Membranous Nephropathy With Transverse Myelitis

Mudasir Mushtaq[1], Maqbool Wani[2], Mushtaq Ahmed Wani[3], Rouf Asimi[4], Sawan Verma[5], Irfan Shah[6]

[1] Senior Resident, [2] Associate prof, [3] Prof. & Head, Additional Prof, [4] Additional Prof, [5]Senior Resident, [6] Senior Resident, neurology, Sher-i-Kashmir Institute of Medical Sciences (SKIMS) Soura Srinagar J&k India

ABSTRACT

MGN is the most common form of glomerulonephritis causing nephritic syndrome in adults. Cases have been reported depicting association between MGN and neurological diseases like guillain-barre syndrome, multiple sclerosis and chronic inflammatory demyelinating polyneuropathy . However only one case has been reported with MGN and inflammatory myelopathy. Our patient was 36 yrs old female admitted with paraparesis and paresthesias in bilateral hands with bowel and bladder involvement and she had a biopsy documented episode of membranous nephropathy.

Key words: Membranous Nephropathy, Transverse Myelitis

Corresponding author address: Dr Mudasir Mushtaq, Boys Hostel SKIMS Soura Srinagar Jammu And Kashmir India -190011 **Phone:** 09797064543 **M:** 9086474543 **E-Mail :** drshah.mudasir@gmail.com

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INTRODUCTION

Membranous glomerulonephritis (MGN) is characterised by irregular proteinaceous deposits containing IgG, along the outer aspects of the glomerular capillary wall. The capillary wall thickens with increase in the basement membrane thickness. MGN is the most common form of glomerulonephritis causing nephrotic syndrome in adults. Cases have been reported depicting association between MGN and neurological diseases like guillain-barre syndrome, multiple sclerosis, and chronic inflammatory demyelinating polyneuropathy. However only one case has been reported with MGN and inflammatory myelopathy. MGN results from immune-complex deposits in basement membrane and inflammatory myelopathy results from disease of Schwann cells and myelin. The myelin sheath is primarily destroyed, usually leaving axons intact and interrupting nerve conduction. Here we report a case of membranous nephropathy and transverse myelitis; and both conditions responded to steroid therapy.

CASE REPORT

A 36 Years old housewife with two living issues was admitted with 15 days history of weakness of both lower limbs. This was associated with paresthesias in her hands and feet

that gradually ascended to the trunk. There was history of bowel and bladder incontinence. There was past history of biopsy documented membranous glomerulonephritis (FIGURE 1) 2 yrs back that was treated with steroids with good response. Her urea and creatinine was normal that time and 24 – hrs urinary protein was 7 grams per 24 hrs. Her vasculitic profile was negative (ANA, anti-dsDNA, ANCA). After 2 months of steroid therapy (1 mg/dl prednisolone), her 24 hr urinary protein was < 300 mg (normal <150 mg/24 hrs) and urea and creatinine was 39 mg/dl (7-18 mg/dl) and 0.9 mg/dl (0.7-1.1 mg/dl). Her steroids were gradually tapered and she was last to follow-up for almost 2 yrs.

On admission, pulse 88/min, blood pressure 130/84 mm Hg, respiratory rate 14/min. she had mild pallor. There was no icterus and lymphadenopathy. JVP was not raised and pedal edema was present. Her chest and cardiovascular system was normal. CNS examination revealed patient was conscious, cooperative, and oriented. There was no cranial nerve deficit. There was grade 3 power lower limbs and grade 4 – power in the upper limbs. Reflexes were brisk all over with clonus bilateral ankles and planters were extensor bilaterally. Touch and pinprick were diminished in legs and arms.

Investigations revealed hemoglobin 11.5 g/dl (11.5-16.5 g/dl), total leukocyte count 9.8 x 109/l (4.0-11.0 x 109 cells/L) and platelets 2.1 x 109/l (150-400 x 109 cells/L). renal function was normal with urea 28 mg/dl and creatinine 1.12 mg/dl. Her serum albumin was 2.9 g/dl (3.5-5.0 g/dl) and 24-hrs urinary protein was 3.7 gm. Ultrasonography abdomen revealed normal kidneys. Patient was reluctant for renal biopsy this time and her immunological screen was negative including ANA, anti-dsDNA, ANCA, and anticardiolipin antibodies.

Magnetic resonance imaging of the cervical spine revealed cord was swollen at C4-6 with increased T2 signal (FIGURE 2). There was minimal contrast enhancement. MRI brain was normal. CSF analysis revealed slightly raised protein (50 mg/dl). CSF was negative for oligoclonal bands.

Visual evoked potential and brainstem evoked auditory responses were normal.
Her viral serology HIV and hepatitis B, C were negative.

A neurological diagnosis of transverse myelitis associated with nephrotic syndrome was made. She was treated with three doses of intravenous methylprednisolone (500 mg) followed by 1 mg/kg of oral prednisolone for 6 weeks. Her repeat 24 hrs urinary protein came down to 500 mg/ 24 hrs and her neurodeficit also improved (good bowel and bladder control and ambulatory with minimal support) at 6 weeks. Her urea and creatinine was also normal (30/0.98). During her follow-up no steroid related adverse effect was noted by the time we published this case.

Figure 1 : Pathological Report Showed Diffuse Thickening Of The Gbm With Presence Of Lucent Holes In En Face Sections Of Gbm And Focal Sub Epithelial Spikes. Mild Mesangial Expansion Is Seen And There Is No Increase In Cellularity. Two Vessels Of The Calibre Of Interlobular Arteries As Well As Small Arteries And Arterioles Appear Unremarkable (H&E Stain With 400 Magnification).

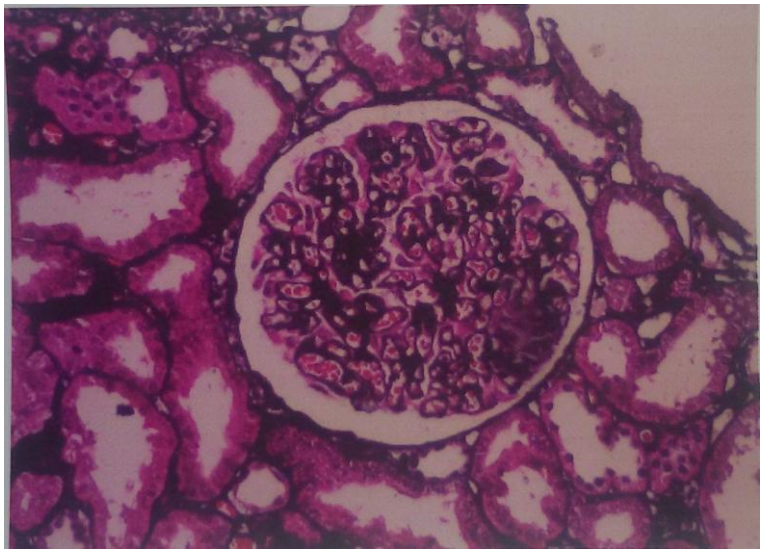
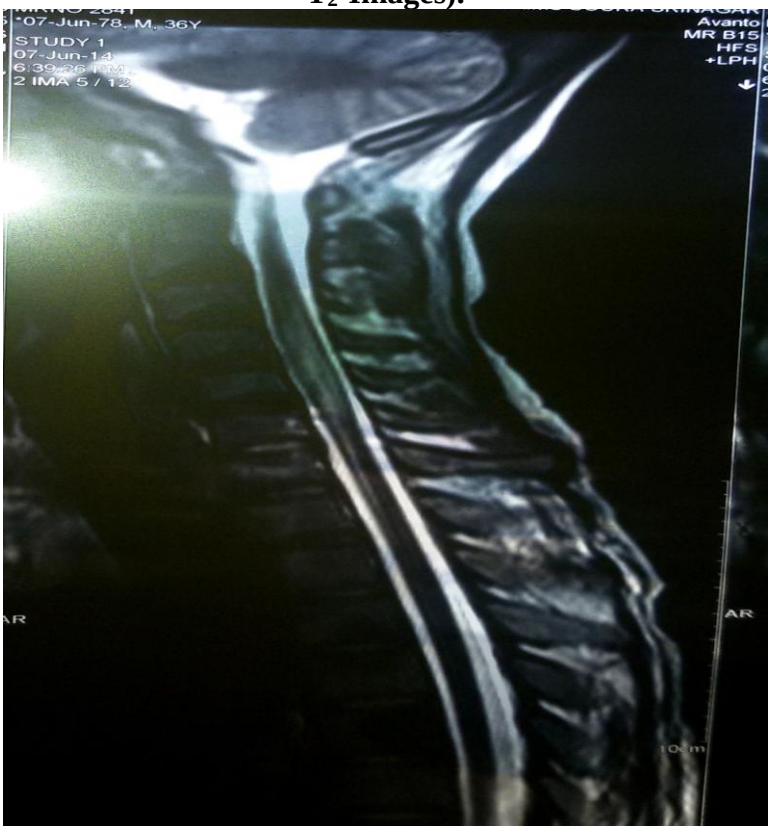


Figure 2: Mri Cervical Spine Showing Swollen Cord With Hyperintensity From C_{4-6} (T_2 Images).



DISCUSSION

Membranous glomerulonephritis (MGN) is characterised by IgG deposits along the outer aspects of the glomerular capillary wall 1 resulting in thickening of capillary wall. Approximately 80% of cases present with overt nephritic syndrome1, while the reminder have isolated proteinuria. Idiopathic MGN is the most common cause of nephrotic syndrome in adults1. Causes of secondary MGN include SLE, chronic infections, solid tumors of the lung and gastrointestinal system and drugs such as penicillamine and captopril1,2. It has been postulated that idiopathic MGN is associated with auto antibodies directed against antigens in the basement sub epithelial spaces. The prevalence of secondary forms of MGN among biopsy documented MGN is approximately 23 % 2. Spontaneous remission is a common finding in children3 and up to 50 % of cases in adults show either complete or partial remission. Steroids have been found to be effective in MGN in many trials4.

Many miscellaneous disorders have been noted in association with MGN. Whether these are chance associations or some etiological relationship exists remains uncertain. Several case reports of MGN in association with neurological diseases like guillain-barre syndrome5,6, multiple sclerosis7 and chronic inflammatory demyelinating polyneuropathy8,9 have been reported. In our case patient had two episodes of nephritis with cord involvement at second time; and both diseases responded to immunosuppression. The only other reported case of inflammatory myelopathy and MGN is by A.W Crowe et al10. It is possible that patients susceptibility to inflammatory myelopathy and MGN may be related to genetic predisposition7. In a study by Honkanen E et al11 it was seen that at 10 yrs 46 % were in complete or partial remission. Ponticelli et al12 described a treatment regimen for idiopathic MGN consisting of methylprednisolone and chlorambucil and stated that this combination may protect renal function and increase the chance of remission.

CONCLUSION

It is important in view of renal and neurological associations that neurological patients are screened for renal disease and it is also important to look for a secondary cause of MGN as this has prognostic and therapeutic implications.

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