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Silent Lupus Nephritis: Unmasking the Subtle Burden of Renal Involvement in Systemic Lupus Erythematosus – A Case Report

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Abstract

Systemic Lupus Erythematosus (SLE) is a chronic autoimmune disease characterized by the involvement of multiple organs, including the kidneys. Lupus nephritis (LN) is a common and severe manifestation of SLE, often leading to renal dysfunction and disease progression. However, some cases of LN can present with minimal or no clinical symptoms, making early detection and intervention challenging. We present a case report of a 26-year-old lady from Bangladesh with "silent" LN, emphasizing the importance of vigilant monitoring and timely diagnosis. The patient initially presented Swelling of lower limbs but did not exhibit overt urinary symptoms or signs of renal dysfunction. Laboratory investigations revealed elevated antinuclear antibodies (ANA), anti-double-stranded DNA (anti-dsDNA) antibodies, and reduced complement levels. Despite the absence of classical clinical features, a renal biopsy was performed due to persistent laboratory abnormalities, which confirmed the presence of LN. The patient's treatment regimen included high-dose corticosteroids and immunosuppressive agents, resulting in clinical improvement and normalization of laboratory parameters. Regular monitoring of renal function and disease activity was crucial in maintaining disease control and preventing further renal damage.

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Introduction

Silent Lupus Nephritis (LN) is a significant complication associated with Systemic Lupus Erythematosus (SLE), a chronic autoimmune disease characterized by multisystemic involvement. LN is a common manifestation of SLE, affecting approximately 50-60% of patients with the condition, and it is a leading cause of morbidity and mortality in this population. However, the term "silent" refers to the fact that LN can often be asymptomatic or have subtle clinical manifestations, making early detection and intervention challenging.[1]

The kidneys are commonly targeted by the immune system in SLE, resulting in the development of LN. The presence of immune complexes and autoantibodies in the kidneys triggers a cascade of inflammatory processes, leading to renal damage and dysfunction. If left untreated or undiagnosed, silent LN can progress to end-stage renal disease (ESRD), necessitating dialysis or kidney transplantation.[2,3]

The challenge in diagnosing silent LN lies in the absence of overt clinical symptoms. Patients may have normal renal function, making routine laboratory tests, such as serum creatinine and urinalysis, unreliable for early detection. Instead, kidney biopsy remains the gold standard for

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diagnosing LN and assessing its severity. Histopathological findings, including the presence of immune complex deposits and cellular infiltration, help guide therapeutic decisions and predict disease progression.[4]

The management of silent LN requires a multidisciplinary approach, involving rheumatologists, nephrologists, and other healthcare professionals. Treatment strategies aim to suppress the immune system, reduce inflammation, and preserve renal function. Immunosuppressive agents, such as corticosteroids, immunomodulators, and biologic therapies, are commonly used. Early intervention with appropriate therapy has shown promising outcomes in preventing or delaying renal damage and improving patient prognosis.[5]

In this article, we will explore the characteristics, diagnostic challenges, and management strategies of silent LN in patients with SLE. Our main focus is to highlight the importance of early detection and intervention in improving patient outcomes with SLN. By increasing awareness of silent LN and its implications, healthcare professionals can enhance their ability to diagnose and manage this complex condition effectively.

Case Summary

In December 2017, a 26-years old lady came to the Department of Nephrology, Community Based Medical College, Bangladesh (CBMC,B) Hospital, presenting with Swelling of lower limbs for one month (Fig: 1,2).

According to the statement of the patient, she was reasonably well 2 months back. Since then, she had been suffering from pain in multiple joints of both lower and upper limbs for 2 months. The pain involves mostly wrists and ankles and small joints of feet. It was not migratory but associated with morning stiffness, usually persists 2 to 3 hours. Pain was more with activity and reduces with rest.

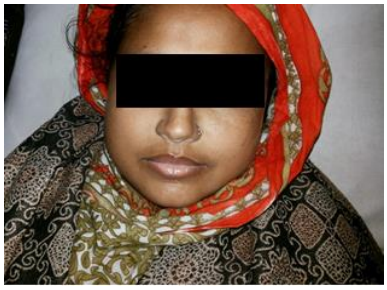

Fig. 1: 26-years old lady presenting with SLN

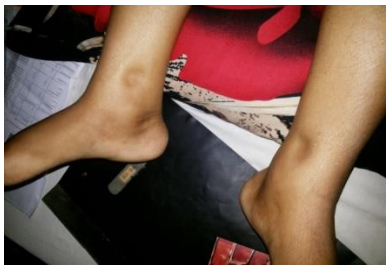

Fig. 2: Swelling of lower limbs

From the last one month, she noticed swelling of both lower limbs and her face which was gradually increasing then it became generalized. She also complained of passage of scanty reddish urine for one month but there was no burning sensation during micturition. She also developed oral ulcer for same duration, first on upper hard palate then it spreads on both inner chicks & lastly on tongue but no history of sore throat.

She also noticed low grade fever for last one month which was irregular in nature, decreased at evening and increased at night with sweating; her highest recorded temperature was 100°F. Fever was not associated with chills and rigors and subsided by taking paracetamol.

She also developed vomiting and vomitus was white coloured containing mucous with food particles & projectile in nature. With those above complaints, she got herself admitted in this hospital for better management. She had no history of any chronic medical illness like diabetes mellitus, hypertension, or tuberculosis. Her parents were healthy. She had one sister who was also suffering from this type of illness. Her sibling also had occasional complaints of arthralgia. She was immunized as per EPI schedule and also completed TT vaccine schedule. Examination of all the systems was quite normal, except for the presence of oedema and anaemia. We did some investigations before admission and found RA test result was negative; Routine examination of urine revealed presence of albumin (+++), and her hemoglobin was found 6 gm/dl.

Routine investigations done at her admission revealed hemoglobin 6.6 gm/dl, while serum creatinine was 1.4 mg/dL, ESR 140 mm in 1st hour and serum albumin 2.5 gm/dl. Her Anti-nuclear antibody (ANA) came out positive, while Anti-ds DNA was >250.00 IU/ml. The patient was further investigated and found low complement levels (C3: 0.25 gm/L, C4: 0.25 gm/L). Ultrasonogram of the renal system revealed bilateral glomerulonephritis. Renal biopsy report revealed diffuse proliferative lupus nephritis, ISN/RPS class IV. Our final diagnosis was SLE with lupus nephritis (Class IV variety) with AKI Stage-I.

According to the findings, we suggested complete bed rest and oxygen inhalation when needed. Medication was started with hydroxychloroquine and prednisolone. After 6 weeks, follow-up of the patient revealed a remarkable improvement with stable renal functions.

Discussion

Lupus nephritis is a severe complication of systemic lupus erythematosus and can lead to irreversible renal damage if left untreated.[6] The clinical presentation of LN can range from asymptomatic proteinuria to overt renal failure. However, cases of silent LN, where patients lack typical renal symptoms, are not uncommon.[7] In our case report, the absence of classical urinary symptoms delayed the diagnosis of LN, emphasizing the need for vigilance among healthcare professionals.

The diagnosis of LN requires a high index of suspicion, especially in patients with SLE. Laboratory investigations play a crucial role in identifying renal involvement. Elevated serum creatinine levels and abnormal urinary findings, such as proteinuria and hematuria, should raise suspicion for LN, even in the absence of overt renal symptoms.[8] In our case, routine renal function tests and urinalysis were instrumental in identifying the silent LN.

Renal biopsy remains the gold standard for confirming the diagnosis of LN and determining the disease activity and histopathological class.[9] In our patient, the renal biopsy revealed class IV LN, characterized by diffuse proliferative glomerulonephritis. Early detection of LN is crucial as it allows for timely intervention, including the initiation of immunosuppressive therapy to prevent further renal damage. Treatment of LN involves a multidisciplinary approach, with a combination of immuno-suppressive agents, such as corticosteroids, and disease-modifying anti-rheumatic drugs (DMARDs).[6]

Prompt initiation of therapy can help achieve disease remission and prevent progression to end-stage renal disease. Regular monitoring of renal function and urinary findings is essential to assess treatment response and prevent relapses.

Conclusion

This case report highlights the importance of considering silent LN in patients with SLE, even in the absence of typical renal symptoms. Early detection through routine laboratory investigations, including renal function tests and urinalysis, is crucial for timely intervention and prevention of irreversible renal damage. Healthcare professionals should maintain a high index of suspicion for renal involvement in SLE patients and promote regular monitoring to improve outcomes. Further research is needed to better understand the underlying mechanisms of silent LN and develop strategies for early detection and management.

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