Case Report

Lupus Podocytopathy in an Adolescent Nigerian Girl: A Rare Renal Manifestation of SLE

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ABSTRACT

Lupus podocytopathy (LP) is a rare renal manifestation of systemic lupus erythematosus (SLE), characterized by nephrotic syndrome and minimal change disease or focal segmental glomerulosclerosis in the absence of immune complex deposition. It is often under-recognized, especially in low-resource settings due to limited access to advanced diagnostic tools. We report the case of a 15-year-old Nigerian female who presented with a 10-month history of recurrent body swelling without other systemic features of SLE. Initial evaluation revealed nephrotic-range proteinuria (6.94 g/24h), hypoalbuminemia (13.1 g/L), positive ANA (1:160), and anti-dsDNA (1:172), with normal renal function. Renal biopsy showed features consistent with minimal change disease and no immune deposits on immunohistochemistry. A diagnosis of lupus podocytopathy was made. She was treated with high-dose prednisolone and mycophenolate mofetil (MMF), achieving partial remission by 6 months and complete remission by 12 months. Maintenance therapy with azathioprine was commenced thereafter, with sustained remission. In Conclusion, Lupus podocytopathy should be considered in young patients presenting with nephrotic syndrome and positive lupus serologies even in the absence of other systemic manifestations. Early diagnosis and prompt immunosuppressive therapy can result in favourable outcomes. This case underscores the importance of renal biopsy and serologic testing in evaluating atypical lupus presentations in African clinical settings.

Keywords: Adolescent, Immunosuppression Lupus podocytopathy, Minimal Change Disease, Nephrotic Syndrome, Nigeria, Systemic Lupus Erythematosus.

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CASE PRESENTATION

A 15-year-old secondary student presented to our centre 4 years ago with 10 months history of recurrent body swelling. There was no associated breathlessness and no history of yellow eyes. Also there was no decline in urine output nor gross haematuria with no history of prodromal illness. No prior ingestion of any medication or strange foods. No preceding history of sore throat, body rashes, joint pains or swellings. She was not a known Hypertensive or Diabetic and she had no family history of such including kidney disease. She had a normal childhood history. She has had multiple courses of Prednisolone 5 to 20mg all the while, and Frusemide with no lasting response.

Clinical examination essentially showed residual body swelling otherwise it was unremarkable. Her blood pressure then was 100/70mmHg. Urinalysis showed 3+ protein. Serum albumin was 13.1g/L. Urine microscopy had no active urinary sediments.

An impression of Nephritic Nephrotic syndrome was made to exclude the cause. Further investigations included 24h urine protein of 6.94g, Repeat Serum albumin 26g/L. Complete blood count showed Haemoglobin of 12.5g/dL, white blood cells of 4.7 x 10^9/L, Platelet count 276 x 10^9/L. Clotting profile was normal. Urea electrolyte and Creatinine were normal (Creatinine 61 µmol/L). Lipid profile (mmol/L): Total Cholesterol 6.9 mmol, LDL 4.7, HDL 1.7, TG 1.2. Antinuclear antibody was positive (1:160). Anti double stranded DNA (1:172) was also positive. Serology to Hepatitis B and C, and HIV were all non-reactive.

She had renal biopsy done and histology was as followed: Essentially normal microscopy. Immunohistochemistry for IgG, IgA, and IgM and C3 also stained negative. Findings were consistent with Minimal change disease. Patient was managed as per protocol for Lupus Podocytopathy.

She had Prednisolone 40mg daily and Mycophenolate mofetil (MMF) 1g bid, along with frusemide and Irbesartan, but only showed evidence of partial remission after 6 months course. 24hour urine protein became 1.38g. Prednisolone was then tapered down over subsequent two months and maintained on MMF 500mg bid to complete 1 year course. By the end of 1 year she attained complete remission, 24hour urine protein became 0.39g. By the next follow 3 months later 24hour urine protein became 0.14g. MMF was converted to Azathioprine 50mg daily for which she is still currently maintained on them with excellent outcome so far.

DISCUSSION

Lupus podocytopathy (LP) is a rare but recognized manifestation of systemic lupus erythematosus (SLE), characterized by nephrotic syndrome and minimal change disease (MCD) or focal segmental glomerulosclerosis (FSGS) patterns on renal biopsy in the absence of immune complex deposition on immunofluorescence or electron microscopy. It accounts for approximately 1% to 2% of lupus nephritis cases globally and is classified under the ISN/RPS 2018 lupus nephritis revisions as a non-immune complex-mediated podocytopathy associated with SLE[1].

Our patient, a 15-year-old girl, presented with classical features of nephrotic-range proteinuria and hypoalbuminemia in the absence of hypertension, or renal dysfunction. The renal histology findings of minimal change disease (MCD), absence of immune deposits, and a seropositive ANA and dsDNA profile strongly supported the diagnosis of lupus podocytopathy. This aligns with reports in paediatric and adolescent populations where LP can often mimic idiopathic nephrotic syndrome at onset[2].

A Nigerian study by Okafor et al. highlights that while lupus nephritis remains a significant cause of morbidity among young females with SLE, the diagnosis of lupus podocytopathy may be under-recognized due to limited access to renal biopsy and immunohistochemistry[3]. In a retrospective review at a tertiary centre in Ibadan, minimal change disease was the predominant lesion seen in young lupus patients with nephrotic syndrome, raising suspicions of podocytopathy but without definitive electron microscopy confirmation[4].

In other African contexts, such as Ghana and South Africa, reports similarly identify the challenge of diagnosing LP due to overlapping features with idiopathic nephrotic syndrome and limited

diagnostic facilities[5]. Asian studies, particularly from China and India, have been pivotal in characterizing LP. Hu et al. documented a series of patients with biopsy-proven MCD or FSGS patterns and serologic evidence of active SLE, some without other organ manifestations[6]. Indian literature also supports that LP often presents with isolated nephrotic syndrome and responds variably to steroids and calcineurin inhibitors[7].

Therapeutic response in LP has been found to be better with combined immunosuppression. In our patient, the use of high-dose corticosteroids and mycophenolate mofetil (MMF) resulted in partial remission within six months and complete remission by one year. This mirrors findings in several cohorts where MMF and steroids have been used successfully to induce remission in lupus podocytopathy[8]. A shift to azathioprine for maintenance therapy is consistent with protocols suggested in western and Asian studies to sustain remission and reduce steroid burden[9].

In the West, studies from the United States and Europe suggest that while LP is relatively rare, its prognosis is favourable with early diagnosis and appropriate immunosuppression[10]. A multicentre study in France observed that a significant proportion of lupus patients with nephrotic syndrome and no proliferative or membranous lesions had underlying podocytopathy, often showing prompt response to corticosteroids[11].

It is worth noting that while response to treatment is generally favourable, relapse can occur, and long-term follow-up is essential. The absence of other systemic lupus manifestations in our patient also aligns with some literature reports suggesting that LP may precede or remain isolated from other SLE-related organ involvement[12].

CONCLUSION

Lupus podocytopathy management can be tenacious and challenging in both diagnosis and treatment.

Recommendations

Some of the MCD might not be Idiopathic as often assumed, further evaluation might reveal more aetiology with or without systemic disease. Treatment of Lupus podocytopathy might require beyond steroid only treatment even as first line. There might be a need to extend expected time for remission beyond the maximum four months in adolescents and adults, as recommended by KDIGO guidelines.

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