RESEARCH PAPER

A Rare Case of Acute Kidney Injury in an Adult Male: Coexistence of Acute Tubular Injury with Minimal Change Disease

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ABSTRACT
Minimal Change Disease (MCD) is an uncommon cause of nephrotic syndrome in adults and rarely presents as acute kidney injury (AKI). This case report describes a 58-year-old male who developed AKI in the context of MCD, confirmed through renal biopsy. His clinical course was marked by oliguric renal failure necessitating dialysis, and subsequent recovery was achieved through high-dose corticosteroids, immunosuppression with mycophenolate mofetil (MMF), and supportive care. This case highlights the critical role of renal biopsy in diagnosing atypical presentations of AKI in adults and underscores the therapeutic benefits of early immunosuppressive treatment
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INTRODUCTION

Acute Kidney Injury (AKI) is a common and serious clinical problem encountered across healthcare settings, with Acute Tubular Necrosis (ATN) being one of its predominant intrinsic causes. ATN is typically triggered by prolonged hypotension, sepsis, or exposure to nephrotoxins such as aminoglycosides and contrast agents, resulting in the death of tubular epithelial cells and a subsequent decline in renal function [1,2].

Minimal Change Disease (MCD), while the leading cause of nephrotic syndrome in children, is far less common in adults. It is characterized by podocyte foot process effacement on electron microscopy with minimal or no abnormality seen on light microscopy [3]. MCD is highly responsive to corticosteroids, but adult cases can present with atypical features, including acute kidney injury [4,5].

CASE

A 58-year-old male with no known history of chronic illness presented with markedly reduced urine output over three days, following a two-day febrile illness that resolved with self-medication. He denied symptoms such as vomiting, hematuria, abdominal pain, or breathlessness. Clinical examination revealed a hemodynamically stable and afebrile patient with no signs of volume overload.

Laboratory investigations revealed elevated serum urea (106mg/dl) and creatinine (7.2mg/dl) levels, along with significant hyponatremia (122meq/l) and mild hypokalemia (3.51meq/l). Platelet count was notably reduced, and ESR was elevated. Infectious etiologies such as dengue and scrub typhus were ruled out, although urine culture showed growth of Acinetobacter species. Blood cultures remained sterile. Immunologic screening for ANA, ANCA, and anti-GBM antibodies was negative. Cardiac function was preserved as shown on echocardiography.

Despite fluid resuscitation, urine output did not improve, necessitating the initiation of hemodialysis. A renal biopsy was performed, which revealed features of both acute tubular injury and Minimal Change Disease (fig.1). These findings confirmed the diagnosis of AKI with coexisting

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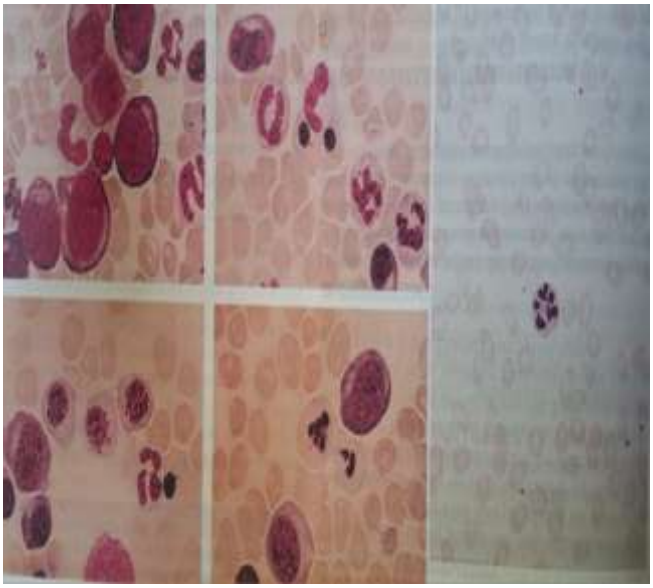
glomerular and tubular pathology.



The patient was managed with high-dose intravenous methylprednisolone for three days, followed by oral corticosteroids. Hemodialysis was continued on alternate days during the acute phase. Immunosuppressive therapy was escalated with mycophenolate mofetil (MMF) 360 mg once daily for a total duration of 16 weeks, alongside corticosteroids. An ACE inhibitor was added for renal protection and reduction of proteinuria. Supportive measures included fluid restriction, electrolyte correction, and infection control guided by culture sensitivity.

Over the course of treatment, the patient gradually showed significant clinical improvement. Urine output increased progressively, allowing discontinuation of dialysis. Serial follow-ups showed a steady decline in serum creatinine to within normal limits (urea – 23mg/dl , creatinine – 0.87mg/dl). Urine protein levels significantly decreased, indicating improvement in glomerular function. The patient tolerated the treatment regimen well, with no major adverse effects. At the end of 16 weeks, renal function had normalized, proteinuria had resolved, and the patient remained asymptomatic and stable.

	UREA	CREATININE
AT PRESENTATION	106mg/dl	7.2mg/dl
POST DIALYSIS/PRE BIOPSY	62mg/dl	4.9mg/dl
AT DISCHARGE	91mg/dl	4.8mg/dl
AT REVIEW AFTER 2 WEEKS	29mg/dl	0.82mg/dl
AT REVIEW AFTER 4 WEEKS	23mg/dl	0.87mg/dl

DISCUSSION

Minimal Change Disease presenting with AKI in adults is rare and often underrecognized without a renal biopsy. In most adult presentations, MCD is associated with nephrotic syndrome and preserved renal function [4]. However, when it presents as AKI, the underlying mechanisms may include volume depletion, hemodynamic instability, and direct podocyte injury [3,6].

In this case, the coexistence of acute tubular injury and MCD complicated the clinical picture. Acute tubular injury likely resulted from transient hypoperfusion during the febrile episode or localized infection, while the underlying podocytopathy contributed to glomerular dysfunction. This overlap has been previously documented in select cases and emphasizes the necessity of histological confirmation [6,7].

Treatment involved corticosteroids, which are the mainstay of therapy in MCD, even when associated with AKI. The addition of mycophenolate mofetil in this case provided extended immunosuppression, helping reduce the risk of relapse and taper steroid use. Serial monitoring confirmed functional recovery, and the patient remained dialysis-free at discharge and on follow-up.

CONCLUSION

This case underscores the importance of considering Minimal Change Disease in the differential diagnosis of AKI in adults, particularly in the absence of systemic autoimmune or infectious markers. Renal biopsy plays a central role in such diagnostic dilemmas. Timely initiation of corticosteroids, extended immunosuppression with MMF, and supportive care can result in full recovery, even in complex presentations involving dual pathologies...

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