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Successful Treatment of Acute Kidney Injury on Chronic Kidney Disease Due to Tubulointerstitial Nephritis and Acute Tubular Necrosis With Steroids and Mycophenolate Mofetil: A Case Report

Iqra Ahmad, Merina Khan, Syed Nayer Mahmud

Department of Nephrology, Shifa International Hospital, Islamabad, Pakistan.

Abstract:

Acute tubulointerstitial nephritis causes interstitial inflammation and tubular injury leading to acute kidney injury which can present as rapid decline in renal function. Early diagnosis and prompt treatment is essential. After taking informed consent from our patient we present a case report where a patient with chronic kidney disease presented with rapid decline of renal function. Furthermore, renal biopsy revealed the diagnosis of tubulointerstitial nephritis. She was later started on steroids and mycophenolate mofetil, to which she responded adequately. From this case we conclude that steroids and mycophenolate mofetil are helpful in treatment in acute tubulointerstitial nephritis.

KEY WORDS:

Tubulointerstitial nephritis, acute kidney injury, acute tubular injury, steroids, mycophenolate mofetil.

Corresponding Author

Dr. Merina Khan
Department of Nephrology,
Shifa International Hospital, Islamabad, Pakistan
Email: khanmerina@yahoo.com
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Introduction:

Acute tubulointerstitial nephritis is one of the significant contributors to acute kidney injury. It causes interstitial inflammation and tubular injury, the risk factors of which include infections, drugs, autoimmune diseases and systemic form of diseases. Acute tubulointerstitial nephritis induced acute kidney injury can present as rapid and significant decline in renal function. Early diagnosis and prompt treatment can prevent permanent and irreversible damage to the kidney.

According to kidney biopsy reports and registries the incidence of ATIN ranges between 1% to 3% for all patients biopsied, and 15% to 27% for patients who undergo biopsy due to acute kidney injury.[1] In addition, 2% of chronic kidney disease (CKD) cases are associated with acute tubulointerstitial nephritis.[2] It should however be considered that renal biopsies are usually performed in cases with rapid and significant decline in renal function potentially overlooking the milder and subclinical cases.

Kidney biopsies are gold standard for diagnosing ATIN. It is characterized by interstitial inflammation, tubulitis and cellular infiltration predominantly by T lymphocytes. Under light microscopy the tubulointerstitium shows patchy or diffuse edema with T cell predominant lymphocytic infiltration. Electron microscopy does not reveal any specific findings and immunofluorescence does not reveal any specific staining.[3]

Treatment usually includes stopping the potential offending agent which can cause resolution of signs and symptoms of ATIN but if there is no resolution and renal biopsy is consistent with less severe ATIN then steroids are considered but if the interstitial fibrosis and tubular atrophy on renal biopsy is severe then they are less likely to respond to immunosuppressive therapy and supportive treatment is advised.[4]

Here we present a case where a patient of chronic kidney disease with stable creatinine developed acute tubular interstitial nephritis which was confirmed through renal biopsy and she was then started on immunosuppressive therapy which eventually led to improvement in her renal function tests.

Case Report:

A 77 years old female patient known diabetic, hypertensive and history of thyroidectomy 3 years ago presented to emergency with presenting complaints of nausea, vomiting and bilateral limb swelling for one week. She is a known case of chronic kidney disease secondary to diabetic nephropathy with baseline creatinine of 1.7mg/dl. There was no history of any recent infection or use of any nephrotoxic drugs. Current medications included thyroxine, glimepiride 1 mg, linagliptin 5 mg, fenofibrate 67 mg, nifedipine 30 mg, febuxostat 80 mg and bisoprolol 5 mg.

On examination, she was vitally stable with double positive pitting edema. Laboratory findings revealed a fall in glomerular filtration rate along with significant rise in creatinine from baseline in two months that is from 1.7 mg/dl to 4.3 mg/dl. Urine routine examination showed no urinary sediment and her autoimmune profile which included C3 levels, anti- glomerular basement membrane antibodies, P-ANCA, C-ANCA were negative. Ultrasound kidney ureters and bladder revealed right kidney to be 77 x 36 mm and left kidney to be 96 x 33 mm with grade one renal parenchymal changes. Detailed laboratory findings are mentioned below.

Renal biopsy was done to rule out the cause of sudden deterioration of renal function test. It showed 33 glomeruli out of which 20 glomeruli were globally sclerosed, morphology and immunofluorescence was consistent with diabetic nephropathy class IV. In addition it showed moderate tubulointerstitial nephritis with moderate acute tubular necrosis and tubular atrophy. Biopsy also showed moderate hypertensive

vasculopathy. She was started on steroids which tapered off rapidly and mycophenolate mofetil was started as the patient was diabetic after which her laboratory findings including renal function tests showed significant improvement.

LABORATORY WORK-UP ON ADMISSION AND AFTER STARTING IMMUNOSUPRESSION			
LABORATORY TEST	REFERENCE RANGE	ON DAY OF ADMISSION	AFTER STARTING STEROIDS AND MMF
Hemoglobin	11-17 mg/dl	10.6	9.0
White cell count	4000 –10000/µL	9600	9400
Platelet count	150,000 - 450,000 µL	255000	232000
Blood sugar levels (random)	70-110 mg/dl	110	
Serum creatinine	0.6-1.1 mg/dl	5.2	3.95
Blood urea nitrogen	6-24 ng/dl	54	54
Serum sodium	133-144 mmol/dl	126	127
Serum potassium	3.5-5.4 mg/dl	5.1	3.3
Serum bicarbonate	21-32 mmol/l	16	28
Serum calcium	8.4 - 10.2 mg/dl	8.4	9.2
Serum phosphorus	3.5-5.0 mg/dl	6.3	3.2
Urine albumin creatinine ratio	<30 mg/g	51	
Serum albumin	3.5-5.4 mg/dl	4.0	4.2

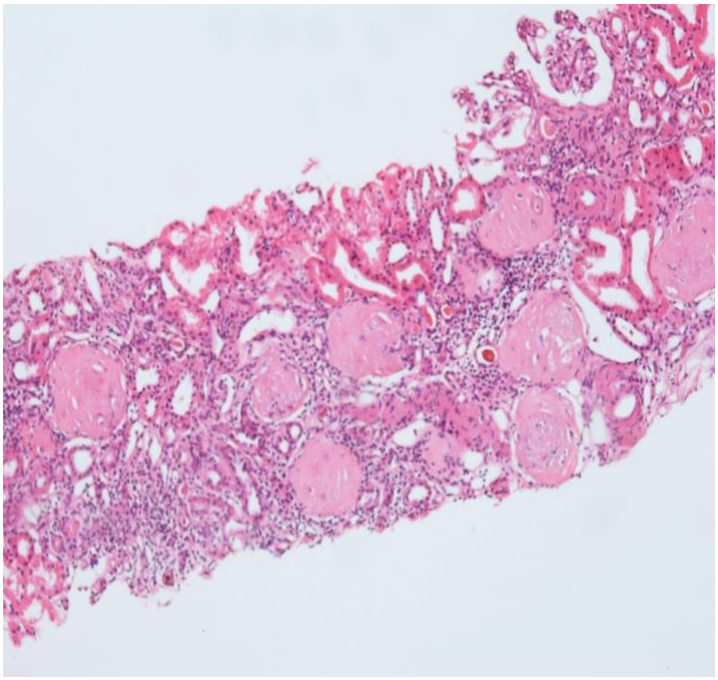


Figure 1: Kidney Biopsy showing sloughing of cells at renal tubules.

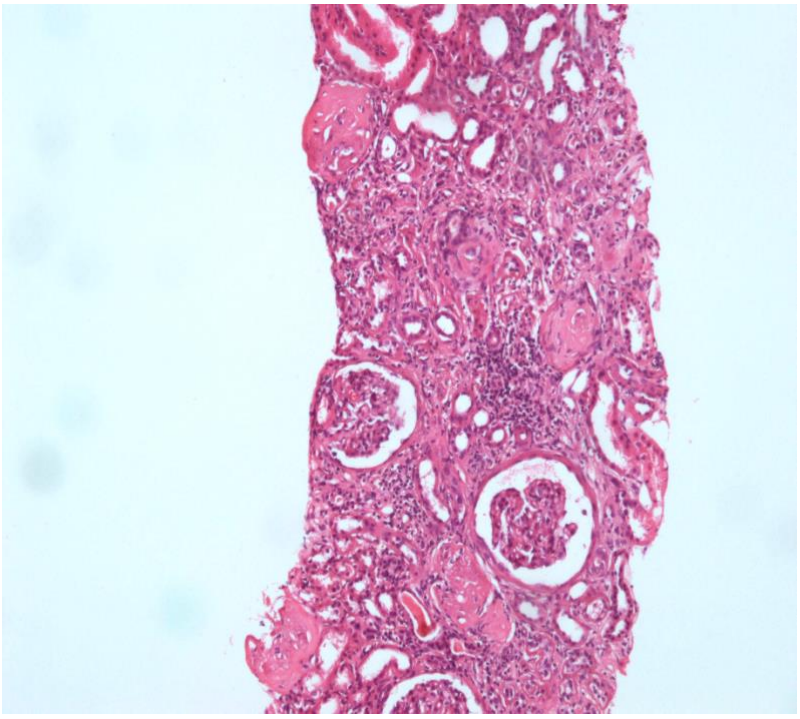
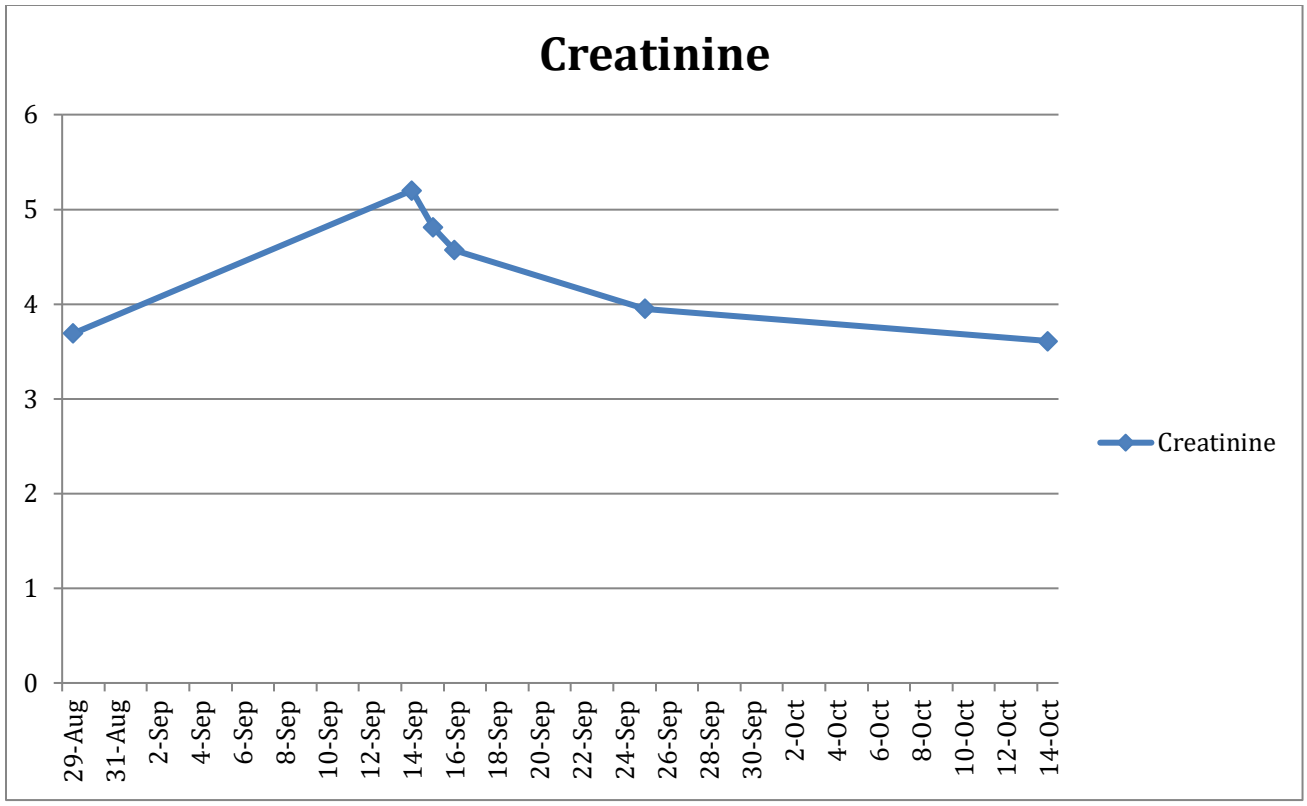


Figure 2: Kidney biopsy showing multiple cellular infiltrates in the Mesangium



Note: Renal biopsy done on 16th Sept 2025 and Steroids and immunosuppressant started on 17th Sept 2025

Figure 3: Line graph showing trend of serum creatinine with introduction of Immunosuppression.

Discussion:

Acute tubulointerstitial nephritis is a clinicopathological entity characterized by acute renal failure, low grade proteinuria and sterile leucocyturia. Pathologically it is characterized by tubular and interstitial inflammation that extends up to the basement membrane eventually leading to tubulitis and tubular degenerative changes. The interstitial infiltrates are predominantly lymphocytes with variable number of plasma cells, eosinophils and neutrophils.[5]

Multiple conditions are associated with acute tubulointerstitial nephritis, the most common being drug induced or allergic. In addition to this, many autoimmune diseases and collagen vascular diseases such as systemic lupus erythematosus, Sjogren's syndrome and mixed connective tissue disorders are linked with causing ATIN. AIN can also be caused by viral or bacterial infections of the renal parenchyma. Despite all these conditions being linked to ATIN, many times no cause of ATIN is identified.[5]

Optimal therapy is by the presumed cause. Stopping the offending agent in drug induced interstitial nephritis remains the mainstay treatment and corticosteroids are reserved for resistant cases. The best

supportive data to treat ATIN with glucocorticorticoids comes from a retrospective study in which 61 with biopsy proven ATIN were considered from which 52 patients were treated with glucocorticoids and the remaining 9 were not. Those treated with steroids had lower frequency of hemodialysis and those patients who were treated within seven days of presentation with ATIN had better outcomes than the others. Other data had similar results.[6, 7] A case series done from 1993 to 2011 included 133 patients with biopsy proven AIN. At 6 months post biopsy, 49% of patients with drug-induced AIN treated with steroids achieved complete recovery; 39%, partial recovery; and 12%, no recovery.[8]

Mycophenolate mofetil has been widely used as immunosuppressive agents. The immunosuppressive properties of MMF are from inhibition of the rate-limiting enzyme in the de novo pathway of purine synthesis. Therefore it reduces lymphocyte proliferation, down regulates the expression of cell surface adhesion molecules, and decreases antibody production.[9] There is limited experience of using immunosuppressive drugs to treat acute interstitial nephritis. Eight cases of steroid resistant, biopsy proven AIN were treated with MMF at an institution for 13-34 months. Patients had a mean decline in serum creatinine from 2.3mg/dl to 1.6mg/dl. Only two patients out of eight showed no improvement in their renal function tests. Therefore it was concluded that MMF is a good therapeutic option for steroid resistant AIN and can be considered as first line treatment.[10]

Similarly our case report showed improvement in renal function tests of a patient with biopsy proven tubulointerstitial nephritis as soon as steroids and MMF was initiated.

Conclusion:

As tubulointerstitial disease has a marked effect on renal function therefore we recommend early treatment with steroids and immunosuppressive agents. Our case report supports this concept but further data is required to refine recommendations.

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