

Significance of Antiphospholipid Antibodies in Lupus Nephritis

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Background: Some autoantibodies have been associated with lupus nephritis but the role of antiphospholipid antibodies (APA) is controversial.

Objective: The present study was to explore the role of APA by comparing demographic profiles and the presence of anticardiolipin antibody (aCL) and lupus anticoagulant (LA) in systemic lupus erythematosus (SLE) patients with and without nephritis.

Material and Method: The cross-sectional study in a tertiary center was conducted in 77 SLE patients. All patients attended our renal or rheumatology clinics between June 2002 and December 2003.

Results: Sixty-three (82%) of the 77 SLE patients had nephritis. The prevalence of antiphospholipid syndrome (APS) was 10% (8 patients), positive aCL (IgG) was 26% (20 patients) and positive LA was 26% (20 patients). The receiver operating characteristic (ROC) method was applied to assess the significance of aCL in both nephritis and non-nephritis groups. Area under the ROC curve was 0.538 (95%CI 0.312-0.765), a cutoff value of 20.5 GPL had a sensitivity of 75% and a specificity of 53%. In univariate analysis, neither positivity for anticardiolipin antibody nor lupus anticoagulant was associated with lupus nephritis. Analyzed in only the lupus nephritis group, LA-positive lupus nephritis patients had higher systolic blood pressure (SBP) (133.7 vs 121.9 mmHg, $p = 0.005$), lower platelet count (209.8 vs $264.4 \times 10^3/\mu\text{L}$, $p = 0.02$) and higher 24-hr urine protein excretion (2.6 vs 1.4 g, $p = 0.02$) than LA-negative lupus nephritis patients. Serum creatinine was higher in LA-positive lupus nephritis than LA-negative (233.0 vs $94.9 \mu\text{mol/L}$), but did not reach statistical significance.

Conclusion: APA are frequently seen in SLE patients, but not associated with lupus nephritis. However, lupus anticoagulant tends to associate with lupus nephritis. Detection of LA in lupus nephritis patients could identify patients who had increased risk to develop bad renal outcomes (elevated SBP and 24-hr urine protein excretion).

Keywords: Antiphospholipid antibodies, Lupus nephritis, Antiphospholipid syndrome

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Anti phospholipid antibodies (APA) are autoantibodies that have activity directed against cardiolipin and are negatively charged phospholipids. They are composed of anticardiolipin antibody (aCL) and lupus anticoagulant (LA). Evidence suggests that the target antigens are plasma proteins such as b₂ glycoprotein I (b₂-GPI) and prothrombin.

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These antibodies have been described to be associated with systemic lupus erythematosus (SLE) patients. The frequency of aCL and LA ranges from 43 to 77% and 7 to 65% respectively⁽¹⁾. Love and Santoro reported an average frequency of 44% for aCL and 34% for LA in the analysis of 29 published series⁽²⁾. Recently the true significance of these antibodies in SLE patients in terms of clinical evident nephritis is still controversial. Some studies demonstrated that the presence of APA in SLE patients was strongly associated with nephritis^(3,4), with more prevalence and

increased in severity of disease⁽⁵⁻⁷⁾. Conflicting results that shows no association between APA and renal histological pattern, or long term renal outcome have been reported in some studies^(8,9).

The present study was to analyze the association between the positivity of either aCL or LA and the appearance of lupus nephritis. The authors also examined the correlation between the presence of APA and clinical and laboratory profiles in SLE patients.

Material and Method

A cross-sectional study was conducted in 77 SLE patients, all of them met the revised criteria of the American College of Rheumatology⁽¹⁰⁾.

All patients attended the Renal or Rheumatology Clinics, Phramongkutklao Hospital between June 2002 and December 2003 and underwent clinical and laboratory assessment. This included complete blood count, urinary protein quantitation, serum Creatinine (Cr), complement levels (C3, C4), AntiNuclear Antibody (ANA) profiles and APA.

Clinical Data

The data of the 77 patients were recorded in a standard collection form. The clinical data of interest were thrombosis of either arterial or venous side including myocardial infarct, stroke, transient ischemic attack, visceral organ vasculitis, pulmonary embolism and deep vein thrombosis.

Venous thrombosis events were documented by ultrasonography and/or venography and/or magnetic resonance venography. Arterial thrombosis events were documented by angiography and/or magnetic resonance angiography and/or computerized tomography.

Pregnancy events were defined as having at least one abortion or premature births (< 34 weeks).

Antiphospholipid Syndrome

Definite antiphospholipid syndrome (APS) was defined by the association of arterial and/or venous thrombosis or obstetrical fetal loss (repeated miscarriages or fetal death) with the presence of LA and/or moderate to high level of aCL, according to the preliminary classification criteria of International Consensus Statement⁽¹¹⁾.

Anticardiolipin Antibody Assay

The conventional enzyme-linked immunosorbent assay (ELISA) using commercial kit (DiaSorin) was used to detect IgG aCL.

Lupus Anticoagulant Assay

The laboratory criteria for LA were followed as described by the Subcommittee on Lupus Anticoagulant Antiphospholipid Antibody of the Scientific and Standardization Committee of the ISTH⁽¹²⁾. The four screening assays used to enhance the overall sensitivity to LA were activated in partial thromboplastin time, kaolin clotting time, tissue thromboplastin inhibitor test and diluted Russell viper venom time. The presence of an inhibitor was documented by the persistence of an abnormal clotting time after mixing the patients' plasma with normal plasma in a ratio of 1:1. After that the authors performed the confirmation test that demonstrated the evidence of phospholipid dependence by platelet neutralization procedure. This corresponded to the abnormal screening test.

Statistical Analysis

Descriptive statistics (median, mean \pm SD) for continuous variables were applied. Patient groups were compared using Chi-square analysis for categorical data and when cell numbers were less than five, Fisher's exact test was used instead. Unpaired t- test was used to analyze continuous variable and Mann-Whitney U test for non-normal distribution.

Diagnostic accuracy of aCL was estimated by calculating the receiver operating characteristic (ROC) area under the curve and classified aCL-positive patients if aCL > 20.5 GPL.

Results

Patients' characteristics are summarized in Table 1. Of the 77 SLE patients, 63 (82%) had nephritis, and 14 (18%) were without nephritis. Their mean ages were 36.5 and 36.4 years old, respectively. The mean duration of disease was longer in SLE with lupus nephritis group but did not reach statistical significance.

The continuous scale used for reporting aCL measurements made it possible to be analyzed by ROC curve analysis. The area under the ROC curve was 0.538 (95%CI 0.312-0.765) as in Fig. 1. A cutoff value of 20.5 GPL had a sensitivity of 75% and a specificity of 53% and aCL level > 20.5 GPL was used to define positivity of IgG aCL.

The prevalence of aCL-positivity in SLE patients was 26% (20/77). The authors found no correlation between the aCL and lupus nephritis ($p = 0.276$). This corresponded to area under the ROC curve that could not be distinguished between SLE patients with and without nephritis. The prevalence of LA positivity in SLE patients was 26% (20 patients). There were 19 SLE patients with nephritis and only one patient without

Table 1. The demographics of the two groups of SLE patients according to the presence of lupus nephritis (LN) (N = 77)

Characteristics	SLE with LN n = 63 (82%)	SLE without LN n = 14 (18%)	p-value
Age (yr) (mean) \pm SD	36.4 \pm 11.0	36.5 \pm 14.1	0.99
Duration of disease (month) (mean) \pm SD	77.5 \pm 78.5	51.0 \pm 87.0	0.31
Gender			
female (%)	60 (95.2)	14 (100.0)	1.00
male (%)	3 (4.8)	0	
Current smoking (%)	2 (3.2)	0	1.00
Contraception use (%)	3 (4.8)	1 (7.7)	0.53
Hypertension (%)	25 (39.7)	2 (14.3)	0.12
Diabetes (%)	4 (6.3)	0	1.00
Aspirin usage (%)	2 (3.2)	1 (7.1)	0.45
Warfarin usage (%)	7 (11.1)	3 (21.4)	0.37

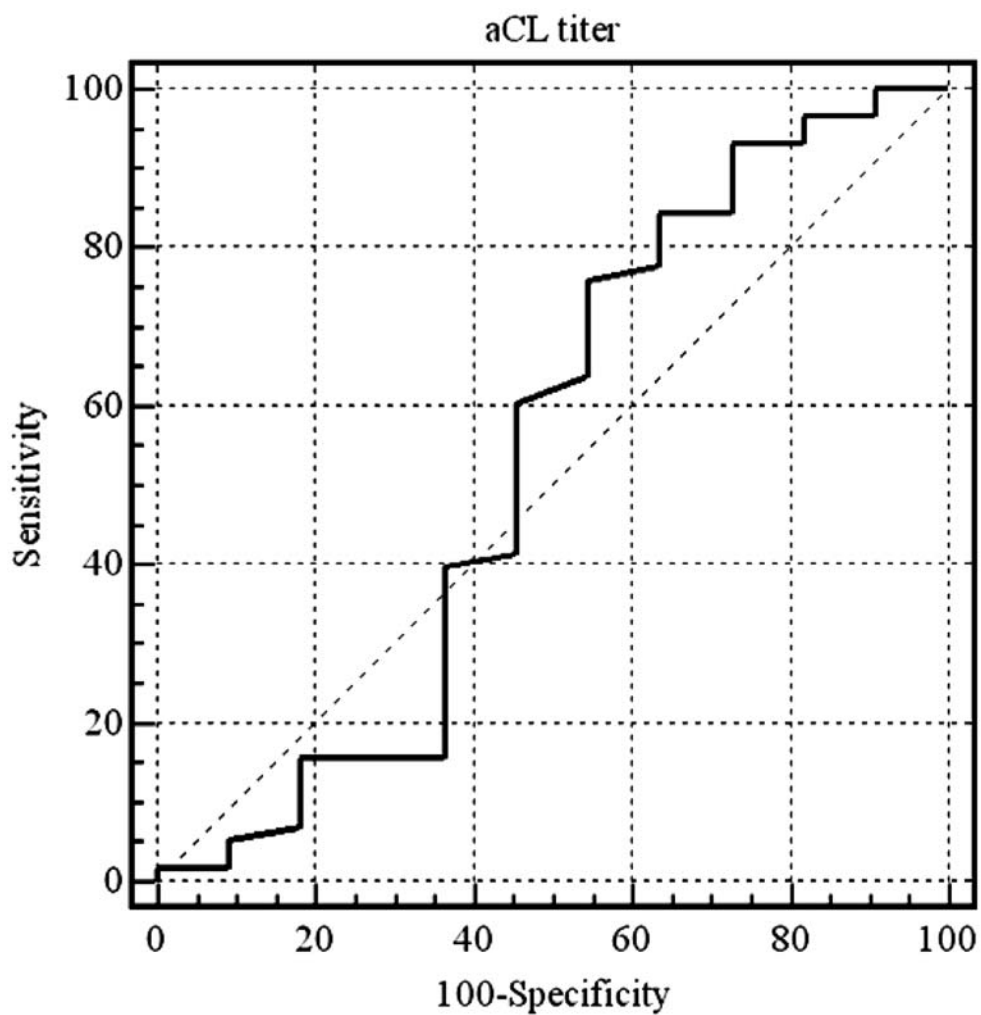


Fig. 1 The ROC curve graphically represents the relationship between sensitivity and specificity for all aCL values

nephritis. The majority of positivity of LA test was abnormal kaolin clotting time (data not shown). However, lupus anticoagulant tended to be correlated with lupus nephritis ($p = 0.095$). Laboratory features of SLE patients with and without lupus nephritis are shown in Table 2.

The prevalence of APS was 10% (8/77), five in SLE patients with nephritis and three in SLE patients

without nephritis. There was no difference in the clinical features of APS between the two groups of patients (Table 3). Despite categorizing the patients into aCL-positive or LA-positive (APA-positive) patients to determine the high risk group and to develop clinical spectrum of APS, there was no significant difference of thrombosis events between APA-positive patients and APA-negative patients (Table 4).

Table 2. Laboratory features of systemic lupus erythematosus patients with and without lupus nephritis (N = 77)

	SLE with LN (n = 63)	SLE without LN (n = 14)	p-value
Systolic blood pressure (mmHg)	126 (15.7)	111.8 (8.7)	0.005
Diastolic blood pressure (mmHg)	78.3 (9.8)	73.0 (8.0)	0.10
Hemoglobin (g/dL)	11.6 (2.7)	11.4 (2.2)	0.78
White blood count (* 10 ⁹ /L)	7.13 (3.8)	6.58 (2.9)	0.58
Platelet (* 10 ³ /μL)	246 (85.0)	229 (18)	0.67
Serum Creatinine (mmol/L)	135 (22.6)	66 (16)	0.02
24 hr urine protein (g)	1.63 (1.9)	0.28 (0.2)	0.02
C3 (g/L)	1.79 (7.4)	0.37 (0.4)	0.36
C4 (g/L)	0.22 (0.2)	0.29 (0.2)	0.28
aCL titer (GPL)	20.69 (21.7)	32.89 (40.1)	0.28
Positivity of aCL	15 (25.9%)	5 (45.5%)	0.27
Positivity of lupus anticoagulant	19 (31.1%)	1 (7.1%)	0.095
Positivity of anti dsDNA	18 (30.0%)	9 (64.3%)	0.02
Prolonged aPTT	18 (30.0%)	9 (64.3%)	0.47

Note: Number in parentheses is standard deviation

Table 3. Clinical features of antiphospholipid syndrome in systemic lupus erythematosus patients with and without lupus nephritis (N = 77)

Clinical-related Antiphospholipid Syndrome	SLE with LN (n = 63) (%)	SLE without LN (n = 14) (%)	p-value
All thrombosis	9 (14.3)	4 (28.6)	0.23
Arterial thrombosis	3 (4.8)	2 (14.3)	0.22
Venous thrombosis	7 (11.1)	2 (14.3)	0.66
Pregnancy events	6 (9.5)	2 (14.3)	0.63
Antiphospholipid syndrome	5 (7.9)	3 (21.4)	0.11

Table 4. Clinical features of antiphospholipid syndrome in systemic lupus erythematosus patients with aCL or LA (+) and with aCL and LA (-) (N = 77)

Clinical-related Antiphospholipid Syndrome	aCL or LA (+) (n = 35) (%)	aCL and LA (-) (n = 42) (%)	p-value
All thrombosis	8 (22.8)	5 (11.9)	0.20
Arterial thrombosis	4 (11.4)	1 (2.3)	0.17
Venous thrombosis	5 (14.2)	4 (9.5)	0.72
Pregnancy events	4 (11.4)	4 (9.5)	1.00

The majority of LA-positive patients in the present study were in lupus nephritis group. The authors then focused only in 63 lupus nephritis patients who had a positivity of LA. The authors excluded two patients who had clinical thrombosis and were taking warfarin at the time of the LA test thus their results could not be interpreted. The authors found that LA-positive lupus nephritis patients had higher systolic blood pressure (SBP) (133.7 vs 121.9 mmHg, $p = 0.005$), lower platelet count (209.8 vs $264.4 \times 10^3/\mu\text{L}$, $p = 0.02$) and higher 24-hr urine protein excretion (2.6 vs 1.4 g, $p = 0.02$) than LA-negative lupus nephritis patients. Serum creatinine was higher in LA-positive lupus nephritis than LA-negative (233.0 vs 94.9 $\mu\text{mol/L}$), but did not reach statistical significance ($p = 0.15$) (Table 5).

The present study did not show the impact of aCL- positivity in lupus nephritis patients as it was not an independent risk factor that contributed to poor renal outcome (data not shown). Among 63 lupus nephritis patients, there were only 56 patients who had both aCL and LA tests performed. Patients who had either aCL- positivity or LA- positivity were three-fold more likely to develop any clinical thrombosis than patients who had aCL and LA-negative tests (18.5% vs 6.9%), although it did not reach statistical significance ($p = 0.24$) (Table 6).

Discussion

The present study demonstrated that the prevalence of aCL- positivity and LA-positivity in Thai SLE patients were 26% each. This is less than that reported number in the literature of Love and Santoro⁽²⁾. It could be explained by the limitation of aCL test that was done only for IgG antibody, not including IgM antibody of the present study. Because of the diversity of assay used to define the anticardiolipin positivity, the authors used ROC analysis and defined patients as aCL- positive SLE patients when aCL titer was more than 20.5 GPL. However, the area under the ROC curve did not predict the risk of developing nephritis in the presented SLE patients. The result was shown to be contradictory to the study of Loizou et al, who suggested that the presence of aCL was associated with lupus nephritis⁽³⁾. The study in terms of anticardiolipin and renal morphology from the United Kingdom⁽⁹⁾ reported 29% of lupus nephritis patients had elevated IgG aCL and there was an association between IgG aCL and glomerular thrombi but not with renal histologic pattern or long-term renal outcome.

The prevalence of APA either positive IgG aCL or LA was 45.5% (35/77). The prevalence of APS was 10% (8/77) in the present study. After the authors categorized the patients into either positivity of aCL or LA to determine the high risk group to develop throm-

Table 5. Clinical and laboratory features of lupus nephritis patients with LA-positive compared to LA-negative (N = 61)

	LA + (n = 19)	LA - (n = 42)	p-value
Systolic blood pressure (mmHg)	133.4 (15.8)	121.9 (14.4)	0.005
Diastolic blood pressure (mmHg)	79.5 (11.3)	77.4 (11.4)	0.44
Hemoglobin (g/dL)	11.7 (4.4)	11.7 (1.8)	0.98
White blood count ($10^9/\text{L}$)	6.80 (2.52)	6.85 (3.33)	0.95
Platelet ($10^3/\mu\text{L}$)	209.8 (67.3)	264.4 (88.7)	0.02*
Serum Creatinine ($\mu\text{mol/L}$)	233.0 (365.5)	94.9 (52.3)	0.15
24 hr urine protein (gm)	2.6 (1.7)	1.4 (1.6)	0.02*

Table 6. Clinical features of antiphospholipid syndrome in lupus nephritis patients with aCL or LA-positive, compared to aCL and LA-negative (N = 56)

Clinical - related Antiphospholipid Syndrome	aCL or LA (+) (n = 27)	aCL and LA (-) (n = 29)	p-value
All thrombosis	5 (18.5)	2 (6.9)	0.24
Arterial thrombosis	2 (7.4)	1 (3.4)	0.61
Venous Thrombosis	4 (14.8)	1 (3.4)	0.18
Pregnancy events	3 (11.1)	2 (6.8)	0.66
Antiphospholipid syndrome	4 (14.8)	1 (3.4)	0.19

bosis events, there was no correlation between positivity of aCL or LA and clinical features of APS. Some studies have shown the presence of aCL conveys a risk factor for thrombosis and hence a risk for APS if they are followed up for a period of time. Alarcon-Segovia et al reported the cohort study of 667 SLE patients followed for a mean of 7.5 months. They found 10% of patients with definite APS. The prevalence increased to 15% in 3-year follow-up and the highest prevalence of 23% at the follow-up interval of 15 to 18 years⁽¹³⁾. Shah et al reported the results of the 10-year follow-up of 52 patients with APS and 21 SLE patients with aCL but without clinical features of APS. During the follow-up period, 52% of patients with aCL, but initially lacking clinical features of APS, developed the syndrome⁽¹⁴⁾.

In the present study, there were 5 SLE patients who had at least one episode of clinical thrombosis, but had neither positivity of aCL nor LA. Two of them had LA test after the commencement of anticoagulant due to serious thrombotic complications, one had dural sinus thrombosis and the other had pulmonary thromboembolism. Three of them had negativity of aCL and LA at the time of thrombosis. Gomez-Pacheco et al found a decrease of IgG or IgM aCL titer occurred during thrombosis in 6 out of 24 patients, compared to the levels before and after the event⁽¹⁵⁾. They also found that anti- β 2-glycoprotein I antibody was strongly associated with thrombosis in SLE patients. These findings led the authors to have concern about the fluctuation of aCL titer overtime. It was not clear whether factors such as duration of disease, the activity of disease or immunosuppressive medications had influenced the results.

The present study has shown that LA tends to associate with lupus nephritis, although the authors recruited fewer patients in the non-nephritis group. The authors could demonstrate that LA-positive lupus nephritis patients had a higher SBP ($p = 0.005$) and higher 24-hr urine protein excretion ($p = 0.02$) than LA-negative patients. It is still unclear whether LA has any direct impact on lupus nephritis outcome. In contrast to the study of Emanuel, they did not find the correlation of LA and renal dysfunction in lupus nephritis patients. A minority of LA-positive patients have thrombotic microangiopathy in renal biopsy⁽⁸⁾. Other studies supported the negative impact of LA on renal outcome. They included lupus nephritis patients who had either positivity for LA or aCL antibody^(5,6). Moss et al followed the patients for a mean of 173 months and found a strong association between APA and the development

of chronic renal insufficiency in the multivariate analysis⁽⁵⁾. Daugas et al demonstrated the association between LA and APS with antiphospholipid nephropathy (APSN) in lupus nephritis patients. In that study, LA had a high predictive ability for vascular lesions identified by kidney biopsy, but they did not find such an association with aCL. Statistical tests revealed significance of APS or LA and the existence of an APSN (odd ratio = 6 and 5.9, respectively; $p = 0.0001$ for both). They also noted that the presence of LA or antiphospholipid syndrome was associated with more severe interstitial fibrosis by univariate analysis and, therefore, probably associated with a worse renal prognosis⁽¹⁶⁾.

The inability to discern the significance of positivity of aCL or LA in all SLE patients in terms of clinical features of APS is due to some limitations. The first is the small number of patients especially in the non-nephritis group. The second problem is the limited time of follow up. Finally, because the present study was cross-sectional, the test of APA was done only once. Some APS patients might have low levels of aCL during the thrombosis and, hence, were classified as aCL-negative patients.

The present study does not allow the authors to recommend the routine APA test performed in all SLE patients. SLE patients who had thrombosis or pregnancy events with LA positivity should be carefully monitored for the development of lupus nephritis. Selected lupus patients who have poorly controlled blood pressure and/or marked proteinuria should be examined for LA as it is the marker of poor renal outcome. However, appropriate treatment for LA-positive patients lacking extra-renal clinical thrombosis depends upon renal morphology⁽¹⁷⁾.

Conclusion

Antiphospholipid antibodies are frequent findings in SLE patients, but not associated with lupus nephritis. However, LA tends to associate with lupus nephritis. Detection of LA in lupus nephritis patients could identify patients who have increased risk to develop bad renal outcomes and contribute to an elevated SBP and 24-hr urine protein excretion.

References

1. Amigo MC, Khamashta MA. Antiphospholipid (Hughes) syndrome in systemic lupus erythematosus. *Rheum Dis Clin North Am* 2000; 26: 331-48.
2. Love PE, Santoro SA. Antiphospholipid anti-

- bodies: anticardiolipin and the lupus anticoagulant in systemic lupus erythematosus (SLE) and in non-SLE disorders. Prevalence and clinical significance. *Ann Intern Med* 1990; 112: 682-98.
3. Loizou S, Samarkos M, Norsworthy PJ, Cazabon JK, Walport MJ, Davies KA. Significance of anticardiolipin and anti-beta(2)-glycoprotein I antibodies in lupus nephritis. *Rheumatology (Oxford)* 2000; 39: 962-8.
 4. Alba P, Bento L, Cuadrado MJ, Karim Y, Tungekar MF, Abbs I, et al. Anti-dsDNA, anti-Sm antibodies, and the lupus anticoagulant: significant factors associated with lupus nephritis. *Ann Rheum Dis* 2003; 62: 556-60.
 5. Moss KE, Isenberg DA. Comparison of renal disease severity and outcome in patients with primary antiphospholipid syndrome, antiphospholipid syndrome secondary to systemic lupus erythematosus (SLE) and SLE alone. *Rheumatology (Oxford)* 2001; 40: 863-7.
 6. Moroni G, Ventura D, Riva P, Panzeri P, Quaglini S, Banfi G, et al. Antiphospholipid antibodies are associated with an increased risk for chronic renal insufficiency in patients with lupus nephritis. *Am J Kidney Dis* 2004; 43: 28-36.
 7. Bhandari S, Harnden P, Brownjohn AM, Turney JH. Association of anticardiolipin antibodies with intraglomerular thrombi and renal dysfunction in lupus nephritis. *QJM* 1998; 91: 401-9.
 8. Farrugia E, Torres VE, Gastineau D, Michet CJ, Holley KE. Lupus anticoagulant in systemic lupus erythematosus: a clinical and renal pathological study. *Am J Kidney Dis* 1992; 20: 463-71.
 9. Frampton G, Hicks J, Cameron JS. Significance of anti-phospholipid antibodies in patients with lupus nephritis. *Kidney Int* 1991; 39: 1225-31.
 10. Hochberg MC. Updating the American College of Rheumatology revised criteria for the classification of systemic lupus erythematosus. *Arthritis Rheum* 1997; 40: 1725.
 11. Wilson WA, Gharavi AE, Koike T, Lockshin MD, Branch DW, Piette JC, et al. International consensus statement on preliminary classification criteria for definite antiphospholipid syndrome: report of an international workshop. *Arthritis Rheum* 1999; 42: 1309-11.
 12. Brandt JT, Triplett DA, Alving B, Scharrer I. Criteria for the diagnosis of lupus anticoagulants: an update. On behalf of the Subcommittee on Lupus Anticoagulant/Antiphospholipid Antibody of the Scientific and Standardisation Committee of the ISTH. *Thromb Haemost* 1995; 74: 1185-90.
 13. Alarcon-Segovia D, Perez-Vazquez ME, Villa AR, Drenkard C, Cabiedes J. Preliminary classification criteria for the antiphospholipid syndrome within systemic lupus erythematosus. *Semin Arthritis Rheum* 1992; 21: 275-86.
 14. Shah NM, Khamashta MA, Atsumi T, Hughes GR. Outcome of patients with anticardiolipin antibodies: a 10 year follow-up of 52 patients. *Lupus* 1998; 7: 3-6.
 15. Gomez-Pacheco L, Villa AR, Drenkard C, Cabiedes J, Cabral AR, Alarcon-Segovia D. Serum anti-beta 2-glycoprotein-I and anticardiolipin antibodies during thrombosis in systemic lupus erythematosus patients. *Am J Med* 1999; 106: 417-23.
 16. Dugas E, Nochy D, Huong du LT, Duhaut P, Beaufils H, Caudwell V, et al. Antiphospholipid syndrome nephropathy in systemic lupus erythematosus. *J Am Soc Nephrol* 2002; 13: 42-52.
 17. Nzerue CM, Hewan-Lowe K, Pierangeli S, Harris EN. "Black swan in the kidney": renal involvement in the antiphospholipid antibody syndrome. *Kidney Int* 2002; 62: 733-44.

ความสำคัญของ Antiphospholipid Antibodies ในผู้ป่วยไตอักเสบเรื้อรัง

จินดารัตน์ เนตรจันทน์, ประเจษฎ์ เรืองกาญจนเศรษฐ์, พจน์ เอมพันธุ์, ถนอม สุภาพร

Antiphospholipid antibody (APA) เป็น antibody ต่อ phospholipid บน endothelial cell membrane และ platelet ประกอบด้วย anticardiolipin antibody (aCL) และ lupus anticoagulant (LA) ยังมีรายงานจำนวนน้อยที่ศึกษาความสัมพันธ์ระหว่าง APA กับความผิดปกติของไต การศึกษานี้เป็น cross-sectional จากการรวบรวมผู้ป่วย SLE 77 คน ระหว่างเดือนมิถุนายน พ.ศ. 2545 ถึงธันวาคม พ.ศ. 2546 พบมีอาการแสดงออกทางไต 63 คน (82%) ความชุกของ antiphospholipid syndrome ในผู้ป่วยมี 10% (8/77) ตรวจพบผลบวกของ IgG aCL และ LA 26 % (20/77) เท่ากัน เมื่อนำค่า aCL มาวิเคราะห์โดยวิธี ROC (receiver operating characteristic) พบว่าพื้นที่ใต้กราฟเท่ากับ 0.538 (95%CI 0.312-0.765) ค่า aCL ที่มากกว่า 20.5 GPL ถือเป็นผลบวก มี sensitivity 75 % และ specificity 53 % พบว่าการตรวจพบ aCL หรือ LA ไม่ได้เป็นปัจจัยเสี่ยงที่ทำให้มีอาการแสดงออกทางไตสูงขึ้นในผู้ป่วย SLE และ เมื่อศึกษาเฉพาะในกลุ่มผู้ป่วย SLE ที่มีการแสดงออกทางไตร่วมกับการตรวจพบ LA พบว่ามีความสัมพันธ์กับการมีความดันโลหิต systolic ที่สูงขึ้น (133.4 และ 121.9 มม.ปรอท, $p = 0.005$) เกล็ดเลือดที่ต่ำกว่า (209.8×10^3 และ 264.4×10^3 /ไมโครลิตร, $p = 0.02$) และโปรตีนในปัสสาวะมากกว่า (2.6 และ 1.4 กรัม/วัน, $p = 0.02$) นอกจากนี้กลุ่มผู้ป่วยที่ตรวจพบ LA ยังมีค่าครีเอตินีนสูงกว่า (233.0 และ 94.9 ไมโครโมล/ลิตร) แต่ไม่มีนัยสำคัญทางสถิติ

โดยสรุป จากการศึกษาพบความชุกของ antiphospholipid antibodies ได้ค่อนข้างสูงในผู้ป่วย SLE แต่ไม่มีความสัมพันธ์กับอาการแสดงออกทางไตอย่างมีนัยสำคัญทางสถิติ อย่างไรก็ตามการตรวจพบ LA ในกลุ่มที่มีการแสดงออกทางไตแล้ว พบว่ามีความสัมพันธ์กับความดันโลหิต systolic ที่สูงขึ้น และโปรตีนรั่วในปัสสาวะมากขึ้น ซึ่งเป็นปัจจัยที่นำไปสู่การทำงานเลวลงของไต