

Successful Management of Adult-Onset IgA Vasculitis-Associated Nephritis Following Influenza B Infection with Rituximab: A Case Report

Abdullah Hussien Alghamdi, MD*

ABSTRACT

This is a case report for 65-year-old male patient diagnosed with influenza B-associated crescentic IgA vasculitis nephritis with successful rituximab-based induction and maintenance. This case report supports the potential role of Rituximab (RTX) as an effective therapeutic option in severe or refractory immunoglobulin A vasculitis nephritis, possibly through B-cell depletion and reduction of IgA immune complex formation. Further larger scale studies are needed to clarify the optimal management strategies and establish the role of RTX in adult patients with immunoglobulin A vasculitis nephritis.

Keywords: *Immunoglobulin A; Vasculitis; Nephritis; Rituximab*

INTRODUCTION

Immunoglobulin A vasculitis is an immune complex-mediated, small-vessel vasculitis that involve the skin, gastrointestinal tract, joints, and kidneys¹. Immunoglobulin A vasculitis-associated nephritis is the term for this condition when it affects the kidneys². Usually, it is indistinguishable from IgA nephropathy³. Immunoglobulin A vasculitis-associated nephritis is one of the most common systemic vasculitis during childhood, with an incidence rate that ranges between 3 and 26.7 per 100,000 children⁴. At the same time, it is less common in adults, with an estimated incidence rate that ranges between 0.1 and 1.8 per 100,000 individuals⁴.

Immunoglobulin A vasculitis-associated nephritis is poorly understood in adults' population⁵. Due to its under-diagnosis most patient receive treatment at a later stage⁶. Consequently, immunoglobulin A vasculitis-associated nephritis in adults' population is associated with more severe disease, which increase the importance of giving more focus for his condition and its early and proper management⁵. In adults' patients, immunoglobulin A vasculitis-associated nephritis has a more severe course and poorer outcomes⁷. Therefore, one of the main priorities for the improvement of patients' outcomes is the prevention of the development of established kidney inflammation.

Current therapeutic approaches are for the management of immunoglobulin A vasculitis-associated nephritis are based on the evolved understanding of the pathophysiology of this condition, which spans kidney inflammation, mucosal immunity, and complement pathway activation^{1,8}. This case report aimed to present the successful management for adult-onset Immunoglobulin A vasculitis-associated nephritis in Saudi Arabia.

CASE PRESENTATION

This is a 65-year-old male patient, who has history of well controlled diabetes mellitus (DM), that was diagnosed 10 years prior to his presentation. His average A1C is <7%, with no history of diabetic retinopathy, nephropathy or neuropathy. The patient medication use history reported that he is too sensitive to steroid. The patient was on dapagliflozin 10 mg daily, and linagliptin 5 mg every other day. Besides, the patient reported a history of well controlled hypertension

and dyslipidemia that were diagnosed three years ago. The patient was on nebivolol 5 mg daily and rosuvastatin 10 mg daily. The patient surgical history reported testicular cancer that was surgical treated, with no evidence of recurrence or metastasis.

The patient presented to the emergency department with flu-like symptoms and diagnosed as influenza B pneumonia treated with Tamiflu for five days, with complete resolving of his symptoms. Then, 10 days later the patient visited the dermatology clinic as the patient started to have bilateral lower leg and feet skin vasculitic rash. Incidentally, that patient diagnosed with acute kidney injury, with serum creatinine at presentation was 117 µmol/L, then raised to more than 200 µmol/L within the first 7 days of his presentation.

Worth to mention, patient has recurrent history of nasal congestion for the last 15 years, with on and off mild epistaxis. No history of abdominal pain, nausea, vomiting, diarrhea or gastrointestinal bleeding. No other symptoms suggestive autoimmune diseases or vasculitis. No constitutional symptoms. On examination, the patient vital signs were within normal, and unremarkable cardiovascular, respiratory, abdominal, and neurological exam. The patient has vasculitic skin rash involving both lower legs and feet, Figure 1 and Figure 2.

The patient initial AKI/glomerulonephritis work up showed creatinine level that ranged between 117 µmol/L to 220 µmol/L during five consecutive days. The urine analysis was positive for protein and blood. The 24-hour urine protein was 2.2 gm/day, repeated 1.8 gm/day. C and P- ANCA was negative. ANA, C3, and C4 were normal. Anti-DS, anti-GBM, HIV, HCV, HBV, cryoglobulinemia were negative with no monoclonal gammopathy. US-KUB was normal apart from mild enlargement of the prostate. Ultrasound guided kidney biopsy done and showed the following:

1-The specimen was received in formalin labeled with patient name and file number only and consists of 1.1 x 0.1 cm single elongated tan core tissue fragment submitted in toto in one cassette.

2-The specimen is received in RPMI solution labeled with patient name and file number only and consists of a tan core tissue fragment (1.0 x 0.2 x 0.1 cm), submitted in toto in one cassette for DIF.

* Department of Internal Medicine, College of Medicine
Imam Mohammad Ibn Saud Islamic University (IMSIU)
Riyadh, Saudi Arabia.
Email: Dr.alhomrani@gmail.com



Figure 1. Right leg vasculitic skin rash



Figure 2. Left leg vasculitic skin rash

3-The specimen is received in glutaraldehyde labeled with patient name and file number only. It is sent for EM.

Light microscopy study: The tissue is examined with H/E, PAS, and Trichrome stains. The biopsy consists of renal cortex. Up to fourteen glomeruli are present for evaluation, one reveals global sclerosis and one shows segmental sclerosis. The glomeruli showed mild mesangial matrix expansion and hypercellularity with normal thickness of the capillary walls and patent capillary lumina. Four glomeruli showed cellular crescents. No definite endocapillary hypercellularity seen. There was mild interstitial fibrosis and tubular atrophy (10% of the cortical tissue), associated with mild interstitial mononuclear inflammatory cell infiltrate admixed with few plasma cells and neutrophils, and eosinophils. The tubules showed mild acute tubular injury with red blood cell casts. Arteries were unremarkable. Arterioles showed mild subendothelial hyalinosis.

Immunofluorescence study: Five glomeruli were present for evaluation. The glomeruli showed focal granular mesangial staining with antisera specific for IgG (trace), IgA (1-2+), and C3 (1-2+). No extraglomerular staining seen.

Electron microscopy study: The semi thin sections reveal three glomeruli. Ultrastructural examination demonstrated glomerular basement membranes with normal thickness and contour. There were few mesangial electron dense deposits. No subepithelial or subendothelial deposits were seen. The podocytes showed focal foot process effacement.

Case management: After diagnosing the patient with IgA vasculitis (kidney and skin), the patient was induced with pulse steroid 500 mg daily for three days then, the patient continued on high dose of steroid 60 mg daily for a month, then dose was decreased by 5 mg weekly, till he was off 12 weeks later. Also, the patient received Rituximab 750 mg (375 mg/m²) four doses, a week apart from one dose to another as induction therapy. Then, the patient maintained on Rituximab maintenance first dose 500 mg q 6 months for another three doses. CD19-20 three months after his first induction dose of maintenance was undetectable (0%). After the kidney function test stabilized, the patient was on prophylactic Septra (Sulfamethoxazole and Trimethoprim) 960 mg every other day. Also, the patient was kept on PPI, calcium and vitamin D as GI and osteoporosis prophylaxis.

The patient course was complicated with varicella zoster on his right thigh buttock and lower back. Treated with acyclovir, with complete remission apart from mild remnant post-herpetic neuroglia.

Also, the patient had mild medication-induced mood disorder possibly related to steroid, treated with Cipralix (escitalopram) which was stopped later after he stopped the steroid. After a month from initiating his immunosuppressive therapy, the patient showed excellent response with improvement on his creatinine, proteinuria and hematuria, with complete remission after six months, that was maintained till two years later.

DISCUSSION

This is a case report that presents a rare adult case of influenza B-associated crescentic immunoglobulin A vasculitis nephritis with successful rituximab-based induction and maintenance. This is a 65-year-old male patient. The patient presented to the emergency department with flu-like symptoms and diagnosed as influenza B pneumonia. After diagnosing the patient with IgA vasculitis, the patient was induced with pulse steroid 500 mg daily for three days then, the patient continued on high dose of steroid 60 mg daily for a month, then

dose was decreased by 5 mg weekly, till he was off 12 weeks later. Also, the patient received Rituximab 750 mg (375 mg/m²) four doses, a week apart from one dose to another as induction therapy. Then the patient maintained on Rituximab maintenance first dose 500 mg q 6 months for another three doses.

The ideal therapeutic approach for immunoglobulin A vasculitis nephritis in adults remained debatable⁹. The goals of the treatment are improvement of acute symptoms, mitigation of short-term morbidity and prevention of chronic renal failure. Though, many patients receive only supportive therapy⁹.

Rituximab (RTX) has been effectively used in systemic lupus erythematosus and vasculitis with pathogenic antibodies or immune complexes deposition or antineutrophil cytoplasmic antibodies (ANCA)-associated vasculitis¹⁰⁻¹². Rituximab is an anti-CD20 monoclonal antibody that was effectively used in vasculitis associated with pathogenic antibodies or immune complex deposition cryoglobulinemia, such as ANCA-associated or cryoglobulinemic vasculitis^{13,14}. The role of ANCA in immunoglobulin A vasculitis nephritis is debatable. Additionally, RTX might reduce the formation of IgA containing immune complexes and limited immunoglobulin A vasculitis nephritis disease activity through depleting B cells⁹. The mechanism of action of rituximab in autoimmune disease is thought to be due to the disruption in B cells' function or due to a decrease in plasma cell production, as CD20-positive B cells are intermediates in the process of their maturation¹⁵. Previous literature reported that RTX to an effective treatment in severe and refractory immunoglobulin A vasculitis nephritis and as a first-line therapy, and with high chance of long-lasting remission⁹.

CONCLUSION

This case report supports the potential role of RTX as an effective therapeutic option in severe or refractory immunoglobulin A vasculitis nephritis, possibly through B-cell depletion and reduction of IgA immune complex formation. Further larger scale studies are needed to clarify the optimal management strategies and establish the role of RTX in adult patients with immunoglobulin A vasculitis nephritis.

Authorship Contribution: All authors share equal effort contribution towards (1) substantial contributions to conception and design, acquisition, analysis and interpretation of data; (2) drafting the article and revising it critically for important intellectual content; and (3) final approval of the manuscript version to be published. Yes.

Potential Conflicts of Interest: None

Competing Interest: None

Acceptance Date: 07 April 2026

REFERENCE

1. Gan M Y, Chua F Z Y, Chang Z Y, et al. Navigating Adult-Onset IgA Vasculitis-Associated Nephritis. *Life (Basel)* 2024;14(8): 1-11.
2. Zhang X, Xie X, Shi S, et al. Plasma galactose-deficient immunoglobulin A1 and loss of kidney function in patients with immunoglobulin A vasculitis nephritis. *Nephrol Dial Transplant* 2020;35(12):2117-23.
3. Wyatt R J, Julian B A. IgA nephropathy. *N Engl J Med* 2013;368(25):2402-14.
4. Gardner-Medwin J M, Dolezalova P, Cummins C, et al. Incidence of Henoch-Schönlein purpura, Kawasaki disease, and

- rare vasculitides in children of different ethnic origins. *Lancet* 2002;360(9341):1197–202.
5. Kelly B G, Stratton D B, Mansour I, et al. Navigating the initial diagnosis and management of adult IgA vasculitis: A review. *JAAD Int* 2022;8(1):71–8.
6. Du L, Wang P, Liu C, et al. Multisystemic manifestations of IgA vasculitis. *Clin Rheumatol* 2021;40(1):43–52.
7. Lai L, Liu S, Azrad M, et al. IgA Vasculitis with Nephritis in Adults: Histological and Clinical Assessment. *J Clin Med* 2021;10(21): 1-10.
8. Williams C E C, Lamond M, Marro J, et al. A narrative review of potential drug treatments for nephritis in children with IgA vasculitis (HSP). *Clin Rheumatol* 2023;42(12):3189–200.
9. Fenoglio R, Naretto C, Basolo B, et al. Rituximab therapy for IgA-vasculitis with nephritis: a case series and review of the literature. *Immunol Res* 2017;65(1):186–92.
10. Isenberg D A. Rituximab-it was the best of times, it was the worst of times. *Autoimmun Rev* 2012;11(11):790–1.
11. Niaudet P, Habib R. Methylprednisolone pulse therapy in the treatment of severe forms of Schönlein-Henoch purpura nephritis. *Pediatr Nephrol* 1998;12(3):238–43.
12. Sinico R A, Di Toma L, Radice A. Renal involvement in anti-neutrophil cytoplasmic autoantibody associated vasculitis. *Autoimmun Rev* 2013;12(4):477–82.
13. Geetha D, Specks U, Stone J H, et al. Rituximab versus cyclophosphamide for ANCA-associated vasculitis with renal involvement. *J Am Soc Nephrol* 2015;26(4):976–85.
14. Audemard-Verger A, Pillebout E, Guillevin L, et al. IgA vasculitis (Henoch-Shönlein purpura) in adults: Diagnostic and therapeutic aspects. *Autoimmun Rev* 2015;14(7):579–85.
15. Randall K L. Rituximab in autoimmune diseases. *Aust Prescr* 2016;39(4):131–34.