



MINIMAL CHANGE DISEASE IN AN ADULT PATIENT: A RARE PRESENTATION FOR THE AGE.

Nephrology

Dr Dhruveeshinh A. Parmar

M.B.B.S. M.D. Medicine., Senior Resident, PIMSR, Parul University.

Dr Pooja Harshitha Karnayina

M.B.B.S. M.D. Medicine., Assistant Professor, Dhiraj Hospital, Sumandeep Vidyapeeth.

Dr Kesha A. Parmar

M.B.B.S. M.D. Pharmacology. Senior Resident, S.B.K.S. MI & RC, Sumandeep Vidyapeeth.

ABSTRACT

Background: Minimal change disease (MCD) is a common cause of nephrotic syndrome in children but is rarely observed in adults. Limited literature exists on its incidence and presentation in the adult population. **Case Presentation:** We report the case of a 20-year-old female who presented with generalized body swelling and reduced urine output for one month. **Investigations:** Physical examination revealed periorbital and pedal edema. Laboratory investigations prompted a renal biopsy. Histopathology and immunofluorescence supported the diagnosis of MCD. **Diagnosis:** Minimal change disease was diagnosed based on light microscopy and immunofluorescence findings. **Treatment:** The patient was initially treated with diuretics and statins. Following biopsy confirmation, she was started on oral prednisolone and torsemide. **Conclusion:** Minimal change disease, though uncommon in adults, should be considered in patients presenting with nephrotic syndrome.

KEYWORDS

Minimal change disease, Nephrotic syndrome, FSGS, Adult MCD

INTRODUCTION

Minimal change disease (MCD) accounts for 70–90% of nephrotic syndrome cases in children but only 10–15% in adults. Characterized by diffuse podocyte foot process effacement on electron microscopy with unremarkable findings on light microscopy, MCD presents with nephrotic syndrome: proteinuria, hypoalbuminemia, edema, and hyperlipidemia.

Adult MCD diagnosis is challenging due to its overlap with conditions such as FSGS, membranous nephropathy, and diabetic nephropathy. Biopsy is essential in adult cases, especially when steroid resistance or atypical features are present. MCD may be idiopathic or secondary to drugs (e.g., NSAIDs, lithium), malignancies (e.g., Hodgkin lymphoma), or infections (e.g., HIV). We report a rare case of MCD in a 20-year-old female without identifiable secondary causes.

Case Report

A 20-year-old female presented with one month of progressive generalized edema, starting periorbitally and progressing to the lower limbs, face, and abdomen. She also reported oliguria for three weeks, along with frothy urine and dysuria. Her history was unremarkable, including a recent healthy vaginal delivery.

Initial lab results (see Table 1) revealed anemia, hyponatremia, hypokalemia, hyperlipidemia, and nephrotic-range proteinuria. Renal function was preserved. USG showed normal-sized kidneys with corticomedullary differentiation, moderate ascites, and bilateral pleural effusion. A 2D echocardiogram was normal.

Table 1: Baseline Laboratory Investigations

Parameter	Value	Normal Range
Hemoglobin	10.6 g/dL	12–16 g/dL
Serum Creatinine	0.9 mg/dL	0.6–1.2 mg/dL
Sodium	129 mEq/L	135–145 mEq/L
Potassium	3.2 mEq/L	3.5–5.1 mEq/L
Triglycerides	170 mg/dL	<150 mg/dL
Urinary Albumin	+4	Negative

Renal Biopsy

Light microscopy revealed seven glomeruli with normal cellularity, open capillary loops, and intact basement membranes. There was no mesangial proliferation, sclerosis, crescents, or amyloid. Tubules showed protein resorption droplets. Interstitium and vessels were unremarkable.

Immunofluorescence on five glomeruli showed no significant IgG, IgA, IgM, C3, C1q, kappa, or lambda deposition. A single glomerulus had weak non-specific IgM staining. These findings were consistent

with minimal change disease.

Treatment And Outcome

The patient was started on oral prednisolone 50 mg once daily and torsemide 10 mg once daily. Supportive management included sodium restriction and NSAID avoidance.

Clinical follow-up showed symptomatic improvement.

DISCUSSION

MCD is a podocytopathy presenting with nephrotic syndrome and characterized by electron microscopic podocyte effacement. Light microscopy typically appears normal. Distinguishing MCD from FSGS is critical due to therapeutic and prognostic implications.

In adults, MCD accounts for fewer cases and presents diagnostic difficulty given overlapping features with other glomerular diseases. Biopsy and immunofluorescence are essential for diagnosis.

Studies suggest T-cell dysfunction, with associations to atopy and Hodgkin lymphoma, contribute to pathogenesis [1,2]. Secondary MCD may arise from drugs (e.g., NSAIDs, lithium) or malignancy. In our case, no secondary cause was identified.

Our findings align with previous adult MCD case reports, including preserved renal function, absence of hematuria, and biopsy confirming lack of sclerosis [3,4]. As per Hogan et al., corticosteroids remain first-line treatment, though 10–30% of adults may require calcineurin inhibitors or rituximab [5].

Our patient responded well to corticosteroids, reinforcing MCD as an important differential even in young adult females.

CONCLUSION

MCD, though uncommon in adults, should be considered in patients presenting with nephrotic syndrome. Early renal biopsy and appropriate therapy are essential for diagnosis and management.

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