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IgA Nephropathy (Burger's Disease): Case Report

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ABSTRACT

IgA nephropathy is a condition characterized by deposition of IgA immunoglobulins in glomeruli. This condition is fairly common in Western countries. The scope of the disease is wide and case-by-case. Cases of IgA nephropathy are rare. Our case report is of a young man who developed rapid-onset IgA nephropathy leading to end-stage renal disease (ESRD). This case report describes a 26 years age young man who presented and eventually presented with microscopic hematuria and severe proteinuria. Hemodialysis for his burned out IgA nephropathy.

KEYWORDS: *IgA nephropathy, Burger's disease, Immunoglobulins, Hematuria and Proteinuria*

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INTRODUCTION

IgA nephropathy, also known as Burger's disease, is a kidney disease caused by the accumulation of an antibody called immunoglobulin A (IgA) in the kidneys. This causes localized inflammation, which over time can affect the kidneys' ability to filter waste products from the blood. IgA nephropathy usually progresses slowly over years, but the course of the disease varies from person to person. Some people lose blood in their urine without developing problems, some eventually achieve complete remission, and some develop end-stage renal disease. There is no cure for IgA nephropathy, but certain drugs can slow its progression. Controlling blood pressure and lowering cholesterol levels also slows disease progression.

Case description:

A 26-year-old man with no history of illness who had bilateral extremity swelling and scrotal swelling for the past 2 weeks. He was more tired than usual and vomited several times during the same period. He denied fever, chills, recent sore throat, rash, change in urinary disposition, or his recent travel history. He took no home remedies. He was a lifelong nonsmoker and had no history of multiple drug abuse. Family history was important for maternal hypertension.

On admission, his physical examination revealed a blood pressure of 210/100 mmHg, other vital signs were within normal limits. He had swollen testicles without tenderness on palpation and his 3 or more bilateral leg edemas extended to the thighs. There was not throat discharge, renal murmur, or unusual skin rash.

His most important blood investigation results were in table 1-3.

Table 1: Serum biochemistry

Test	Result	Normal value
Sodium	139 mmol/L	136-145 mmol/L
Potassium	4.2 mmol/L	3.4-4.7 mmol/L
Chloride	106 mmol/L	98-107 mmol/L
Bicarbonate	25 mmol/L	21-31 mmol/L
BUN	60 mg/dl	7-25 mg/dl
Creatinine	9.85 mg/dl	0.7-1.3 mg/dl
Albumin	2.6 g/dl	3.5-5.0 g/dl
Total protein	4.9 g/dl	6.4-8.3 g/dl

Table 2: Hematology

Test	Result	Normal value
Hemoglobin (Hb)	8.2 g/d	12-16 g/dl
Platelets	71,000 thou/L	130-400 thou/L
White blood cells	8600 cells/uL	4.8-10.8 cells/uL
Mean corpuscular volume	97.5 fL	78-102 fL

Table 3: Lipid profile

Test	Result	Normal value
Cholesterol	209 mg/dl	<200 mg/dl
Low density lipid (LDL)	111 mg/dl	<129 mg/dl
Triglycerides (TG)	113 mg/dl	<150 mg/dl
High density lipid (HDL)	58 mg/dl	>40 mg/dl

His dipstick urine test was significant with large blood volume, >1000 mg/dl protein, and 10-20 red blood cells. Cells (RBC) under the microscope. His urinalysis showed that he had a protein to creatinine ratio of 4.26g/dl. Considering his proteinuria in the Nephrotic range, an extensive work-up was performed to clarify this etiology of Nephrotic syndrome.

His clotting profile was normal, but his cholesterol was increased (Table 3) and he was Hypoalbuminemia (Table 1). His C3 was as low as his 72 mg/dl (normal range 98-140 mg/dl) and normal his C4. Perform serum and urine protein electrophoresis Shows hypogamma globulinemia but no abnormal protein spikes. Anti-nuclear Antibodies and anti-glomerular basement membrane antibodies (anti-GBM) were negative; Antistreptolysin O (ASO) titers were within normal limits. HIV, rapid plasma regeneration and a hepatitis panel was negative. Cytoplasmic-antineutrophil cytoplasmic antibody (C-ANCA) and P-ANCA antibody was also negative. Glycated hemoglobin (HbA1C) was 4.2%. Computed tomography (CT) of the abdomen/pelvis and ultrasonography of the kidneys showed increased renal echogenicity (figure1 & 2). Serum iron panel showed pictures of anemia Chronic disease with ferritin 294 mg/dl and serum iron saturation <10%.

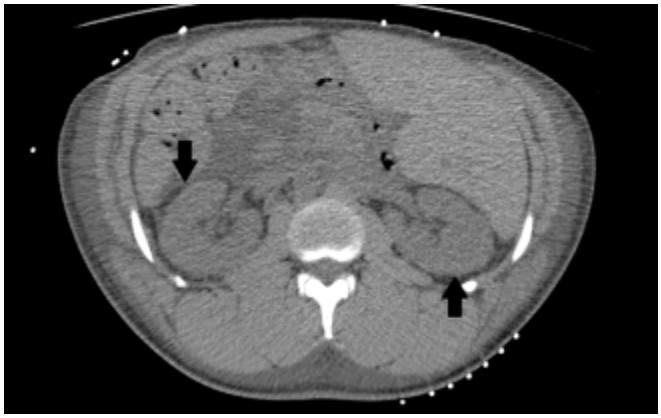


Figure 1: CT abdomen and pelvis

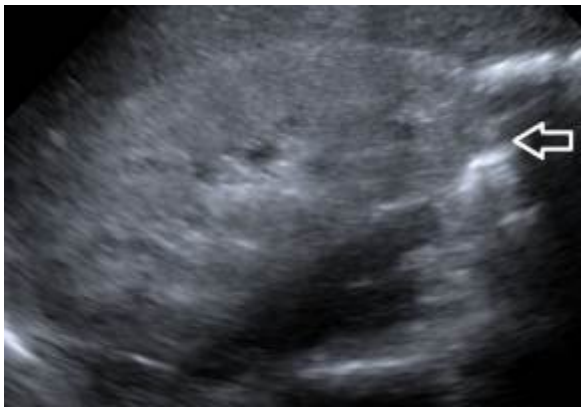


Figure 2: USG Abdomen

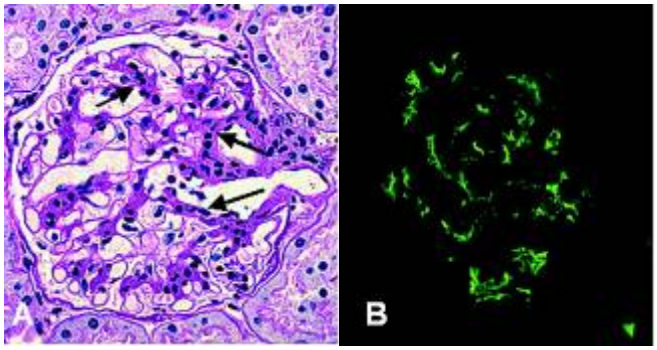
A cause of secondary hypertension was identified as presenting with hypertensive urgency. The patient's plasma metanephrines were found to be normal, Normetanephrine concentration and plasma renin-to-aldosterone ratio and plasma aldosterone concentration was also inconspicuous. During his hospitalization, he was started on a nicardipine drip for aggressive blood he controls his blood pressure and then switches to oral antihypertensive, including carvedilol, hydralazine, isosorbide dinitrate, diltiazem. His urine volume throughout the hospital of course he stayed below his 500 ml/day. So he was oliguric. He was also started on furosemide.

It did not improve his urine output. Only his leg and scrotal edema reduced. He should have a kidney biopsy to determine the exact etiology of Nephrotic drugs syndrome. His anemia was treated with intravenous iron and erythropoietin stimulation agent. As blood pressure decreased, platelet counts also improved significantly hypothesized to be better controlled and reduced with thrombotic microangiopathy with malignant hypertension. Upon biopsy, light microscopy (LM) revealed that the specimen had 29 global sclerotic glomeruli, Up to 3 additional glomeruli per slice level had segmental sclerosis with hyalinosis, two partial cell crescents. For immunofluorescence (IF), hematoxylin and eosin stains were used which revealed that five globally sclerotic glomeruli had granular segmental mesangial staining with antisera specific for IgA, C3, and Kappa and Lambda light chains but negative for C1q, IgM, and IgG antibodies as shown in Figures3. Based on the biopsy results, he was diagnosed to have advanced diffuse sclerosing and proliferative IgA nephropathy.

He was discharged on a beta-blocker, non dihydropyridine calcium-channel blocker, angiotensin-receptor-blocker, and hydralazine for aggressive blood pressure control and high intensity statin therapy for hyperlipidemia. He was set up for hemodialysis three days a week with placement of arteriovenous fistula in the near future. He was also

arranged to meet with transplant nephrologist for evaluation of renal transplant in the future.

Figure 3: Biopsy and Light microscopic examination



A- Light microscopy shows glomerular sclerosis, B- Positive immunofluorescence for lambda chains

Discussion:

Cases of IgA nephropathy have not been commonly reported in the literature, and less than 10% of these patients have Nephrotic syndrome that progresses to hemodialysis. Although the pathophysiology is not fully understood, IgA deposition is known to occur in the glomerular mesangial lambda light chain predominance state. Glomerular deposition of C3 is also common, whereas C1q is usually absent. Compared with healthy subjects, sick patients have abnormally glycosylated IgA1 molecules in their serum. Thrombotic microangiopathy can also be found in cases of IgA nephropathy due to malignant hypertension and indicates a poor renal outcome as seen in our case.

For diagnostic purposes, renal biopsy is usually performed to stage the disease according to Oxford Classification, which also helps in determining the disease prognosis at the time of biopsy. Clinical and laboratory findings at the time of diagnosis can also help stratify the severity of disease and these markers include increased serum creatinine (Cr), reduced glomerular filtration rate (GFR), hypertension (blood pressure > 140/90 mmHg) and persistent proteinuria. The presence of the aforementioned features indicates a worse prognosis. On biopsy, if pathological findings of crescents, glomerular and/or segmental sclerosis, tubular atrophy, interstitial fibrosis, and interstitial cellular infiltrates are discovered, there is worsening of disease state and elevated risk of developing ESRD. For cases of IgA nephropathy, there has been reported faster decline in GFR compared to IgA nephropathy alone.

Our patient was not given a trial of these therapies as he already had developed ESRD by the time of diagnosis. Non-immunosuppressive treatment options include utilization of angiotensin converting enzyme inhibitors (ACE-I) or angiotensin receptor blockers (ARB) for better blood pressure control and proteinuria. A clinical trial suggested the use of ACE-I/ARB is superior to any other anti-hypertensive agent for such patients. The monitoring of disease activity can be performed by regular assessment of GFR, serum Cr, proteinuria, and urinary sediment.

Conclusion:

Our case highlights and suggests coexistence of IgA nephropathy leads to poor prognosis. The patient progressive course leading to ESRD and early diagnosis and treatment are critical.

Conflict of Interest

None

Funding

None

Consent for publication

Informed consent was obtained from the parents of the patients to publish this case in medical journal.

References:

[1] IgA nephropathy. National Kidney Foundation. https://www.kidney.org/atoz/content/iganeph. Accessed March 27, 2019.

[2] Gilbert SJ, et al, eds. Immunoglobulin A nephropathy and related disorders. In: National Kidney Foundation's Primer on Kidney Diseases. 7th ed. Philadelphia, PA.: Elsevier/National Kidney Foundation; 2018. https://www.clinicalkey.com. Accessed March 29, 2019.

[3] Rodrigues JC, et al. IgA nephropathy. Clinical Journal of the American Society of Nephrology. 2017;12:677.

[4] IgA nephropathy. National Institute of Diabetes and Digestive and Kidney Diseases. https://www.niddk.nih.gov/healthinformation/kidney-disease/iga-nephropathy. Accessed March 27, 2019.

[5] Glomerular diseases. National Institute of Diabetes and Digestive and Kidney Diseases. https://www.niddk.nih.gov/healthinformation/kidney-disease/glomerular-diseases. Accessed March 29, 2019.

[6] Warner KJ. All scripts EPSi. Mayo Clinic. May 11, 2021.

[7] Ask Mayo Expert. Glomerular disease. Rochester, Minn.: Mayo Foundation for Medical Education and Research; 2018.

[8] IgA nephropathy. Genetic and Rare Diseases Information Center. https://rarediseases.info.nih.gov/diseases/863/iga-nephropathy. Accessed March 27, 2019.