Acute Interstitial Nephritis following Lenalidomide Therapy in a Patient with AL Amyloidosis

Sir,

Drug-induced acute kidney injury (AKI) is a common phenomenon in patients with monoclonal gammopathies.[1] In such instances, it is often difficult to differentiate whether the worsening kidney function is secondary to the disease or its treatment.

We report a case of a 36-year-old lady who presented to our outpatient department with progressive loss of appetite, generalized weakness, progressive leg swelling, and frothy urine for the last 2 weeks. Her evaluation revealed the presence of normal renal functions, hypoalbuminemia, and massive proteinuria. A kidney biopsy was done, which showed mesangial widening with weakly PAS-positive congophilic deposits. The IF study showed a differential staining with smudgy κ deposits (2+) and λ deposits 1+.

As the biopsy showed light chain restriction, further investigations were done, which confirmed the diagnosis of AL amyloidosis. She was started on steroids, bortezomib, and lenalidomide. After 1 month of therapy, her urinary κ levels were 106.7 mg/L. On follow-up, her serum creatinine started to rise 6 weeks after her treatment (serum creatinine was 3.8 mg/dL from 1.2 mg/dL). Within a week, she needed dialysis as her creatinine levels rose to 6.8 mg/dL and she developed uremic symptoms. Her renal functions showed no signs of improvement, and she underwent a repeat kidney biopsy. The second biopsy confirmed the primary diagnosis of AL amyloidosis. There was an additional finding of moderately dense interstitial infiltrate of lymphocytes, plasma cells, and eosinophils in this biopsy [Figure 1]. A diagnosis of allergic interstitial nephritis (probably drug-induced) was considered. A repeat urinalysis showed the presence of eosinophils. Upon review of possible causes of her renal failure, it was felt that lenalidomide may be the responsible agent. Lenalidomide was withdrawn and the renal functions subsequently normalized in the next 2 weeks. Her further therapy consisted of steroids and bortezomib only. Her urinary κ levels further declined to 61.7 mg/L and her serum creatinine was 0.8 mg/dL on the last follow-up.

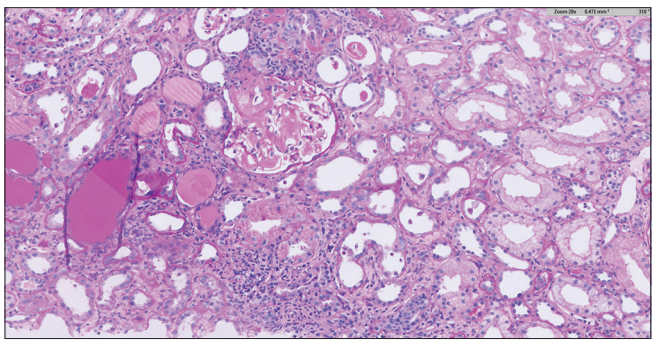

Figure 1: Second kidney biopsy showing intense lymphocytic infiltration in the interstitium (PAS)

AL amyloidosis is a rare clinical presentation in young adults. The kidney is the site of amyloid deposition in 50% of cases[2,3] With the advent of many new drugs the overall prognosis has improved over the last few years.[1] However, in some cases, the therapeutic agent can cause worsening renal failure secondary to allergic interstitial nephritis. The diagnosis of drug-induced interstitial nephritis is often clinically suspected but proven only with a kidney biopsy as noted in our case.[1] With the increasing use of lenalidomide in AL amyloidosis, there have been increasing reports in the literature about its nephrotoxicity. Kidney involvement with lenalidomide included reports of Fanconi syndrome and minimal change disease. One patient had concomitant eosinophilia, rash, and systemic symptoms (DRESS syndrome) with lenalidomide therapy.[4] In a large series, 66% (27 out of 41) of AL amyloidosis patients had kidney dysfunction following treatment with lenalidomide. In these series, kidney failure was severe in 13 (32%) patients, and 4 required dialysis.[5] Patients with severe renal involvement had greater proteinuria and lower serum albumin as was noted in our patient also.

This case report highlights the nephrotoxic potential of lenalidomide and nephrologists should closely monitor renal functions when this drug is used in their patients.

Financial support and sponsorship

Nil.

Conflicts of interest

There are no conflicts of interest.

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Access this article online

Quick Response Code:

Website: www.indianjnephrol.org

DOI: 10.4103/ijn.ijn_101_22

How to cite this article: Anandh U, Vaswani B, Gowrishankar S. Acute interstitial nephritis following lenalidomide therapy in a patient with AL amyloidosis. Indian J Nephrol 2022;32:390-1.

Received: 14-03-2022; **Revised:** 01-05-2022; **Accepted:** 08-05-2022; **Published:** 16-07-2022