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Evolution of minimal change disease to probable focal segmental glomerulosclerosis in a patient with corticosteroid-resistant nephrotic syndrome and mesangial IgM deposition: A case report. Case

Evolution from minimal change disease to probable focal and segmental glomerulosclerosis in a patient with steroid-resistant nephrotic syndrome and mesangial IgM deposition: Case report

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SUMMARY

Corticosteroid-resistant nephrotic syndrome in adults represents a significant diagnostic and therapeutic challenge, especially due to its association with a higher risk of renal progression and recurrence. This report describes the case of a 66-year-old male patient initially diagnosed with minimal change disease, who did not respond adequately to corticosteroid therapy and experienced multiple relapses. After the introduction of tacrolimus, he showed a late partial response. A new renal biopsy performed during a relapse revealed focal mesangial IgM deposition, raising the hypothesis of progression to unsampled focal segmental glomerulosclerosis. This case reinforces the diagnostic difficulty of podocytopathies in adults and highlights the possible prognostic role of mesangial IgM deposition in corticosteroid-resistant patients.
Keywords: nephrotic syndrome; focal segmental glomerulosclerosis; minimal change disease; IgM; tacrolimus; podocytopathy.

ABSTRACT

Steroid-resistant nephrotic syndrome in adults remains a major diagnostic and therapeutic challenge, especially due to its association with a higher risk of renal progression and frequent relapses. We report the case of a 66-year-old male patient initially diagnosed with minimal change disease, who showed no adequate response to corticosteroid therapy and evolved with multiple relapses. After tacrolimus initiation, a partial late response was observed. A second renal biopsy performed during relapse demonstrated focal mesangial IgM deposition, raising the possibility of progression to unsampled focal and segmental glomerulosclerosis. This case highlights the diagnostic complexity of adult podocytopathies and the possible prognostic role of mesangial IgM deposition in steroid-resistant patients.

Keywords: nephrotic syndrome; focal segmental glomerulosclerosis; minimal change disease; IgM; tacrolimus; podocytopathy.

INTRODUCTION

Corticosteroid-resistant nephrotic syndrome (CRNS) in adults remains a significant challenge.

Therapeutic use in nephrology is primarily due to the increased risk of progression to kidney disease.

chronic and with a lower remission rate compared to corticosteroid-responsive cases. Among the

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primary podocytopathies, Minimal Change Disease (MCD), and Segmental Glomerulosclerosis and Focal scleroderma (FSGS) exhibits overlapping clinical and histological characteristics, and may represent different stages of the same glomerular disease.

Although DLM (dystrophic lymphoma) usually responds well to corticosteroids, some patients They develop therapeutic resistance and frequent relapses. In these situations, one should consider the The possibility of FSGS was not initially identified, mainly due to the focal nature of the lesions. sclerotic. The clinical significance of mesangial IgM deposition remains controversial. Some Studies suggest an association with a higher frequency of relapses and a poorer therapeutic response.

OBJECTIVE

To describe the clinical and histopathological evolution of a patient with nephrotic syndrome. corticosteroid-resistant disease initially diagnosed as Minimal Change Disease, with subsequent Mesangial deposition of IgM and probable evolution to FSGS.

CASE REPORT

A 66-year-old male patient began experiencing progressive edema in his lower limbs in 2020. associated with nephrotic-range proteinuria, hypoalbuminemia, and dyslipidemia. It did not present Significant hematuria and no significant impairment of renal function at diagnosis.

An initial renal biopsy was performed, and optical microscopy revealed no glomerular abnormalities. Relevant, without signs of segmental sclerosis or mesangial proliferation. Immunofluorescence The test was negative for immune deposits. The findings were consistent with minimal change disease. The patient received full corticosteroid therapy with prednisone, however, without clinical remission after appropriate treatment, characterizing primary corticosteroid resistance, according to KDIGO criteria. 2021 [7].

In the following years, he experienced multiple nephrotic relapses, maintaining persistent proteinuria and Recurrent episodes of edema. In 2022, tacrolimus was introduced, with a response.

Late partial reduction and progressive reduction of proteinuria.

In 2025, during a new recurrence, a second renal biopsy was performed. Optical microscopy, Again, it did not show significant glomerular changes. However, immunofluorescence demonstrated focal mesangial deposition of IgM, with segmental enhancement, in a glomerulus. Despite the absence of evident segmental sclerosis, the clinical evolution of the case raised the hypothesis. progression to FSGS not sampled. The absence of juxtamedullary glomeruli in the sample may

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which contributed to the diagnostic limitations. In the following years, he experienced multiple relapses.
nephrotic syndrome, maintaining persistent proteinuria and recurrent episodes of edema. In 2022, the decision was made to
through the introduction of tacrolimus, with a delayed partial response and progressive reduction of proteinuria.
In 2025, during a new recurrence, a second renal biopsy was performed. Optical microscopy,
Again, it did not show significant glomerular changes. However, immunofluorescence
demonstrated focal mesangial deposition of IgM, with segmental enhancement, in a glomerulus.
Despite the absence of evident segmental sclerosis, the clinical evolution of the case raised the hypothesis.
progression to FSGS not sampled. The absence of juxtamedullary glomeruli in the sample may
to have contributed to the diagnostic limitation.

DISCUSSION

Differentiation between Minimal Change Disease (MCD) and Focal Segmental Glomerulosclerosis
(GESF) can be challenging, especially in adults with corticosteroid-resistant nephrotic syndrome.
Currently, it is understood that both are part of a spectrum of podocytopathies, sharing
mechanisms related to structural and functional damage to the podocyte [1,2]. In some patients,
Particularly in those with corticosteroid resistance, histological progression may occur.
throughout evolution, with the appearance of focal areas of glomerular sclerosis.
In the present case, corticosteroid resistance since diagnosis is noteworthy, given that DLM
Classic adult rheumatoid arthritis usually responds well to corticosteroid therapy [1]. In addition, maintaining
Persistent proteinuria, associated with multiple relapses over the years, suggests ongoing activity.
of podocyte injury.
Another important point is the possibility of sampling error. In the early stages of FSGS, the lesions
They can be focal and preferentially affect juxtamedullary glomeruli, which makes their detection difficult.
identification in some renal biopsies [2,3]. Thus, the absence of segmental sclerosis
Evidence visible under an optical microscope does not completely rule out the diagnosis of early FSGS.
It is possible that the persistence of subclinical podocyte lesions over the years has contributed to
progressive glomerular remodeling, culminating in a phenotype consistent with early FSGS,
not yet demonstrated histologically.
The late appearance of focal mesangial IgM deposition in the second biopsy also warrants consideration.
highlight. The significance of this finding remains controversial in the literature. Some authors
They interpret IgM only as passive trapping secondary to increased permeability.
glomerular, while others suggest active participation in perpetuating glomerular injury [4–6].
Observational studies demonstrate an association between mesangial IgM deposition and higher frequency.

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relapses and reduced response to corticosteroids, especially in patients with syndrome
corticosteroid-resistant nephrotic syndrome [4,5]. In this context, the finding observed in this case may represent a
An indirect marker of persistent podocyte damage and a worse renal prognosis.
A delayed and partial response to tacrolimus is also a relevant aspect of the clinical course. In addition to...
Due to their immunosuppressive effect, calcineurin inhibitors exert a direct action on podocytes.
promoting the stabilization of the cellular cytoskeleton through the preservation of synaptopodin [8].
This mechanism helps explain the reduction in proteinuria even in patients with the disease.
prolonged and partially refractory.

CONCLUSION

This case reinforces the diagnostic complexity of corticosteroid-resistant podocytopathies in adults.
Prolonged clinical course, associated with mesangial IgM deposition and resistance to
Corticosteroid use suggests possible progression to FSGS, even in the absence of sclerotic lesions.
evident in the renal biopsy.
The case also highlights the diagnostic limitations stemming from the focal nature of the disease and reinforces
The importance of clinicopathological correlation in follow-up. Furthermore, the late and partial response.
The use of tacrolimus suggests a potential benefit of calcineurin inhibitors even in patients with
prolonged illness.

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