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A CASE OF PRIMARY MEMBRANEOUS NEPHROPATHY

General Medicine

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ABSTRACT

Membranous nephropathy is among the most common cause of primary nephrotic syndrome in adults.It accounts for 30% cases in adults.The peak incidence is inbetween 30 and 50 years of age.Male:Female ratio is 2:1.It is extremely rare in childhood. Primary cause is due to Auto antibodies against Phospholipase A2receptors;Secondary cause includes Infections-Hepatitis B and Hepatitis C,Drugs includes Gold ,Penicillamine, NSAIDS, Captopril ,Carcinoma of colon, lung and melanoma, Autoimmune disorder like SLE ,Rheumatoid Athritis.Eighty percent of patients present with nephrotic syndrome and non selective proteinuria.One third of patient have relapsing nephritic syndrome and another one third develops renal failure and death.Stages of 1 to 5 have been described with progression on membraneous lesion,but there is evidence that degree of tubular and interstitial fibrosis have more predictive value.

KEYWORDS

Nephrotic syndrome,Membraneous Glomerulonephritis,Membraneous Nephropathy

INTRODUCTION

Membranous nephropathy is among the most common cause of primary nephrotic syndrome in adults.It accounts for 30% cases in adults.The peak incidence is inbetween 30 and 50 years of age.Male:Female ratio is 2:1.It is extremely rare in childhood. Primary cause is due to Auto antibodies against Phospholipase A2receptors;Secondary cause includes Infections-Hepatitis B and Hepatitis C,Drugs includes Gold ,Penicillamine, NSAIDS, Captopril ,Carcinoma of colon, lung and melanoma, Autoimmune disorder like SLE ,Rheumatoid Athritis.Eighty percent of patients present with nephrotic syndrome and non selective proteinuria.One third of patient have relapsing nephritic syndrome and another one third develops renal failure and death.Stages of 1 to 5 have been described with progression on membraneous lesion,but there is evidence that degree of tubular and interstitial fibrosis have more predictive value.

Case Report

A 24 year old male was admitted to the hospital in view of facial puffiness more marked on early morning, swelling of lower limbs and froathy urine for 1 week. Patient was apparently normal prior to this. He is not a known case Type 1/Type 2 Diabetes Mellitus, Systemic Hypertension,

Bronchial Asthma, Seizure disorder , Thyroid disorders ,Pulmonary tuberculosis, hepatitis B,Rheumatoid disease and SLE. No H/O similar illness in family and no history of autoimmune disorders in family. On reviewing his previous past medical records it is noted that he had not been on any continuous medications.Patient is not a smoker or alcoholic or drug abuse , mixed diet ,normal sleep pattern and has normal bowel and bladder habits.

Examination

On Examination,Patient was Conscious ,oriented and Afebrile. On Physical examination patient had B/L pitting pedal oedema with no pallor / icterus / clubbing / cyanosis / lymphadenopathy .Patient was Normotensive with BP 120/80 mmHg and a pulse rate of 87/min. Systemic examination was unremarkable.

Investigations

All routine investigations were done.

Table-1

Investigations	Value	Reference Value
Hb	14.3	13.0-17.0 g/dl
RBS	99	80-120mg/dl
HbA1C	5.18	4.0-5.6%
ESR	77	0 -15mm/hr

Table-2

Investigations	Value 6/6/19	Value 11/6/19	Value 18/6/19	Reference Value
Urea	44	39	36	12.84-42.8mg/dl
Creatinine	1.1	1.0	1.0	0.6-1.1mg/dl
Na	141	136.7	143	139-146mEq/L
K	4.06	3.54	3.72	3.5-5.1mEq/L
Cl	111.1	105.9	106.5	98-107mEq/L
Albumin	1.4	1.6	1.8	3.5-5.5g/dl
Uric acid	8.2	7.8	7.0	4.0-7.0mg/dl

Table-3

Investigati-ons	Value	Reference Value
Magnesium	8.2	1.6-2.5mg/dl
Phosphorous	4.3	2.5-4.6mg/dl
T.Protein	3.0	6.0-8.0g/dl

PT,APTT,INR was normal.

***Urine Lab Reports*:** Colourless,Ph-6.5 [4.6-8.0],Sugars-Nil,Urine protein +4,Appearance-Turbid[Clear],Urine Rbc-3-4[0-2/hpf],Urine pus-3-4[0-5/hpf],Urine epithelium 1-2[Occasional/Absent],Urine cast-Nil[Absent],Total urine volume-3000ml/day[800-2000ml/day],Urine protein-4440mg/dl[150 mg/dl]

***USG KUB*:** Right Kidney-10.6x5.1cm,Left Kidney-10.5x5.5cm,Right renal Cyst measuring 1.3x0.9 cm

***Chest Xray*:** Normal

***Renal Biopsy*:** Light microscopy- Sections and special stains (PAS,silver and trichrome) include renal cortical tissue. Nine glomeruli are seen in this biopsy. None are globally sclerotic .They are of normal cellularity Capillary loops are open. Glomerular basement membranes show no spikes or double contours. No segmental sclerosis, endocapillary proliferation or crescent formation seen. No significant inflammatory infiltrate or fibrosis are seen in the interstitium .No vascular pathology observed.

Immunoflurescence- The section are stained for IgGIgM, IgA,C3, C1q, Kappa& Lambda light chains. IgG (+3) and C3 (+1) show granular positivity over the capillary loops. No light chain restriction seen. Rest of the antisera are negative.

***Final report*:** This lesion most likely represents stage I Membranous nephropathy. *Anti DS-DNA,C3 and C4,HIV 1,HIV 2,HBsAG ,ANTI-HCV,ANA,RF* was negative.

***Colonoscopy*:** Normal study

***Anti-PLA2R Ab* –Qualitative# (IFA):** Positive

Management

The patient came to hospital with above mentioned complains.All routine investigation was done. BP Charting was Done and showed maximum reading of 150/90 mmHg .Urine Input and Output charting was done and was monitered carefully,No significant inference were made.On view of nephrotic range of proteinuria,patient was Planned for biopsy advised by nephrologist.Patient was advised to avoid high protein diet,patient was started conservative management with anti-coagulants,ACE inhibitors,Lipid lowering agents and inj.human Albumin.After Obtaining biopsy report,patient was started work up for Membraneous nephropathy.Patient was diagnosed with Primary Membraneous nephropathy on confirmation with Anti-PLA2R Ab test.On obtaining nephrologist review,patient was advised to start PONTICELLI Regimen with steroids on 1^{st},3^{rd},5^{th} month and Cyclophophamide on 2^{nd},4^{th},6^{th} month.

On our Last Followup,

Patient had normal Renal function with resolved proteinuria and had no complaints.

DISCUSSION

Membranous nephropathy is among the most common cause of primary nephrotic syndrome in adults.It accounts for 30% cases in adults.The peak incidence is inbetween 30 and 50 years of age.Male:Female ratio is 2:1[1].It is extremely rare in childhood. Primary cause is due to Auto antibodies against Phospholipase A2receptors;Secondary cause includes Infections-Hepatitis B and Hepatitis C,Drugs includes Gold ,Penicillamine, NSAIDS, Captopril ,Carcinoma of colon, lung and melanoma, Autoimmune disorder like SLE ,Rheumatoid Athritis.Eighty percent of patients present with nephrotic syndrome and non selective proteinuria[2].One third of patient have relapsing nephritic syndrome and another one third develops renal failure and death.Stages of 1 to 5 have been described with progression on membraneous lesion,but there is evidence that degree of tubular and interstitial fibrosis have more.Presence of Anti PLA2R is considered as bad prognosis[4].The initial study of the disease was done in Hagmann rat model.In the clinical features part,it follows 70/30 rule in which 70% are symptomatic and 30% are asymptomatic.It is necessary to differentiate the Membraneous nephropathy from minimal change disease in which microhematuria,s.htn and Renal failure are seen in membraneous and to differentiate it from focal segmental glomerulonephritis Thrombosis is the key feature seen in membraneous nephropathy.For Asymtomatic cases the management mainly focussed on conservative management with ACE inhibitors,Lipid lowering drugs,Anti coagulants and avoiding high protein diet[5].For symptomatic cases it is best to start PONTICELLI regimen[6].Some patients may show resistant to this regimen ,hence they can switch over to Rituximab or Calcinurin classes of drugs[5].The prognosis is poor in patients with hypoalbuminia and with patients with primary membraneous nephropathy[6,7].

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