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Original Article

Clinical manifestations of IgA nephropathy combined with thin glomerular basement membrane nephropathy in children

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Abstract

Background: Immunoglobulin A nephropathy (IgAN) and thin glomerular basement membrane nephropathy (TBMN) are the most common causes of persistent hematuria during childhood. The objective of this study is to determine the difference in clinical features and laboratory findings between pediatric patients with IgA deposited TBMN and IgAN alone.

Methods: Between January 2000 and March 2009, 95 children were diagnosed with IgAN by renal biopsy. Clinical features and laboratory findings of patients with isolated IgAN and with IgAN plus TBMN were compared; the children diagnosed with IgAN were compared to 127 children who had been diagnosed with TBMN alone during the same period.

Results: There were 71 (74.7%) of a total 95 patients that were diagnosed with isolated IgAN (Group1); in 24 (25.3%) of the 95 patients IgAN was combined with TBMN (Group 2). There was marked difference in the gender distribution between Group 2 and isolated TBMN patients. The degree of proteinuria and pathologic severity was higher in Group 1 compared with Group 2. Gross hematuria was present in both groups. There were no distinguishing features in the other laboratory parameters.

Conclusion: Patients with both IgAN and TBMN seem to have similar clinical features to patients with isolated IgAN; however, the latter tend to have better pathologic and laboratory findings, compared to the patients with IgAN alone.

Introduction

Thin glomerular basement membrane nephropathy (TBMN) and IgA nephropathy (IgAN) are the most common causes of primary glomerular disease with persistent hematuria in children [1,2]. Since January 1998 in Korea, all school children undergo a mandatory urine screening test for the early detection of chronic kidney disease. Park et al. [3] reported that more than 72.5% of children showing isolated microscopic hematuria in mass school urine screening test were diagnosed with IgAN or TBMN.

Although these two diseases are similar in their clinical features, patients with IgAN tend to have gross hematuria and proteinuria; it is also known that renal impairment and renal failure can occur during the course of the IgAN [4,5].

Some studies have reported patients with TBMN and IgAN, coincidently. These patients showed heavy proteinuria and progression of renal impairment that have similar characteristics to the patient with a prognosis of IgAN alone [6].

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We conducted this study to compare the differences of clinical manifestations between IgAN with TBMN patients and isolated IgAN or TBMN patients during childhood.

Methods

On a retrospective basis, we performed a cross-sectional study. We reviewed the medical records of 222 patients who had been diagnosed with IgAN or TBMN from January 2000 to March 2009 at Kyungpook National University Hospital. We divided 95 patients diagnosed with having IgAN into two groups: patients with IgAN alone were classified into Group 1 and patients with thinning of glomerular basement membrane on electron microscopy, as well as mesangial IgA deposition, were classified into Group 2. We compared the clinical manifestations between these two groups. In addition, we compared these two groups with isolated TBMN patients, who had been diagnosed at our institution during the same period (Group 3).

The diagnosis of TBMN was based on the criteria proposed by Yoshikawa et al. [7] in 1984. Based on this criterion, biopsy of the kidney reveals thinning of the glomerular basement membrane at <250 nm on electron microscopy with normal findings on light microscopy and on immunofluorescence microscopy. The thickness of the glomerular basement membrane was measured as at least more than five capillary loops and is regarded as diagnostic when $>50\%$ of total visible glomeruli are affected. IgAN was classified based on criteria of the Haas' classification [8].

We compared male to female ratio and average age of each group; in addition, we investigated clinical features, laboratory findings, existence of gross hematuria, degree of proteinuria, and blood pressure at the time of renal biopsy. Because of the cross-sectional design, it was not possible to find results of the spot urine protein to creatinine ratio in every patient, so we used the results of a dipstick test using first morning urine.

Statistical analysis was performed using SPSS version 12.0 (SPSS Inc., Chicago, IL, USA) and the difference of frequency was analyzed by Chi-square test and analysis of variance (ANOVA). A P value <0.05 was considered to be statistically significant.

Results

Of the 222 children enrolled, there were 95 IgAN patients, and 127 TBMN patients. Of 95 children with IgAN, 71 children (Group 1, 74.7%) were diagnosed with isolated IgAN and in 24 (Group 2, 25.3%) IgAN was accompanied by TBMN. The mean age of Groups 1 and 2 were 11.0 ± 2.95 (range, 5.3–16.3) years and 10.9 ± 3.04 (range, 6.9–16.4) years, respectively. Male to female ratio was 7:3 in both groups. In Group 3, the mean age was 7.9 ± 2.84 (range 0.5–14.1) years and the male to female ratio was 5:5; the results of Group 2 resembled Group 1 in age and male predominance (Table 1).

Although no patient had gross hematuria in Group 3, all children in Groups 1 and 2 had an accompanying history of gross hematuria.

The average thickness of the glomerular basement membrane in Group 2 was 205.8 ± 31.1 nm. It was thicker than that of Group 3 (183.8 ± 35.7 nm).

There were 33 (46.5%) children with proteinuria of >100 mg/dL in Group 1 and five (20.8%) in Group 2; thus, the patients with isolated IgAN showed more severe proteinuria ($P=0.04$).

Mean systolic blood pressure of Group 1, Group 2, and Group 3 was 115.2 ± 11.40 mmHg, 113 ± 11.79 mmHg, and 109.9 ± 10.0 mmHg, respectively. Mean diastolic blood pressure of Group 1, Group 2, and Group 3 was 63.7 ± 9.26 mmHg, 65.3 ± 5.95 mmHg, and 59.6 ± 7.85 mmHg, respectively ($P<0.01$).

Histologic types of each group by Haas′ classification were as follow: Class I was the most common type (47%), and Class III accounted for 37% in Group 1 (Fig. 1). In addition, in Group 2, Class I was the most common histologic type (63%)

Table 1. Clinical features and laboratory findings of the patients

Findings	IgAN (Group 1)	IgAN+TBMN (Group 2)	TBMN (Group 3)	P^*
Demographic characteristics				
Number	71	24	127	
Sex (M:F)	49:22	18:6	59:68	
Age at biopsy (y)	11.0 ± 2.95	10.9 ± 3.04	7.9 ± 2.84	
Clinical parameters				
Gross hematuria	71 (1 0 0)	24 (1 0 0)	0%	0.92
Proteinuria (<100 mg/dL: >100 mg/dL)	38 (53.5):33 (46.5)	19 (79.2):5 (20.8)		0.04
SBP (mmHg)	115.2 ± 11.40	113 ± 11.79	109.9 ± 10.0	<0.01
DBP (mmHg)	63.7 ± 9.26	65.3 ± 5.95	59.6 ± 7.85	<0.01
Pathologic findings				
Hass Class I–II:III–V	36 (50.7):35 (49.3)	17 (70.8):7 (29.2)		0.08
Thickness of GBM (nm)		205.8 ± 31.1	183.8 ± 35.7	0.04
Laboratory findings				
Hemoglobin (g/dL)	12.7 ± 1.30	12.8 ± 1.10	12.4 ± 0.99	NS
BUN (mg/dL)	11.9 ± 3.54	12.5 ± 6.32	10.3 ± 2.71	NS
Creatinine (mg/dL)	0.7 ± 0.67	0.7 ± 0.32	0.6 ± 0.12	NS
Sodium (mmol/L)	138.6 ± 3.05	138.7 ± 4.93	138.5 ± 2.50	NS
Potassium (g/dL)	4.1 ± 0.35	4.16 ± 0.32	4.2 ± 0.33	NS
Protein (g/dL)	6.8 ± 0.82	7.1 ± 0.35	7.1 ± 0.48	NS
Albumin (g/dL)	4.1 ± 0.56	4.3 ± 0.29	4.4 ± 0.22	NS
Calcium (mg/dL)	9.3 ± 0.66	9.4 ± 0.43	9.4 ± 0.41	NS
Phosphate (mg/dL)	4.6 ± 0.68	4.7 ± 0.73	4.5 ± 0.63	NS

* P is the statistical value between Group 1 and Group 2.
Data are presented as mean $\pm$ SD or n (%).
BUN, blood urea nitrogen; DBP, diastolic blood pressure; F, female; GBM, glomerular basement membrane; IgAN, immunoglobulin A nephropathy; M, male; NS, nonspecific; SBP, systolic blood pressure; TBMN, thin glomerular basement membrane nephropathy.