
Case Report

Lupus Nephritis in a Postpartum Patient - Case Report

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Abstract

Systemic lupus erythematosus (SLE) is a chronic multisystem autoimmune disorder characterized by auto-antibody production and immune complex-mediated organ damage. The predominant clinical signs include fatigue, fever, weight loss, arthritis, pericarditis, anemia, and pulmonary complications. Diagnosing SLE requires a combination of clinical evaluations and laboratory testing, including antinuclear antibodies and anti-double-stranded DNA antibodies, among others. Lupus nephritis is a serious manifestation of SLE and is particularly challenging during pregnancy and the post-partum period.

We present a case of a previously healthy woman, who developed severe proteinuria, hypertension, and extra-renal lupus manifestations, eight weeks after delivery. Laboratory and immunological findings confirmed new-onset SLE. Renal biopsy established the diagnosis of lupus nephritis. The patient was treated with high-dose corticosteroids, cyclophosphamide, and therapeutic plasmapheresis, resulting in significant clinical and biochemical improvement. This case highlights the importance of an early recognition of lupus nephritis in the post-partum period, when immune rebound phenomenon may precipitate autoimmune activation.

In conclusion, we can say that the early recognition of lupus nephritis in the post-partum period is crucial for the treatment of the lupus nephritis. This patient achieved complete clinical remission following immunosuppressive therapy and continues under nephrology follow-up due to risk of relapse.

Keywords: systemic lupus erythematosus, lupus nephritis, post-partum, renal biopsy, cyclophosphamide, plasmapheresis

Introduction

Systemic lupus erythematosus (SLE) is a chronic multisystem autoimmune disorder characterized by auto-antibody production and immune complex-mediated

organ damage [1,2]. It predominantly affects young women and is more common in Black, Hispanic, and Asian populations. However, its onset involves a complex interplay of genetic predispositions, environmental stimuli, hormonal factors, and immune system abnormalities [1,2]. The predominant clinical signs include fatigue, fever, weight loss, arthritis, pericarditis, anemia, and pulmonary complications [2,3]. Diagnosing SLE requires a combination of clinical evaluations and laboratory testing [2,4], including antinuclear antibodies and anti-double-stranded DNA antibodies, among others [4]. Renal involvement occurs in approximately 50% of patients and typically develops within the first five years after diagnosis [3,5,6]. Lupus nephritis must be considered in patients presenting with unexplained proteinuria, hematuria, or renal dysfunction-even when SLE has not been previously identified. The post-partum period is a known trigger for autoimmune disease exacerbations [7] due to the immunological rebound following pregnancy-related immune modulation. Herein, we present a case of a patient with hypertension during pregnancy and diagnosis of SLE in the post-partum period.

Case Presentation

A previously healthy woman, gravida 2 para 1, developed elevated blood pressure at six weeks of gestation during her second pregnancy and was started on methyldopa. She delivered a healthy infant, but post-partum she continued to experience persistent hypertension and progressive bilateral lower-extremity edema. Eight weeks after delivery, she was presented for nephrology evaluation. Antihypertensive therapy was switched to nifedipine 20 mg. Because of bacteriuria, ciprofloxacin was prescribed.

Laboratory tests revealed proteinuria ++++ and hematuria +++, with serum creatinine 183 $\mu\text{mol/L}$. The other laboratory findings are presented in Table 1. Renal ultrasound showed bilaterally enlarged hyperechoic parenchyma with calyceal dilatation, and no lithiasis or obstruction. Due to persistent heavy proteinuria and edema, the patient was hospitalized.

During hospitalization, echocardiography revealed EF≈43%. The echocardiographic report showed left ventricular systolic and diastolic dysfunction, with in-

Table 1. Laboratory findings in patient with SLE and Lupus nephritis during hospitalization

Analyse	Results	References
Erythrocytes	3,9	4.2 – 5.9 ×10 ¹² /L
Hemoglobin	120	♂ 135–175 · ♀ 120–155 g/L
PLT	203	150 – 400 ×10 ⁹ /L
Glycemia	5.3	3.9 – 5.6 mmol/L (fasting)
Urea	5.5...9.3	2.5 – 7.8 mmol/L
Creatinine	107...182	♂ 64–104 µmol/L · ♀ 49-90 µmol/L
Uric acid	541...593	♂ 210–420 µmol/L · ♀ 150-360 µmol/L
Total Protein	45	64 – 83 g/L
Albumin	23	35 – 52 g/L
AST	45	< 40 U/L
ALT	41	< 41 U/L
LDH	312	135 – 225 U/L
NA	135	136 – 145 mmol/L
K	3.7	3.5 – 5.1 mmol/L
Ca	1.7	2.15 – 2.55 mmol/L
P	1.9	0.8 – 1.5 mmol/L
Triglycerides	3.7	< 1.7 mmol/L
HDL	0.8	♂ >1.0 · ♀ >1.2 mmol/L
C3	0.4	0.9 – 1.8 g/L
C4	0.1	

creased internal dimensions of the left-sided cardiac chambers and globally reduced left ventricular wall motion. A small circumferential pericardial effusion was present, measuring approximately 8 mm to the posterior wall of the left ventricle, 5 mm anterior to the right ventricle, and 4 mm anterior to the apex. A small bilateral pleural effusion was also present.

Dermatologic examination showed malar erythematous-desquamative plaques. Face with “butterfly rash”. Immunological tests demonstrated ANA positivity, high anti-dsDNA (225.5 IU/mL), positive anti-Sm (123 IU/mL), and p-ANCA +++, with negative antiphospholipid antibodies.

A renal biopsy performed on the day 3, confirmed lupus nephritis (LUPUS NEPHRITIS class IV/V, with small part of class VI).

Histopathology Report: small blood vessels show changes consistent with "onion-skin" lesions. The remaining tubular epithelium shows changes consistent with protein reabsorption. Considering the DIF (full house pattern), the findings are consistent with Lupus Nephritis, Class IV + V (±VI).

The Activity Index was 10 and Chronicity Index 2.5, How it relates to Classes: **Class I**-Normal light microscopy; *mesangial immune deposits* seen only by IF/EM; **Class II**-Mesangial hypercellularity/deposits visible in light microscopy; **Class III / IV**-Focal (III) or diffuse (IV) proliferative lesions with *subendothelial deposits*; and **Class V**-Membranous pattern with *subepithelial deposits*.

Treatment included a three days course of high-dose intravenous methylprednisolone (500 mg) followed by tapering, after that with a peroral corticosteroid therapy for a prolonged period. The patient received three doses of cyclophosphamide overall, during hospitalization and three doses after that, repeated every 3 weeks for a total 6 doses. Five plasmapheresis sessions were performed with satisfactory outcome [8,9]. There was no need for hemodialysis. The consultation with rheumatologists included a therapy with hydroxychloroquine.

Follow up during the next month showed improvement of the results and stable state. The results are presented on Table 2.

Table 2. Laboratory findings one month after hospitalization

Analyse	Results	References
Erythrocytes	3.7	4.2 – 5.9 ×10 ¹² /L
Hemoglobin	121	♂ 135–175 · ♀ 120–155 g/L
Hct	0.34	
PLT	368	150 – 400 ×10 ⁹ /L
Urea	8.2	2.5 – 7.8 mmol/L
Creatinine	90	♂ 64–104 µmol/L · ♀ 49-90 µmol/L
Total Protein	72	64 – 83 g/L
Albumin	42	35 – 52 g/L
NA	140	136 – 145 mmol/L
K	5.0	3.5 – 5.1 mmol/L
Ca	2.4	2.15 – 2.55 mmol/L
P	1.9	0.8 – 1.5 mmol/L
C3	1.6	0.8-1.4
C4	0.2	0.2-0.5

Discussion

SLE is a complex autoimmune and idiopathic condition that affects connective tissues. It is characterized by the formation of autoantibodies, the accumulation of immune complexes, inflammation, and, ultimately, irreversible organ damage. Managing SLE patients involves various approaches and lifestyle recommendations, including avoiding direct or reflected sunlight. The diverse clinical manifestations of SLE present a challenge to the clinician. Several mechanisms lead to a loss of self-tolerance and organ dysfunction. Impressive progress has been made over the last several years toward understanding how almost every aspect of the immune system contributes to the expression of systemic autoimmunity. The kidney is involved in more than half of patients with SLE and contributes significantly to its morbidity. The contribution of autoantibodies with a number of reactivities and of immune complexes in the expression of kidney inflammation has been reviewed extensively over the years. Studies of the involved organs, including skin, the kidney and the brain, have revealed local processes that are responsible for tissue damage. Without ignoring the role of the autoimmune response in the organ damage, it is

possible that the local inflammatory processes proceed independently with little or no outside input. Better understanding of these processes should open new approaches to the disease treatment [8,9].

This case illustrates new-onset SLE presenting with lupus nephritis in the post-partum period. Immune rebound following pregnancy has likely contributed to the disease activation [7,10]. The combination of a nephrotic-range proteinuria, hematuria, malar rash, reduced ejection fraction, and positive autoantibodies pointed strongly toward lupus nephritis, that was biopsy confirmed [6]. Early intervention with corticosteroids, cyclophosphamide [8,9], and plasmapheresis resulted in significant improvement.

Conclusion

Early recognition of lupus nephritis in the post-partum period is crucial for the treatment of the lupus nephritis. This patient achieved complete clinical remission following immunosuppressive therapy and continues under nephrology follow-up due to the possible risk of relapse.

Conflict of interest statement. None declared.

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