ORIGINAL RESEARCH PAPER

Nephrology

IgA NEPHROPATHY: MASQUERADING AS AKI IN YOUNG PATIENT

KEY WORDS: IgA Nephropathy, Acute Kidney Injury.

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ABSTRACT

The most prevalent primary glomerulonephritis in world, IgA nephropathy usually manifests as moderate proteinuria and asymptomatic hematuria. This case describes a young male's atypical presentation of acute kidney injury (AKI) and systemic symptoms, which was diagnosed as IgA nephropathy with acute tubular damage and global tuft sclerosis characteristics. The immune complexes are nephritogenic, c directly to cause glomerular inflammation and mesangial proliferation. Activation of the local and systemic renin angiotensin system and complement activation also ultimately contribute to glomerulosclerosis and tubulo-interstitial fibrosis, leading to loss of renal function.

INTRODUCTION

IGA nephropathy is characterized by episodic hematuria and deposition of iga deposits in the mesangium. Iga nephropathy has male preponderance occuringin 2nd and 3rd decade of life. Its close differential is Henoch Scholein Purpura mainly abdominal complaints. which is distinguished by the prominent systemic illness. It occurs due to the synthesis of poorly galactosylated Iga 1. Renal biopsy for the diagnosis is compulsory even if there is significant rise of Iga levels in serum and skin biopsy.

Case Report

A 25-year-old male presented with a 3-day history of vomiting and abdominal pain, a 7-day history of rash, and lower limb swelling for 2 months. He reported decreased urine output for the past 5 days and a history of frothy urine, suggestive of nephrotic-range proteinuria(3.36g/day). Laboratory findings revealed elevated serum creatinine, indicating acute renal impairment. Urinalysis showed proteinuria and microscopic hematuria. Given the unexplained AKI and systemic symptoms, a renal biopsy was performed for definitive diagnosis.

On Examination

General examination: Pallor, rash and lower limb swelling. Respiratory: Bilateral basal crepitations.

Cardiovascular, abdominal, neurological: No abnormalities.

Investigations

Urine output: Decreased over 5 days

- HB-9.7/WBC-7.49/ PLT-310
- Na-134/k-5.4/cl-105
- Ca-8.5/Mg-3.17/ Hco3-14.1
- ESR-70
- CRP-1.92
- Serum creatinine: 7.89mg/dl
- C3-128
- ANA BY IF NEGATIVE
- C-ANCA and P-ANCA- NEGATIVE
- Ferritin-133/iron-107/tsat-42
- Urinalysis: Proteinuria(3.36g/day) and microscopic hematuria(10-12 rbc/hpf)
- Renal biopsy: Performed for definitive diagnosis

Biopsy report : The renal biopsy confirmed a diagnosis of IgA nephropathy, revealing:

- Global tuft sclerosis in 5 of 14 (35%) glomeruli
- Mild mesangial matrix expansion and cellularity
- Acute tubular injury with multifocal chronic interstitial inflammation
- Moderate tubulointerstitial chronicity

Thus the Final Diagnosis as
IgA nephropathy with global sclerosis, acute tubular injury, and features of nephrotic syndrome and AKI.

MANAGEMENT

Patient had received steroids pulse therapy of MPS 1 g for 3 days after which pt. had decrease in the proteinuria his 24 hour urine proteins post treatment had reduced to approximately 650-700mg/day.

DISCUSSION

Two most common clinical presentations are asymptomatic hematuria and progressive kidney disease. Clinical manifestations of Iga nephropathy vary from asymptomatic with microscopic or intermittent macroscopic haematuria and stable kidney function to rapidly progressive glomerulonephritis. This case underscores the importance of considering IgA nephropathy in the differential diagnosis of young patients presenting with unexplained AKI and systemic manifestations. Early recognition and renal biopsy are crucial for accurate diagnosis and management

CONCLUSION

This case highlights an atypical presentation of IgA nephropathy in a young male with systemic symptoms, acute kidney injury and massive proteinuria in nephrotic range. Therefore clinicians should maintain a high index of suspicion for IgA nephropathy in similar presentations to facilitate timely diagnosis and management.

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