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Systemic Lupus Erythematosus and Rapid Progressive Glomerulonephritis with History of Hypothyroidism: A Case Report

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Abstract
Systemic lupus erythematosus (SLE) is a multisystem autoimmune disease with highly variable clinical manifestations. This report discusses a case of SLE that occurred in a patient with a history of hypothyroidism, accompanied by severe renal complications in the form of rapid progressive glomerulonephritis (RPGN), highlighting the complexity of diagnosis and management of these overlapping conditions. This research aims to report the case of a young woman with hypothyroidism who subsequently developed SLE and RPGN, emphasizing the importance of a high clinical index of suspicion, early diagnosis, and comprehensive management. This case report uses a descriptive study approach for a single patient. Data were collected through history taking, physical examination, and a series of ancillary tests such as laboratory investigations (including renal function tests, ANA-IF), imaging studies (chest X-ray and ultrasound), and specialist consultations. The diagnosis of SLE was established based on the 2019 ACR/EULAR criteria. The patient was diagnosed with new-onset SLE and RPGN, characterized by a rapid decline in renal function, accompanied by pulmonary and cardiac manifestations. The patient also had a history of hypothyroidism. Management consisted of immunosuppressive therapy (pulse methylprednisolone), antibiotics for intercurrent infections, and hemodialysis to support kidney function. This case reminds clinicians to consider SLE in patients with other autoimmune diseases, such as hypothyroidism, who experience systemic symptoms. Early diagnosis and intervention are crucial to prevent permanent organ damage and improve patient outcomes.

INTRODUCTION

Systemic lupus erythematosus (SLE) is a complex autoimmune disease characterized by multiorgan involvement with highly variable clinical presentations . The spectrum of disease manifestations ranges from mild mucocutaneous involvement to severe, life-threatening systemic complications, including central nervous system involvement (Tayem et al., 2022; Tian et al., 2023; Tsokos, 2011). The global burden of SLE demonstrates significant geographic and ethnic variation. In the United States, the prevalence ranges from 72.1 to 74.4 per 100,000 persons, with the highest rates observed among African Americans. Globally, the estimated prevalence of SLE is 43.7 per 100,000 persons, affecting approximately 3.41 million individuals worldwide. Women are disproportionately affected, with prevalence estimates in Indonesian women reaching 131,214 cases compared to only 15,617 cases in men (Andreoli et al., 2017; Choe et al., 2010; Fischer-Betz & Specker, 2017; Tian et al., 2023). The disease typically manifests at an earlier age, predominantly affecting women during their reproductive years, with an overall female-to-male ratio of approximately 9:1. Although SLE risk decreases after menopause, postmenopausal women still demonstrate twice the risk compared to age-matched men (Ruacho et al., 2022; Tabarkiewicz et al., 2015).

The etiology of SLE remains incompletely understood, although immunological dysregulation, hormonal influences, genetic predisposition, and environmental factors are believed to play significant roles in disease pathogenesis (Mukkera et al., 2022; Piga & Arnaud, 2021; Rees et al., 2017). Female sex and hormonal influences constitute primary risk factors for SLE development. Estrogen, in particular, has been shown to induce macrophage activation, promote the production of specific cytokines including interleukin-1 (IL-1), stimulate $CD4^+$ T cells and B cells, and upregulate human leukocyte antigen (HLA) expression along with endothelial cell adhesion molecules such as vascular cell adhesion molecule (VCAM) and intercellular adhesion molecule (ICAM) (Emorinken et al., 2021; Fanouriakis et al., 2024; Hahn et al., 2012). Estrogen enhances autoimmunity through multiple mechanisms, including increased B-cell activation and modulation of lymphocyte function. Hormonal contraceptives containing estrogen have been associated with higher SLE incidence and can precipitate disease flares in patients with established SLE. Conversely, androgens demonstrate immunosuppressive properties. Various environmental exposures and certain medications have been identified as potential triggers for drug-induced lupus-like syndromes through mechanisms involving DNA demethylation and modification of self-antigens. SLE represents a multifaceted autoimmune disorder with complex pathogenesis that continues to be elucidated through ongoing research. The cascade of immune dysfunction involves cellular damage from infections or other triggers that activate both T cells and B cells, initiating an autoimmune response directed against self-antigens. This process encompasses complement system activation, cytokine release, and subsequent autoantibody production, ultimately contributing to tissue and organ injury (Aringer et al., 2019; Barber et al., 2021; Crow, 2023; Lin et al., 2023).

The diagnosis of SLE is established based on comprehensive clinical evaluation and immunological testing. The ACR/EULAR 2019 classification criteria recommend initial screening for antinuclear antibodies (ANA) when SLE is clinically suspected, followed by systematic scoring based on specific clinical and immunological criteria. The primary goals of SLE treatment include prevention of irreversible organ damage, achievement of disease remission or low disease activity, and improvement in patient quality of life. Treatment strategies are individualized based on the organs involved and disease severity, encompassing a spectrum of interventions from conservative management with antimalarials and nonsteroidal anti-inflammatory drugs for mild disease, to aggressive immunosuppressive therapy including corticosteroids and cytotoxic agents for severe manifestations. Patient education, promotion of regular physical activity, and provision of psychosocial support play essential roles in comprehensive SLE management.

Several previous case reports have documented the co-occurrence of autoimmune thyroid disease and SLE. A nationwide population-based cohort study by Lin et al. (2023) demonstrated that Hashimoto's thyroiditis significantly increases the risk of new-onset SLE, suggesting a potential pathogenic link between these autoimmune conditions. However, cases reporting the simultaneous presentation of untreated hypothyroidism and newly diagnosed SLE with rapid progressive glomerulonephritis remain relatively rare in the medical literature, particularly among young female patients from Southeast Asian populations.

Despite increasing recognition of autoimmune disease clustering, several knowledge gaps persist. The precise mechanisms underlying the association between autoimmune thyroid disease and SLE development remain incompletely characterized. Furthermore, the clinical implications of pre-existing hypothyroidism on SLE disease severity, particularly regarding renal manifestations, require further investigation. Additionally, there is limited evidence regarding optimal management strategies for patients presenting with multiple concurrent autoimmune conditions in resource-limited settings, where advanced diagnostic procedures such as renal biopsy may not be readily available.

This case report presents a unique clinical scenario of a 20-year-old female with inadequately treated hypothyroidism who subsequently developed SLE complicated by RPGN. The novelty of this case lies in (1) the temporal relationship between untreated hypothyroidism and SLE onset, (2) the severe renal manifestation as RPGN in a young patient, (3) the clinical management approach in a resource-limited setting without renal biopsy, and (4) the contribution to understanding autoimmune disease clustering patterns in Indonesian populations.

The objectives of this case report are (1) to describe the clinical presentation, diagnostic workup, and management of a young female patient with concurrent hypothyroidism and SLE with RPGN; (2) to highlight the importance of systematic clinical evaluation in patients with multiple autoimmune manifestations; (3) to emphasize the critical role of early diagnosis and prompt intervention in preventing irreversible organ damage; and (4) to contribute clinical evidence regarding the potential association between hypothyroidism and SLE development. The clinical implications of this report extend to improving awareness among healthcare providers regarding autoimmune disease clustering, reinforcing the importance of compliance with thyroid hormone replacement therapy, and providing guidance for diagnostic and therapeutic approaches in resource-limited settings. This case underscores that early diagnostic recognition and adequate multidisciplinary intervention are crucial factors in achieving positive clinical outcomes for patients with SLE and severe systemic complications.

RESEARCH METHOD

This study employed a qualitative research design, specifically a case report format, to conduct an in-depth analysis of a single patient's clinical presentation, diagnostic journey, and management. The research was conducted retrospectively, as data was collected from an existing patient encounter to illustrate a unique and instructive clinical scenario. This methodological approach is ideal for documenting rare disease associations, detailing complex diagnostic processes, and generating hypotheses for future investigation, providing a rich, contextual understanding of the case.

The data population for this research was defined as the complete medical record of the single patient in question. The data sample consisted of the entirety of the documented clinical information for this case, including the patient's history, physical examination findings, laboratory results, and diagnostic imaging reports. A purposive sampling technique was used, as the case was intentionally selected based on its intrinsic value in demonstrating the co-occurrence of hypothyroidism, Systemic Lupus Erythematosus (SLE), and Rapid Progressive Glomerulonephritis (RPGN). The primary research instruments were the patient's electronic

medical record (EMR) and standardized hospital forms for laboratory and radiology reports, which provided the raw data for analysis.

Data analysis was performed using a descriptive analytical technique. The patient's clinical data were systematically extracted from the EMR and chronologically organized to reconstruct the timeline of symptom onset, diagnostic workup, and therapeutic interventions. The findings were then interpreted and discussed in the context of established diagnostic criteria, specifically the ACR/EULAR 2019 classification criteria for SLE, and compared with the existing scientific literature to draw conclusions about the case's significance and implications for clinical practice.

RESULTS AND DISCUSSION

Case Presentation

A 20 years old female come to ER with fever and significant multiple joint pain, particularly in hands and feet. Symptoms accompanied by dry cough without blood, and weight loss in the last few months. Upon examination, no other symptoms were noted. Patient had history of thyroiditis induced by hypothyroidism since two years prior, with irregular medication.

Laboratory examination revealed anemia, leukocytosis, and elevated ESR (Erythrocyte Sedimentation Rate). Upon further examination indicated acute hypothyroidism with elevated TSH levels. Treatment was initiated with daily administration of Levothyroxine 100mcg once daily. Additionally, the patient received blood transfusion for the anemia.

After several days of treatment, patient developed hemopytysis, followed by nausea and vomitting. Chest X-ray revealed cardiomegaly and pneumonia, and urine culture identified Klebsiella pneumoniae. Echocardiography confirmed pericardial effusion. Comprehensive blood tests showed a rapid deterioration in kidney function, marked by elevated urea and creatinine levels. ASTO and ANA-IF tests were conducted, with the latter returning positive. Based on ACR/EULAR 2019 criteria, the patient was diagnosed with newly onset systemic lupus erythematosus (SLE) followed by rapid progression in deterioration of kidney suspected for lupus nephritis, Due to logistical constraints, renal biopsy was not feasible.

Further evaluation by the pulmonologist suggested interstitial lung disease as a complication of SLE. Treatment involved antibiotics targeting the identified infection; Meropenem was chosen based on culture results. Given the acute kidney injury, hemodialysis was initiated to preserve renal function. The patient also received pulse doses of methylprednisolone for three days, followed by oral prednisone thrice daily. Multiple sessions of hemodialysis and regular renal function monitoring ensued.

After five sessions of hemodialysis, the patient's condition show advanced progression, and she was permitted to be discharged from the hospital. Plans were made to refer her to a larger hospital for renal biopsy to further guide management.

Figure 1. Chest x-ray shows cardiomegaly, penumonia and scoliosis

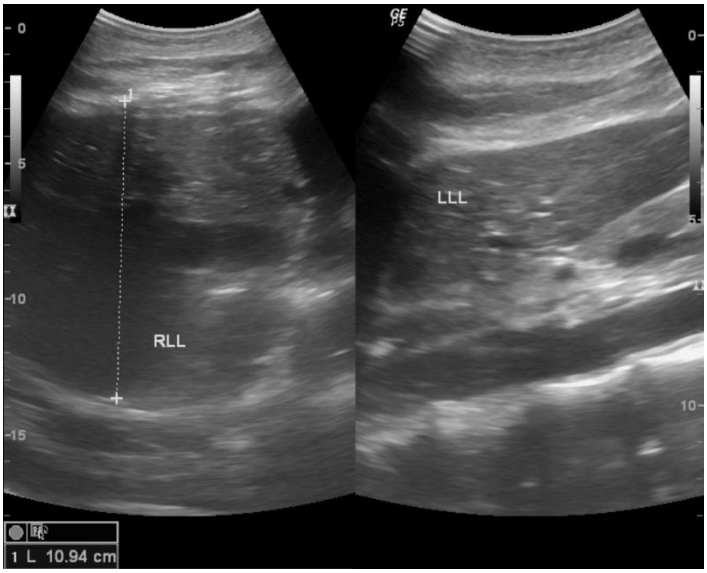

Figure 2. Abdominal Ultrasonography shows bilateral parenchymal kidney disease with pleural effusion and ascites

SLE, ofter referred to as "disease with a thousand faces", is characterized by its diverse array of symptoms and variability in clinical test results among individuals. Furtherly, various characteristics may shift over time and often mimic symptoms of distinct diseases. Common manifestations of lupus include exhaustion, muskuloskeletal pain, athralgia, skin rashes (such as the characteristic malar butterfly rash), chest pain, fever, and kidney failure but not only limited to these. SLE is one of many autoimmune disease, formerly known as skin disease from mallar buterfly rash, during technology evolvement, SLE become known to affect multigorgan with various clinical presentations.

Initially recognized as a skin disease with the malar butterfly rash, advancements in technology have revealed that SLE is a systemic autoimmune disease that can affect multiple organs, presenting with varied clinical features. In our patient's case, there were no cutaneous manifestations observed; instead, systemic manifestations, primarily renal involvement and a history of hypothyroidism with unknown antibody involvement, were predominant. This underscores the importance for clinicians to be vigilant regarding the diverse symptoms that may present in SLE cases.SLE predominantly affects young women, with estimates indicating a prevalence of 131,214 affected women compared to 15,617 affected men in Indonesia. Diagnosis of SLE can be made using the ACR/EULAR 2019 criteria, which are detailed below.

Entry criterion
Antinuclear antibodies (ANA) at a titer of ≥1:80 on HEp-2 cells or an equivalent positive test (ever)

↓

If absent, do not classify as SLE
If present, apply additive criteria

↓

Additive criteria
Do not count a criterion if there is a more likely explanation than SLE.
Occurrence of a criterion on at least one occasion is sufficient.
SLE classification requires at least one clinical criterion and ≥10 points.
Criteria need not occur simultaneously.
Within each domain, only the highest weighted criterion is counted toward the total score§.

Clinical domains and criteria	Weight	Immunology domains and criteria	Weight
Constitutional		***Antiphospholipid antibodies***	
Fever	2	Anti-cardiolipin antibodies OR	
Hematologic		Anti-β2GP1 antibodies OR	
Leukopenia	3	Lupus anticoagulant	2
Thrombocytopenia	4	***Complement proteins***	
Autoimmune hemolysis	4	Low C3 OR low C4	3
Neuropsychiatric		Low C3 AND low C4	4
Delirium	2	***SLE-specific antibodies***	
Psychosis	3	Anti-dsDNA antibody* OR	
Seizure	5	Anti-Smith antibody	6
Mucocutaneous			
Non-scarring alopecia	2		
Oral ulcers	2		
Subacute cutaneous OR discoid lupus	4		
Acute cutaneous lupus	6		
Serosal			
Pleural or pericardial effusion	5		
Acute pericarditis	6		
Musculoskeletal			
Joint involvement	6		
Renal			
Proteinuria >0.5g/24h	4		
Renal biopsy Class II or V lupus nephritis	8		
Renal biopsy Class III or IV lupus nephritis	10		

Total score:

↓

Classify as Systemic Lupus Erythematosus with a score of 10 or more if entry criterion fulfilled.

Touma Z, Cervera R, Brinks R, Lorenzoni V, Tani C, Hoyer BF, Costenbader KH, Sebastian GD, Navarra SV, Bonfa E, Ramsey-Goldman R, Tedeschi SK, Dörner T, Johnson SR, Aringer M, Mosca M. 2019. ACR/EULAR group. Associations among classification criteria items within systemic lupus erythematosus.

Figure 3. ACR/EULAR Criteria

Previous studies have consistently indicated that patients with SLE are predisposed to developing hypothyroidism. However, recent research by Hong et al. suggests that hypothyroidism may additionally corelate with a greater risk of SLE. The underlying reasons for this relationship require further clarification in future studies. Previous research has highlighted that impairment of T cells might induced the increases risk of autoimmune disease including SLE. Loss of regulatory T cells function has been observed in both autoimmune hypothyroidism and SLE patients. Another studies identified Th 17 and IL-17 as key contributors to inflammation in autoimmune disease including autoimmune hypothyroidism and SLE. Elevated IL-17 and Th 17 can exacerbate hypothyroidism and promote SLE progression. Presence of one autoimmune disease is another risk for developing another autoimmune condition. 5,6 These findings underscore the complex interplay between immune dysregulation and the development of autoimmune diseases like hypothyroidism and SLE, highlighting the need for further research to elucidate the underlying mechanisms.

Clinical features of SLE can range from a very mild disease (cutaneous) to severe form (systemic). Constitutional symptoms are prevalent in over than 90% patients with SLE and

often described as initial presenting features. Common constitutional symptoms include exhaustion, malaise, anorexia, fever, and weight loss. Mucocutaneus involvement is also frequent, affecting more than 80% of patients. Skin abnormalities in SLE can be categorized as lupus-specific sush as acute cutaneous (ACLE), subacute cutaneous (SCLE), and chronic cutaneous lupus erythematosus (CCLE). Muskuloskeletal manifestations usually found in aprroximately 80-90% of the patients, extending from mild join discomfort to more severe manifestations like deforming arthritis. Lupus arthritis which primarily affecting small joints (hands, knees, and wrists) usually presents as symmetrical inflammatory polyarthritis, non-erosive forms.

Anemia is a common finding in approximately over 50% of patients with SLE, often attributed to anemia chronic disease (ACD) Other causes can varies from iron deficiency anemia, coomb's autoimmune hemolytic, and microangiopathic hemolytic anemia, sometimes associated with antiphospolipid antibody syndrome. Leukopenia, (neutropenia, lymphopenia) and thrombocytopenia (characterized by low platelet count) are also commonly found and can be severe in SLE patients. SLE can affect various layers of heart (pericardium, myocardium, and endocardium) including coronary arteries. The most common cardiac manifestation is pericarditis which related with exudative pericardial effusion. Myocarditis though uncommon, can occur and is associated with anti-RO antibodies. Gastrointestinal disturbances can present in SLE patients, with esophageal dysmotility being a common feature. Other gastrointestinal manifestations may include mesenteric vasculitis, lupus enteritis, peritonitis, and ascites. Pulmonary manifestations of SLE can include pleuritis, pleural effusion and acute lupus pneumonitis. Intersitial lung disease (ILD), which may manifest as nonspecific intersitial pneumonia (NSIP) can lead to diffuse alveolar hemmorhage linked to capillaritis, pulmonary embolism, or pulmonary arterial hypertension.

Kidney involvement in SLE (lupus nephritis), is common and serious complications varies from mild (subnephrotic proteinuria) to severe form (progressive glomerulonephritis), potentially leading to chronic kidney injury. Lupus nephiritis can manifest early in the proggression of SLE and affects various structures of kidney, including glomeruli, intersitium, tubules, and vessels with immune complex deposition. World Health Organization (WHO) classification categorizes for lupus nephritis into in six classes, featuring various pathological features and clinical outcomes. Biopsy plays a crucial role in staging lupus nephritis, as histopathological findings guide treatment decisions. Prognosis varies significantly among the classes with lower classes (I and II) generally have a better prognosis, while higher classes (III and IV) are associated with poorer outcomes. Class V usually have a generally better prognosis with the exception of nephritis related complications like tromboembolism. Management of lupus nephritis involves a multidisciplinary approach, including immunosuppressive therapy, to control inflammation and preserve kidney function. Regular monitoring and early intervention are essential to mitigate progression and improve long-term outcomes for patients with SLE-associated kidney disease.

Rapid progressive glomerulonephritis (RPGN) can indeed be a notably severe form of lupus nephritis. RPGN is marked by a rapid deterioration in renal function over a brief period, ranging from days to weeks. Clinical features include nephritic urine analysis with findings such as microscopic or macroscopic hematuria, RBC casts, proteinuria, and dysmorphic red

blood cells (RBCs). Histopathological examination of renal from the biopsy specimens often reveals cellular cresent formation in the glomerulli. These crescents are composed of proliferating epithelial and inflammatory cells and signify severe glomerular injury. The crescent formation can lead to irreversible loss of renal function if left untreated. One of glomerulonephritis etiology is immune complex glomerulonephritis is believed to account 25-30% of RPGN cases. Biopsy is essential to confirm the diagnosis of RPGN diagnostic and to classify it based on histopathological findings.

The EULAR guidelines recommend a stepwise approach to the treatment of SLE aimed at achieving disease control while minimizing side effects. Hydroxychloroquine is advised as a first-line therapy for all SLE patients due to its immunomodulatory effects and potential to reduce disease activity. Glucocorticoids are used as adjunctive therapy based on the type and severity of organ affected, with the goal of tapering and discontinuing them when feasible to minimize long-term complications. Immunomodulatory / immunosupresive agents such as methotreaxate, azathioprine, mycophenolate and biologic agents such as belimumab, and anifrolumab can be considered in patient who showed no response in hydroxychloroquine or steroid dependent patient. The treatment approach is tailored to each patient's specific clinical presentation and disease course, with regular monitoring and adjustments as needed to optimize outcomes and minimize treatment-related risks.

CONCLUSION

This case report details a rare presentation of a young female with hypothyroidism who developed Systemic Lupus Erythematosus (SLE) complicated by Rapid Progressive Glomerulonephritis (RPGN), highlighting diagnostic challenges posed by atypical manifestations and the predominance of renal and constitutional symptoms over classic cutaneous signs. The case underscores the necessity for heightened clinical suspicion and timely multidisciplinary intervention, including immunosuppressive therapy, renal support, and infection management, even in resource-limited settings lacking advanced diagnostics such as renal biopsy. Future research should employ large-scale, longitudinal cohort studies to clarify the temporal and pathogenic relationship between hypothyroidism and SLE, explore shared mechanisms such as dysregulated T-cell responses, Th17/IL-17 pathways, and specific autoantibody profiles, and develop validated screening protocols for SLE in patients with autoimmune thyroid disease, ultimately informing standardized treatment guidelines for complex multi-autoimmune presentations.

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