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Simultaneous Presentation of Anti- GBM Antibody Disease and IgA Nephropathy in a Case of Rapidly Progressive Glomerulonephritis

Saxena S[1]*, Dawale P[2], Agrawal V[3] and Khande M[4]

[1]Senior Resident, Department of Nephrology, DKSPGI Raipur, India
[2]Associate Professor and Head of Department, Department of Nephrology, DKSPGI Raipur, India
[3]Associate Professor, Department of Nephrology, DKSPGI Raipur, India
[4]Associate Professor, Department of Nephrology, DKSPGI Raipur, India

Case Report
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***Corresponding author:** Soumya Saxena, Senior Resident, Department of Nephrology, DKSPGI Raipur, India

Abstract

Anti-GBM antibody disease is a rare entity characterised by immunoglobulin deposition along the glomerular basement membrane (visualised on IF as bright linear staining of the GBM), along with circulating IgG anti-GBM antibodies on serology and a typical diffuse crescentic nephritis on renal histopathology. While it has often been reported to be associated with other glomerulopathies such as ANCA-associated vasculitis, membranous nephropathy, and diabetic nephropathy, case reports on its association with IgA nephropathy are scarce. Herein, we describe a rare case presenting with rapidly progressive glomerulonephritis and a renal biopsy suggestive of features of both anti-GBM antibody disease and IgA nephropathy.

Keywords: Anti-GBM Disease; IgA Nephropathy; Diabetic Nephropathy; Rapidly Progressive Glomerulonephritis; Crescentic Glomerulonephritis

Introduction

The diagnosis of Anti-GBM antibody disease in a patient presenting with clinical features of Rapidly Progressive Glomerulonephritis is considered to be relatively straightforward in the presence of positive Anti-GBM antibody serology and biopsy findings of diffuse crescentic nephritis and linear IgG staining over the GBM. The presence of this disease warrants urgent therapy in the form of immunosuppression and plasmapheresis to remove the pathogenic antibody. However, the approach to the diagnosis and management of patients presenting with atypical forms of this disease is still unclear. In this report, we hope to shed light on the diagnostic and management considerations in a patient presenting with seronegative Anti-GBM antibody disease with concurrent IgA Nephropathy on renal biopsy.

Case Presentation

A 14-year-old male presented to our outpatient department (OPD) with generalised body swelling and cola colored urine since 2 months, without oliguria or hemoptysis. There was no history of sore throat, cough, loose motions, fever, rash, oral ulcers, arthralgia or photosensitivity; past history was non-contributory. On admission, he was hemodynamically stable with a blood pressure of 120/80 mmHg; general and systemic examination was unremarkable except for grade 2+ edema over both ankles.

Investigations

Laboratory investigations were significant for anemia (Hb – 9.1 g/dL), with raised white blood cell (WBC) counts

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(14,500 cells/mm3) and normal platelet count (2.8 lakh/mm3), deranged renal function (serum creatinine of 4.1 mg/dL, blood urea levels of 55 mg/dL), urinalysis showing proteinuria (3+ protein) and hematuria (580-600 RBC's/hpf) with no casts or crystals, and hypoalbuminemia (Serum albumin – 2.9 gm/dL). 24-hour urine protein revealed protein excretion of 4.1 gm in 24 hours (volume collected – 1500 mL). Serology showed negative ANA, ds-DNA, and anti-neutrophil cytoplasmic antibodies (both PR-3 and MPO), along with negative Anti-GBM antibodies (by ELISA). DCT was negative, while complement C3 levels were decreased with normal complement C4 levels. Abdominal ultrasound showed normal-sized kidneys, with no evidence of obstruction. With a syndromic diagnosis of RPGN, pulse steroids were commenced, and a kidney biopsy was performed.

Light microscopy showed 15 glomeruli, of which 2 were globally sclerosed, and 7 (46.6%) revealed cellular crescents (Figure 1), and two revealed segmental tuft necrosis. Tubular atrophy and interstitial fibrosis involved 10-15% of the sampled cortex, and tubules showed hyaline, RBC, and granular casts with evidence of severe acute tubular injury. Immunofluorescence (IF) showed strong (3+) linear staining for IgG (subclass IgG1 only) along the glomerular basement membranes, along with 3+ mesangial granular staining for IgA. C1q and IgM immunostaining was negative, and mild (1+) linear C3 staining could be seen over glomerular capillaries.

Figure/Video Captions

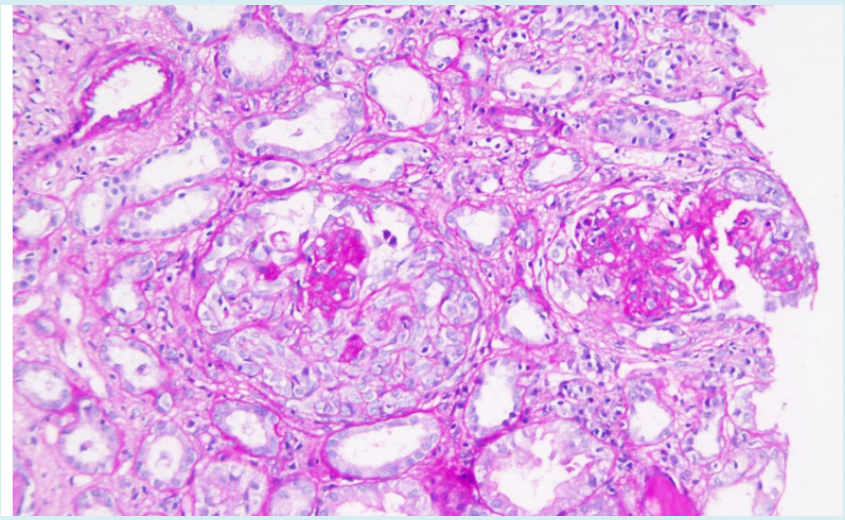


Figure 1: Periodic Acid Schiff Stain Shows Two Glomeruli with Cellular Crescents and Tuft Compression and Segmental Fibrinoid Necrosis (Light Microscopy, 40x Objective).

Differential Diagnosis

The Renal Biopsy report revealed 1) Similarly staged, cellular crescents in almost 50% of sampled glomeruli, and 2) Strong linear staining of IgG1 over the GBM, strongly suggestive of Anti-GBM antibody nephritis despite the absence of Anti-GBM antibodies on serology. The presence of codominant staining of IgA over the mesangium led us to consider the presence of IgA nephropathy along with Anti-GBM antibody disease.

Treatment

Overall, the biopsy report was suggestive of focal necrotising and crescentic glomerulonephritis, likely due to concurrent presentation of Anti-GBM disease and IgA nephropathy. After three doses of pulse methylprednisolone (given at a dose of 500 mg/day), plasmapheresis(PP) was initiated (5 sessions of alternate-day PP with an exchange volume of 40 mL/kg), and intravenous cyclophosphamide at a dose of 15 mg/kg was given. After 5 doses of IV cyclophosphamide, laboratory reports suggested an improvement in renal function (serum creatinine 1.0 mg/dL) with an improvement in proteinuria and hematuria (urinalysis – 2+ protein, 8-10 RBC's/hpf; Urine PCR – 2.1 (ref range < 0.2 mg/mg). Medications at discharge include oral prednisolone 25 mg once a day (0.5 mg/kg/day), telmisartan 40 mg once a day, and amlodipine 5 mg once a day.

Outcome and Follow-Up

The patient was clinically stable during his last OPD follow up (5 months after initial hospitalization), with a blood pressure on 120/82 mmHg on two antihypertensive

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medicines (Telmisartan 80 mg and amlodipine 5 mg once a day), and laboratory investigations showing stable renal function (Serum Creatinine of 0.94 mg/dl), normal serum albumin levels (3.73 gm/dl), and reduction in proteinuria to near normal levels (UPCR – 0.2 mg/mg)

Discussion

This case report describes a patient presenting with the typical features of acute glomerulonephritis, with renal biopsy showing the presence of similarly aged crescents with diffuse linear staining for IgG along the glomerular basement membrane, without any detectable anti-GBM antibody titre on serology (by ELISA). Presence of codominant mesangial staining by IgA on immunofluorescence further broadens the scope of the differential diagnosis.

Anti-GBM antibody disease is considered the prototypical crescentic nephritis. It is often associated with dialysis dependency on presentation (in around 55% of cases) [1], linear IgG deposition over the GBM, diffuse (>50%) crescents on biopsy, and a fulminant clinical course with a high incidence of ESRD despite therapy. The presence of strong (>3+) linear staining of the GBM with immunoglobulin is highly suggestive of this pathology, especially if electron microscopy does not show any corresponding electron-dense deposits [2]. Differential diagnosis for linear Immunoglobulin staining over GBM includes Diabetic nephropathy, MIDD (Monoclonal Immunoglobulin Deposition Disease), Fibrillary Glomerulonephritis, and Idiopathic Nodular Glomerulosclerosis (also known as Smoking-Associated Glomerulosclerosis). However, the absence of kappa/lambda light chain restriction, normal sugars at presentation, and absence of DNAJB55 positivity on immunohistochemistry suggests against any other alternative pathology/diagnosis in this case. The presence of strong (3+) granular mesangial IgA immunostaining also suggests the coexistence of IgA nephropathy along with Anti-GBM antibody disease. A literature search revealed scattered case reports and case series with similar clinicopathological features [3-5], which will be discussed below.

Atypical Anti-GBM antibody disease is an entity characterised by immunoglobulin deposition along the glomerular basement membrane without circulating IgG anti-GBM antibodies on serology, in the absence of other causes [6]. Hypothesised causes of negative serology include differences in target or target antibody leading to non-detection of the pathogenic Anti-GBM antibody by the ELISA assay, or the presence of a high-affinity Anti-GBM antibody that binds completely to the GBM leading to very low titres in the circulation [6]. Ideally, serological testing should be done using alternative laboratory tests to detect such outlier cases and confirm the diagnosis [7]. Unlike classical Anti-GBM antibody disease, which is characterised by a short clinical history and an acute presentation, Atypical Anti-GBM disease may have a milder presentation and a more indolent clinical course [6]. Histopathologically, it is more likely to be associated with focal (rather than diffuse) crescents, and with the presence of mesangioproliferative, endocapillary proliferative, or membranoproliferative patterns on light microscopy [6]. Atypical Anti-GBM disease is also more likely to be associated with other concurrent glomerular disease, such as IgA nephropathy, FSGS, Membranous Nephropathy, TMA, and ANCA-associated vasculitis [5].

In our case, the patient presented with an insidious onset and gradually progressive swelling over the face and feet, in contrast to the fulminant, rapidly progressive course associated with Classical Anti-GBM disease. We may consider this to be a case of atypical Anti-GBM nephritis; however, additional investigations for anti-GBM antibodies using alternative methods (such as Indirect Immunofluorescence) should be performed to confirm the diagnosis. Additional steps to be considered (which could not be done in our case due to logistical constraints) include assessing the biopsy sample with electron microscopy to investigate for the presence of electron-dense deposits and/or fibrils over GBM, and further serological testing for Anti-GBM antibody titres using immunofluorescence assay, which uses kidney tissue sections and may aid in the detection of non-classical Anti-GBM antibodies.

Learning Points/Take Home Messages

1. Anti-GBM antibody disease may be present even in the absence of circulating Anti-GBM antibodies, and in conjunction with other glomerulopathies
2. It is important to identify such cases and tailor immunosuppression appropriately – consideration for plasmapheresis/cyclophosphamide protocols for organ/life threatening disease
3. In cases of suspected Anti-GBM antibody nephritis with negative serology, it is worthwhile to perform alternative laboratory tests such as Indirect immunofluorescence or biosensor assay to prevent a missed diagnosis

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