UPDATE IN DIAGNOSIS AND MONITORING OF SYSTEMIC LUPUS ERYTHEMATOSUS AND LUPUS NEPHRITIS

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ABSTRACT

Objective:The aim of this study is to investigate the role of some newly reported immunological markers(IL-10& antiC1q antibodies) in the diagnosis and monitoring of systemic lupus erythermatosus and lupus nephritis in correlation with the old used ones(ANA, Anti- dsDNA&C3).

Methods: Sixty systemic lupus erythermatosus patients and 30 healthy individuals were enrolled in this study. Antinuclear antibodies by indirect immunofluorescence and western blotting techniques, anti-dsDNA antibodies by indirect immunofluorescence technique, C3 by single radial immunodiffusion were all performed. In addition to performance of serum anti-C1q antibodies using ELISA & IL-10 assay after stimulation by immune complexes

Results: *Patients with lupus nephritis had a statistically significant higher titers of anti-dsDNA and anti-C1q antibodies levels compared with both SLE patients without lupus nephritis and control group. On comparing IL-10 levels produced from PBMC after immune complexes stimulation, a significant difference between control group and all other groups was found. In follow up, significant difference was found regarding anti-dsDNA antibodies titers and antiC1q antibodies levels.*

Conclusion: *Anti-C1q antibodies may play a role in diagnosis and monitoring of Systemic lupus erythematosus & lupus nephritis. Combined markers to be detected would give better results in diagnosis of Systemic lupus erythematosus and lupus nephritis.*

Keywords: Systemic lupus eryteramatosus,diagnosis, monitoring.ANAs, anti-dsDNA, antiC1q, lupus nephritis.IL-10.

INTRODUCTION

Systemic lupus erythematosus (SLE) is a systemic autoimmune disease immunologically characterized by B cell hyperreactivity, production of a multitude of different antibodies and immune complex formation[1]

SLE predominantly affects women, especially from ethnic groups with African and Asian ancestry. This disorder is a chronic illness that can be life threatening when serious complications such as lupus nephritis occurs[2].

Besides exploring more effective but less toxic treatment modalities that will further improve the remission rate, early detection and treatment of disease activity may spare patients from intensive immunosuppressive therapies and reduce renal damage [3]

Laboratory investigations for disease activity as estimation of routine inflammatory markers may be elevated in any inflammatory condition, including SLE, so further markers have been investigated. One of these markers is interleukin (IL-10) as many reports have described a relationship between increased serum IL-10 levels and disease activity suggesting a potential role of this cytokine in the pathoigenesis of SLE and disease activity. [4]

Several autoantibodies, especially those against anti-dsDNA, are believed to play a major role in the induction of glomerular inflammation. Raised titres of anti-dsDNA and hypocomplementaemia are reported to be associated with the activity of the disease. However, the lack of sensitivity of these biological markers for renal exacerbations has led to the search for other autoantibodies that might contribute to nephritis and help in the diagnosis of a renal flare[3].

Anti-C1q antibodies have received increasing interest as a diagnostic tool in SLE patients, it was further strengthened by the observation that increasing titers of anti-Clq seemed to precede renal flares by 2-6 months. In addition, after the successful treatment of a renal flare, anti-Clq mostly decreases or becomes undetectable [5]

PATIENTS AND METHODS

This study was conducted in "Medical Microbiology and Immunology Department", Faculty of Medicine, Zagazig University, Egypt, where sixty hospitalized patients at

Zagazig University Hospitals fulfilling the 1982 criteria for the diagnosis of SLE were incorporated into the study[6,7].

Disease activity was monitored using modified-SLEDAI (M-SLEDAI) score, in which immunological laboratory items were omitted. A lupus flare was defined as M-SLEDAI score ≥6 [8]. The patients were divided according to M-SLEDAI into 2 main groups: active and inactive lupus.

According to the results of 24 hours urinary protein provided from the hospital laboratory, active and inactive lupus groups were subdivided into patients with nephritis and patients without nephritis.

As controls, sera from 30 donors who were healthy, apparently free from any relevant diseas and matched to SLE patient group by age and sex were used.

All patients were subjected to the following investigations:

- ***Antinuclear antibodies (ANAs)' detection by indirect immunofluorescent (IIF) technique*:** on Human Epithelial (HEP-2) cells **(ANAFLUOR Test System, DiaSorin, Stillwater, Minnesota, U.S.A.)** in which diluted serum samples were overlaid onto HEp-2 cells grown on a microscope slide. The resultant positive reaction was observed as apple-green fluorescence of the nuclei when viewed with a properly equipped fluorescence microscope determining the pattern and the titer.
- ***ImmuBlot™ ANA Western Blot Immunoassay* (IMMCO Diagnostics, Buffalo, New York, USA);** antigen containing strips were incubated with serum samples. If present in the serum, anti-nuclear antibodies bind to the specific antigen on the strip. Positive reactions were indicated by a blue violet banding on the strip. The specificity of the antibody was defined by the identification of the positive bands in comparison with the provided positive & negative control templates, when aligned with 3 MW markers used as internal control bands.
- ***Anti-dsDNA autoantibodies detected by indirect immunofluorescent technique*** on *Crithidia luciliae* **(INOVA Diagnostics, San-Diego, USA)** determining its titers
- ***C3 assay by single radial immunodiffusion* (Astra, RID plates, Milano, Italy)**
- ***AntiC1q antibodies levels by ELISA* (ORG 549, Orgentec, USA).**
- ***IL-10 assay after stimulation by immune complexes***:

i. Polyethylene glycol (PEG) precipitation of immune complexes according to Mathsson et al. (2007)[9].

ii. Preparation of peripheral blood mononuclearPBMC according to Hay and Westwood (2002)[10]

iii. Cell culture Freshly prepared PEG precipitates were added to the PBMC cultures of 1x106 PBMC/ml (10% vol/vol) within two hours of preparation [11].

iv. Supernatants were harvested after 20 hours' incubation, and IL10 concentrations were determined by Human IL-10 ELISA kit, pink-One **(Koma biotechnology)**

Statistical Analysis

All Data were entered and analyzed by using **EPI-INFO (2005)**[12] soft ware computer package. Mann-whitney, ANOVA and F or Kruskal-Wallis tests were used. Correlations between serologic results were determined by the Pearson coefficient correlation. Variables were expressed as mean ±standard deviation. ROC curve analyses were used to determine optimal cut-off points. For these cut-off points, sensitivity and specificity were given. $P< 0.05$:significant & $P< 0.001$: highly significant.

RESULTS

This study comprised 60 patients suffering from SLE, they were 56 (93.3%) females and 4 (60.7%) males, their ages were with a mean value ±SD (31.4±10.3). Also the study included 30 normal persons apparently free from any relevant disease, age and sex matched with patients as a control group. They were 26 females (86.7%) and 4 males (13.3%), their ages were with a mean value ±SD (34.4±6.4). From these results, we found non-significant differences as regard age and sex between the studied groups.

ANAs were tested by IIF test on HEp-2 cells by which 58(96.7%) of SLE patients were positive and 2 (3.3%) were negative. Comparing ANAs titers between active and inactive lupus groups a statistically highly significant difference was detected. However, no statistically significant difference between lupus with nephritis and without nephritis groups was found .

Different ANA patterns were revealed. The homogenous pattern was the most common ANA pattern detected in 31 out of 58 (53.45%) of SLE patients**(fig.1)**. Coarse speckled, fine speckled & mixed patterns were seen in 7 (12.1%), 2 (3.45%) &18 (31%) patients respectively.

On studying ANA by western blotting technique, different Extractable nuclear antigen (ENAs) were detected **(fig. 2)**, anti-RNP antibodies were detected in 30 (50%), anti-Sm(B,B') in 22 (36.7%), anti-Ro in 13 (21.7%), anti-La in 13 (21.7%) and antiScl-70 in 1 (1.7%). Some patients had one or more ENAs.

On comparing ANA-IIF pattern results with western blotting results, homogenous pattern showed an association with anti-RNP (N= 10), Anti-Sm (N= 6), anti-Ro & anti-La (N= 4 each), the coarse speckled pattern showed an association with anti-RNP in all cases in varying combinations with anti-Sm, anti-Ro and anti-La, the fine speckled pattern showed an association with anti-Ro and anti-La in the two cases and one of them showed also combination with anti-Sm and anti-RNP, while the only one nucleolar ANA pattern showed positivity to anti-Ro, anti-La and anti-Scl-70.

On studying anti-dsDNA antibodies by IIF test on *Crithidia Lucilliae,* 47 out of 60 (78.3%) of SLE patients were found to be positive, 26 out of 47 (53.3%) were lupus nephritis **(fig. 3)**.

Analysis of variance (ANOVA) test was used to compare between anti-dsDNA titers among the studied groups. It has to be mentioned that on excluding nephritis as a factor for elevating anti-dsDNA titers (as in groups II&IV); a highly significant difference was reported indicating that activity alone had a highly significant role. On the other hand, on excluding activity as a factor (as in groups III&IV); a significant difference was reported indicating that nephritis had a significant role.

On studying C3 values among the studied groups by ANOVA test, each of activity and nephritis had a highly significant role.

Anti C1q antibodies were detected in 49 (80.3%) SLE patients and 11 (37.9%) of normal controls. 67.9% patients with lupus nephritis and only 6.3% of SLE patients without lupus nephritis were positive for anti-C1q antibodies. On studying antiC1q antibodies values by ANOVA test, it has to be mentioned that on excluding nephritis as a factor for elevating antiC1q antibodies values (as in Groups II & IV); a significant difference was reported indicating that activity alone had a significant role. On the other hand, on excluding activity as a factor (as in Groups III & IV); a highly significant difference was reported indicating that presence of nephritis had a highly significant role in elevating antiC1q antibodies **(Tables 1&2)**.

The sensitivity and specificity of antiC1q antibodies for lupus nephritis were 90.5% and 76.9% respectively compared with 66.7% and 100% for anti-dsDNA antibodies. Both assays combined had a sensitivity of 95.2% and specificity 76.9% **(Table 3)**. For evaluating the activity of SLE, Spearman rank correlation was used and it was found that ANA titers were best correlated with antidsDNA titers. However, it had negative correlation with C3 **(Table 4)**.

As regarding the nephritis, anti-C1q titers were correlated anti-dsDNA antibodies titers. There was significant negative correlation with C3 levels, while there was no correlation with ANA titers **(Table 5, fig 4a&b).**

On comparing IL-10 levels produced from PBMC after immune complexes stimulation, a significant difference between control group and all other groups was found. So ANOVA test was used to compare between IL-10 levels among the studied groups. It has to be mentioned that on excluding nephritis as a factor for elevating IL-10 levels (as in Groups II&IV); a highly significant difference was reported indicating that activity alone had a highly significant role. On the other hand, on excluding activity as a factor (as in Groups III&IV); a significant difference was reported indicating that nephritis had a significant role **(Table 6)**.

Follow up assay: Seven of our patients (active lupus with nephritis, anti-dsDNA +ve) were followed up for 24 hours urinary proteins, anti-dsDNA titers and antiC1q antibodies levels after 2 months treatment. No significant difference was found regarding proteinuria, however significant difference was found regarding anti-dsDNA antibodies titers and antiC1q antibodies levels **(Table 7)**.

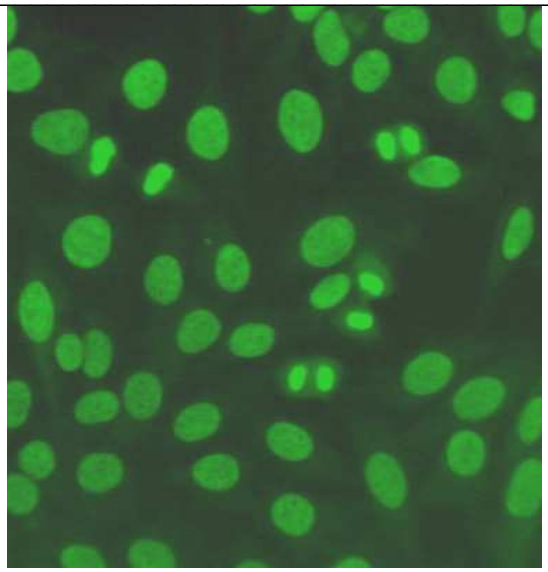


Figure (1): ANA-IIF on HEp-2 cells slide showing homogeneous pattern.

1 2 3 4 5 6 7

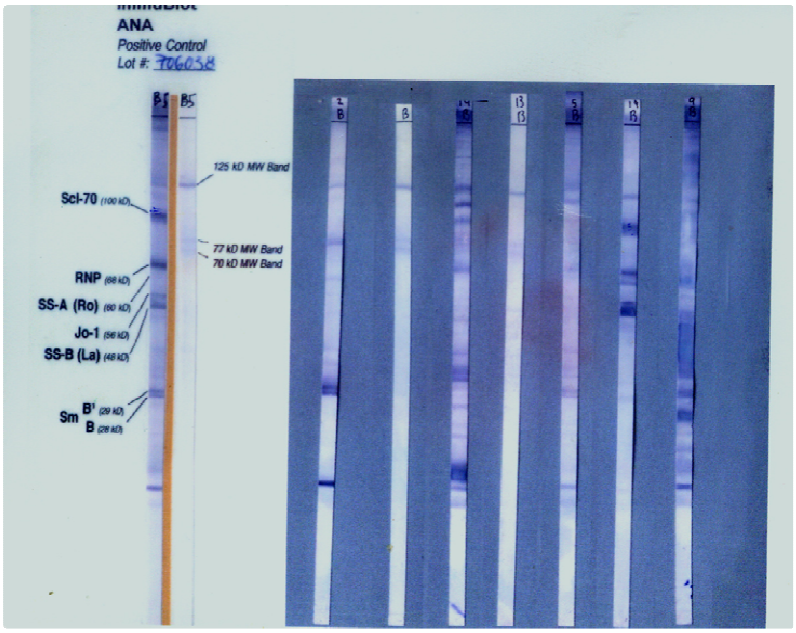


Figure (2): ENAs detected by western blot

	No.	Interpretation
1	2	Anti-Sm(B,B')
2	1	Negative
3	14	Anti-RNP
4	13	Negative
5	5	Anti-Sm(B,B')
6	19	Anti-La, Anti-Ro, Anti-Scl-70
7	9	Anti-Sm(B,B') Anti-RNP

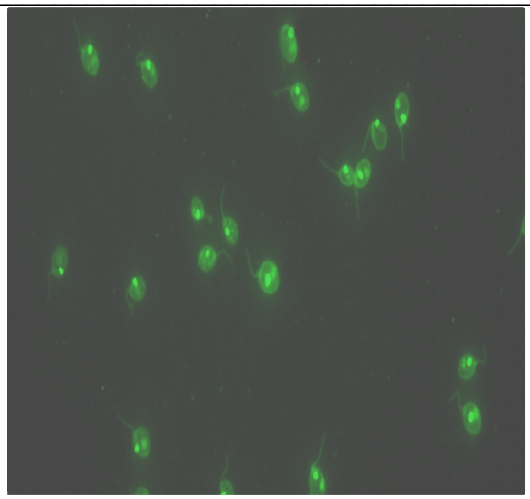


Figure (3): *Crithidia luciliae* hemoflagellates showing positive reaction

Table (1): Mean± SD of antiC1q antibodies values among SLE patients and control groups.

	Active SLE patients		**Inactive SLE patients**		**Control**
	GroupI (20) with nephritis	GroupII (14) without nephritis	GroupIII (12) with nephritis	GroupIV (14) without nephritis	GroupV(30)
Mean± SD	68.6±32.2	47.1±18.89	61.7±26.35	37.04±25.1	8.9±5.7

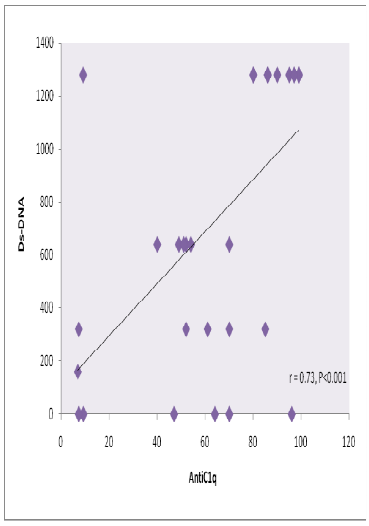
F = 26.79P<0.001

Table (2): Least significant difference (LSD) for comparison between means of Anti-C1q antibodies:

Group	**Active with nephritis I**	**Active Without nephritis II**	**Inactive with nephritis III**	**Inactive without nephritis IV**
Active Without nephritis II	<0.001			
Inactive with nephritis III	0.45	0.22		
Inactive without nephritis IV	<0.001	0.004	<0.001	
Control V	<0.001	<0.001	0.002	0.23

Table (3): Sensitivity and specificity of antiC1q antibodies and anti-dsDNA antibodies as determinants of lupus nephritis.

	sensitivity	specificity	PPV	NPV	Kappa	P
AntiC1q	90.5%	76.9%	67.9%	93.8%	0.62	P<0.001
Anti-dsDNA	66.7%	100%	100%	84.8%	0.72	P<0.001
AntiC1q and anti-dsDNA	95.2%	76.9%	69%	96.8%	0.66	P<0.001



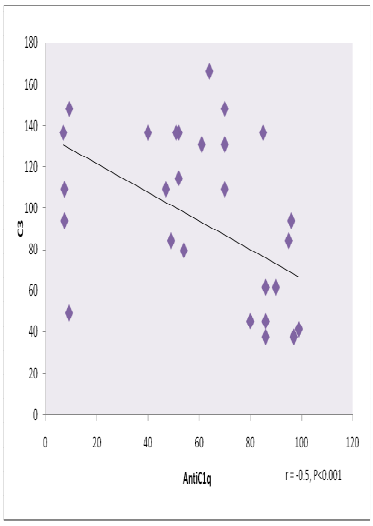


Table (4): Spearman rank correlation coefficients (r) between different laboratory parameters in diagnosis of SLE.

			ANA	ds-DNA	C3
ds-DNA	**r**	**P**			
			0.53		
			0.03		
C3	**r**	**P**			
			-0.64	-0.91	
			0.03	<0.001	
AntiC1q	**r**				
P			0.18	0.33	-0.28
			0.43	0.2	0.23

Figure (4): a) Correlation between AntiC1q antibodies levels and anti-dsDNA antibodies titers. **b)** Correlation between AntiC1q antibodies and C3 levels

Table (5): Spearman rank correlation coefficients (r) between different laboratory parameters in diagnosis of lupus nephritis

			ANA	Ds-DNA	C3
Ds-DNA					
r			0.32		
P			0.026		
C3					
r			-0.31	-0.82	
P			0.019	<0.001	
AntiC1q					
r		**P**	0.11	0.73	-0.5
			0.4	<0.001	<0.001

Table (6): Mean ±SD of IL-10 detected by ELISA after ICs stimulation (pg/ml) among SLE patients and control group

	Active SLE patients		Inactive SLE patients		
	Group I(20) with nephritis	Group II(14) without nephritis	Group III (12) with nephritis	Group IV(14) without nephritis	Control Group V(30)
After IC+*	95.8±30.2	64.8±18.9	57.7±21.7	57.7±17.9	47.1±24.7

*F = 12.7 P <0.001

Table (7): The median and range of 24 hs urinary proteins (g/day), anti-dsDNA titers and anti-C1q antibodies values (U/ml) in patients with lupus nephritis before and after 2 months treatment.

	Before treatment	After treatment	Mann-Whitney test	P
24hs urinary proteins				
Median	0.96	0.75	1.52	0.129
Range	0.86-1.8	0.8-1.6		
Anti-dsDNA				
Median	1/1280	1/640	2.63	0.008
Range	1/320-1/1280	1/160-1/640		
Anti-C1q				
Median	85	35	2.81	0.005
Range	31-99	22-52		

DISCUSSION

Systemic lupus erythematosus is a multisystem, autoimmune, connective-tissue disorder with a broad range of clinical presentations. This disorder is a chronic illness that can be life threatening when serious complications such as lupus nephritis occurred[13]

Parameters to assess renal disease included persistent proteinuria >0.5 g in a 24-h period, the presence of cellular casts, and/or elevated serum creatinine (>2.0 mg%). Patients were considered to have nephritis if at least two of these criteria were present. However, these parameters are not predictive of the classification and severity of nephropathy seen at biopsy nor have they been shown to be reliable in evaluating treatment response. Renal biopsy remains the "gold standard" to assess disease severity but multiple biopsies to gauge treatment efficacy are not feasible due to their invasive nature. It has thus become clear that there is a real need for surrogate markers that can predict the degree of renal inflammation[14]

ANAs are a specific class of autoantibodies that have the capability of binding and destroying certain structures within the nucleus of the cells. ANAs are considered one of the laboratory hallmarks of SLE according to the revised ACR criteria, having a frequency of 95% or greater[15]**.** In this study, ANAs were positive in 58(96.7%) of SLE patients and negative in 2 (3.3%) patients, when detected by IIF, this is in agreement with results of **Al-Jabri *et al.* (2009)** [16] and of **Laustrup *et al.* (2010)** [17] and higher than that obtained by **Farhat (2006)** [18] and **Qin *et al.* (2009)** [19].

In this study, it is observed that ANA reflect the level of disease activity, whereas they have no role in nephritis. This is in consistent with results obtained by **Paz et al. (2007)** [20]**.**

In this study, Different patterns of ANA were detected.These results are in consistent with results obtained by **Al-Jabri *et al.* (2009)** [16]. Although some IIF patterns strongly suggest distinct specificities, additional tests are requested to demonstrate antibody reactivities against specific nuclear antigens. These tests are used to either support the diagnosis (disease specificity) or to identify subsets of patterns that are prone to particular disease manifestation (prognostic marker) [21]**.**

Western blotting technique was used in the present study and different ENAs were identified. Several studies reported different ENAs among SLE patients, **Al Arfaj and Khalil (2009)** [22] in study done in Saudi Arabia, **AlSaleh *et al.* (2008)** in study done in Dubai [23] .Also, comparing IIF-ANA pattern results

with western blotting results, were in agreement with results reported by **Peene *et al.* (2001)** [24] and **Vos *et al.* (2006)** [25] indicating that anti-dsDNA antibodies should be incorporated as one of the antibodies tested by western blotting.

In the current study, the two IIF-ANA negative SLE patients revealed anti-Ro and anti-La antibodies. This confirms the conclusion that the combination of two or more testing methods can enhance the accuracy of the results [19]. Anti-double-stranded DNA antibodies are a useful tool for the diagnosis of SLE and may reflect the disease activity serving as a predictor of disease exacerbation and are suitable for monitoring therapy. The correlation of serum antibodies levels with disease activity and the isolation of DNA immune complexes from site of renal damages suggest also their involvement in the etiopathogenesis of lupus nephritis[26].

In the present study, results of anti-dsDNA antibodies agreed with results of **Manson *et al.* (2009)** [27] **and Mok *et al.* (2010)** [28] and were also consistent with the study done in Egypt by **Farhat (2006)** [18] and with **Al-Jabri *et al*. (2009)** [16]. On the other hand, **Al Attia *et al*. (1998**) [29] reported anti-dsDNA antibodies in 90% of SLE patients but with no statistical significant difference between lupus patients with nephritis and those without nephritis which could be explained by the low number of the patients they studied.

C3 concentration is one of important measures especially in lupus nephritis as it reflects the level of the circulating component irrespective of their functional state [30]. In the present study, results coincided with the results obtained by **Sinico *et al*. (2005)** [31]**, Hussain *et al*. (2008)** [32] and **Moura *et al.* (2009)** [33]

Anti-C1q antibodies have received increasing interest as a diagnostic tool in SLE patients, it was further strengthened by the observation that increasing titers of anti-Clq seemed to precede renal flares by 2-6 months. In addition, after the successful treatment of a renal flare, anti-Clq mostly decreases or becomes undetectable 5Anti-C1q autoantibodies could be detected in several disease conditions other than as well as in healthy individuals and its presence in lupus patients might be associated with evident renal affection. This can be explained as the threshold of transient C1q in the kidney in these persons is too low to cause significant and overt immunological damage and therefore no renal injury [34]

Titers of anti-C1q correlate with global SLE disease activity scores. In patients with lupus nephritis, anti-C1q titer strongly correlates with renal disease activity [3]**.** In this study, results of anti-C1q antibodies were in concordance with this study results as there was significant statistical difference between active and inactive lupus patients whereas, there was a highly significant statistical difference between lupus patients with nephritis and those without nephritis. Results of a study performed by **Sinico *et al.* (2005)** confirmed that presence of anti-C1q antibodies and levels correlate with disease activity in particular during renal flares-up[31].

Trendelenburg *et al*. (2005) reported 97.2% of patient with lupus nephritis were positive for anti-C1q compared with the 35% of SLE patients with inactive lupus nephritis and 25% of SLE patients without lupus nephritis[35]. Anti-C1q antibodies were detected in 32/81 (39.5%) of Brazilian SLE patients by **Moura *et al.* (2009)[33].** Also, similar results obtained by **Fang *et al.* (2009)** [36].

The differences in the prevalence of anti-C1q antibodies among the studies might be attributed to differences in the assay used and the definition of a positive test result. Also, the timing of blood sampling in relation to the renal flare should be considered.

As both anti-dsDNA and antiC1q antibodies were reported to be associated with lupus activity, several studies compared between sensitivity and specificity of both assays. In this study, the sensitivity and specificity of antiC1q antibodies for lupus nephritis were 90.5% and 76.9% respectively compared with 66.7% and 100% for anti-dsDNA antibodies. Both assays combined had sensitivity of 95.2% and specificity 76.9%.

Sinico *et al*. (2005) reported that the sensitivity and specificity of antiC1q antibodies for lupus nephritis were 86% and 95% respectively compared with 79% and 84% for anti-dsDNA antibodies. Both assays combined had sensitivity of 91% and specificity 90%[32].

Moroni *et al*. (2009) found that the sensitivity and specificity of antiC1q antibodies for lupus nephritis were 80.5% and 71% respectively and of anti-dsDNA were 70% and 67% respectively[37].

IL-10 might play a critical role in SLE with regard to B cell activation as IL-10 functions as a potent B cell stimulator that enhances activation, proliferation, and differentiation of B cells. In SLE, high levels of autoantibodies generate immune complexes causing tissue damage. 4 Depletion of IL-10 by anti-IL-10 mAb in vitro treatment of SLE patient-derived PBMC significantly decreased autoantibody production[38] and IL-10 antagonists may be beneficial in the treatment of human SLE [39]. Therefore, this cytokine was investigated in our study. Consistent with the putative implication of IL-10 in the pathophysiology of SLE, the present study showed that SLE is characterized by an increased production of IL-10 compared with healthy individuals and there was a significant statistical difference between active and inactive lupus patients. **Park *et al*. (1998)** [40] and **Waszczykowska *et al.* (1999)** [41] reported similar results. The study showed a significant statistical difference between active and inactive lupus patients and that was also in agreement with those obtained by **Gröndal et al. (2000)** [42] . In addition**, Suh and Kim (2008)** [43] **& El Sayed *et al.* (2008)** [44] agreed with these results.

Seven of our patients (active lupus with nephritis, anti-dsDNA +ve) were followed up after 2 months treatment. No significant difference was found regarding proteinuria, however significant difference was found regarding anti-dsDNA antibodies titers and antiC1q antibodies levels and antiC1q antibodies were more significant than anti-dsDNA antibodies in follow up of lupus nephritis. **Grootscholten *et al*. (2007)** reported that a comparable rapid decline in the levels of anti-dsDNA and anti-C1q autoantibodies was detected[45]. **Meyer *et al*. (2009)** found that antiC1q antibodies levels were significantly decreased with treatment[46].

AntiC1q antibody is a valuable non invasive biological marker for evaluation of renal involvement and lupus prognosis owing to being more sensitive and specific clinical marker for the onset or relapse of renal disease activity in patients with SLE [3].

To conclude: Owing to its complex aetiopathogenesis, heterogeneous presentation and unpredictable course, SLE remains one of the greatest challenges to both investigators and physicians. Anti-C1q antibodies may allow earlier institution of treatment and even preventive strategies, also the usefulness of anti-C1q level in monitoring of lupus activity in patients with negative anti-dsDNA antibodies has to be considered. We investigated some markers in this study however, no single test is sufficiently sensitive and specific to be diagnostic or prognostic. So, combined markers to be detected would give better results in diagnosis of SLE and lupus nephritis.

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الجديد في تشخيص و متابعة الذئبة الحمراء والتهاب الكلى الذئبي

إن الذئبة الحمراء مرض مناعي يتسم بزيادة نشاط الخلايا الليمفاوية ب ، إنتاج عدد وافر من الأجسام المضادة المختلفة وتكوين المركبات المناعية.
إن التهاب الكلي الذئبي، وهو مرض الكلى المصاحب لحالات الذئبة الحمراء، هو السبب الأساسي في زيادة نسبة المرض و معدل الوفيات.
العديد من الأجسام المضادة، خاصة تلك المضادة للحمض النووي الديؤكسى ريبوزى تلعب دورا كبيرا في الحث على التهاب الكلى. إن ارتفاع نسب الأجسام المضادة للحمض النووي ونقص نسبة المكملات في الدم مصاحب لزيادة نشاط المرض، لكن عدم كون هذه العلامات الحيوية مخصصة لتفاقم التهاب الكلى أدى إلى البحث عن أجسام مضادة أخرى من الممكن أن تساهم في حدوث التهاب الكلى وتساعد على تشخيص زيادة نشاط التهاب الكلى. كما ان نسب ضام الخلايا ١٠ تزيد و تتلازم مع مؤشرات المرض.
لذلك تم اقتراح أن وجود الأجسام المضادة للمكمل ١ كيو مطلوب لحدوث التهاب الكلي الذئبي كما إن متابعة الأجسام المضادة للمكمل ١ كيو قد تكون قيمة كعلامة حيوية مفيدة في حالات الذئبة الحمراء حيث وجودها متلازم مع التهاب الكلى واحتمال مع زيادة نشاط المرض.
الهدف من البحث:
تقييم دور بعض الدلائل المناعية التى ذكر جديدا انها تشارك فى تشخيص و متابعة حالات الذئبه الحمراء و التهاب الكلي الذئبي و علاقتها بالدلائل المستخدمة قريبا.

طرق البحث:

تم إجراء هذا البحث على ٦٠حالة من المصابين بمرض الذئبة الحمراء وتم تقسيمهم الى ٥ مجموعات وذلك حسب نتائج نسب البروتين فى البول المجمع ٢٤ ساعه ومؤشر نشاط مرض الذئبه الحمراء كما تم الاستعانه بمجموعه ضابطه من ٣٠ شخص.
تعرض مجموعة المرضى والمجموعه الضابطه إلى:
الكشف عن الأجسام المضادة للنواة باختبار التألق المناعى

- قياس نسبة المكمل ٣
- قياس الأجسام المضادة للمكمل ١ كيو
- تم الكشف عن الأجسام المضادة للنواة بطريقه الويسترن بلوت والأجسام المضادة للحمض النووي الديؤكسى ريبوزى باختبار التألق المناعى فى مجموعه المرضى
- قياس نسبة ضام الخلايا ١٠ بالارتباط الانزيمى للامتصاص المناعى(اليزا)التى تم انتاجها من الخلايا وحيدة النواة عند تعرضها للمركبات المناعية المفصولة من مصل المريض.

نتائج البحث:

بعد عمل التحليل الأحصائى، أظهر البحث النتائج التالية:
نسب قياس الأجسام المضادة للمكمل ١ كيو ، الأجسام المضادة للحمض النووي الديؤكسى ريبوزى ، ضام الخلايا- ١٠ اعلى فى الحالات النشطه واعلى فى حالات التهاب الكلى الذئبى.

الاستنتاج :

أن نسب قياس الأجسام المضادة للمكمل ١ كيو و الأجسام المضادة للحمض النووي الديؤكسى ريبوزى لهما دور فى تشخيص و متابعه التهاب الذئبه الحمراء والتهاب الكلى الذئبى. كما ينصح باستخدام أكثر من طريقة معملية عند التشخيص للحالات .