Journal of Neonatal Surgery
ISSN(Online): 2226-0439
Vol. 14, Issue 25s (2025)
https://www.jneonatalsurg.com

OPEN ACCESS

Management of Systemic Lupus Erythematosus with Lupus Nephritis and Subclinical Hypothyroidism: a Case Report

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Cite this paper as: Tedo Briantono Basuki, Awalia, (2025) Management of Systemic Lupus Erythematosus with Lupus Nephritis and Subclinical Hypothyroidism: a Case Report. *Journal of Neonatal Surgery*, 14 (25s), 379-381.

ABSTRACT

Systemic Lupus Erythematosus (SLE) is a chronic autoimmune disease characterized by diverse clinical manifestations affecting multiple organ systems, often complicating diagnosis and management. Lupus nephritis (LN) and thyroid dysfunction, notably subclinical hypothyroidism, are common comorbidities that influence disease outcomes. We report the case of a 29-year-old female diagnosed with SLE presenting lupus nephritis and subclinical hypothyroidism. Clinical features included joint pain, edema, hair loss, and foamy urine, supported by laboratory findings of thrombocytopenia, proteinuria, and elevated thyroid-stimulating hormone (TSH). Management involved corticosteroids, hydroxychloroquine, mycophenolate mofetil, and levothyroxine replacement therapy. Despite ongoing immunosuppressive treatment, persistent proteinuria highlighted challenges in disease management. Regular monitoring of renal and thyroid function is crucial to reduce morbidity and mortality.

Keywords: *Systemic Lupus Erythematosus, Lupus Nephritis, Subclinical Hypothyroidism, Immunosuppressive Therapy, Levothyroxine*

1. INTRODUCTION

Systemic Lupus Erythematosus (SLE) is an autoimmune disease with an unclear etiology involving genetic, immunological, hormonal, and environmental factors. Predominantly affecting females of reproductive age, it manifests variably across ethnicities. Common manifestations include arthritis, nephritis, mucocutaneous lesions, and hematologic disorders. Hypothyroidism frequently co-occurs with SLE, potentially complicating its clinical presentation and management. This report aims to describe the clinical course, diagnostic findings, and integrated management of a patient with SLE complicated by lupus nephritis and subclinical hypothyroidism to illustrate the importance of multidisciplinary monitoring in improving patient outcomes.

2. CASE PRESENTATION

A 29-year-old Indonesian female presented with a six-month history of intermittent polyarthralgia predominantly affecting finger joints, bilateral leg edema, hair loss, and fatigue. Additional complaints included headaches and irregular bowel movements. The patient was first diagnosed with SLE and hypothyroidism six months prior to this admission and had been treated intermittently with methylprednisolone, hydroxychloroquine, furosemide, and levothyroxine.

Physical examination identified mild conjunctival anemia, bilateral pitting edema in the lower extremities, and ecchymosis on both elbow flexures. Laboratory findings showed significant thrombocytopenia (18,000/mm^3), anemia, hypoalbuminemia, and severe proteinuria. Immunological evaluation revealed high ANA titers (>400 AU/mL). Thyroid function tests indicated subclinical hypothyroidism (TSH 36.75 µIU/mL, FT4 0.73 ng/dL).

Initial management involved methylprednisolone (48 mg/day), hydroxychloroquine (200 mg/day), mycophenolate mofetil (720 mg/day), and levothyroxine (50 mcg/day). Subsequent follow-up demonstrated improved symptoms, although

persistent knee pain and proteinuria remained. Treatment was adjusted to include aspirin and lisinopril, increasing mycophenolate mofetil to 1080 mg/day and initiating folic acid supplementation. Follow-up laboratory tests showed moderate improvement in thyroid parameters (TSH 7.6 µIU/mL, FT4 0.83 ng/dL). **DISCUSSION**

Systemic lupus erythematosus (SLE) is a chronic autoimmune disease characterized by widespread organ involvement and significant immunological abnormalities, notably antinuclear antibodies (ANA)[1]. It predominantly affects women of reproductive age, with a higher prevalence among African-Americans, followed by Caucasians, Asians, and Hispanics[2]. Clinical manifestations are highly variable, ranging from joint pain, skin lesions, and kidney involvement to hematological disorders and neurological symptoms, contributing to diagnostic challenges. Our patient, a young Asian woman, presented with joint pain, lower limb swelling, and hair loss exacerbated by physical and psychological stress. Diagnosis was established using the 2019 ACR/EULAR criteria, demonstrating a high ANA titer (>1:400), arthritis, and thrombocytopenia, totaling a score of 14. Disease activity was classified as moderate (SLEDAI score of 8).

Severe thrombocytopenia (platelets <50,000/mcL) in SLE typically results from immune thrombocytopenic purpura (ITP), characterized by increased platelet destruction and reflected by an elevated immature platelet fraction (IPF)[3]. Clinically, patients may present with petechiae, purpura, or ecchymosis[4], as observed in our case, where the patient exhibited petechiae with a platelet count of 18,000/mcL and an elevated IPF (31.5%). Lupus nephritis (LN) is a critical complication, indicated by significant proteinuria (>0.5 g/day). Histological evaluation through renal biopsy is important to guide prognosis and management[5]. Our patient presented with marked proteinuria (>2000 mg/dL, P:C ratio ≥0.50) and clinical features consistent with LN, necessitating aggressive immunosuppressive therapy.

An association between SLE and thyroid disorders is well-documented, with hypothyroidism being the most common[6]. Hypothyroidism may exacerbate disease activity and delay remission, particularly in female patients[7,8]. Subclinical hypothyroid conditions (elevated TSH with normal FT4) that are inadequately treated may delay remission in SLE for approximately six months. SLE patients with thyroid dysfunction tend to experience longer durations of active disease compared to those with normal thyroid function. The presence of anti-Smith antibodies in SLE is also associated with a higher likelihood of hypothyroidism, indicating a possible immunological link[9]. Subclinical hypothyroidism, as seen in our patient (TSH 36.75 µIU/mL, normal FT4), may further complicate the disease course.

There is evidence suggesting that hypothyroidism in SLE may be caused by the autoimmune process itself. Structural abnormalities of the thyroid may result from immune-mediated damage in SLE, leading to dysfunction in other organ systems. SLE can activate the immune system to secrete chemokines and cytokines, which circulate and infiltrate the thyroid gland, triggering inflammation, tissue damage, and a worsening immunological response, ultimately resulting in hypothyroidism. Deposition of immune complexes along the walls of small and medium-sized vessels leads to inflammation, necrosis, thrombosis, ischemia, hypoxia, and organ dysfunction. Vasculitis is considered a central pathophysiological mechanism in SLE, and when it involves the thyroid, it may cause hypothyroidism.

Moreover, patients with SLE and concomitant organ dysfunction (such as lupus nephritis or hepatic impairment) may have disrupted thyroid hormone metabolism due to impaired binding, distribution, or excretion, further contributing to hypothyroid states. The relationship between hypothyroidism and SLE is bidirectional—hypothyroidism can affect the course of SLE, and SLE can lead to thyroid dysfunction[10,11]. This interplay underscores the necessity for integrated management strategiesTherapeutic interventions aim to control disease activity, preserve organ function, and improve quality of life. Management includes immunosuppressants

such as corticosteroids, hydroxychloroquine, and mycophenolate mofetil, alongside supportive treatments like ACE inhibitors and aspirin[5]. Hormone replacement therapy with levothyroxine is essential in managing hypothyroidism, tailored to clinical response and periodic monitoring of thyroid function[12,13]. Regular follow-up involving clinical assessment, laboratory evaluation (urinalysis, renal function tests, complement levels), and thyroid function tests (TSH, FT4, anti-TPO) is crucial for achieving optimal outcomes and preventing complications[5,13].

3. CONCLUSION

Comprehensive management of SLE complicated by lupus nephritis and subclinical hypothyroidism involves coordinated multidisciplinary care, regular disease monitoring, and personalized treatment strategies to ensure favorable patient outcomes. This case highlights the importance of integrated renal-thyroid evaluation in SLE and underscores the benefit of early recognition and treatment of subclinical hypothyroidism to support disease control and improve quality of life.

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