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Original Article

Clinico-pathological association of Henoch-Schoenlein purpura nephritis and IgA nephropathy in children

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Abstract: Objective: Henoch-Schonlein purpura nephritis (HSPN) and IgA nephropathy (IgAN) are similar syndromes. We aimed to determine whether the crescent formation/immunocomplex in glomeruli is associated with the differences of the biochemical indexes between HSPN and IgAN. Methods: We investigated the medical records of 137 HSPN cases and 41 IgAN cases from January 2009 to April 2014 in Nanjing Children's Hospital of Nanjing Medical University. The clinical and pathological data were analyzed and compared between HSPN and IgAN. Results: HSPN patients had markedly higher levels of blood white blood cell (WBC), hemoglobulin (Hb) and platelet (PLT), lower levels of hematuria, blood nitrogen (BUN) and C4 compared with IgAN cases. Crescents formation and C3 deposition in the kidney did not affect these differences. Significantly lower levels of hematuria, blood IgG, IgM and C4 in HSPN compared with IgAN cases were observed among patients with IgG deposition. Markedly higher levels of WBC and Hb, lower levels of hematuria, creatinine (Cr), C4 in HSPN compared with IgAN cases were observed among patients with IgM deposition. No marked differences of the biochemical indexes were noted between HSPN and IgAN cases among patients with C1q deposition. Markedly higher levels of WBC and Hb, lower level of blood C4 in HSPN compared with IgAN cases were observed among patients with fibrogen deposition. Conclusions: The different levels of biochemical indexes at presentation between HSPN and IgAN may be associated with the deposition of IgG, IgM, C1q and fibrogen in the kidney.

Keywords: Henoch-Schonlein purpura nephritis, IgA nephropathy, children, retrospective

Introduction

Henoch-Schonlein purpura nephritis (HSPN) is the most common secondary glomerular disease in children [1], while IgAN is one of most common causes of primary glomerulonephritis in adult and children [2]. HSP is a self-limited condition with nearly 40% of pediatric patients develops nephritis within 4 to 6 weeks after disease onset [3]. Most IgAN patients experienced persistent microscopic hematuria and intermittent episodes of gross hematuria [4]. HSPN and IgAN have a lot in common in many clinical, histological, and immunological features. Both of these two diseases are characterized by the presence of mesangial IgA deposits and hematuria [5]. Also, it is reported that IgAN patient evolutes into HPSN, and HSP and IgAN coexisted in different members of the same family [6, 7]. Hence, HSPN is usually considered a systemic form of IgAN, and they are even considered a single disease.

In spite of their similar pathophysiology, the clinical manifestations and laboratory findings of IgAN and HSPN at presentation are somewhat different. Furthermore, the renal pathological changes between HSPN and IgAN are slightly different. It is imperative to identify the association between the clinical indexes and pathological changes. Previous studies showed that HSPN was an acute disease and prognosis was associated with the severity of glomerular changes, while IgAN was a chronic, slowly progressive glomerular disease [8]. HSPN was associated with more extra-renal manifestations while IgAN has more severe renal involvement [9]. HSPN and IgAN have similar immune abnormalities and long-term prognosis [10, 11]. Also, significant clinicopathological difference was found between HSPN and IgAN [10]. However, whether the pathological changes, including crescents formation, IgG, IgM, C3, C4, C1q and fibrogen deposition in the kidney were associated with

Table 1. Baseline characteristics of HSPN and IgAN in children

HSPN (137)	IgAN (41)		P
Age (years)	8.6 ± 2.7	9.5 ± 2.5	0.058
Boy (n)	92	31	0.661
SBP (mmHg)	112.9 ± 11.2	112.8 ± 11.6	0.960
DBP (mmHg)	70.5 ± 11.0	68.8 ± 10.9	0.385
BMI (Kg/M^2)	17.6 ± 3.0	17.0 ± 2.1	0.233
WBC ($X10^9/L$)	11.9 ± 4.6	8.3 ± 3.3	$< 10^{-4}$
Hb (g/L)	134.1 ± 13.4	124.9 ± 10.7	$< 10^{-4}$
PLT ($X10^9/L$)	304.6 ± 92.5	251.2 ± 69.4	$< 10^{-4}$
Hematuria (/uL)	530.3 ± 822.5	4309.6 ± 9326.3	$< 10^{-4}$
Proteinuria/cr ratio	2.1 ± 2.4	1.8 ± 1.9	0.463
BUN (mmol/L)	4.2 ± 1.4	5.5 ± 4.5	0.004
Cr (umol/L)	44.4 ± 23.9	52.6 ± 29.8	0.071
Alb (g/L)	40.9 ± 6.6	38.7 ± 6.9	0.065
Chol (mmol/L)	5.4 ± 1.8	5.0 ± 1.7	0.207
IgA (g/L)	2.3 ± 1.1	2.4 ± 1.1	0.610
IgG (g/L)	7.7 ± 3.5	7.6 ± 2.7	0.866
IgM (g/L)	1.2 ± 0.7	1.2 ± 0.5	1.000
C3 (g/L)	1.2 ± 0.3	1.2 ± 0.2	1.000
C4 (g/L)	0.2 ± 0.1	0.3 ± 0.1	$< 10^{-4}$
Crescent formation	206/3393	40/832	0.189
IgA deposition	137	41	1.000
IgG deposition	26	7	0.820
IgM deposition	79	26	0.739
C3 deposition	68	30	0.156
C1q deposition	8	3	0.746
Fibrogen deposition	97	19	0.904

HSPN: Henoch-Schonlein purpura nephritis; IgAN: IgA nephropathy.

the differences of clinical biochemical indexes remain elusive.

To have an in-depth understanding of the association between the crescents formation, immunoglobulin (Ig), complement and fibrogen deposition in the kidney and the differences of biochemical indexes between HSPN and IgAN, we compared the laboratory findings between HSPN and IgAN and investigate the influence of crescents formation, Ig, complement and fibrogen.

Materials and methods

Patient population

The study population undergoing renal biopsy at Nanjing Children's Hospital between January 2009 and April 2014 were screened. The cases were enrolled when who were diagnosed as HSPN/IgAN at presentation. HSPN was diagnosed when hematuria and/or proteinuria was associated with HSP. HSP was diagnosed according to the criteria, based on the American College of Rheumatology (ACR) database and methodology [12]. The diagnosis of IgAN was based on the presence of IgA as the predominant immunoglobulin in the glomerular mesangium according to the Oxford classification [13]. The patients were excluded if they were with other systemic diseases or underwent a repeated renal biopsy.

Data collection

We earnestly extracted the clinical and laboratory findings of the patients from the medical records. Demographic and clinical data were reviewed respectively for age, gender, systolic blood pressure (SBP), diastolic blood pressure (DBP) and body mass index (BMI). Laboratory data included blood WBC, Hb, PLT, BUN, Cr, albumin (Alb), cholesterol (chol), IgA, IgG, IgM, C3, C4, ratio of proteinuria/cr and hematuria. Renal pathology data included information on the number of crescentic glomeruli, deposition of IgA, IgG, IgM, C3, C4, C1q and fibrogen in kidney.

Statistical analysis

Categorical variables were expressed as cases/total, OR (odds ratio) was used to compare the differences of categorical variables between HSPN and IgAN groups.

Continuous variables were expressed as means ± standard deviation (SD), Standard mean deviation (SMD) was used to measure the differences in continuous variables between HSPN and IgAN groups. The analyses were conducted using STATA version 12.0 (Stata Corp, College Station, TX). $P < 0.05$ was considered statistically significant, except where otherwise specified.

Results

Patient characteristics

A total of 137 HSPN (92 bodys) and 41 IgAN (31 boys) cases were recruited in our study. The

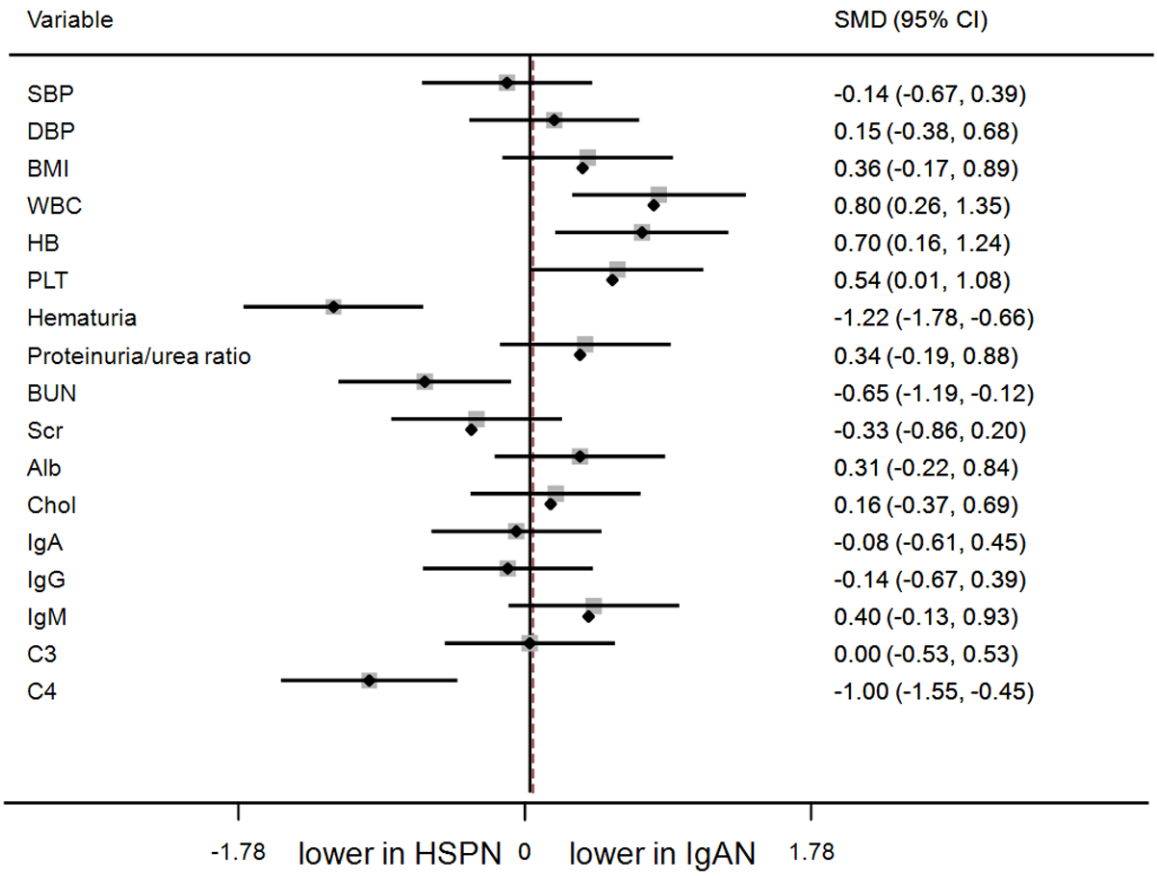

Figure 1. Comparison between HSPN and IgAN with crescents.

mean age was 8.6 ± 2.7 and 9.5 ± 2.5 years for HSPN, IgAN cases, respectively. No marked differences of SBP, DBP, BMI, ratio of proteinuria/cr, Cr, Alb, Chol, serum IgA, IgG, IgM, C3, crescents formation, deposition of IgA, IgG, IgM, C3, C1q and fibrogen in the kidney were observed between HSPN and IgAN cases (**Table 1**). HSPN patients had markedly higher levels of blood WBC, Hb, and PLT than those in IgAN cases (**Table 1**). HSPN patients had markedly lower levels of hematuria, blood BUN and C4 than those in IgAN cases (**Table 1**).

HSPN and IgAN with crescents formation in glomeruli

A total of 72 HSPN and 17 IgAN cases were enrolled. There were no significant differences of SBP, DBP, BMI, ratio of proteinuria/cr, Cr, Alb, Chol, blood IgA, IgG, IgM, and C3 between HSPN and IgAN cases (Table S1; **Figure 1**). HSPN patients had significantly higher levels of blood WBC, Hb, and PLT than those in IgAN patients (Table S1; **Figure 1**). HSPN patients had markedly lower levels of hematuria, blood BUN and C4 compared with those in IgAN cases (Table S1; **Figure 1**).

HSPN and IgAN with IgG deposition in glomeruli

A total of 26 HSPN and 7 IgAN patients were included. No significant differences of SBP, DBP, BMI, WBC, Hb, PLT, ratio of proteinuria/cr, BUN, Cr, Alb, Chol, blood IgA, and C3 between HSPN and IgAN patients (Table S2; **Figure 2**). HSPN patients had markedly lower levels of hematuria, blood IgG, IgM and C4 in comparison to those in IgAN cases (Table S2; **Figure 2**).

HSPN and IgAN with IgM deposition in glomeruli

A total of 79 HSPN and 26 IgAN cases were enrolled. There were no significant differences of SBP, DBP, BMI, PLT, ratio of proteinuria/cr, BUN, Alb, Chol, blood IgA, IgG, IgM, and C3 between HSPN and IgAN cases (Table S3; **Figure 3**). HSPN patients had significantly

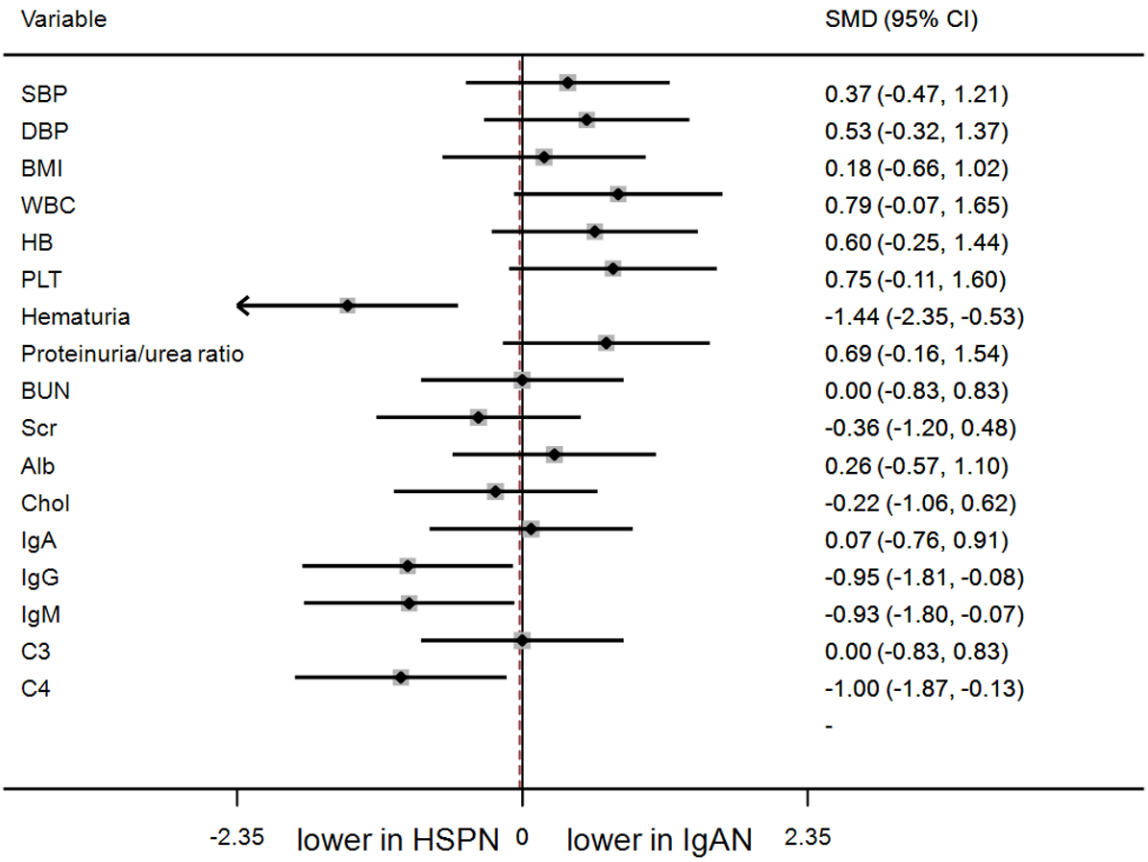

Figure 2. Comparison between HSPN and IgAN with IgG deposition.

higher levels of blood WBC and Hb than those in IgAN patients (Table S3; **Figure 3**). HSPN patients had markedly lower levels of hematuria, blood Cr and C4 compared with those in IgAN cases (Table S3; **Figure 3**).

HSPN and IgAN with C3 deposition in glomeruli

A total of 68 HSPN and 30 IgAN cases were enrolled. There were no significant differences of SBP, DBP, BMI, ratio of proteinuria/cr, Cr, Alb, Chol, blood IgA, IgG, IgM, and C3 between HSPN and IgAN cases (Table S4; **Figure 4**). HSPN patients had significantly higher levels of blood WBC,Hb, and PLT than those in IgAN patients (Table S4; **Figure 4**). HSPN patients had markedly lower levels of hematuria, blood BUN and C4 compared with those in IgAN cases (Table S4; **Figure 4**).

HSPN and IgAN with C1q deposition in glomeruli

A total of 8 HSPN and 3 IgAN cases were enrolled. There were no significant differences of SBP, DBP, BMI, WBC, Hb, PLT, hematuria, ratio of proteinuria/cr, BUN, Cr, Alb, Chol, blood IgA, IgG, IgM, C3 and C4 between HSPN and IgAN cases (Table S5; **Figure 5**).

HSPN and IgAN with fibrogen deposition in kidney

A total of 97 HSPN and 19 IgAN cases were enrolled. There were no significant differences of SBP, DBP, BMI, PLT, ratio of proteinuria/cr, BUN, Cr, Alb, Chol, blood IgA, IgG, IgM, and C3 between HSPN and IgAN cases (Table S6; **Figure 6**). HSPN patients had significantly higher levels of blood WBC and Hb than those in IgAN patients (Table S6; **Figure 6**). HSPN patients had markedly lower levels of hematuria and serum C4 compared with those in IgAN cases (Table S6; **Figure 6**).

Discussion

Increasing attention has been paid to the differences between HSPN and IgAN.

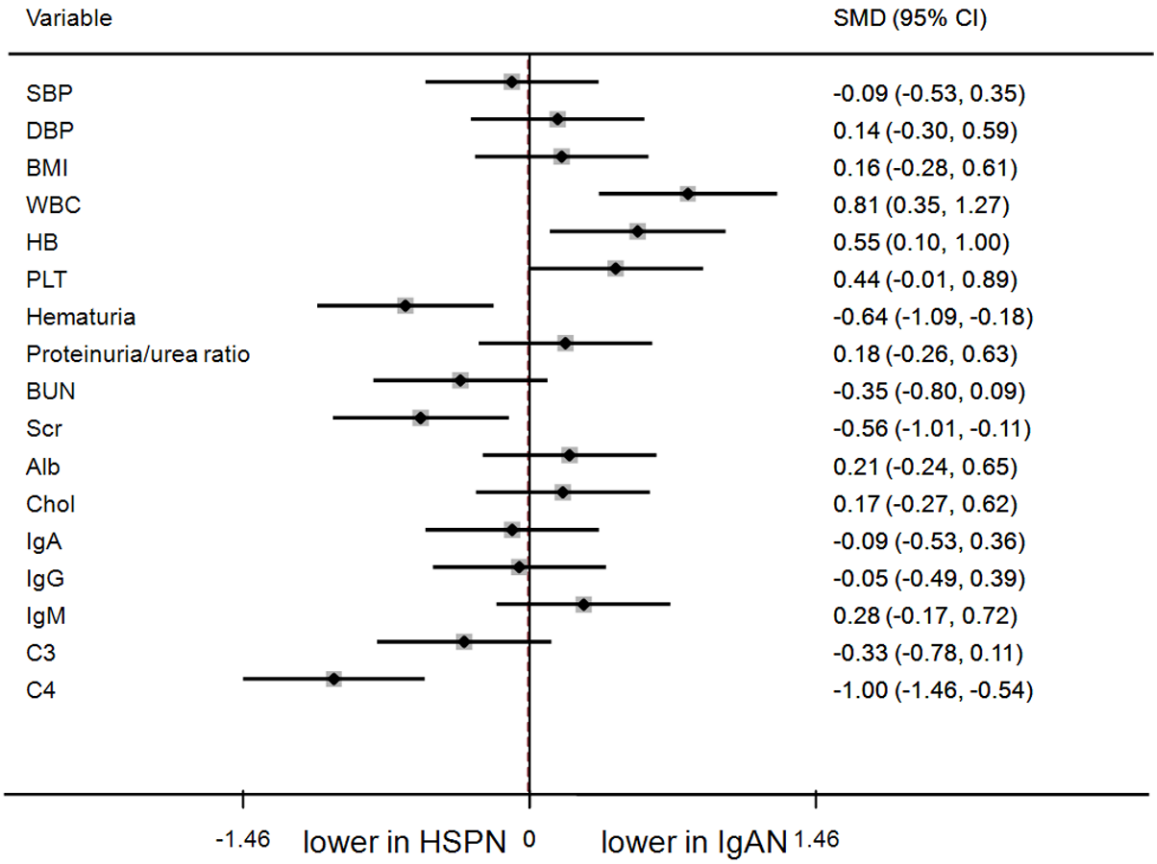

Figure 3. Comparison between HSPN and IgAN with IgM deposition.

To our knowledge, this is the first investigation identifying the association between crescents formation, deposition of Ig, complement and fibrogen in the kidney and the differences of biochemical indexes at presentation between HSPN and IgAN. Our study showed that HSPN patients had markedly higher levels of blood WBC, Hb and PLT, lower levels of hematuria, BUN and C4 compared with IgAN cases. IgG, IgM, C1q and fibrogen deposition in the kidney were associated with these differences, which indicated that the similar laboratory findings among HSPN and IgAN cases may reflect different pathological changes.

Several mechanisms may account for the association between clinical biochemical indexes and pathological changes among HSPN and IgAN. First, HSPN, a leukocytoclastic vasculitis, is often associated with the increase of eosinophilic granulocyte [14]. HSPN is also usually associated with bacterial infection [15], which may lead to the elevation of serum WBC level. On the other hand, the steroid use in HSPN may result in the increase of blood WBC level. IgAN is characterized by intermittant macrohematuria. Due to the loss of blood, Hb decreased more seriously in IgAN than HSPN. The PLT may decrease because of the consumption. IgAN is a chronic, progressive glomerulonephropathy. The renal function in IgAN is more likely to be worse than that in HSPN. Hypocomplementemia is closely associated with the infection and immune reaction [16]. The above-mentioned evidence may explain the differences of biochemical indexes between HSPN and IgAN. Second, mesangial IgG deposition may play a role in the pathogenesis of renal damage in HSPN and IgAN [17, 18]. IgM deposition was associated with glomerular obsolescence and tuft adhesions in IgAN [19]. Fibrinogen deposition was more severe in earlier histological changes of IgAN [20]. Mesangial C1q deposition in the glomerulus was associated with a poor renal outcome and severe pathologic features in IgAN [21]. These data indicated that

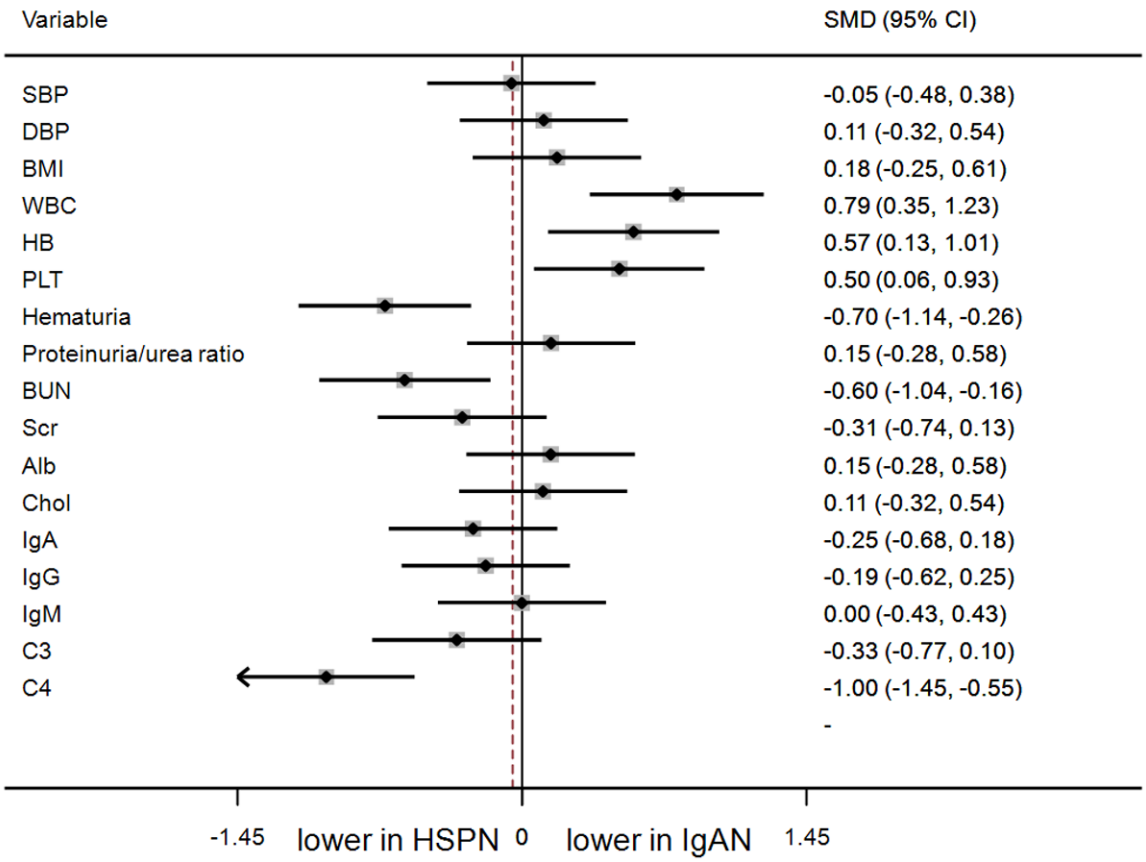

Figure 4. Comparison between HSPN and IgAN with C3 deposition.

the deposition of Ig and fibrogen might be closely associated with the severity of HSPN/IgAN. Finally, the biochemical indexes in glomerulopathy may predict the histopathological diagnosis. Also, the severity of glomerulopathy is usually associated with the changes of the biochemical indexes. Hence, it is reasonable to speculate the existence of the close relationship between biochemical indexes levels and the deposition of Ig, complement and fibrogen.

Our findings also give rise to two questions. First, although some studies demonstrated that HSPN and IgAN had a similar prognosis, the deposition of Ig, complement and fibrogen may have different influence on the prognosis of HSPN and IgAN. In terms of the association between biochemical indexes and the deposition of Ig, complement and fibrogen, this speculation seems reasonable. Regrettably, due to the time limit, we do not perform a prospective follow-up. Further studies should be focused on this point. Second, the deposition of Ig, complement and fibrogen may attenuate or even disappear with the remission of glomerulopathy. It is unclear that which biochemical indexes are most closely with these depositions. It is very helpful for guiding the therapy by clarifying this question.

In the past, several studies were performed to investigate the differences between HSPN and IgAN. Oh et al [11] reported that HSPN patients had a higher level of c-reactive protein than that in IgAN cases. Guo et al [4] reported that HSPN cases had higher ratio of fibrogen deposition than IgAN patients. Li et al [22] reported that significant pathological difference was observed between HSPN and IgAN. Takemura et al [23] reported that IgA was frequently noted with deposition of IgG, IgM and C3 both in HSPN and IgAN. Calvo-Rio et al [9] and Yoshikawa et al [8] reported that IgAN and HSPN were different syndromes with different clinical presentations. All these previous find-

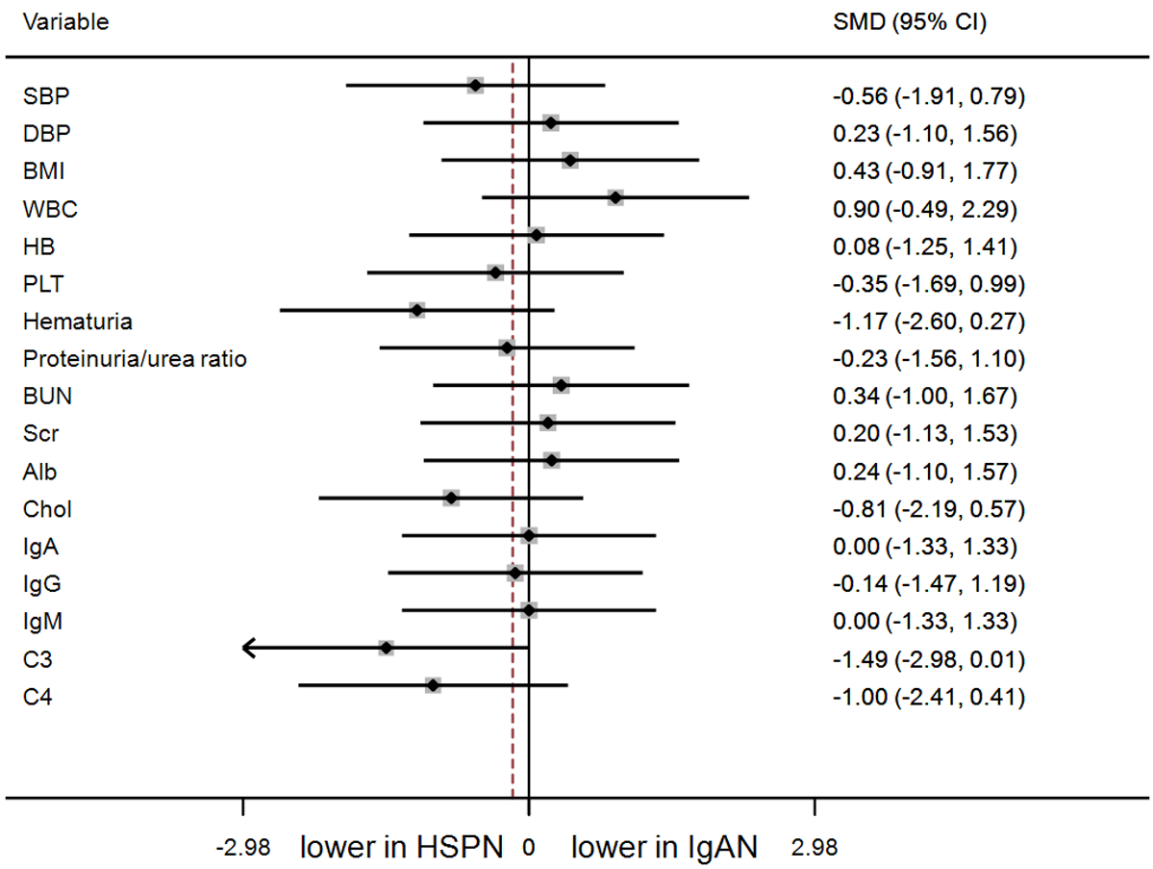

Figure 5. Comparison between HSPN and IgAN with C1q deposition.

ings supported the idea that HSPN and IgAN were not totally same. Our findings further clarified the association between biochemical indexes levels and the deposition of Ig, complement and fibrogen, which were consistent with the above- mentioned evidence.

Several limitations should be considered in our study. First, the retrospective study design might induce recall bias, we have to seek the reliable laboratory data. Second, due to the limit of time and loss of follow-up of some patients, we did not observe the long-term renal outcome of patients. Finally, the limited number of study participants reduced the statistical power. Hence, a long-term, continuous, lager number of study should be performed.

Taken together, our investigation suggests that HSPN and IgAN may have different levels of biochemical indexes at presentation, which may be associated with the deposition of IgG, IgM, C1q and fibrogen in kidney. However, larger number of studies should be conducted to validate our findings.

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Disclosure of conflict of interest

None.

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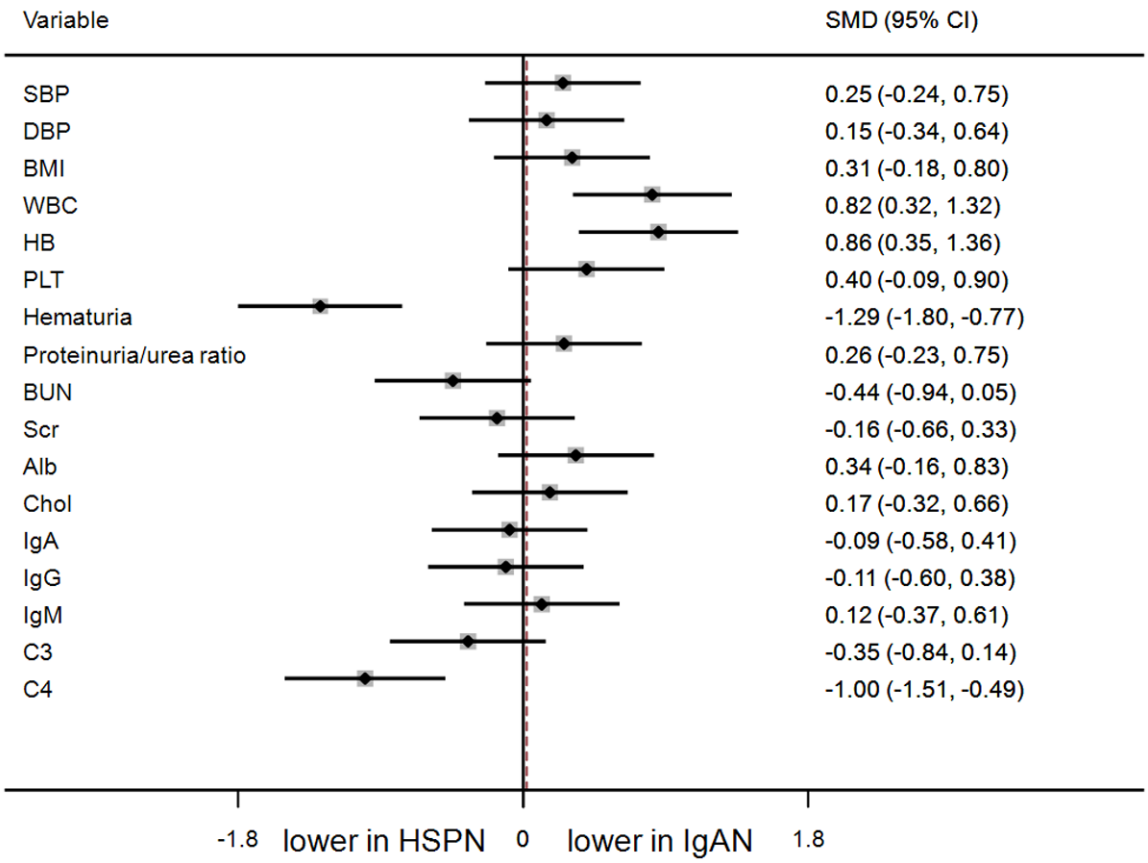

Figure 6. Comparison between HSPN and IgAN with fibrogen deposition.

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Table S1. Comparison between HSPN and IgAN in children with crescents formation in glomeruli

	HSPN (72)	IgAN (17)	P
SBP (mmHg)	113.0 ± 10.2	114.5 ± 12.3	0.601
DBP (mmHg)	70.1 ± 10.5	68.5 ± 10.7	0.574
BMI (Kg/M^2)	17.8 ± 3.2	16.7 ± 2.3	0.184
WBC (X10^9/L)	11.7 ± 4.6	8.1 ± 3.9	0.004
Hb (g/L)	133.0 ± 14.2	123.4 ± 11.6	0.011
PLT (X10^9/L)	301.1 ± 95.6	250.3 ± 82.3	0.046
Hematuria (/uL)	593.4 ± 884.6	7627.8 ± 13292.3	< 10^{-4}
Proteinuria/cr ratio	2.5 ± 2.8	1.6 ± 1.5	0.203
BUN (mmol/L)	4.4 ± 1.4	6.3 ± 6.1	0.017
Cr (umol/L)	45.2 ± 28.9	55.6 ± 40.5	0.221
Alb (g/L)	41.1 ± 6.9	39.3 ± 5.9	0.249
Chol (mmol/L)	5.4 ± 1.9	5.1 ± 1.7	0.551
IgA (g/L)	2.4 ± 1.2	2.5 ± 1.2	0.757
IgG (g/L)	7.4 ± 3.0	7.8 ± 2.6	0.613
IgM (g/L)	1.4 ± 0.8	1.1 ± 0.5	0.142
C3 (g/L)	1.2 ± 0.2	1.2 ± 0.2	1.000
C4 (g/L)	0.2 ± 0.1	0.3 ± 0.1	< 10^{-4}

HSPN: Henoch-Schonlein purpura nephritis; IgAN: IgA nephropathy.

Table S2. Comparison between HSPN and IgAN in children with IgG deposition in glomeruli

	HSPN (26)	IgAN (7)	P
SBP (mmHg)	111.8 ± 11.9	107.1 ± 15.0	0.382
DBP (mmHg)	70.8 ± 12.3	64.7 ± 7.4	0.219
BMI (Kg/M^2)	17.2 ± 3.0	16.7 ± 1.6	0.674
WBC (X10^9/L)	11.7 ± 5.4	7.7 ± 3.3	0.071
Hb (g/L)	130.7 ± 13.9	122.1 ± 16.4	0.168
PLT (X10^9/L)	309.8 ± 80.2	254.0 ± 46.3	0.087
Hematuria (/uL)	520.2 ± 670.9	12716.8 ± 19221.7	0.002
Proteinuria/cr ratio	3.1 ± 2.5	1.5 ± 1.3	0.112
BUN (mmol/L)	3.9 ± 1.0	3.9 ± 0.9	1.000
Cr (umol/L)	38.1 ± 11.6	41.9 ± 3.9	0.401
Alb (g/L)	40.8 ± 6.2	39.2 ± 5.6	0.538
Chol (mmol/L)	4.7 ± 1.8	5.1 ± 1.9	0.606
IgA (g/L)	2.4 ± 1.5	2.3 ± 0.8	0.866
IgG (g/L)	6.9 ± 2.5	9.1 ± 1.4	0.033
IgM (g/L)	1.0 ± 0.3	1.3 ± 0.4	0.035
C3 (g/L)	1.2 ± 0.2	1.2 ± 0.2	1.000
C4 (g/L)	0.2 ± 0.1	0.3 ± 0.1	0.024

HSPN: Henoch-Schonlein purpura nephritis; IgAN: IgA nephropathy.

Table S3. Comparison between HSPN and IgAN in children with IgM deposition in glomeruli

	HSPN (79)	IgAN (26)	P
SBP (mmHg)	113.1 ± 11.1	114.1 ± 11.4	0.692
DBP (mmHg)	70.5 ± 11.5	68.9 ± 10.1	0.527
BMI (Kg/M^2)	17.5 ± 3.3	17.0 ± 2.0	0.467
WBC ($X10^9/L$)	12.4 ± 4.9	8.7 ± 3.3	0.001
Hb (g/L)	133.7 ± 13.9	126.4 ± 10.7	0.016
PLT ($X10^9/L$)	316.2 ± 96.3	275.5 ± 78.4	0.053
Hematuria (/uL)	546.6 ± 831.9	3258.7 ± 8536.5	0.006
Proteinuria/cr ratio	2.2 ± 2.2	1.8 ± 2.1	0.417
BUN (mmol/L)	4.3 ± 1.4	4.9 ± 2.4	0.120
Cr (umol/L)	41.6 ± 13.1	52.3 ± 31.5	0.015
Alb (g/L)	39.8 ± 7.3	38.3 ± 7.3	0.364
Chol (mmol/L)	5.4 ± 1.7	5.1 ± 1.9	0.449
IgA (g/L)	2.4 ± 1.2	2.5 ± 0.9	0.697
IgG (g/L)	7.7 ± 4.2	7.9 ± 2.6	0.819
IgM (g/L)	1.3 ± 0.8	1.1 ± 0.4	0.223
C3 (g/L)	1.1 ± 0.3	1.2 ± 0.3	0.142
C4 (g/L)	0.2 ± 0.1	0.3 ± 0.1	$< 10^{-4}$

HSPN: Henoch-Schonlein purpura nephritis; IgAN: IgA nephropathy.

Table S4. Comparison between HSPN and IgAN in children with C3 deposition in glomeruli

HSPN (68)	IgAN (30)		P
SBP (mmHg)	113.2 ± 11.0	113.8 ± 11.2	0.805
DBP (mmHg)	70.8 ± 11.2	69.6 ± 9.7	0.611
BMI (Kg/M^2)	17.4 ± 3.1	16.9 ± 1.9	0.415
WBC ($X10^9/L$)	11.9 ± 5.0	8.3 ± 3.3	$< 10^{-4}$
Hb (g/L)	131.7 ± 14.6	124.1 ± 9.8	0.011
PLT ($X10^9/L$)	301.9 ± 86.2	261.2 ± 71.1	0.025
Hematuria (/uL)	446.1 ± 757.8	4406.8 ± 10228.5	0.002
Proteinuria/cr ratio	2.3 ± 2.9	1.9 ± 2.1	0.497
BUN (mmol/L)	4.2 ± 1.3	6.0 ± 5.1	0.007
Cr (umol/L)	45.9 ± 29.8	55.3 ± 32.9	0.165
Alb (g/L)	40.6 ± 6.8	39.6 ± 6.9	0.505
Chol (mmol/L)	5.2 ± 1.9	5.0 ± 1.8	0.626
IgA (g/L)	2.3 ± 1.2	2.6 ± 1.2	0.256
IgG (g/L)	7.7 ± 2.7	8.2 ± 2.7	0.399
IgM (g/L)	1.1 ± 0.5	1.1 ± 0.5	1.000
C3 (g/L)	1.1 ± 0.3	1.2 ± 0.3	0.131
C4 (g/L)	0.2 ± 0.1	0.3 ± 0.1	$< 10^{-4}$

HSPN: Henoch-Schonlein purpura nephritis; IgAN: IgA nephropathy.

Table S5. Comparison between HSPN and IgAN in children with C1q deposition in glomeruli

HSPN (8)	IgAN (3)		P
SBP (mmHg)	117.4 ± 4.4	120.7 ± 9.5	0.419
DBP (mmHg)	74.8 ± 14.8	71.7 ± 7.0	0.735
BMI (Kg/M^2)	19.9 ± 6.0	17.6 ± 1.3	0.528
WBC ($X10^9/L$)	15.1 ± 6.5	9.7 ± 3.6	0.203
Hb (g/L)	135.1 ± 19.0	133.7 ± 12.9	0.908
PLT ($X10^9/L$)	310.9 ± 99.8	343.3 ± 62.1	0.609
Hematuria (/uL)	349.7 ± 327.2	1326.1 ± 1665.9	0.110
Proteinuria/cr ratio	2.2 ± 1.8	2.6 ± 1.5	0.735
BUN (mmol/L)	5.2 ± 1.9	4.6 ± 1.3	0.622
Cr (umol/L)	48.6 ± 23.5	44.5 ± 1.7	0.771
Alb (g/L)	39.5 ± 8.9	37.5 ± 6.9	0.729
Chol (mmol/L)	4.9 ± 1.6	6.3 ± 2.1	0.248
IgA (g/L)	1.9 ± 0.5	1.9 ± 0.5	1.000
IgG (g/L)	7.7 ± 4.4	8.3 ± 3.3	0.832
IgM (g/L)	1.3 ± 0.7	1.3 ± 0.4	1.000
C3 (g/L)	0.8 ± 0.3	1.2 ± 0.1	0.051
C4 (g/L)	0.2 ± 0.1	0.3 ± 0.1	0.163

HSPN: Henoch-Schonlein purpura nephritis; IgAN: IgA nephropathy.

Table S6. Comparison between HSPN and IgAN in children with fibrogen deposition in kidney

HSPN (97)	IgAN (19)		P
SBP (mmHg)	113.4 ± 11.1	110.5 ± 13.2	0.314
DBP (mmHg)	70.4 ± 11.4	68.7 ± 11.3	0.552
BMI (Kg/M^2)	17.5 ± 3.0	16.6 ± 2.3	0.218
WBC ($X10^9/L$)	11.9 ± 4.8	8.1 ± 3.7	0.001
Hb (g/L)	133.2 ± 13.0	122.5 ± 9.2	0.001
PLT ($X10^9/L$)	306.2 ± 94.2	268.7 ± 88.9	0.111
Hematuria (/uL)	496.0 ± 815.2	2964.7 ± 4453.2	$< 10^{-4}$
Proteinuria/cr ratio	2.0 ± 2.0	1.5 ± 1.5	0.303
BUN (mmol/L)	4.1 ± 1.3	4.7 ± 1.6	0.079
Cr (umol/L)	44.1 ± 25.8	48.1 ± 14.1	0.513
Alb (g/L)	41.4 ± 6.5	39.2 ± 6.9	0.183
Chol (mmol/L)	5.2 ± 1.8	4.9 ± 1.6	0.500
IgA (g/L)	2.4 ± 1.2	2.5 ± 1.0	0.734
IgG (g/L)	7.9 ± 3.8	8.3 ± 2.8	0.663
IgM (g/L)	1.3 ± 0.9	1.2 ± 0.4	0.636
C3 (g/L)	1.2 ± 0.3	1.3 ± 0.2	0.166
C4 (g/L)	0.2 ± 0.1	0.3 ± 0.1	$< 10^{-4}$

HSPN: Henoch-Schonlein purpura nephritis; IgAN: IgA nephropathy.