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Ig M nephropathy: A clinicopathological study

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Abstract
Introduction: Most studies of IgM nephropathy have consisted of patients with nephrotic syndrome (NS). However, IgM nephropathy is also associated with hematuria and asymptomatic proteinuria. In the long-term follow-up approximately one-third of the NS patients found to develop renal failure of some degree, half the patients experienced hypertension and some developed focal segmental glomerular sclerosis (FSGS) [1-2]. There are only few studies have been reported on the population-based incidence and prevalence, mode of presentation, immunopathological features, pattern of steroid response, and the long-term prognosis of IgM nephropathy in either children or adults.
Objective: To describe the clinical and immunopathological features of IgM nephropathy and its response to steroids and its progression to renal failure.
Method: This is a descriptive, retrospective study carried out in SVIMS and Katuri hospitals, where the clinical records of patients with IgMN were analysed.
Results: From a total number of 2000 renal biopsies, performed during this period, we encountered 45 patients (2.25%) of IgM nephropathy with renal biopsy proof. The number of adults were 35 (77.7%), the number of males were 24 (53.3%). The mean age was 28 years and age range was one year to 64 years; male to female ratio was 1.2:1. The renal syndrome presentation at the first instance was NS in all 45. In addition to NS the other clinical manifestations included hypertension in six, hematuria in eight, and elevated serum creatinine in four patients.
Conclusion: In conclusion, IgM nephropathy is an important cause of NS in both children and adults. It shows a spectrum of morphologic changes ranging from minor changes to FSGS. The diagnosis depends on IF. Although a poor clinical response to steroid therapy distinguished this disease from minimal change disease, in our patients the steroid sensitivity was greater.

Keywords: Ig M nephropathy, clinicopathological, SVIMS

Introduction
Most studies of IgM nephropathy have consisted of patients with nephrotic syndrome (NS). However, IgM nephropathy is also associated with hematuria and asymptomatic proteinuria. It appears that there are two sub- groups of IgM nephropathy in presentation: a sub-group with predominance of men usually manifest NS associated with progressive disease and the other group mainly seen in women with microscopic hematuria with favorable prognosis. In the long-term follow-up approximately one-third of the NS patients found to develop renal failure of some degree, half the patients experienced hypertension and some developed focal segmental glomerular sclerosis (FSGS) [1-2]

Material and Methods
Study Design
A retrospective descriptive study was done from the records of authors' institution.

Study Population
The patients information obtained from the review records between 1994 and 2019. From a total number of 2000 renal biopsies performed during this period, we identified 45 patients of IgM nephropathy.

Analysis of the parameters
Clinical history and findings of age, sex, hypertension, quantitative proteinuria, and serum creatinine (SCr) were considered for evaluation. The diagnosis of IgM nephropathy was determined by the presence of typical light microscopy features and glomerular IgM deposition on immunofluorescence (IF). Electron microscopy is not available at authors' institution.

Follow ups in case records also evaluated to know the response to treatment and progression of disease.

Study Procedure

The patients information obtained from the review records between 1994 and 2019.

From a total number of 2000 renal biopsies performed during this period, we identified 45 patients of IgM nephropathy.

Clinical history and findings of age, sex, hypertension, quantitative proteinuria, and serum creatinine (SCr) were considered for evaluation. The diagnosis of IgM nephropathy was determined by the presence of typical light microscopy features and glomerular IgM deposition on immunofluorescence (IF). All patients managed as follows, the first episode was treated with prednisolone of 0.5 to2.0 mg/kg/day for six months. If necessary, the prednisolone dose was reduced to 0.5 mg/kg/ day, but only after three months. The treatment of steroid-dependent IgM nephropathy was with cyclophosphamide, 2 mg/kg/d for 12 weeks. The treatment of steroid-resistant IgM nephropathy was with cyclosporine at 6 mg/kg/day for children or 5 mg/kg/day for adults for at least six months. All patients were treated with levamisole 2.5 mg/kg/alternate day for 18 months after remission. Follow ups in case records also evaluated to know the response to treatment and progression of the disease.

Results

From a total number of 2000 renal biopsies, performed during this period, we encountered 45 patients (2.25%) of IgM nephropathy with renal biopsy proof. The number of adults were 35 (77.7%), the number of males were 24 (53.3%). The mean age was 28 years and age range was one year to 64 years; male to female ratio was 1.2:1. The renal syndrome presentation at the first instance was NS in all 45. In addition to NS the other clinical manifestations included hypertension in six, hematuria in eight, and elevated serum creatinine in four patients.

The pattern of response to steroids in our patients included, steroid-sensitive NS (SSNS) in 33, steroid resistant NS in five, steroid- dependent NS in seven. Among 33 patients of SSNS, 19 were infrequent relapsers, seven were frequent relapsers and another seven never had a relapse.

The details of renal biopsy findings were tabulated (Table 1). At the end of follow-up for a mean duration of 3.4 ± 0.6 years, 40 patients achieved remission and five patients had reached chronic kidney disease with serum creatinine 3.8, 4.0, 4.5 and 5.1 mg/dL.

Table 1: Renal biopsy findings in 45 patients of IgM nephropathy

Biopsy findings	Number of patients (%) (*n* = 45)
Light microscopy	
Normal glomeruli	6 (13.3)
Mild focal and segmental mesangial hypercellularity	6 (13.3)
Diffuse mesangial hypercellularity	15 (33.3)
Mesangial matrix expansion	13 (28.8)
Interstitial inflammation	22 (48.8)
Interstitial fibrosis	0
Tubular atrophy	0
Hyaline arteriosclerosis	4 (8.8)
Immunofluorescence microscopy	
IgM	35 (77.7)
IgM + C3	6(13.3)
IgM + IgG	4(8.8)
IgM + IgA	0

Discussion

There are only few studies have been reported on the population-based incidence and pre- valence, mode of presentation, immunopathologic features, pattern of steroid response, and the long-term prognosis of IgM nephropathy in either children or adults. There is a wide variation in the prevalence of IgM nephropathy and its long-term prognosis. The reason for this is unclear. Two studies found that frequencies of 2% and 6.1%, respectively in their biopsy series [3-4].

In our study, the adults were more in number than children and a similar observation was reported in the earlier studies [2-7]. We did not find sex predilection. In earlier studies, predomi- nance of either sex was reported.

In the earlier reports, steroid resistance was reported in 28% [5] and steroid dependence in about 33% [1]. While, in our study, among the NS patients, steroid-sensitive were 74%, ste- roid resistance in 10% and steroid dependence in 16%. In our study, 15% had raised SCr at presentation while it was reported as 23% and 27% in earlier studies [1].

In renal histopathology, the majority had matrix expansion, interstitial inflammation with mono-nuclear infiltration with IgM deposits on IF. FSGS was found in only one patient. Similar observation was reported in earlier studies [1, 8-10].

The clinical course and prognosis of IgMN nephropathy were very variable. In part the variation in these studies was due to variable follow-up duration In the largest and longest follow-up study, renal failure was observed in 35% of cases at 15 years after initial biopsy, and 23% of patients went on to develop end- stage renal disease [2, 11-16].

Conclusion

In conclusion, IgM nephropathy is an important cause of NS in both children and adults. It shows a spectrum of morphologic changes ranging from minor changes to FSGS. The diagnosis depends on IF. Although a poor clinical response to steroid therapy distinguished this disease from minimal change disease, in our patients the steroid sensitivity was greater.

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