Correlation Between Serum Anti-C1q Antibody Levels and Renal Pathological Characteristics and Prognostic Significance of Anti-C1q Antibody in Lupus Nephritis

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ABSTRACT. ***Objective.*** To investigate the relationship between serum anti-C1q antibody levels and renal pathological characteristics in lupus nephritis as well as the prognostic significance of serum anti-C1q antibody.

Methods. Seventy-three patients with biopsy-proven lupus nephritis were enrolled. Anti-C1q antibody was measured in serum samples taken within 7 days before renal biopsy and remeasured at the end of the first and the third month after treatment. All patients were followed at least once a month for 3 months. A cross-sectional study analyzed the relationship between serum anti-C1q antibody levels and renal histopathology and nephritic activity, while a longitudinal study evaluated the prognostic significance of anti-C1q antibody levels in lupus nephritis.

Results. Fifty-eight of 73 patients (79.5%) were reported as having positive baseline serum anti-C1q antibody, with a mean level of 95.3 (± 55.2) U/ml. Significant differences were found in serum anti-C1q antibody levels between each World Health Organization (WHO) classification of lupus nephritis. The serum anti-C1q antibody level of WHO class IV was the highest. Serum anti-C1q antibody was positively correlated with the active and chronic indices in renal pathology. Patients with persistent high levels or increased titers of serum anti-C1q antibody tended to develop delayed remission in nephropathy. Serum anti-C1q antibody levels before and after treatment were relevant to renal remission, but serum anti-C1q antibody at the end of the third month after treatment was a stronger predictor for the prognosis after adjustment in the Cox's proportional hazards regression model.

Conclusion. Serum anti-C1q antibody is a valuable noninvasive biological marker for evaluation of renal involvement and lupus prognosis. (J Rheumatol First Release March 1 2010; doi:10.3899/jrheum.090779)

Key Indexing Terms:
LUPUS NEPHRITIS ANTI-C1q ANTIBODY RENAL BIOPSY PROGNOSIS

Nephropathy is considered one of the main risks and prognostic determinants of systemic lupus erythematosus (SLE)[1]. Markers clarifying different symptoms, histopathology, therapeutic responses, and prognoses in lupus nephritis require attention. Autoimmune antibody to C1q, a subcomponent of C1 in the classical pathway of complement activation, described as an immune deposition in lupus nephritis[2], has been thought to be a strong predictor of renal involvement in patients with SLE[3,4]. In addition, serum anti-C1q antibody elevation is associated with renal flare of lupus nephritis[5]. Anti-C1q antibody-negative patients may have low risk of developing severe lupus nephritis[6]. Similar response was found in murine models such as accumulation of anti-C1q antibody in glomeruli and rising titers in sera[7,8]. Anti-C1q antibody was also found to recognize a neoepitope on C1q and specifically targeted C1q bound on early apoptotic cells[9], suggesting that SLE, apoptosis, and C1q are linked.

Although it is suggested that anti-C1q antibody might play an important role in lupus nephritis[10], its biological function is unclear. The prevalence of anti-C1q antibody in patients with active lupus nephritis and its predictive value for renal disease remain controversial[11,12]. Thus, the possible pathogenic role of anti-C1q antibody in lupus nephritis

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and the relevance of assessing it in clinical practice deserve further investigation.

We investigated the prevalence of anti-C1q antibody in a group of Chinese patients with lupus nephritis, and evaluated the association of the anti-C1q antibody with renal pathological characteristics and disease activity. We also investigated whether anti-C1q antibody serves as a novel noninvasive biological marker of renal involvement and whether monitoring anti-C1q antibody serves as a prognostic indicator in the management of lupus nephritis.

MATERIALS AND METHODS

Demographic and clinical characteristics of the patients. Seventy-three patients with biopsy-proven lupus nephritis were enrolled, including 65 women and 8 men with a mean age 31.0 (± 13.8) years (Table 1). The mean duration of SLE was 2.3 (± 1.6) years. All patients presented with proteinuria (> 0.5 g/24 hours) and 30 (41.1%) with hematuria. Renal function was impaired in 16 cases (21.9%). Hypoalbuminemia (serum albumin < 30 g/l) was shown in 65 cases (89.0%).

Study design and patients. In addition to biopsy-proven lupus nephritis, the patients met the American College of Rheumatology 1997 revised criteria for the diagnosis of SLE[13] and had evidence of active renal involvement (proteinuria, hematuria, or increasing serum creatinine level). The cases of biopsy-proven lupus nephritis were identified at the Department of Rheumatology, the First Municipal People's Hospital and the First Affiliated Hospital of Sun Yat-Sen University, Guangzhou, China, between June 2002 and December 2006. The study group included incipient patients, regarded as having no prescription of corticosteroids and/or immunosuppressants before the entry point, and recurrent patients, regarded as having a prescription of prednisone ≤ 10 mg/day and no immunosuppressants for at least 3 months but with a treatment history. Patients taking large doses of corticosteroids and immunosuppressants within 3 months, patients with overlap syndrome (coexistence of lupus with other connective tissue diseases), and patients with other renal diseases (such as diabetic nephropathy, arteriolar nephrosclerosis, etc.) were excluded. Patients were evaluated both clinically and biochemically at least once a month.

Table 1. Demographic and clinical characteristics of patients with lupus nephritis at baseline.

Characteristic	Study Subjects, n (%), or mean ± SD
Women	65 (89)
Age, yrs	31.0 ± 13.8
SLE duration, yrs	2.3 ± 1.6
Incipient cases	49 (67.1)
Disease activity (SLEDAI)	12.1 ± 1.8
Renal manifestations	
Proteinuria	73 (100)
Hematuria	30 (41.1)
Elevation of serum creatinine	16 (21.9)
Hypoalbuminemia	65 (89.0)
Extrarenal manifestations	
Fever	32 (43.8)
Arthritis/arthralgia	44 (60.3)
Malar rash	23 (31.5)
Polyserositis	19 (26.0)
Hematologic disorders	28 (38.4)
Neurologic disorders	2 (2.7)

SLE: systemic lupus erythematosus; SLEDAI: SLE Disease Activity Index.

Sera samples from 73 patients with biopsy-proven lupus nephritis were taken within 7 days before renal biopsy, and at the end of the first and the third month after treatment. As controls, 40 patients with active SLE without clinical evidence of renal involvement and 40 healthy donors matched for gender and age were included at the beginning of the study.

All data went into the prospective cohort study. A cross- sectional study analyzed the relationship between serum anti-C1q antibody levels and renal histopathology/nephritic activity. A longitudinal study evaluated the prognostic significance of anti-C1q antibody in lupus nephritis. Our study was approved by the hospital research ethics committee (No. 2002013), and informed consent was obtained from all patients.

Determination of serum anti-C1q antibody level. Aliquots of sera were stored at –70°C until needed. Serum anti-C1q antibody levels were tested using enzyme-linked immunosorbent assay (ELISA) kits according to the assay procedure (IMTEC Immundiagnostika GmbH, Berlin, Germany). The test is based on the immobilization of the C1q to a solid phase (polystyrene) that has been chemically activated and the subsequent binding of the anti-C1q antibodies from patient serum. The bound antibodies are detected with a peroxidase-labeled secondary antibody that is directed against human IgG. Briefly, C1q-coated microtiter strips (IMTEC) were used. Sera at room temperature (30 min) were diluted 1:100 with sample buffer. Serum dilution at 100 μl or undiluted standard (inked according to rising concentration) or control serum were added into each well, sealed, and incubated for 1 hour at room temperature. The wells were then rinsed 3 times with washing buffer. As a secondary antibody, 100 μl horseradish peroxidase conjugate was pipetted into each well and sealed. After 30 min incubation, the wells were washed 3 times. Then 100 μl trimethylbenzene solution was pipetted into each well and incubated in the dark. Finally, 100 μl stopping solution was pipetted into each well. Absorbence was read at 450 nm within 30 min. The cutoff for a positive test result was 20 U/ml, which was determined by the manufacturer.

Evaluation of renal involvement. Percutaneous renal biopsy was performed at the beginning of the study. The World Health Organization (WHO) classification of lupus nephritis[14] was used to define the histological lesions, and mixed forms were classified in keeping with the identified proliferative lesion. In addition to the histological classification, lupus nephritis activity was scored according to activity and chronicity indices of the US National Institutes of Health (NIH) reports[15]. The activity index (from 1 to 24)[15] was obtained by adding the scores attributed to endocapillary hypercellularity, glomerular leukocyte infiltration, hyaline deposits, fibrinoid necrosis, interstitial inflammation, and cellular or fibrocellular crescents. The chronicity index (from 0 to 12)[15] was calculated by scoring the following measurements: glomerular sclerosis, fibrous crescents, interstitial fibrosis, and tubular atrophy. The presence of glomerular C1q, C3, IgG, IgA, and IgM was also examined in immunofluorescent microscopy. All histopathology was subsequently reevaluated by a single senior pathologist.

Clinical and other laboratory data. Clinical and other laboratory features including clinical manifestations, urinalysis, 24-hour urinary protein excretion, serum creatinine level, erythrocyte sedimentation rate (ESR), C3, C4, IgG, IgA, IgM, anti-dsDNA antibodies, and more were recorded. Disease activity (both renal and extrarenal) was evaluated using the Systemic Lupus Erythematosus Disease Activity Index (SLEDAI)[16].

Therapy. The therapy for individual patients was determined by disease activity and renal histopathology. The medications mainly included corticosteroids, cyclophosphamide, mycophenolate mofetil, and azathioprine. All patients were given oral glucocorticoid. Prednisone dosage varied from 0.5 to 1.0 mg/kg of body weight. Patients with crescentic glomerulonephritis or severe extrarenal diseases were given intravenous pulse methylprednisolone for 3 consecutive days. Some of the patients with class II lupus nephritis were given combined azathioprine (50–100 mg per day). Patients with class III or IV lupus nephritis were generally treated with intravenous cyclophosphamide at a dosage of 0.5–1 g/m^2 of body surface area month-

ly, or oral mycophenolate mofetil at a dosage of 1.5–2.0 g/day. Patients with class V lupus nephritis were given combined cyclophosphamide, mycophenolate mofetil, or azathioprine if glucocorticoid alone was inadequate. Treatment protocols were adjusted based on clinical response.

Followup and outcome measures. Each patient was followed up every month. During every visit, signs and symptoms and changes in drug treatment were recorded. Routine laboratory tests were performed. Levels of serum anti-C1q antibody were remeasured at the end of the first and the third month after active treatment.

Endpoint assessment. The primary endpoint was the disappearance of proteinuria, defined as proteinuria reduced to ≤ 0.3 g/day[17]. The secondary endpoint was complete remission of lupus nephritis, according to Chan, *et al*[17] with slight modification: 24-hour urinary protein excretion ≤ 0.3 g, inactive urinary sediment (absence of hematuria, < 5 red blood cells/high power field, and cellular casts), and normal serum creatinine and serum albumin levels.

The treatment and followup of patients and the measurement of anti-C1q antibody were conducted by specialists and technicians who were blinded to the study procedures.

Statistical analyses. Bivariate correlations between variables in cross-sectional analyses were determined using Spearman's correlation coefficient. Means and SD between 2 groups were calculated and compared using Student's unpaired t-test. The chi-squared test and Fisher's exact test were employed to assess the statistical relationship between 2 proportions. Comparisons between serum anti-C1q antibody levels in each group of WHO classification of lupus nephritis were determined using ANOVA. In the longitudinal study, comparison of the time to disappearance of proteinuria was calculated by the Kaplan-Meier method and log-rank test. The influence factors for complete remission of lupus nephritis were assessed using Cox's proportional hazards regression model. All data were analyzed using Stata 7.0. Statistical significance required $p < 0.05$.

RESULTS

Prevalence and level of serum anti-C1q antibody. Fifty-eight of 73 (79.5%) patients with lupus nephritis and 11 of 40 (27.5%) patients with nonrenal SLE were positive for anti-C1q antibody, and the 40 healthy controls were negative. The mean serum anti-C1q antibody level in lupus nephritis (95.3 ± 55.2 U/ml) was much higher than that in nonrenal SLE (26.0 ± 11.7 U/ml; $p < 0.001$; Figure 1).

The patients with lupus nephritis with positive anti-C1q antibody were subdivided into those with high levels (≥ 100 U/ml) and those with low levels (< 100 U/ml). Comparison of disease activity and extrarenal clinical manifestations including fever, arthritis/arthralgia, malar rash, polyserositis, and hematologic and neurologic disorders showed no statistical difference between the 2 groups except for SLEDAI scores (13.5 ± 1.2 vs 11.6 ± 1.7, respectively; $p < 0.001$).

Association between serum anti-C1q antibody and renal pathological characteristics. Based on the WHO classification of lupus nephritis, 12 cases were classified as WHO class II, 19 as class III, 26 as class IV, and 16 as class V. Serum anti-C1q antibody difference between each class was measured by ANOVA; class IV was the highest ($p < 0.001$ compared to class II and $p = 0.008$ compared to class V), followed by class III ($p = 0.024$ compared to class II; Table 2).

Immunofluorescence revealed glomerular C1q, C3, IgG, IgA, and IgM deposition in biopsy specimens. Sixty-four (87.7%) patients had a positive staining for glomerular C1q. Serum anti-C1q antibody was positively associated with glomerular C1q deposition (serum anti-C1q antibody with glomerular C1q ≥ ++ compared to C1q ±; $p < 0.001$; Table 3).

Lupus nephritis activity was scored according to the activity index and chronicity index of renal histopathology. Serum anti-C1q antibody level was found to be associated with activity index ($r = 0.555$, $p < 0.001$) and chronicity index ($r = 0.390$, $p < 0.001$). The presence of anti-C1q antibodies was also positively correlated with glomerular leuko-

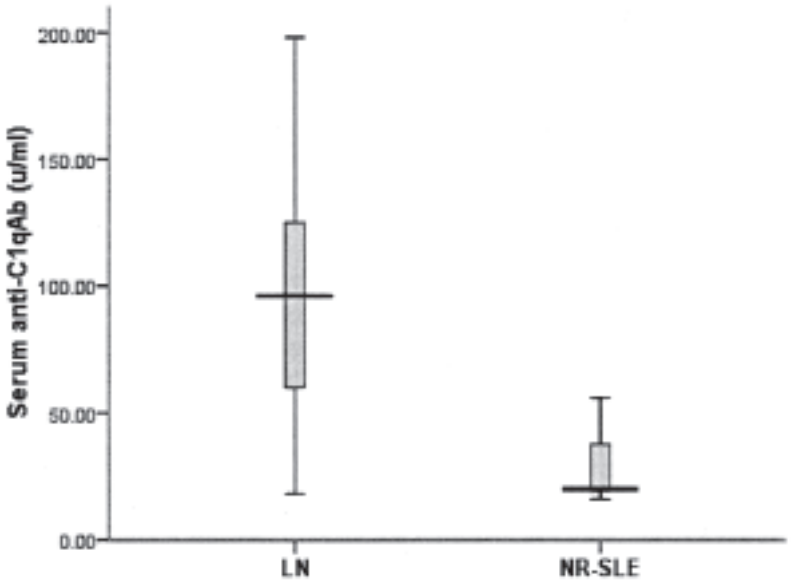

Figure 1. Mean levels of serum anti-C1q antibody in patients with lupus nephritis (LN, n = 73) are higher than in patients with nonrenal SLE (NR-SLE, n = 40; $p < 0.001$). The graph shows maximum, minimum, mean, and SD values.

Table 2. Correlation between serum anti-Clq antibody levels and histological lesions in renal biopsies of lupus nephritis.

WHO Classification of Lupus Nephritis	No. of Patients (%)	Levels of Serum anti-Clq antibody, U/ml
II	12 (16.4)	45.8 ± 32.9
III	19 (26.0)	98.1 ± 39.7**
IV	26 (35.7)	127.6 ± 56.8*
V	16 (21.9)	76.7 ± 49.1

ANOVA test: * $p < 0.001$ compared with WHO class II and $p = 0.008$ compared with WHO class V lupus nephritis. ** $p = 0.024$ compared with WHO class II lupus nephritis.

Table 3. Correlation between serum anti-Clq antibody levels and glomerular deposition of Clq in biopsy specimens.

Glomerular Deposition of Clq	No. of Patients, (%)	Levels of Serum anti-Clq antibody, U/ml
Clq +/–	19 (26.0)	46.4 ± 40.1
Clq ≥ ++	54 (74.0)	112.5 ± 49.4*

* Student unpaired t-test: $p < 0.001$.

cyte infiltration ($r = 0.316$, $p = 0.006$), endocapillary hypercellularity ($r = 0.324$, $p = 0.005$), fibrinoid necrosis ($r = 0.344$, $p = 0.003$), and formation of cellular/fibrocellular crescents ($r = 0.448$, $p < 0.001$) or fibrous crescents ($r = 0.499$, $p < 0.001$; Table 4).

In the cross-sectional analysis of the association between serum anti-C1q antibody and proteinuria, C3, C4, and SLEDAI, proteinuria generally represented the lupus nephritis lesion entity. C3, C4, and SLEDAI determined disease activity. Serum anti-C1q antibody was positively correlated to 24-h urinary protein excretion ($r = 0.419$, $p < 0.001$) and SLEDAI ($r = 0.792$, $p < 0.001$), but negatively correlated to serum C3 ($r = –0.639$, $p < 0.001$) and C4 ($r = –0.519$, $p < 0.001$). No significant correlation was found with serum creatinine (Table 5).

Longitudinal analysis: prognostic value of serum anti-C1q antibody measurements. Treatment was determined by disease activity and renal histopathology. After active treatment

Table 4. Correlations between serum anti-Clq antibody and renal histopathologic measurements.

Histopathologic Measurements	r_s	p
Activity indices	0.555	< 0.001
Glomerular leukocyte infiltration	0.316	0.006
Endocapillary hypercellularity	0.324	0.005
Fibrinoid necrosis	0.344	0.003
Cellular or fibrocellular crescents	0.448	< 0.001
Chronicity indices	0.390	< 0.001
Fibrous crescents	0.499	< 0.001

r_s: Spearman rho.

Table 5. Correlations between serum anti-Clq antibody levels and other laboratory data.

Laboratory Data	r_s	p
24-hour urinary protein excretion	0.419	< 0.001
SLEDAI	0.792	< 0.001
C3	–0.639	< 0.001
C4	–0.519	< 0.001

r_s: Spearman rho; SLEDAI: Systemic Lupus Erythematosus Disease Activity Index.

for 3 months, a marked decrease of anti-C1q antibody levels (the extent of reduction exceeding 50%) or being negative was found in 41 of 73 (56.2%) patients. These patients were classified as group 1, while the others were classified as group 2, including the patients with persistent high levels of serum anti-C1q antibody and the patients with increased titers (from negative to positive or from low levels to high levels) after treatment.

Routine examinations were done at least once a month. Regarding proteinuria, the Kaplan-Meier method estimate and the log-rank test established the significant difference between patients in group 1 and group 2. The median time of disappearance of proteinuria as the primary endpoint was longer in patients with persistent high levels or increased titers of serum anti-C1q antibody after treatment (chi-squared = 9.58, $p = 0.002$; Figure 2).

In the longitudinal study, another endpoint was the time to achievement of complete remission of lupus nephritis. To determine the prognostic factors of remission of lupus nephritis, a Cox's proportional hazards regression model was created. The following were used as input variables in a repeated measures mixed model: age, sex, course of disease, C3, C4, IgG, IgA, IgM, ESR, 24-h urinary protein excretion, hematuria, serum creatinine, activity index, chronicity index, anti-dsDNA antibody, serum anti-C1q antibody levels at baseline and at the end of the first and third months after treatment, SLEDAI, WHO classification of lupus nephritis, and cyclophosphamide therapy. In multivariate analysis, among the clinical, laboratory, and histological features, only WHO classification, chronicity index, serum anti-C1q antibody levels at the end of the third month after treatment, and cyclophosphamide therapy were significantly associated with the endpoint. Severe histological forms of lupus nephritis (WHO classes IV and V), high levels of serum anti-C1q antibody at the end of the third month after treatment, and high chronicity index were found to prevent complete remission of lupus nephritis. Cyclophosphamide therapy was a protecting factor that promoted remission of lupus nephritis. Levels of serum anti-C1q antibody at baseline and the end of the first and third months after treatment were all associated with remission of lupus nephritis in the univariate analysis, but only the level of serum anti-C1q antibody at the end of the third month after treatment was a

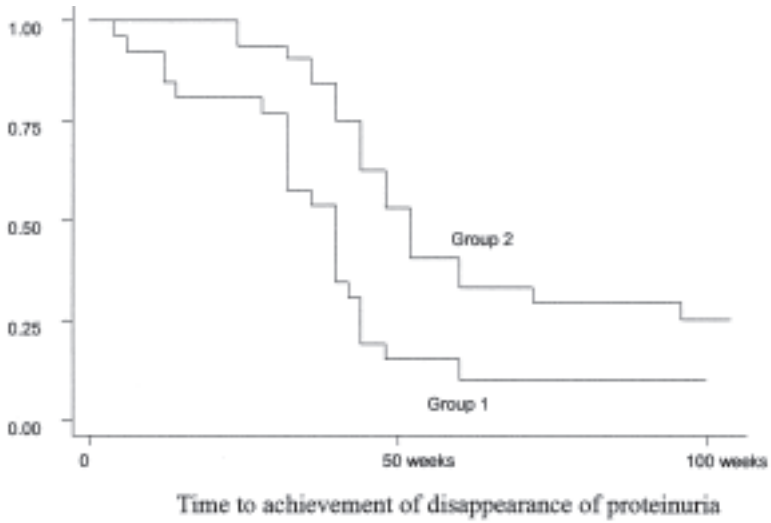

Figure 2. Comparison of the time to disappearance of proteinuria shows significant difference between 2 groups by Kaplan-Meier estimate and log-rank test (chi-squared = 9.58, p = 0.002). Group 1: Patients with marked decrease of anti-C1q antibody levels (the extent of reduction exceeding 50% or being negative) at the end of the third month after treatment. Group 2: Patients with persistent high levels or increased titers of anti-C1q antibody after treatment for 3 months.

stronger prognostic factor after adjustment in the Cox proportional hazards regression model. The crude and adjusted hazard ratios of variables associated with complete remission of lupus nephritis are shown in Table 6.

DISCUSSION

Autoantibodies against C1q, the first component of the classical pathway of complement, have stirred much interest[18-20]. Earlier investigations suggested that anti-C1q antibodies are found in patients with SLE, particularly those with renal involvement, and development or recurrence of nephritis was associated with rising titers of anti-C1q antibody in the preceding 6 months[5]. From a clinical point of view, anti-C1q antibody might be an important biomarker in lupus nephritis. This possibility led us to further explore the potential role of the anti-C1q antibody in the evaluation of renal lesion and its prognostic significance in lupus nephritis.

A prevalence of 79.5% of anti-C1q antibody was found in patients with active lupus nephritis, higher than in Sinico, *et al*[21] (60%) and Fang, *et al*[22] (56%). High levels of serum anti-C1q antibody were correlated with proliferative lupus nephritis. Levels of anti-C1q antibody in WHO class IV and III were much higher than in class II. Although the difference between class III and V was not significant, a high level of anti-C1q antibody may be a reliable predictor of proliferative lupus nephritis. Serum anti-C1q antibody levels were found to be associated with both activity index and chronicity index. Further, anti-C1q antibodies were capable of indicating histopathological damage such as glomerular leukocyte infiltration, endocapillary hypercellu-

Table 6. Crude and adjusted hazard ratios of variables associated with complete remission of lupus nephritis.

	Crude Hazard Ratios (95% CI)	p	Adjusted Hazard Ratios* (95% CI)	p
Serum anti-Clq antibody levels (100 U/ml)				
At baseline	0.398 (0.230–0.689)	0.001	—	NS
At 1st month after treatment	0.246 (0.126–0.481)	< 0.001	—	NS
At 3rd month after treatment	0.242 (0.121–0.483)	< 0.001	0.229 (0.103–0.513)	< 0.001
WHO classification				
III	0.447 (0.202–0.987)	0.046	0.379 (0.129–1.115)	0.078
IV	0.158 (0.068–0.368)	< 0.001	0.176 (0.061–0.507)	0.001
V	0.146 (0.056–0.377)	< 0.001	0.109 (0.037–0.322)	< 0.001
Chronicity index	0.524 (0.410–0.669)	< 0.001	0.590 (0.445–0.783)	< 0.001
Cyclophosphamide	2.806 (1.531–5.143)	0.001	2.441 (1.179–5.054)	0.016

* Adjusted with Cox proportional hazards regression model. WHO: World Health Organization.

larity, fibrinoid necrosis, and crescent lesions (cellular/fibrocellular and fibrous crescents). A similar observation that showed a causal link between anti-C1q antibody and target organ damage was also made in murine SLE[7,8]. These data suggest that in patients with lupus nephritis, anti-C1q antibody measurement could serve as a noninvasive tool for assessing renal lesions in lupus nephritis.

Proteinuria is the main mode of expression of lupus nephritis. Moroni, *et al*[5] reported that titers of anti-C1q antibody were significantly greater during flares of renal activity than in quiescent disease, and titers were elevated in all cases of proteinuric flares and in only two-thirds of the cases of nephritic flares. We demonstrated a serum anti-C1q antibody level strongly correlated to proteinuria. Further, the changes of serum anti-C1q antibody levels after treatment appeared to be more important in determining outcome than their initial levels. The longer the serum anti-C1q antibody level remained with high or increased titer, the less likely that proteinuria would recess after treatment. This suggests that patients with lupus nephritis with persistently high levels or increased titer of anti-C1q antibody should be prescribed intensive treatment, such as induction therapy of high-dose immunosuppressants or rituximab.

Serum anti-C1q antibody levels increased significantly with more C1q deposition in glomerular basement membrane. C1q is deposited in the glomerular basement membrane as part of the immune complexes. However, with respect to all the phenomena discovered, how anti-C1q antibody contributes to the development of lupus nephritis remains puzzling. Anti-C1q antibodies were detected in numerous autoimmune diseases such as Felty's syndrome, rheumatoid vasculitis, mixed connective tissue disease, primary Sjögren's syndrome, and other diseases such as hepatitis C virus infection, and even in healthy controls[23,24]. The presence of anti-C1q antibodies is not absolutely specific for and not necessary for glomerulonephritis. In one study[25], anti-C1q antibodies could be isolated from glomerular basement fragments of patients with proliferative lupus nephritis, and the deposition seemed to occur through binding to deposited C1q. Interestingly, administration of anti-C1q antibodies to naive mice resulted in glomerular deposition of C1q and anti-C1q antibodies but not in overt renal damage. However, the combination of anti-C1q antibodies and C1q-fixing antiglomerular basement membrane antibodies enhanced renal disease[8]. Trouw, *et al*[8,23] showed that anti-C1q antibodies could be pathogenic to the kidney but only in the context of C1q-containing glomerular immune complexes, as found in SLE. Anti-C1q antibodies might attenuate the physiological functions of C1q, including the capacity to activate the classical pathway of complement and to clear immune complexes and apoptotic bodies[26]. As well, anti-C1q was not strongly correlated to nonrenal manifestations regardless of disease activity.

In order to determine the prognostic value of serum anti-C1q antibody measurement, we observed serum anti-C1q antibody levels and other possible factors associated with the complete remission of lupus nephritis, in longterm followup. When the anti-C1q antibody was detected before and after treatment and put into univariate analysis and a Cox's proportional hazards regression model together with other measurements, we found that a persistent high level of anti-C1q antibody was a risk factor for nephritis progression, and after successful treatment, serum anti-C1q antibody had a tendency to decrease. Anti-C1q antibody consuming the early component of the classical pathway C1q may be related to secondary hypocomplementemia in active SLE. Its prolonged existence may amplify complement activation, resulting in a vicious cycle. Thus, serial determinations of serum anti-C1q antibody would identify treatment responders and nonresponders, and combined with monitoring of other clinical measurements would help to determine prognosis and further treatment strategy.

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