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CASE REPORT

Systemic Lupus Erythematosus – from trigger to prognostic

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Abstract

Background. Systemic lupus erythematosus (SLE) is a chronic multisystem autoimmune disease with complex and not fully understood pathogenesis, typically triggered by environmental factors in genetically susceptible individuals [1][2]. Among its many clinical manifestations, gastrointestinal involvement remains underdiagnosed and often overlooked [3]. Furthermore, immunosuppressive treatment increases the risk of serious infections, posing a significant challenge in distinguishing disease flares from infectious complications [4].

Case Report. We present the case of a 38-year-old woman with autoimmune thyroid disease who developed flu-like and digestive symptoms following a suspected insect bite, later confirmed to be due to Rickettsia conorii infection. Despite antibiotic treatment, she experienced persistent symptoms and a purpuric rash, eventually leading to a diagnosis of SLE based on clinical and immunological criteria. The patient experienced multiple disease flares with gastrointestinal and systemic involvement, including lupus enteritis, nephritis, and severe anemia. Her condition was further complicated by sepsis, MRSA endocarditis with mitral valve destruction, pulmonary edema, and multiple organ dysfunction syndrome. Despite initial critical illness and prolonged intensive care, the patient gradually recovered after multidisciplinary interventions, including immunosuppressive therapy, antimicrobial treatment, and renal support.

Conclusion. This case underscores the diagnostic and therapeutic complexity of SLE, particularly when gastrointestinal and infectious complications co-exist [3][4]. It highlights the importance of distinguishing between disease flare and infection in immunocompromised patients. Timely imaging, vigilant monitoring, and interdisciplinary collaboration are essential for appropriate management. The evolving understanding of microbiome alterations and immune dysregulation in SLE may offer future therapeutic insights [5]. Ultimately, this case illustrates the need for a personalized, holistic approach to autoimmune disease management.

Keywords: systemic lupus erythematosus; lupus enteritis; Rickettsia conorii; sepsis; infectious endocarditis; immunosuppression; MRSA; autoimmune disease; microbiome; multiorgan dysfunction.

Abbreviations: BP – Blood pressure, CRP – C reactive protein, CSF – Cerebrospinal fluid, C3 – Complement fraction 3, CT – computed tomography, dsDNA – double stranded deoxyribonucleic acid, Hb – haemoglobin, ICU – intensive care unit, MRSA – methicillin-resistant Staphylococcus aureus, NTproBNP – N-terminal pro-B-type natriuretic peptide, SLE – Systemic Lupus Erythematosus.

Introduction

Systemic lupus erythematosus (SLE) is a chronic, multisystemic autoimmune disorder with complex and incompletely understood ethiopathogenic mechanisms. It is widely accepted that SLE results from a multifactorial interplay between genetic predisposition and environmental triggers such as infections, medications, and ultraviolet light [1][2]. Among these, infections have long been hypothesized to not only exacerbate disease activity but potentially serve as initiating factors for autoimmune dysregulation in genetically susceptible individuals. Despite advances in immunology, the exact mechanisms by which these environmental factors induce autoimmunity remain elusive [1][2].

SLE can present with a broad spectrum of clinical features affecting virtually any organ system. While joint, skin, and renal involvement are well-recognized, gastrointestinal manifestations remain underdiagnosed and frequently overlooked due to their nonspecific nature [3]. Adding to the diagnostic challenge, lupus patients often receive immunosuppressive therapy, which increases their vulnerability to opportunistic and severe infections [4][6]. This duality—autoimmune flare versus infection—poses a critical diagnostic and therapeutic dilemma. In this context, we report the case of a 38-year-old woman who developed SLE following a Rickettsia conorii infection, a rare trigger for autoimmune disease [1][2]. Her clinical course was marked by life-threatening complications, but ultimately an impressive recovery,

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highlighting both the dangers and the resilience seen in complex autoimmune syndromes.

Case presentation

This is the case of a 38-year-old woman, previously diagnosed with autoimmune thyroid disease, who presented one month earlier to the "Matei Balș" National Institute of Infectious Diseases with flu-like symptoms (fever, myalgias, arthralgias, and fatigability) and nonspecific digestive complaints (epigastric pain and diarrhea). The patient reported that the symptoms began, apparently, after a bug bite. During hospitalization, *Rickettsia conorii* was identified as the causative agent, and the patient received antibiotic therapy with doxycycline and ceftriaxone. While some symptoms improved, myalgias, arthralgias, diarrhea, and a newly developed purpuric skin rash persisted. At this stage, an autoimmune etiology was suspected, and the patient was referred to our clinic, where investigations revealed Coombs-positive anemia, low C3 complement levels, positive anti-dsDNA, anti-Sm, and anti-Ro antibodies, and nephritic-range proteinuria (546 mg/24h). Based on these findings, a diagnosis of systemic lupus erythematosus (SLE) was established.

The patient later returned to our clinic with generalized arthralgias and myalgias, diarrhea, low-grade fever, and persistent fatigue. Clinical examination revealed altered general condition, a rash affecting the face, chest, and calves, as well as oral aphthae. At the time of assessment, the patient was afebrile. Muscle strength was decreased, and inflammation was noted in the second and third right metacarpophalangeal joints and both knees. Additional findings included hypertension (BP = 150/70 mmHg), normochromic, normocytic anemia (Hb = 9.3 g/dL), elevated inflammatory markers (CRP = 15.19 mg/L), low complement fractions, hypoalbuminemia, proteinuria, hematuria, and elevated NT-proBNP (938 pg/mL). The patient was receiving methylprednisolone 32 mg/day, hydroxychloroquine 400 mg/day, and thyroid hormone replacement therapy.

Treatment and evolution

Taking into consideration all clinical and paraclinical data, we concluded that the patient was experiencing an activity flare. Therefore, we initiated pulse therapy with intravenous methylprednisolone at 500 mg/day for three days, followed by oral methylprednisolone at 64 mg/day. Initially, the evolution was favorable, with partial improvement of symptoms. However, after three days of oral treatment, the patient again developed myalgias, arthralgias, fatigability, and abdominal pain.

It is worth noting that during her hospitalization at the "Matei Balș" National Institute of Infectious Diseases, two echocardiograms were performed to rule out infectious endocarditis. After stool analysis excluded digestive hemorrhage or infection, an abdominal CT scan revealed diffuse circumferential edema of the terminal ileum and a moderate to large amount of pelvic fluid. Following consultation with a gastroenterologist, the patient was scheduled for an upper digestive endoscopy, which ultimately could not be performed. Together, we concluded that the patient had systemic lupus erythematosus (SLE) with gastrointestinal involvement. Consequently, we administered another three-day course of intravenous methylprednisolone pulse therapy.

After this second course, the patient's symptoms resolved, but her CRP remained elevated—five times above normal. Soon after, the patient developed fever, photophobia, neck stiffness, confusion, and psychomotor agitation. Laboratory investigations showed neutrophilia, lymphopenia, severe anemia, nitrogen retention, and elevated procalcitonin and inflammatory markers. In collaboration with neurologists, we initiated empirical antibiotic therapy with piperacillin-tazobactam and trimethoprim-sulfamethoxazole. Following their recommendations, a lumbar puncture and cerebral angio-CT scan were performed, which revealed no acute ischemic lesions and patent cerebral vessels. CSF analysis was negative for meningoencephalitis. After discussion with an infectious disease specialist, we suspected sepsis of unknown origin with multiple organ dysfunction syndrome (neurological and renal). Antibiotic therapy was adjusted to ceftriaxone, ampicillin, and trimethoprim-sulfamethoxazole.

Over the following days, the patient was closely monitored by infectious disease and ICU specialists. Cardiological evaluation, including repeated echocardiography, was performed. Initially, there were no signs of endocarditis, but blood cultures returned positive for Methicillin-resistant Staphylococcus aureus (MRSA). A subsequent echocardiogram revealed mitral valve vegetation, confirming the diagnosis of infectious endocarditis.

Shortly after this finding, the patient develops increasing psychomotor agitation, with dyspnea, tachypnea, and hypoxemia (SpO_2 = 80%). On auscultation, subcrepitant rales were noted. The patient was also tachycardic (120 bpm) and hypertensive (BP = 180/100 mmHg). We diagnosed acute pulmonary edema and transferred the patient to the ICU. Repeated echocardiography revealed a mitral valve abscess and posterior cusp perforation, with severe mitral regurgitation. Despite efforts to transfer the patient to a cardiovascular surgery unit, surgical teams deemed her too unstable for intervention.

During her ICU stay, the patient developed multiple complications. Neurologically, she presented with reversible cerebral vasoconstriction syndrome, bilateral hemispheric ischemic lesions, and subarachnoid hemorrhage. She also developed several infectious complications, including nosocomial pneumonia with Acinetobacter baumannii progressing to sepsis, a urinary tract infection with Candida glabrata, pulmonary aspergillosis, and an infected lumbosacral eschar colonized by Candida parapsilosis and Acinetobacter baumannii.

Despite severe mitral regurgitation, the patient maintained a consistent left ventricular ejection fraction of 45%. Multiple transfer attempts to a cardiovascular surgery unit were unsuccessful.

The patient's condition gradually improved. Neurologic, respiratory, hemodynamic, and hematologic functions stabilized; however, renal dysfunction persisted. The patient became oligoanuric and required dialysis. She was subsequently transferred to the nephrology department at the Fundeni Clinical Institute.

One month later, the patient no longer required dialysis, showed no signs of nitrogen retention, was fully conscious, and was able to mobilize independently. She was later transferred to the plastic surgery unit for management of the lumbosacral eschar.

Discussion

Gastrointestinal involvement in systemic lupus erythematosus is often underrecognized, yet it may be the initial or predominant manifestation in some patients. Literature reports suggest that up to 40% of SLE patients experience abdominal symptoms during disease flares [3]. These symptoms range from mild gastrointestinal discomfort to life-threatening conditions such as lupus enteritis, bowel ischemia, and perforation. The underlying pathophysiology often includes vasculitis or immune complex deposition in the intestinal vasculature, resulting in edema, ischemia, and inflammation. Imaging, particularly contrast-enhanced CT, is vital for diagnosis, identifying features such as bowel wall thickening, mesenteric edema, and ascites. Prompt initiation of high-dose corticosteroids is the cornerstone of treatment, while immunosuppressants or surgery may be required in severe cases [3].

Immunosuppressed SLE patients are significantly more susceptible to infections. These infections often mimic or coexist with disease flares, making early recognition particularly challenging [4][7][8]. Recent studies show that sepsis accounts for up to 18% of SLE-related hospitalizations, with over half requiring ICU care [4]. In these settings, sepsis is a leading cause of mortality [4]. Our patient, who was already on immunosuppressive therapy, developed multiple infectious complications, including MRSA endocarditis and sepsis caused by Acinetobacter baumanii and fungal pathogens. These pathogens further compromised her already weakened immune system and complicated her clinical course.

Of particular interest in this case is the association with Rickettsia conorii, a rare but documented infectious agent capable of inducing autoimmune phenomena [1]. Rickettsial infections are known to stimulate robust inflammatory responses, including activation of the complement cascade and release of cytokines such as IL-1β, IL-6, IL-8, and TNF-α [2]. In genetically predisposed individuals, such immune activation may initiate or unmask autoimmune diseases, including lupus [1][2]. While causality cannot be definitively proven, the temporal relationship between Rickettsia conorii infection and lupus onset in this patient raises the possibility of a triggering role.

Furthermore, emerging research into the gut microbiome has revealed significant dysbiosis in patients with SLE. One study found increased levels of Candida spp.—notably Candida glabrata—and decreased levels of fungal genera such as Malassezia and Rhizopus [5]. Though fungi make up less than 0.1% of gut microbes, they play a disproportionate role in immune homeostasis. Alterations in the gut mycobiome may contribute to chronic immune activation and autoimmunity [5]. Our patient's multiple fungal infections, including Candida glabrata and Candida parapsilosis, may reflect an underlying dysregulation of her gut and systemic immune responses.

Despite her critical condition—including acute pulmonary edema, mitral valve perforation, neurological impairment, and renal failure—the patient showed remarkable resilience and recovery. This speaks to the potential for recovery even in the most severe cases of SLE when managed with timely, coordinated, and multidisciplinary care. It also highlights the unpredictable course of autoimmune disease, where periods of severe deterioration can be followed by unexpected improvement.

Conclusions

This case highlights the multifaceted challenges posed by systemic lupus erythematosus, particularly when triggered by a rare infection such as Rickettsia conorii. It underscores the diagnostic complexity that arises when autoimmune flares and infectious processes co-occur, especially in immunocompromised patients. The patient's disease course was marked by severe gastrointestinal involvement, hematological abnormalities, and overwhelming infectious complications including MRSA endocarditis and sepsis—yet she experienced a slow but substantial recovery.

The unpredictability and severity of her condition reinforce the importance of high clinical suspicion, timely imaging, and aggressive interdisciplinary management. Infectious triggers and complications remain a significant cause of morbidity and mortality in lupus, necessitating careful monitoring, judicious use of immunosuppressants, and rapid response to signs of systemic infection.

Moreover, this case contributes to the growing recognition of environmental and microbial factors in the etiology and modulation of autoimmune diseases [1][2][5]. The potential role of pathogens such as Rickettsia conorii in triggering autoimmune processes [1][2], alongside evolving research into the microbiome and immune system interactions [5], may offer future therapeutic avenues. Ultimately, this case illustrates the need for a personalized, holistic approach in managing autoimmune patients—balancing immunosuppression with infection risk,

addressing multi-organ involvement, and supporting recovery even from the most critical stages of illness.

Conflicts of Interest: The authors declare no conflicts of interest.

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