

Acute Kidney injury caused by IgA nephropathy induced by Anti-TNF- α therapy (Adalimumab): A case report

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Introduction

Adalimumab is a biologic agent currently established for the management of different immune diseases e.g. rheumatoid arthritis, psoriatic arthropathy and Crohn's diseases. It exerts its immunological effect as an anti-tumour necrosis alpha (TNF α). Case reports points at using TNF α inhibitors as a potential cause of autoimmunity induction (e.g. systemic lupus erythematosus and vasculitis). In literature, both new IgA nephropathy and exacerbation of IgA nephropathy are reported. We are presenting a case with new-onset IgA nephropathy – biopsy-proven who was on adalimumab for psoriatic arthropathy.

Method

A 51-year-old lady had a medical background of psoriatic arthritis, hypertension, fibromyalgia, vertigo and asthma. She has been treated with sulphasalazine and adalimumab for her psoriatic arthritis under the rheumatology team. She had been on adalimumab (Humira) for 4 years before she was switched to a new brand called Imraldi in April 2019. She was referred to the renal team during hospital admission in July 2019 for a petechial rash with headache and throat pain. During this admission, she had acute kidney injury (AKI) as her serum creatinine increased from a baseline 73 to 131 $\mu\text{mol/l}$. Her CRP was raised and she was treated with antibiotics initially. She had Ultrasound of the urinary tract which was unremarkable and an immune screen came back negative.

Sulphasalazine and omeprazole were stopped during this admission as her AKI was thought to be due to interstitial nephritis related to antibiotics with a rash that was suspected to be related to Imraldi which was stopped as well.

Her serum creatinine improved slightly to 119 $\mu\text{mol/l}$ but she was found to have significant proteinuria of 277 mg/mmol. The rheumatology team decided to restart her on adalimumab (Imraldi) again.

After restarting adalimumab her kidney function deteriorated as serum creatinine worsened to 208 $\mu\text{mol/l}$ and reached a peak of 248 $\mu\text{mol/l}$. Her urine showed worsening urine PCR of 397 mg/mmol. Renal biopsy showed IgA nephropathy with acute tubular injury and element of tubulointerstitial nephritis on a background of moderate chronic tubular interstitial damage.

We decided to start her on prednisolone at this stage and we contacted her rheumatology team to stop treatment with adalimumab (Imraldi) as it was suspected to be the cause for her renal pathology.

Results

Since then her kidney function started to improve and her creatinine on her latest result shows an improving creatinine reaching 187 $\mu\text{mol/l}$. Her nephrotic range proteinuria showed a marked improvement to reach 39 mg/mmol.

Conclusion

This patient who developed AKI and IgA nephropathy showed marked improvement of her nephrotic range proteinuria and gradual improvement of her kidney function by stopping adalimumab and steroid treatment. IgA nephropathy is the commonest type of glomerulonephritis in the world and it is usually idiopathic. Deterioration of the kidney function after restarting adalimumab and the improvement of massive proteinuria to normal after drug withdrawal suggests a possible causal relationship between adalimumab and IgA nephropathy.