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Case Report

Management and clinical challenges of lupus nephritis presenting with hypotension: a case report

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ABSTRACT

Lupus nephritis (LN), a grave sequel of systemic lupus erythematosus (SLE), precipitates substantial renal impairment and engenders noteworthy clinical morbidity. This case report delineates the presentation of a 39-year-old female with antecedent diagnoses of SLE and rheumatoid arthritis, who presented with severe dyspnea (Grade 4), retrosternal chest pain radiating posteriorly, and oliguria. Physical examination unveiled profound hypotension, while initial diagnostic evaluations were congruent with acute kidney injury indicative of LN. Despite robust fluid resuscitation, the patient hypotension persisted, necessitating intricate clinical deliberations. This case encapsulates the multifaceted challenges inherent in the management of lupus nephritis, particularly when complicated by refractory hypotension and multi-organ involvement. It accentuates the imperativeness of prompt diagnostic acuity and an integrative therapeutic paradigm. Additionally, it elucidates the rationale underpinning pivotal clinical decisions while addressing the nuances of such a formidable clinical scenario.

Keywords: Lupus nephritis, Systemic lupus erythematosus, Rheumatoid arthritis, Hypotension, Acute kidney injury, Immunosuppressive therapy

INTRODUCTION

Systemic lupus erythematosus (SLE) stands as a hallmark autoimmune disease characterized by a multifaceted interplay of defective apoptotic clearance, heightened activity of both innate and adaptive immunity, complement system activation, immune complex deposition, and tissue inflammation. These processes collectively drive a perpetuating cycle of autoimmunity. Various pathogenic pathways may converge to produce the diverse clinical presentations grouped under the term SLE. While the disease can affect multiple organs and tissues, the specific pattern of symptoms and autoimmune responses varies widely among patients and can evolve over time in the same individual. "Lupus" Once thought to be limited to a dermatological manifestation, lupus is now recognized as a comprehensive multisystemic illness.[1] Lupus nephritis (LN) is the most common severe complication observed in individuals with SLE. Affecting up to 60% of SLE patients, LN substantially influences their quality of life and overall prognosis. It is crucial to assess kidney function in SLE patients because prompt identification and treatment of renal impairment can significantly enhance renal outcomes. After tile commences lupus nephritis usually develops three years later, frequently within five years.[2] Most SLE patients have histological evidence of lupus nephritis, even if they do not exhibit clinical signs of renal illness. Urinalysis, urine albumin-to-creatine ratio, estimated glomerular filtration rate (eGFR) and serial creatinine are used to

monitor the onset of lupus nephritis.[3] These aids in determining whether there has been an increase in serum creatinine from the baseline value and whether proteinuria, which is frequently seen in lupus nephritis, is present. A kidney biopsy is essential for diagnosing and classifying LN. It is advised for SLE patients with proteinuria exceeding 0.5 g/24 hours (or uPCR >0.5 g/g). The presence of hematuria, leukocyturia, urinary casts, and/or unexplained renal function impairment further strengthens the indication for biopsy. For patients without proteinuria but presenting with active urinary sediment and/or renal function impairment, a thorough evaluation is necessary to exclude causes unrelated to SLE prior to proceeding with a kidney biopsy.[4] Given the elevated morbidity associated with lupus nephritis, therapy is crucial in halting the advancement of the condition toward end-stage renal disease (ESRD).[5]

Post-diagnosis monitoring of lupus nephritis

Monitoring the extrarenal systemic manifestations is essential for all patients with LN. The The systemic lupus erythematosus disease activity index (SLEDAI) scoring system provides a systematic and reproducible method to assess such activity.[6]

Proteinuria serves as the key marker for both prognosis and treatment response in LN. Accurate measurement, typically expressed as 24-hour urinary protein excretion or uPCR from an isolated urine sample, is crucial. Automated techniques have replaced traditional manual methods for urinary sediment analysis, and these have been validated for routine patient monitoring. The chronic kidney disease epidemiology collaboration formula is recommended for calculating eGFR. However, it's important to note that not all changes in laboratory parameters correlate with immunological lupus activity. For example, fluctuations in body weight can significantly affect proteinuria levels. If there is a decline in renal function without increases in proteinuria or changes in urinary sediment, it suggests that factors unrelated to LN activity should be considered. Similarly, alterations in urinary sediment without changes in proteinuria or kidney function require evaluating potential non-LN causes.[7]

We present a case of a 39-year-old female patient with lupus nephritis. This case is noteworthy because, although hypertension is a common presenting symptom of lupus nephritis, this patient presented with hypotension instead.

CASE REPORT

A 39-year-old female patient presented to the general medicine department in Gandhi Hospital, Secunderabad. Complaining of shortness of breath (grade 4), chest pain radiating to the back for 3 days, and decreased urine output. The patient had a history of rheumatoid arthritis and systemic lupus erythematosus for 7 years. The patient took hydrocortisone 5 mg for 5 years (not consistent). On physical examination, the patient was conscious and oriented, with pallor, her blood pressure was found to be 80/60 mmHg and her pulse rate was 96 bpm. The routine laboratory workup showed hemoglobin of 9 g/dl (reference range (RR), 14-16 g/dl), leukocytes 3000/mm3 (RR, 4000-11000/mm3), and platelets 3.5×105 /mm3 (RR, 1.5×105-4.5×105 / mm3). ESR was 10 mm in the first hour. The biochemical examination showed random blood sugar level of 5.0 mmol/l (RR; < 7.0 mmol/l), serum creatinine 3.7 mg/dl (RR, 0.5-1.1 mg/dl), urea -22.4 mmol/l (RR;1.8-7.1mmol/l), uric acid -19.2(RR; women -2.4-6.0 mg/dl) Na+1.52 mmol/l (RR; 1.35 – 1.48 mmol/l), K+ 2.7 mmol/l (RR; 3.50 – 5.2 mmol/l), and Cl- 96 mmol/l (RR; 95 - 105 mmol/L). arterial blood gases po2-79 (RR;83.0-108), pco2.48 (RR;32.0-45.0), pH-7.250 (RR;7.350-7.450), antinuclear antibody test (ANA) -positive. There was no family or close contact history with SLE or rheumatoid arthritis. Based on the above data a diagnosis of lupus nephritis was given. The treatment was initiated with nor adrenaline-4cc to manage the hypotension and continued for 5 days followed by sodium bicarbonate, N-acetylcysteine, hydroxychloroquine, and febuxostat.

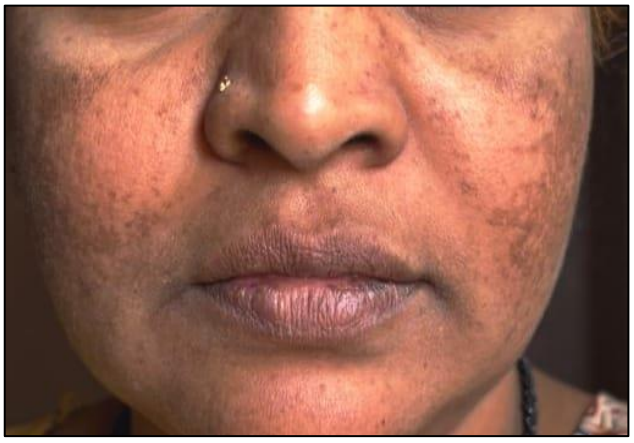

Figure 1: Systemic lupus erythematosus.

DISCUSSION

In cases of lupus nephritis, patients typically present with hypertension due to kidney damage and fluid retention. However, in this particular instance, the patient exhibited hypotension, which is uncommon in lupus nephritis indicating a more severe underlying condition. However, when hypotension occurs, it can signal serious complications which were managed with noradrenaline. Noradrenaline is primarily used in critical care settings to treat hypotension, especially in conditions such as septic shock. It exerts its effects by inducing vasoconstriction, thereby raising blood pressure and ensuring adequate perfusion to vital organs, including the heart and brain.[8]

Hypotension can occur as a result of hydrocortisone use; the patient was administered hydrocortisone 5 mg to manage lupus erythematosus. Hydrocortisone is a corticosteroid commonly prescribed to manage conditions

such as adrenal insufficiency, autoimmune disorders, and inflammatory diseases like lupus erythematosus. One of the mechanisms by which hypotension may arise is through inadequate dosage in patients with primary or secondary adrenal insufficiency, where insufficient cortisol levels can lead to low blood pressure. Additionally, while corticosteroids typically cause fluid and sodium retention, they can also induce hypokalemia, potentially disrupting blood pressure regulation and contributing to hypotension.[9] Abrupt withdrawal of hydrocortisone or inadequate adjustment during periods of physiological stress, such as illness or surgery, can lead to an adrenal crisis characterized by severe hypotension and shock.[10]

Sodium bicarbonate is used to manage metabolic acidosis, a condition where the kidneys become less efficient at excreting hydrogen ions. It neutralizes excess acid in the bloodstream, restoring a balanced pH level.[11] N-acetylcysteine (NAC) is an antioxidant and mucolytic agent that may be beneficial in managing lupus nephritis by replenishing glutathione levels and reducing oxidative stress and inflammation in the kidneys. NAC can be used as an adjunctive therapy alongside standard treatments like corticosteroids and immunosuppressants.[12]

In patients with LN class I, the use of hydroxychloroquine (HCQ) was recommended it act as general nephroprotection, and overall management of SLE in accordance with the treatment of any extrarenal manifestations. It acts as an immunomodulator, reducing the immune response and inflammation.[13] Febuxostat is primarily used to manage hyperuricemia in conditions like gout. It inhibits xanthine oxidase, an enzyme involved in uric acid production, reducing uric acid levels in the blood, and preventing gout flares and related complications. Controlling hyperuricemia may have renal protective effects, as high uric acid levels can contribute to kidney injury.[14]

CONCLUSION

In summary, this case underscores the intricate challenges of managing lupus nephritis, particularly when it manifests with atypical and severe symptoms like persistent hypotension. The treatment approach employed combining vasopressor support with noradrenaline to stabilize blood pressure alongside adjunctive therapies such as sodium bicarbonate, N-acetylcysteine, hydroxychloroquine, and febuxostat exemplifies a comprehensive and targeted strategy. This multidisciplinary approach highlights the critical importance of early identification and proactive intervention to address systemic complications effectively.

By addressing both the acute and underlying aspects of the disease, this case emphasizes the necessity of vigilant monitoring and personalized treatment plans to safeguard kidney function and improve patient outcomes in those suffering from lupus nephritis.

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