colon, approximately 3 cm in diameter. Biopsy specimens were taken for histopathological examination. The patient fulfilled the 2019 EULAR/ACR (European League Against Rheumatism/American College of Rheumatology) classification criteria for SLE. He was treated with intravenous methylprednisolone (1.5 g), followed by oral prednisone from 20 to 5 mg/day and hydroxychloroquine 200 mg/day. Following treatment, skin and mucosal lesions as well as symptoms of arthritis significantly improved, and laboratory test results showed marked improvement. In October 2025, the histopathology results were received: descending colon - a tubular adenoma of the large intestine with high-grade dysplasia and a focus of invasion (adenocarcinoma NOS, low grade, ICD-O 8140/3); transverse colon - tubular adenoma of the large intestine with high-grade dysplasia (ICD-O 8211/2). In December 2025, a partial resection of the large intestine was performed. Currently (March 2026) , the patient is feeling well and has no cutaneous, mucosal, or joint symptoms. Laboratory test results are normal. Immunosuppressive medications have been discontinued. Paraneoplastic SLE has been diagnosed.

Discussion

We presented the case of a patient with a history of skin and mucosal lesions, arthritis and pancytopenia for several months, who was diagnosed with SLE and colorectal cancer. Initial treatment with high-dose corticosteroids and hydroxychloroquine resulted in significant improvement in skin lesions and hematological parameters. Following surgical removal of the tumor, the patient achieved complete remission of SLE, which may indicate a paraneoplastic nature of the disease. The pathomechanism of rheumatic symptoms in the course of neoplastic diseases is not fully understood. Some symptoms may result from the direct action of toxins produced by cancer cells, while others may stem from the activation of an immune response directed

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against cancer cells, which can damage healthy tissues and organs [14,15].

In most cases, paraneoplastic rheumatological syndromes improve or resolve completely following treatment for the underlying malignancy. To date, only a few cases of paraneoplastic SLE have been reported, including just three cases in patients with gastrointestinal cancers.

Rees et al. described the case of a 64-year-old man with nephrotic syndrome and suspected lupus nephritis (LN), as well as a diagnosed colorectal cancer. Following surgical treatment of the cancer, the symptoms of nephrotic syndrome completely resolved. Several months later, cutaneous and mucosal lesions, symptoms of vasculitis, and positive results for SLE serological markers occurred. At the same time, a recurrence of the cancer was confirmed [3].

Jenkins et al. reported the case of a 38-year-old woman diagnosed with bile duct cancer and skin lesions consistent with subacute cutaneous lupus erythematosus (SCLE), which appeared several weeks after the cancer diagnosis. Additionally, the patient was found to have pancytopenia and anti-Ro-60 antibodies. A reduction in tumor mass was associated with the resolution of SCLE symptoms [5].

Murad et al. described a 74-year-old man with advanced, poorly differentiated squamous cell carcinoma of the base of the tongue and pharynx, in whom SLE was diagnosed. The clinical presentation was characterised by SCLE-type skin lesions and the presence of anti-Ro antibodies. Following palliative radiotherapy and corticosteroids, significant improvement in the skin lesions was achieved [4].

In most cases of paraneoplastic SLE, SCLE-like skin lesions were present: erythematous, scaly papules and infiltrates, mostly located on the face, neck, and scalp, which exhibited marked sensitivity to UV radiation. These were often accompanied by mucosal ulcers and anti-Ro antibodies [2-6,10,11]. The skin lesions in our patient were consistent with SCLE; there were erosions of the oral mucosa and the vermilion border of the lips, making it difficult to eat; anti-Rib P were present.

The ribosomal P complex, comprising RPLP0, RPLP1, and RPLP2, serves as a critical regulatory nexus in ribosome function, bridging protein synthesis to oncogenic signaling pathways. Dysregulated expression of these proteins is a recurrent feature in cancers including breast, lung, liver, gastrointestinal, gynecological, and hematological malignancies, strongly correlating with aggressive clinicopathological features [12]. Anti-Rib P are mainly known as highly specific markers for SLE, but research has indicated they can also be elicited in certain cancer types, particularly gastrointestinal and breast cancers, potentially resulting from tumor-induced immunogenicity [13,16].

The ribosomal P0 protein is typically overexpressed in colon cancer tissues, making it an immunogenic target. Colon cancer patients exhibit a significant humoral response to this protein, producing circulating serum antibodies that target P-antigens. In vitro studies have demonstrated that targeting the C-22 P0 epitope can actively inhibit cancer cell growth, highlighting the protein's potential as both a diagnostic biomarker and an immunological target [13].

Conclusions

In summary, any systemic connective tissue disease, including SLE, can present as a paraneoplastic syndrome. The coexistence of SLE and colon cancer is extremely rare. Particular attention should be given to the possibility of cancer in cases involving severe skin lesions, general symptoms and an older age at onset of autoimmune disorders. It cannot be excluded that P protein / anti-Rib P may become a new potential diagnostic or prognostic biomarker, or a promising therapeutic target, in colon cancer.

Author Contribution Statement: All authors take full responsibility for the integrity and accuracy of all aspects of the work. The authors confirm contribution to the paper as follows: study conception and design: DS, BTS; data collection: DS, MP, KP, FP; analysis and interpretation of results: DS, GK, MT, WP, AM, AS; draft manuscript preparation: DS, MP, KP. All authors reviewed the results and approved the final version of the manuscript.

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Ethics statement and patient consent: Written informed consent was obtained from the patient for the publication of clinical details and images. All features were anonymised. As the article is a case report, no ethical approval was required.

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Figure Legends

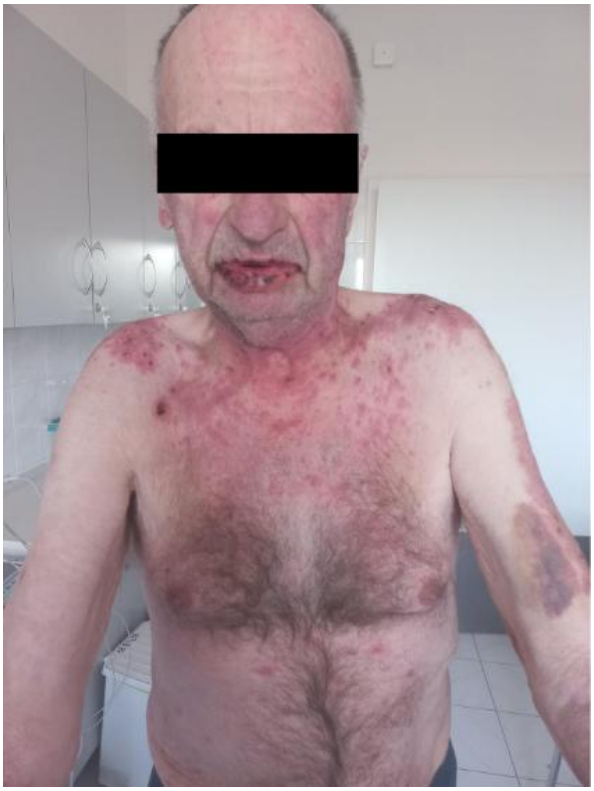

Photo 1.: Inflammatory rash on the skin of face, neck, and erosions of the lips.

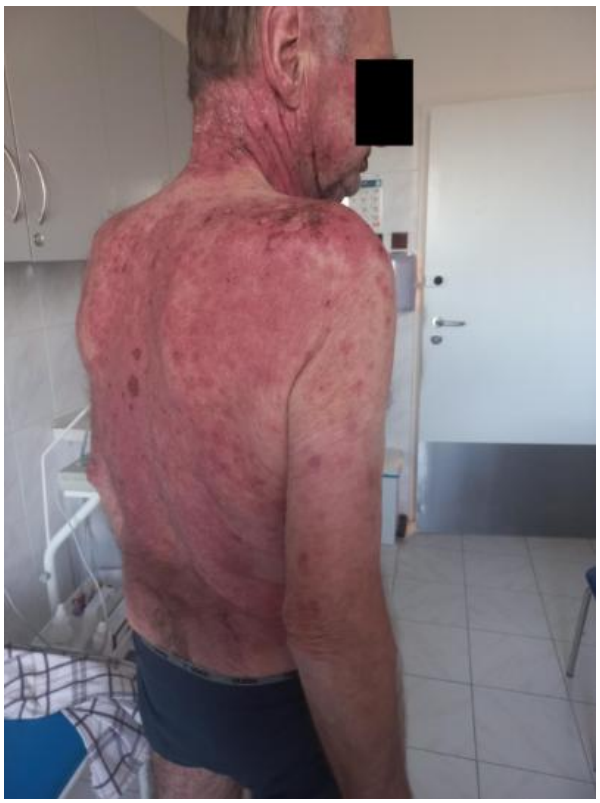

Photo 2. Massive erythematous-scaly lesions on the skin upper limbs, back, and neck.

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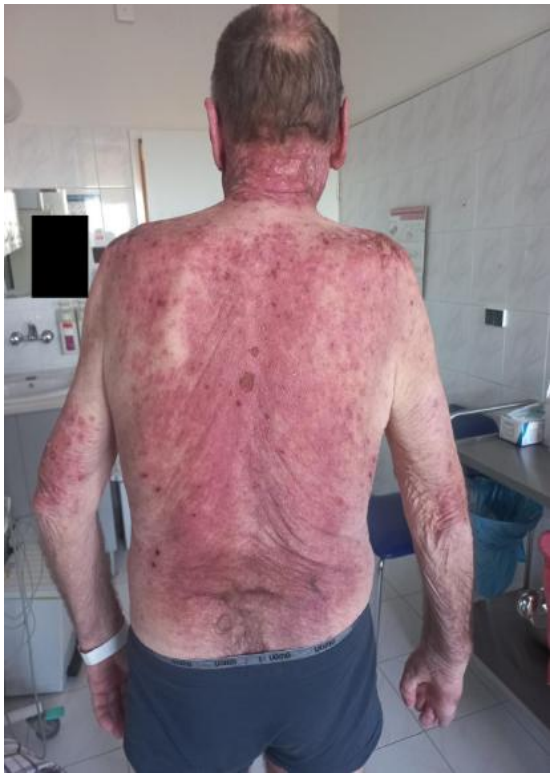

Photo 3. Massive erythematous-scaly lesions on the back.

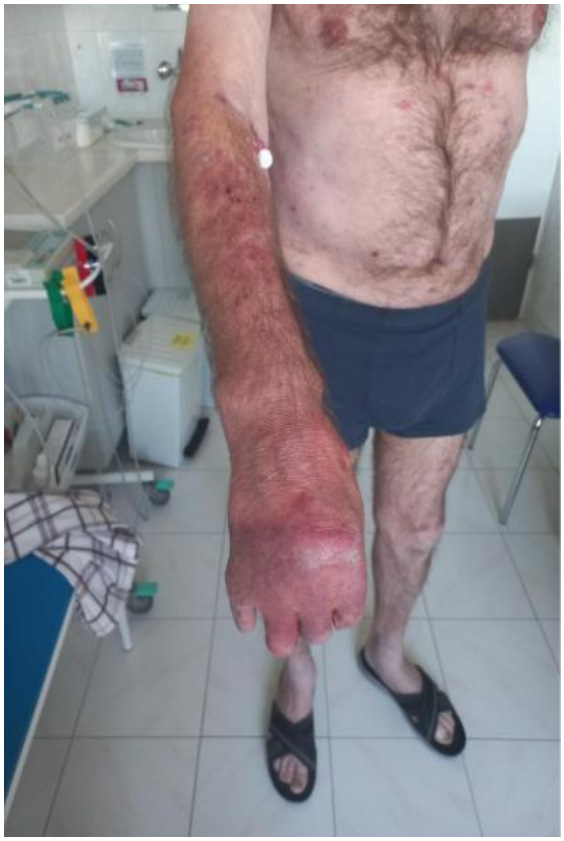

Photo 4. Inflammation of the right wrist.

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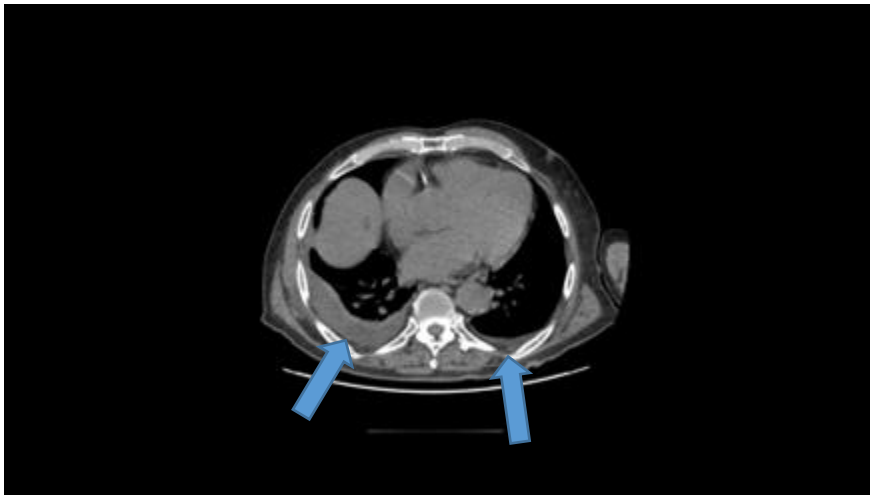

Photo 5. Fluid in the pleural cavities (blue arrows).

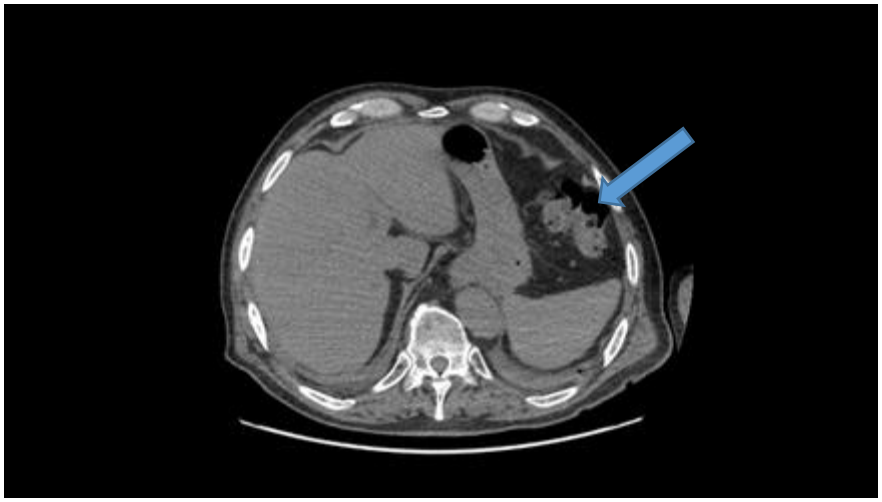

Photo 6. Abdominal and pelvic CT showing thickening of the wall of the splenic flexure of the colon (blue arrow).

Table Legends

Table 1. Laboratory data

Parameters	Results
WBC (4000 - 10 000 cells/µl)	1300
RBC (4 200 000 - 5 400 000 cells/µl)	3 700 000
Hgb (12-16 g/dl)	11.1
Hct (40-54%)	32.8
PLT (200 000-450 000 cells/µl)	81 000
IgG (700-1600 mg/dl)	1319

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Journal of Dermatological Case Reports

Parameters	Results
IgM (40-230 mg/dl)	90
CRP (0-5 mg/L)	67.6
ESR (<10 mm/1h)	16
Ferritin (22-322 ng/mL)	707
Procalcitonin (<0.5 ng/mL)	0.30
Creatinine (0.67-1.17 mg/dl)	0.86
eGFR (>90 ml/min/1.73m2)	88
Alanine aminotransferase (<50 U/L)	64
Aspartate aminotransferase (<50 U/L)	46
Albumin (3.5-5.2 g/dl)	2.4
Total Protein (6.6-8.3 g/dl)	5.5
CEA (<3 ng/dl)	3.8
Urinalysis	
Protein	50 mg/dL
Erythrocytes in urine sediment (<17 /uL)	81
Leukocytes in urine sediment (<28 /uL)	9
24-hour urine protein (<200 mg/24h)	441.00

CEA: carcinoembryonic antigen; CRP: C-reactive protein; eGFR: estimated glomerular filtration rate; ESR: erythrocyte sedimentation rate; Hct: hematocrit; Hgb: hemoglobin; IgG: immunoglobulin G; IgM: immunoglobulin M; PLT: platelets; RBC: red blood cells; WBC: white blood cells.

Table 2. Immunological parameters

Immunological tests	Results
anti-CCP	negative
RF in IgM class	negative
ANA screen	1:1000 homogeneous pattern
Nucleosome antibodies	++
Histone antibodies	+
anti-Rib P	+++
anti-dsDNA (ELISA)	negative
aCL in IgM class	negative
aCL in IgG class	negative
anti-β2-GPI IgG	negative
anti-β2-GPI IgM	negative
Lupus anticoagulant	negative
pANCA (<1:10)	1:100
cANCA (<1:10)	negative
C3 complement (80-92 mg/dl)	58

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Journal of Dermatological Case Reports

Immunological tests	Results
C4 complement (10-14 mg/dl)	10
LKM	negative
AMA	negative
SMA	negative
anti-TPO (0-34 IU/ml)	71.8
anti-TG (0-110 IU/ml)	3.2

anti-CCP: anti-cyclic citrullinated peptide; aCL: anti-cardiolipin antibodies; AMA: anti-mitochondrial antibodies; ANA: anti-nuclear antibodies; anti-dsDNA: anti-double-stranded DNA antibodies; anti-Rib P: anti-ribosomal P protein antibodies; anti-TG: anti-thyroglobulin antibodies; anti-TPO: anti-thyroid peroxidase antibodies; anti-β2-GPI: anti-beta-2 glycoprotein I antibodies; cANCA: cytoplasmic anti-neutrophil cytoplasmic antibodies; LKM: liver-kidney microsomal antibodies; pANCA: perinuclear anti-neutrophil cytoplasmic antibodies; RF: rheumatoid factor; SMA: smooth muscle antibodies.