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Case Report

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IGA NEPHROPATHY WITH ULCERATIVE COLITIS WITH VENOUS THROMBOEMBOLISM-UNDERFLEDGED HSP OR AN OVERFLEDGED IGA NEPHROPATHY

[1]Dr. Karmveer Singh Godara, [2]Dr. Rajendra Singh Tanwar, [3]*Dr. Nishad Raveendran, [4]Dr. Pankaj Beniwal, [5]Dr. Dhananjai Agarwal and [6]Dr. Vinay Malhotra

[1,2,3]Senior Resident, Department of Nephrology, SMS Medical College, Jaipur, Rajasthan, India.
[4]Associate Professor, Department of Nephrology, SMS Medical College, Jaipur, Rajasthan.
[5]Professor, Department of Nephrology, SMS Medical College, Jaipur, Rajasthan.
[6]Professor and Head, Department of Nephrology, SMS Medical College, Jaipur, Rajasthan.

***Corresponding Author: Dr. Nishad Raveendran**

Senior Resident, Department of Nephrology, SMS Medical College, Jaipur, Rajasthan, India.

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ABSTRACT

IgA Nephropathy is the most common form of glomerulonephritis worldwide and is often idiopathic but its association with Ulcerative colitis is reported in various case reports. Henoch-Schonlein purpura (HSP) is a systemic disorder with renal, gastrointestinal, articular and cutaneous manifestations in which renal involvement is in the form of IgA Nephropathy. Here we report a case of 45 year old patient with IgA nephropathy without nephrotic syndrome, with Ulcerative Colitis and Venous Thromboembolism in the absence of any other hypercoagulable state which may be considered as a form of less severe HSP or an extended form of IgA Nephropathy.

KEYWORD: Henoch-Schonlein purpura, hypercoagulable, nephrotic.

INTRODUCTION

Ig A nephropathy (IgA N) is characterized by the deposition of Ig A in the mesangium. The renal histology of Henoch-Schonlein purpura (HSP) is indistinguishable from IgA N.HS nephritis is differentiated from IgA N by the presence of extra renal manifestations. IgA N is known to exist in association with numerous diseases and association with Ulcerative colitis (UC) is also reported in a number of patients. Venous thromboembolism(VTE) along with infections are considered as two most common cause of mortality in patients with nephrotic syndrome.[3,4] The most important risk factors for DVT is the severity of proteinuria and serum albumin levels.[6] The incidence of nephrotic syndrome in IgA N is less than 5%[2] and so the occurrence of VTE is also rare in patients with IgA N.In this communication we report a case of IgAN with subnephrotic proteinuria , associated with UC and Deep Venous Thrombosis(DVT).

CASE HISTORY

A 45 year old male was presented to us with history of abdominal pain and dysentery of 1weeks duration associated with low grade fever. He was complaining of progressively increasing facial puffiness for the past 2 months. This was followed by the development of bilateral pedal edema.He was evaluated else where for these complaints and was found to be hypertensive. Investigations at that time showed 2+ proteins and 10-15 RBC/HPF in urine and his serum creatinine was found to be 2.1gm/dl. The patient was adviced for a kidney biopsy, but as he refused he was started on antihypertensives and diuretics. But there was no symptomatic improvement and he noticed disproportionate swelling of left lower limb for about 2 weeks prior to presentation to us.

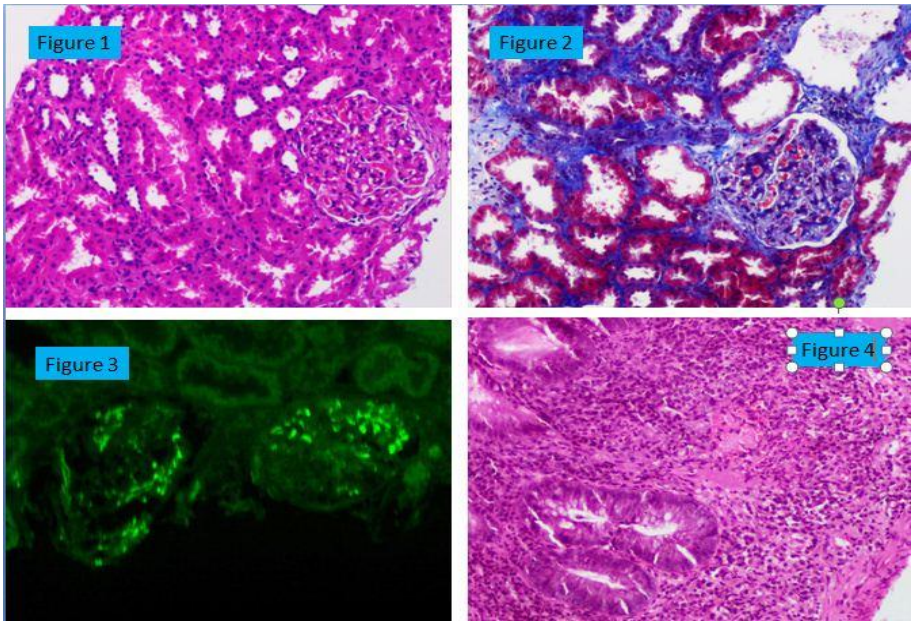
There is no past history of Tuberculosis, Rheumatoid arthritis, Diabetes Mellitus, Hypertension, Coronary Artery Disease, any substance abuse or blood transfusions .General Physical examination was unremarkable except for the presence of edema and ascitis. Vitals – he was afebrile with Pulse rate of 80beats/minute and B.P of 140/90mmHg at the time of admission with anthypertensives.

Investigations: Hb:12.2gm/dl,TLC:6570cells/cmm, DLC:P66L18,PLC:2.46lakhs, B.Urea:125 mg/dl, S. Creatinine: 3.1mg,S.Na:135meq/l, S.K:4.3meq/l, S.bilirubin 1.0mg, SGOT:32, SGPT :22, S.Albumin:3.3gm, S.Globulin:3.0gm/dl, S.Cholesterol:146 mg/dl,S.Triglycerides:134mg/dl, RA factor: negative, Urine: Albumin- ++, RBC: 10-15RBC/HPF, 24hour urine protein:1.2gms, ANA: negative cANCA :negative, p ANCA :negative, HIV: negative, HBs Ag: negative, Anti HCV: Negative, Lupus anticoagulant: negative, Anticardiolipin antibody: negative, Protein C and S levels were normal. Chest X

ray: normal, USG Abdomen: right kidney: 9.3*3.6 cms, left kudney: 9.4*3.7 cms, Blood and Urine cultures were sterile, Sputum AFB: Negative, ECG and 2D Echocardiography was normal, Left lower limb Doppler showed.

A Kidney biopsy was performed. Under light microscopy, H&E Stain (Fig 1) and Silver methenamine stain (Fig 2) shows segmental mesangial proliferation. IF (Fig 3) shows patchy mesangial deposition of Ig A. Sigmoidoscopy and biopsy: shows mucodepletion, crypt distortion, cryptitis and crypt abscess (Fig 4) suggestive of ulcerative colitis.

With the diagnosis of IgA Nephropathy and ulcerative colitis, the patient was started on oral steroids and mesalasin. He was started on Inj. Low molecular weight Heparin overlapped with warfarin for DVT and he is under follow up now.



DISCUSSION

Immunoglobulin A nephropathy (IgAN) is the most common form of glomerulonephritis in whites and in Asian population and is defined by the predominant deposition of IgA in the glomerular mesangium.

Secondary forms of IgAN have been described, most commonly in the setting of liver disease.

The association between IgAN and UC is well established and the first report on the association between IBD and Ig A N was by Hubert et al in 1984.[1] Considering the relatively high frequency of subclinical IgAN in general population this association was once thought to be a chance finding. But further studies showed a significantly higher incidence of IgAN in patients with IBD than in those without IBD. Majority of patients in these case reports had IgAN during onset or exacerbation of IBD, as well as clinical remission of kidney disease in conjunction with successful treatment of bowel inflammation suggesting a possible pathophysiological relationship between the two conditions.[7] At present there is no evidence of any mechanism explaining the pathological association of IgAN and UC. But it may be due to a complex interplay of mucosal inflammation, loss of antigenic exclusion, and tolerance, chronic immune stimulation, and dysregulated IgA production and transport. Additional studies are needed to determine the causal relationship between the two diseases.[8]

The cumulative incidence of VTE in nephrotic syndrome ranges between 3 to 60% in various studies.[5,9] Unlike the studies in earlier days which showed renal vein thrombosis as the most common type of VTE in nephrotic patients recent studies showed that DVT of limbs are the most common type of VTE. The histological diagnosis of glomerular disease is also found to be an independent predictor of the risk of VTE. Various studies have showed that the maximum incidence of VTE is seen with Membranous nephropathy followed by FSGS and the least risk is seen with IgAN.[9] Despite a relatively higher incidence of IgAN in general population, the incidence of VTE in these patients is approximately 5%.[2] Various disease specific factors related to the histological diagnosis may have a role in the pathogenesis of nephrotic syndrome and that could be the reason behind the varying incidence of DVT.[9]

CONCLUSION

IgA Nephropathy and HSP represents two ends of a spectrum of diseases with varying degree of systemic involvement. Our patient doesn't fit in to the diagnostic criteria of HSP,but he has systemic involvement more than what is usually seen in IgA Nephropathy. In conclusion with extra renal manifestations in the form of

VTE and Ulcerative Colitis we may consider that this patient is having a form of underfledged HSP or an overfledged IgA Nephropathy.

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