

Results

This study was conducted on 30 SLE patients (**Group I**) fulfilling at least 4 of the American College of Rheumatology (ACR) criteria who visited the outpatient rheumatology clinic or admitted in rheumatology department in Benha University Hospital, they were females and their ages ranged from 16 to 45 years with mean age = 27.9 ± 8.1 years. In addition to 20 apparent healthy volunteers (**Group II**) who were females and their ages ranged from 18 to 40 years with mean age = 27.4 ± 6.9 years.

Group I was classified according to clinical presentation of lupus nephritis to two groups:-

-**Group Ia:-** SLE patients with renal manifestations [14 (46.6%)].

-**Group Ib:-** SLE patients without renal manifestations [16 (53.3%)].

And Kidney biopsy was taken from all patients for histological examination. According to the results of kidney biopsy Group Ib was further classified to two subgroups:

-**Group Ib1:-** SLE patients without clinical renal manifestations but histological proven L.N (10).

-**Group Ib2:-** SLE patients without clinical renal manifestations nor histological proven L.N (6).

♦**Table (8)** shows comparison between SLE patients (group I) & controls (group II) as regards age that found no statistically significant difference between two groups ($p>0.05$).

♦**Table (9)** shows the clinical presentation of SLE patients (group I) in order of frequency was done and revealed that **Organic brain syndrome** was found in 36.7% of study cases, **Lupus headache** 23.3% ,**Arthritis** 16.7%,**New rash** 60% , **Alopecia** 50% , **Mucosal ulcer** 13.3% , **Pleurisy** 36.7% .

♦**Table (10)** shows SLEDAI score in study group, In **group Ia**, it revealed [4 severe, 8 moderate and 2 mild disease activity], while in **group Ib1**, it revealed [1 severe, 4 moderate and 5 mild disease activity] and in **group Ib2**, it revealed [4 moderate and 2 mild disease activity].

♦**Table (11)** shows SLICC damage index score in study group , In **group Ia** , it was [0 in 8 patients, 1 in 4 patients and 2 in 2 patients], while in **group Ib1**, it was [0 in 6 patients and 1 in 4 patients] and in **group Ib2**, it was [0 in 4 patients and 1 in 2 patients].

♦**Table (12)** shows BILAG index grades in study group, In **group Ia** it revealed [10 patients grade A & 4 patients grade B] , while in **group Ib1** it revealed [10 patients grade B] and in **group Ib2** it revealed [1 patient grade C & 5 patients grade E].

♦**Table (13)** shows Laboratory investigation of group Ia and group Ib including Complete blood picture (RBCs, WBCs, Hb concentration and Platelet count), kidney profile (proteinuria, serum creatinine, blood Urea), Anti-C1q antibody titer, C3, C4 and ESR.

♦**Table (14)** shows the presence of ANA in group I patients sera that was positive in all cases 100%.

♦**Table (15)** shows the presence of anti ds-DNA Abs in group I patients sera that was positive in 83.3%.

♦**Table (16)** shows the presence of anti-C1q Abs in group Ia, group Ib1, group Ib2 and group II patients sera which was 100%, 90%, 50%, and 10% respectively, also shows the presence of anti-C1q Abs in patients with lupus nephritis (group Ia and group Ib1) that was positive in 95.83%.

♦**Table (17)** shows comparison between patients and control as regards to anti C1q Abs titer with statistically significant difference ($p < 0.05$) *figure 11*.

♦**Table (18)** shows the comparison between group Ia and control (group II) according to anti C1q Abs titer and there was a statistically significant difference between SLE patients in group Ia and control group in serum level of anti C1q antibodies being higher in group Ia cases (mean = 126.4 ± 100.01) than in control (mean = 13.7 ± 6.1), $p < 0.05$ *figure 12*.

♦ **Table (19)** shows a comparison between group Ib1 and control according to anti C1q Abs titer is shown and there was a statistically significant difference between SLE cases in group Ib1 and control group in serum level of anti C1q antibodies being higher in group Ib1 cases(mean= 27.7 ± 9.1) than in control (mean= 13.7 ± 6.1), $p < 0.05$ *figure 13*.

♦ **Table (20)** shows a comparison between group Ib2 and control according to anti C1q Abs titer was done and there was a statistically significant difference between SLE cases in group Ib2 and control group in serum level of anti C1q antibodies being higher in group Ib2 cases(mean= 26.1 ± 7.01) than in control (mean= 13.7 ± 6.1), $p < 0.05$ *figure 14*.

♦ **Table (21)** shows comparison between group Ia & group Ib as regards Anti C1q Abs titer and there was a statistically significant difference between group Ia & group Ib ($p < 0.05$) *figure 15*.

♦ **Table (22)** shows Comparison between +ve and -ve cases according to clinical presentation as regards to Anti C1q Ab titer with no statistically significant difference was detected except **Pleurisy** ($p = 0.004$).

♦ **Table(23)** shows the correlation between anti-C1q Abs titer & SLEDAI (Systemic Lupus Erythematosus Activity Index) and SLICC damage index (systemic Lupus International Collaborating Clinics damage index), in the study group (group I) ,There was a statistically significant positive correlation between Ani- C1q antibody titer and SLEDAI ($r = 0.76$, $p < 0.05$)

figure 16, But no significant negative correlation SLICC damage index ($r = -0.76$, $P > 0.05$).

As we mentioned in patients and methods, British Isles Lupus Assessment Group Index (BILAG Index) categorizes renal disease activity into 5 different levels, from grade A to grade E, here in our study and to obtain a relation between anti-C1q Abs titer and these different grades we classified these 5 grades into two groups, the first is **Severe** (that involves A and B grades which present the more severe renal disease activity) and the second is **Mild** (that involves C, D and E grades which present the mild renal disease activity).

♦So, **Table (24)** shows there is a comparison between severe and mild BILAG index as regards to anti C1q Abs titer that revealed a statistically significant difference between them being higher in severe group (85.5 ± 90.2) than in mild group (25.4 ± 8.1), ($p < 0.05$) *figure 17*.

Correlations between anti-C1q Abs titer and laboratory data of study group (group Ia cases and group Ib cases) were done,

♦**Table (25)** shows correlation between antiC1q Abs titer & kidney profile (proteinuria, serum creatinine, blood Urea) in group Ia with a statistically significant positive correlation between Anti-C1q antibody titer and proteinuria ($r = 0.6$, $p < 0.05$) *figure 18*, but there was no statistically significant correlation between Anti-C1q antibody titer and other kidney profile parameters, $p > 0.05$.

♦**Table (26)** shows the correlation between antiC1q Abs titer& kidney profile (protiurea , serum creatinine, blood Urea) in group Ib1, There was statistically significant positive correlation between Ani- C1q antibody titer and protinurea ($r= 0.67$, $p<0.05$) *figure 19*, but there was no statistically significant correlation between Ani- C1q antibody titer and other kidney profile parameters, $p>0.05$.

♦while **Table (27)** shows the correlation between antiC1q Abs titer & kidney profile (protiurea , s. creat., bl. Urea) in group Ib2 revealed no statistically significant correlation between Ani- C1q antibody titer and kidney profile parameters, $p>0.05$.

♦**Table (28)** shows the correlation between antiC1q Abs titer& anti-ds DNA , C3, C4 , ESR in group Ia, There was a statistically significant positive correlation between Ani- C1q antibody titer & Anti-DNA antibody titer ($r=0.76$, $p<0.05$) *figure 20* and ESR ($r=0.74$, $p<0.05$) *figure 23*, also there was a statistically significant negative correlation between Anti C1q antibody titer and C3 ($r= - 0.59$, $p<0.05$) *figure 21*,and C4 ($r=-0.57$, $p<0.05$) *figure 22*.

♦**Table (29)** shows the correlation between antiC1q Abs titer& anti-ds DNA, C3 , C4, ESR in group Ib1, There was a statistically significant positive correlation between Ani-C1q antibody titer and Anti-DNA antibodies titer ($r=0.74$, $p<0.05$) *figure 24* and ESR ($r=0.69$, $p<0.05$) *figure 27*, also there was a statistically significant negative correlation

between Anti C1q antibody titer and C3 ($r = - 0.66$, $p < 0.05$) *figure 25* and C4 ($r = - 0.65$, $p < 0.05$) *figure 26*.

◆While **Table (30)** shows the correlation between antiC1q Abs titer& anti-ds-DNA, C3, C4, ESR in group Ib2 with no statistically significant correlation ($p > 0.05$).

◆**Table (31)** shows the correlation between antiC1q Abs titer& Activity Index and Chronicity Index of kidney biopsy in group Ia, that revealed a statistically significant positive correlation between Ani- C1q antibody titer and activity index ($r = 0.7$, $p < 0.05$) *figure 28*,

On the other hand, there was a statistically significant negative correlation between Ani- C1q antibody titer and chronicity index ($r = - 0.57$, $p < 0.05$) *figure 29*.

◆While **Table (32)** shows the correlation between anti-C1q Abs titer& Activity Index and Chronicity Index, in group Ib1 and there was a statistically significant positive correlation between Ani- C1q antibody titer and activity index ($r = 0.7$, $p < 0.05$) *figure 30*,

On the other hand, there was no statistically significant negative correlation between Ani- C1q antibody titer and chronicity index ($r = - 0.12$, $p > 0.05$).

◆**Table (33)** shows that the sensitivity of anti-C1q Abs for lupus nephritis was 95.8%, the specificity was 80.76%, the positive predictive value (PPV) of anti-C1q Abs was 92.8% and the negative predictive value (NPV) of anti-C1q Abs was 81.8%.

♦ The cut off value of anti-C1q Abs was set at 21 U/ml.

♦ **Figure (31)** shows a case of diffuse proliferative lupus nephritis (WHO Class IV), there is moderate chronicity, evidenced by moderate diffuse interstitial fibrosis, glomerulosclerosis, and fibrocellular crescents. There is still remaining activity of this proliferative process, evidenced by endocapillary proliferation and tubulointerstitial lymphocytic infiltrate.

♦ **Figure (32)** shows a case of segmental nature of proliferation in WHO Class III lupus nephritis. Some glomeruli appear to have only minor mesangial expansion, whereas others have marked segmental endocapillary proliferation and even necrosis. No tubulointerstitial nephritis or fibrosis is seen in this biopsy. There is also no sclerosis. Thus, activity is moderate, indicated by proliferation and necrosis but relatively few crescents, and chronicity is minimal, with absence of significant fibrosis or sclerosis.

♦ **Figure (33)** shows a case of proliferative lupus nephritis (the process was focal in this biopsy, WHO III, which involves less than 50% of glomeruli). There is segmental necrosis and proliferation with mild increase in extracapillary cells, not reaching the level of a crescent. There is surrounding early interstitial fibrosis and lymphoplasmocytic infiltrate with thickening, fibrosis, and rupture of Bowman's capsule in the glomerulus on the right.

♦Table (8) Comparison between SLE patients (group I) & controls (group II) as regards age :-

	Cases	Control	P
Mean \pm SD	27.9 \pm 8.1	27.4 \pm 6.9	>0.05

♦Table (9) the clinical presentation of SLE patients (group I) in order of frequency:-

Clinical data	Number of patients		Percent
Organic brain syndrome	+ve	(11)	36.7%
	-ve	(19)	63.3%
Lupus headache	+ve	(7)	23.3%
	-ve	(23)	76.7%
Arthritis	+ve	(5)	16.7%
	-ve	(25)	83.3%
New rash	+ve	(18)	60%
	-ve	(12)	40%
Alopecia	+ve	(15)	50%
	-ve	(15)	50%
Mucosal ulcer	+ve	(4)	13.3%
	-ve	(26)	86.3%
Pleurisy	+ve	(11)	36.7%
	-ve	(19)	63.3%

♦Table (10): SLEDAI score in study group:-

Patients groups	SLEDAI score		
	Mild	Moderate	Severe
group Ia	2	8	4
group Ib1	6	4	0
group Ib2	2	4	0

♦Table (11): SLICC damage index in study group :-

Patients groups	SLICC damage index score		
	0	1	2
group Ia	8	4	2
group Ib1	6	4	0
group Ib2	4	2	0

♦Table (12): BILAG index Grades in study group :-

Patients groups	BILAG index grades				
	A	B	C	D	E
group Ia	10	4	0	0	0
group Ib1	0	10	0	0	0
group Ib2	0	0	1	0	5

♦Table (13): Laboratory investigation of group Ia and group Ib:-

Laboratory data	Group	N	Mean
RBCs (thousands/cmm)	group Ia	14	3.99
	group Ib	16	4.25
WBCs (thousands/cmm)	group Ia	14	5.7
	group Ib	16	5.54
Platelets (thousands/cmm)	group Ia	14	257.29
	group Ib	16	272.38
HB (gm/dl)	group Ia	14	10.2
	group Ib	16	11.42
ESR (mm/hr)	group Ia	14	70.64
	group Ib	16	59.38
Serum creatinine (mg/dl)	group Ia	14	0.94
	group Ib	16	0.74
Blood urea (mg/dl)	group Ia	14	32.43
	group Ib	16	29.56
SGOT (IU/dl)	group Ia	14	46.93
	group Ib	16	34
SGPT (IU/dl)	group Ia	14	46.79
	group Ib	16	32.62
Antic1q-Ab (U/ml)	group Ia	14	126.35
	group Ib	16	27.10
C3 (mg/dl)	group Ia	14	71.86
	group Ib	16	112.89
C4 (mg/dl)	group Ia	14	15.91
	group Ib	16	31.66
Protine/24 hours urine collection (mg/24 hr)	group Ia	14	2.09
	group Ib	16	207.75

♦Table (14): study group (group I) according to ANA Abs results:-

ANA	Number of patients	Percentage
+ ve	30	100 %
– ve	–	–

♦Table (15): study group (group I)according to anti ds-DNA Abs results:-

Anti ds-DNA Abs	Number of patients	Percentage
+ ve	25	83.3 %
– ve	5	16.7 %

♦Table (16): group I and groupII according to anti C1q Abs results:-

Group	Anti-C1q Abs	Number of patients	Percentage
group Ia	+ ve	14	100%
	– ve	–	–
group Ib1	+ ve	9	90%
	– ve	1	10%
group Ib2	+ ve	3	50%
	– ve	3	50%
groupII	+ ve	2	10%
	– ve	18	90%
groupIa & group Ib1	+ ve	23	95.83%
	– ve	1	4.16%
groupI (SLE patients)	+ ve	26	86.6%
	– ve	4	13.3%

♦Table (17): comparison between cases and control as regards to anti C1q Abs titer:-

	cases (n=30)	Control (n=20)	P
X±SD	73.4±83.9	13.7±6.1	*<0.05

*<0.05 = significant

> 0.05 = non significant

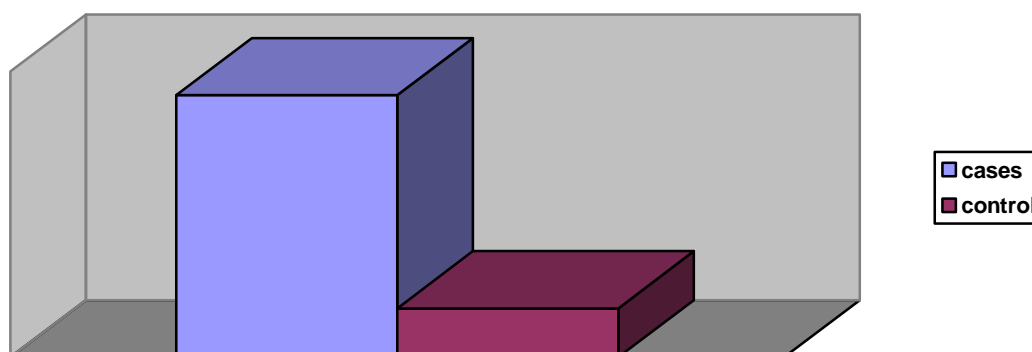
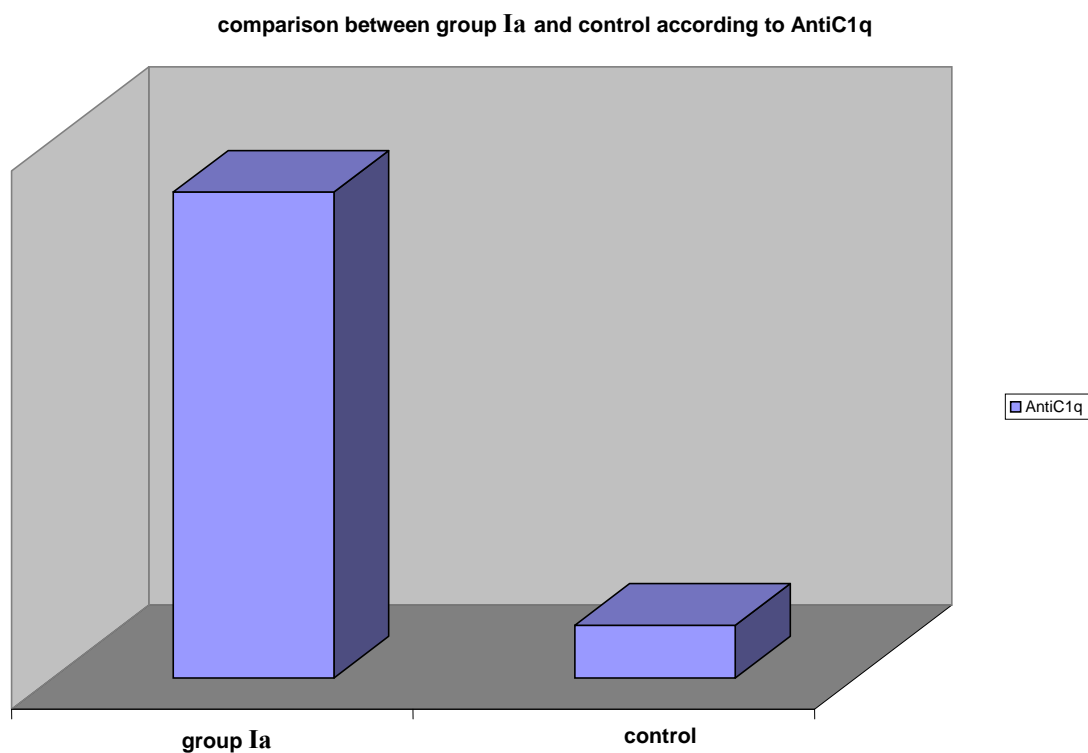


Figure (11): comparison between cases and control as regards to anti C1q Abs titer

♦Table (18): comparison between group Ia and control group II according to anti C1q Abs titer:-

	group Ia (n=14)	Control (n=20)	t	p
$\bar{X} \pm SD$	126.4 \pm 100.01	13.7 \pm 6.1	5.1	*<0.05



Figure(12): comparison between group Ia and control group II according to anti C1q Abs titer

♦Table (19): comparison between group Ib1 and control according to anti C1q Abs titer:-

	group 1b1 (n=10)	Control (n=20)	t	p
$\bar{X} \pm SD$	27.7 \pm 9.1	13.7 \pm 6.1	5.01	*<0.05

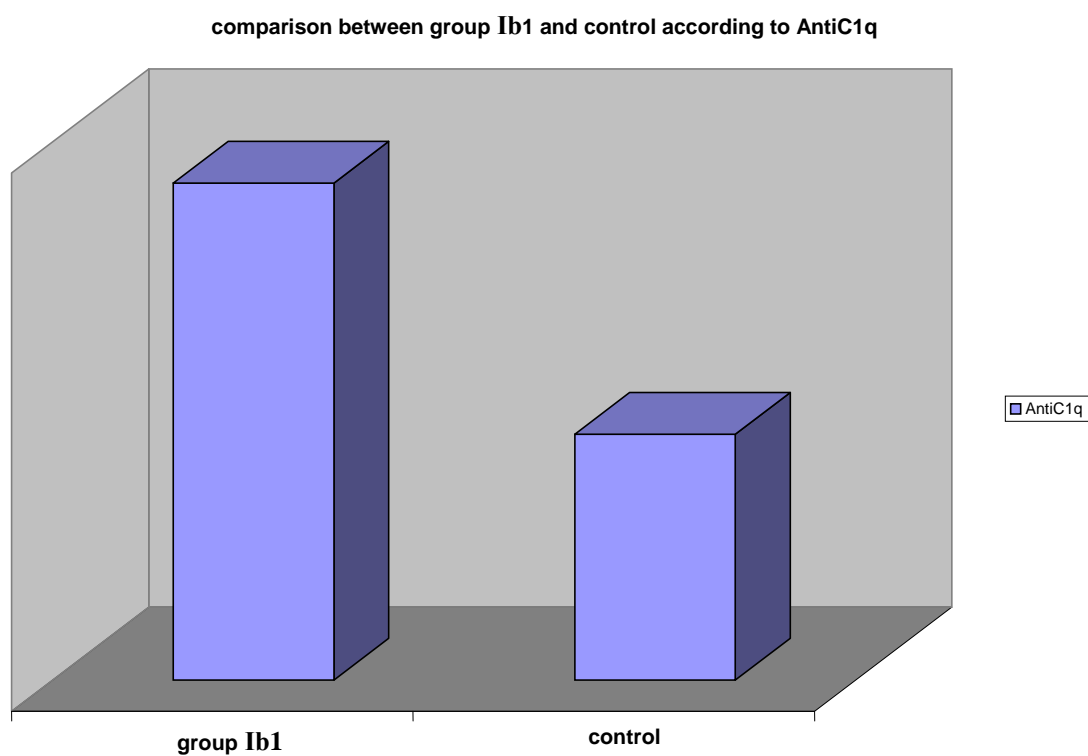


Figure (13): comparison between group Ib1 and control according to anti C1q Abs titer

♦Table (20): comparison between group Ib2 and control according to anti C1q Abs titer:-

	group 1b2 (n=6)	Control (n=20)	T	p
$\bar{X} \pm SD$	26.1 \pm 7.01	13.7 \pm 6.1	4.2	*<0.05

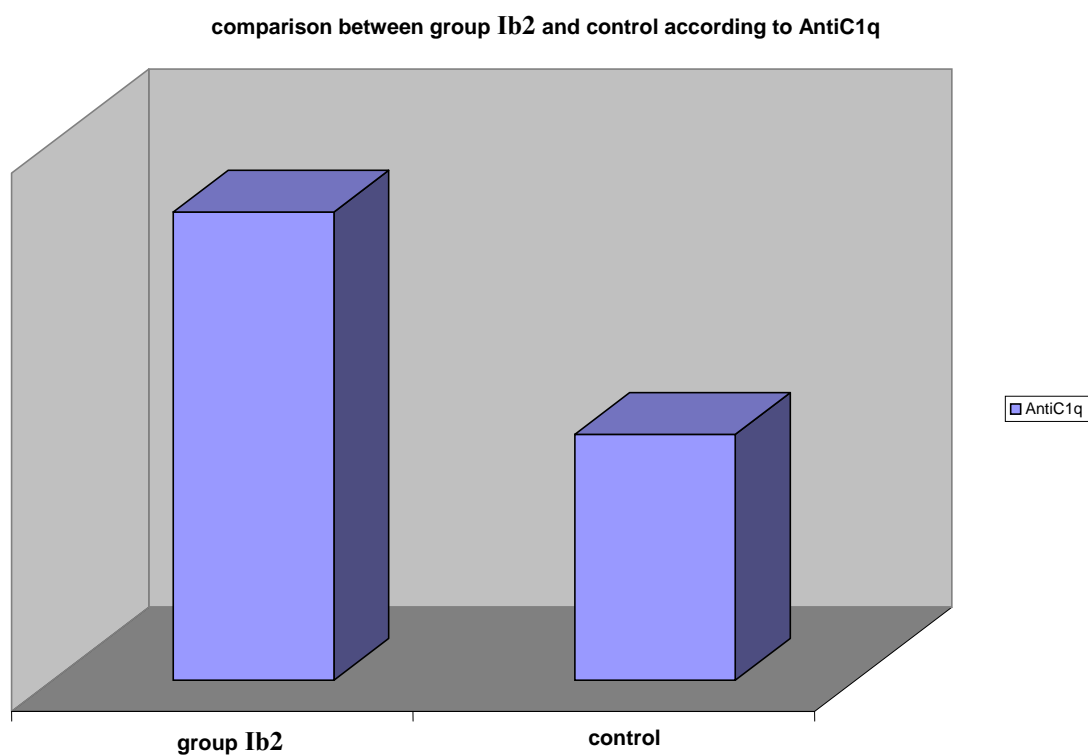


Figure (14): comparison between group Ib2 and control according to anti C1q Abs titer

♦Table (21): comparison between group Ia and group Ib according to Anti C1q Abs titer:-

Laboratory data	Group	N	Mean	St. Deviation	t	p
Antic1q-Ab (U/ml)	group Ia	14	126.35	100.01	3.9	*<0.05
	group Ib	16	27.10	8.16		

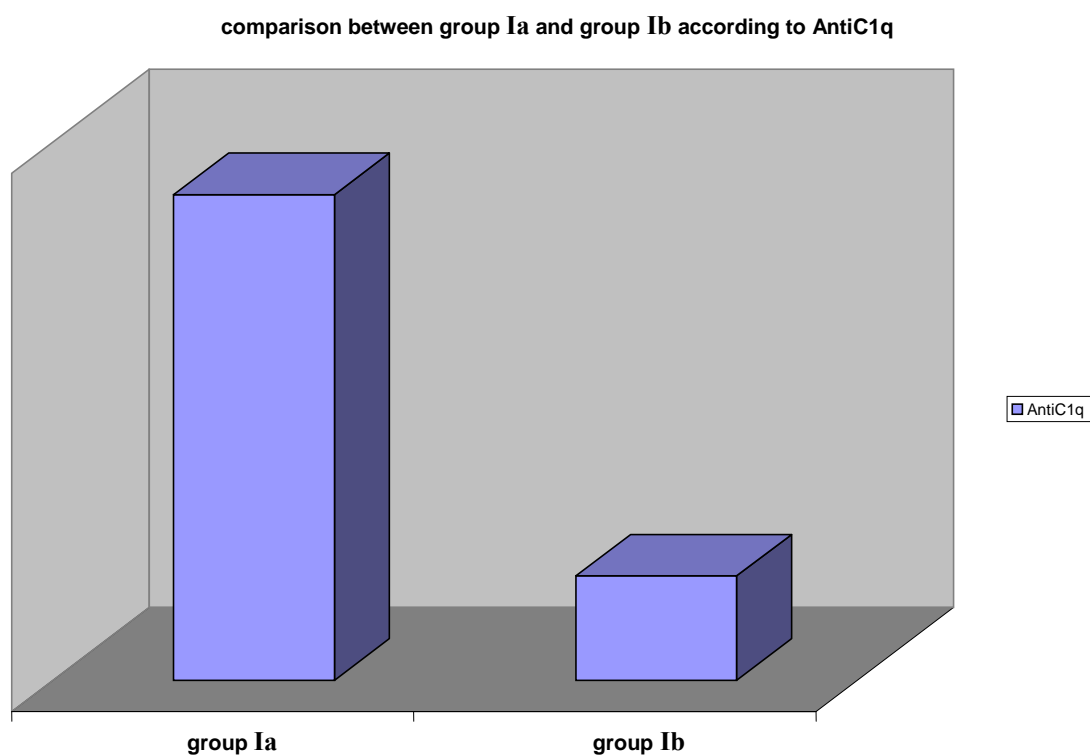


Figure (15): comparison between group Ia and group Ib according to AntiC1q Abs titer

♦Table (22): comparison between +ve and –ve cases according to clinical presentation as regards to Anti C1q Ab titer:-

Clinical data	Number of patients		Median (anti-C1q Ab)	P
Organic brain syndrome	+ve	(11)	47.4	0.9 (NS)
	-ve	(19)	34.4	
Lupus Headache	+ve	(7)	61.7	0.2(NS)
	-ve	(23)	34.4	
Arthritis	+ve	(5)	51.4	0.6(NS)
	-ve	(25)	38.8	
New rash	+ve	(18)	33.02	0.2(NS)
	-ve	(12)	47.4	
Alopecia	+ve	(15)	48.6	0.09(NS)
	-ve	(15)	30.6	
Mucosal ulcer	+ve	(4)	29.6	0.5(NS)
	-ve	(26)	47.4	
Pleurisy	+ve	(11)	86.3	*0.004(S)
	-ve	(19)	30.6	
Pericarditis	+ve	(4)	129.8	0.06(NS)
	-ve	(24)	35.6	

♦Table (23):correlation between antiC1q Abs titer& SLEDAI and SLICC damage index in the study group (group I):-

Index	r	p
SLEDAI	0.76	*<0.05
SLICC damage index	0.76	>0.05

correlation between anti C1q Abs titer & SLEIDAI in study group

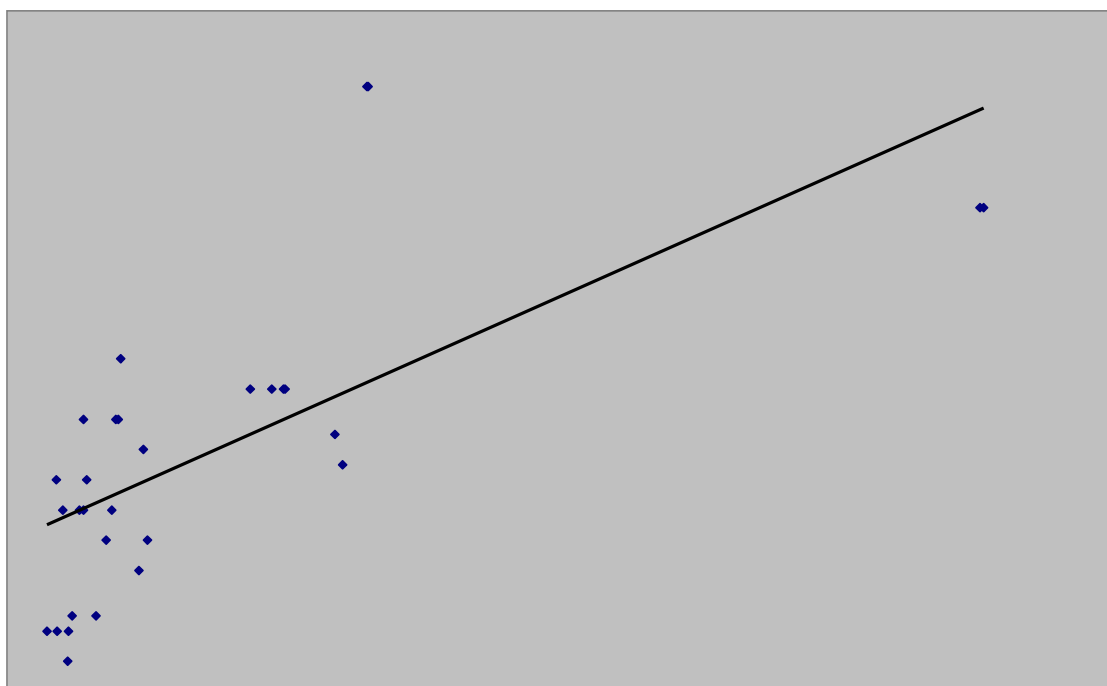


Figure (16):- correlation between anti- C1q Abs titer & SLEIDAI in study group (group I)

♦Table (24): comparison between sever and mild renal part of BILAG index as regards to anti C1q Abs titer:-

	sever (n=24)	mild (n=6)	p
$\bar{X} \pm SD$	85.5 \pm 90.2	25.4 \pm 8.1	*<0.05

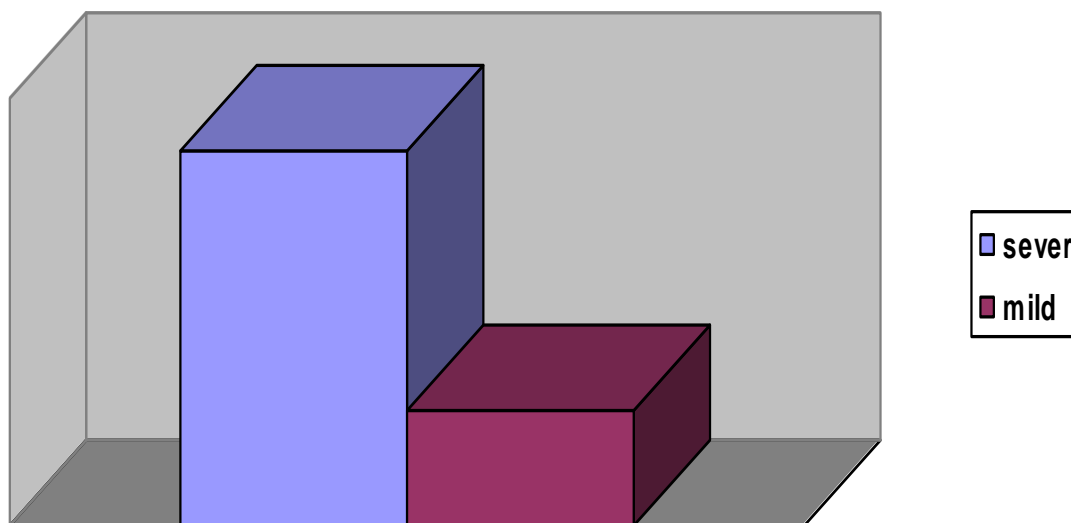
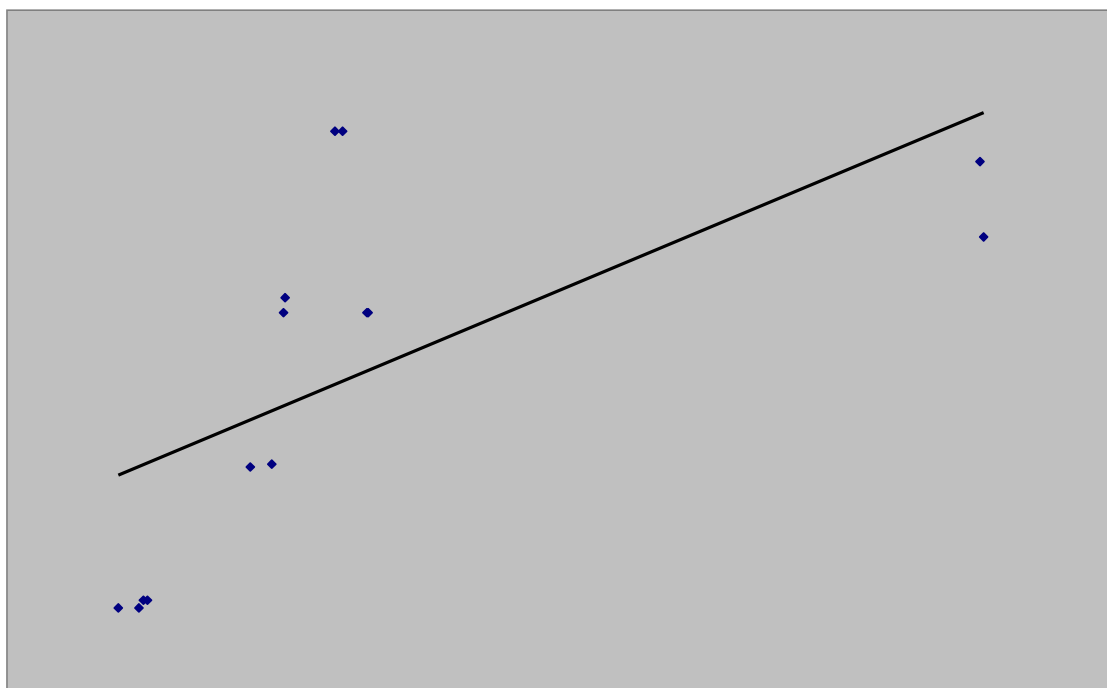


Figure (17): comparison between sever and mild BILAG as regards to anti C1q Abs titer

♦Table (25): correlation between antiC1q Abs titer& kidney profile (protiurea , serum creatinine, blood Urea) in group Ia:

Laboratory data	r	p
protiurea	0.6	*<0.05
Serum creatinine	0.3	>0.05
Blood Urea	0.5	>0.05

correlation between antic1q Abs titer& protinurea in group Ia



Figure(18):correlation between antic1q Abs titer& protinurea in group Ia

♦Table (26):correlation between antiC1q Abs titer& kidney profile(protiurea , serum creatinine, blood Urea) in group Ib1:-

Laboratory data	r	p
Protiurea	0.67	*<0.05
Serum creatinine	-0.7	>0.05
Blood Urea	0.54	>0.05

correlation between anti-C1q Abs titer& protiurea in group Ib1

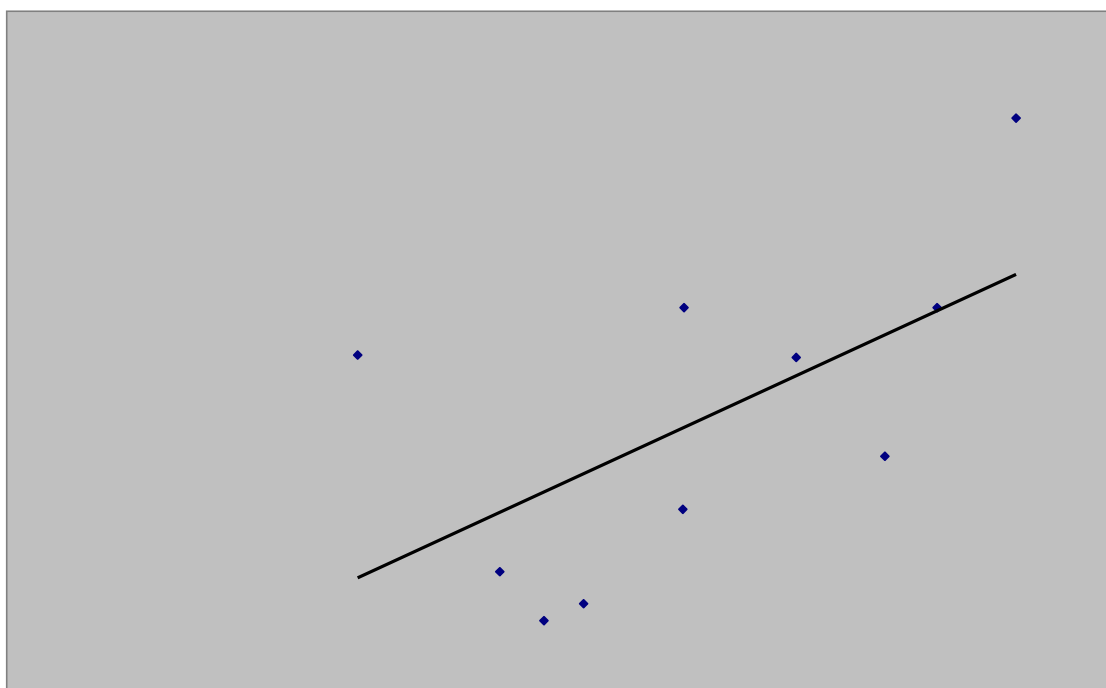


Figure (19) :correlation between antic1qAbs titer& protiurea in group group Ib1

♦Table (27):correlation between antiC1q Abs titer & kidney profile(protiurea , s. creat., bl. Urea) in group Ib2:-

Laboratory data	r	p
protiurea	0.77	>0.05
Serum creatinine	- 0.47	>0.05
Blood urea	0.41	>0.05

♦Table (28):correlation between antiC1q Abs titer& anti-ds DNA , C3, C4 , ESR in group Ia:-

Laboratory data	r	P
Anti-ds DNA	0.76	*<0.05
C3	- 0.59	*<0.05
C4	- 0.57	*<0.05
ESR	0.74	*<0.05

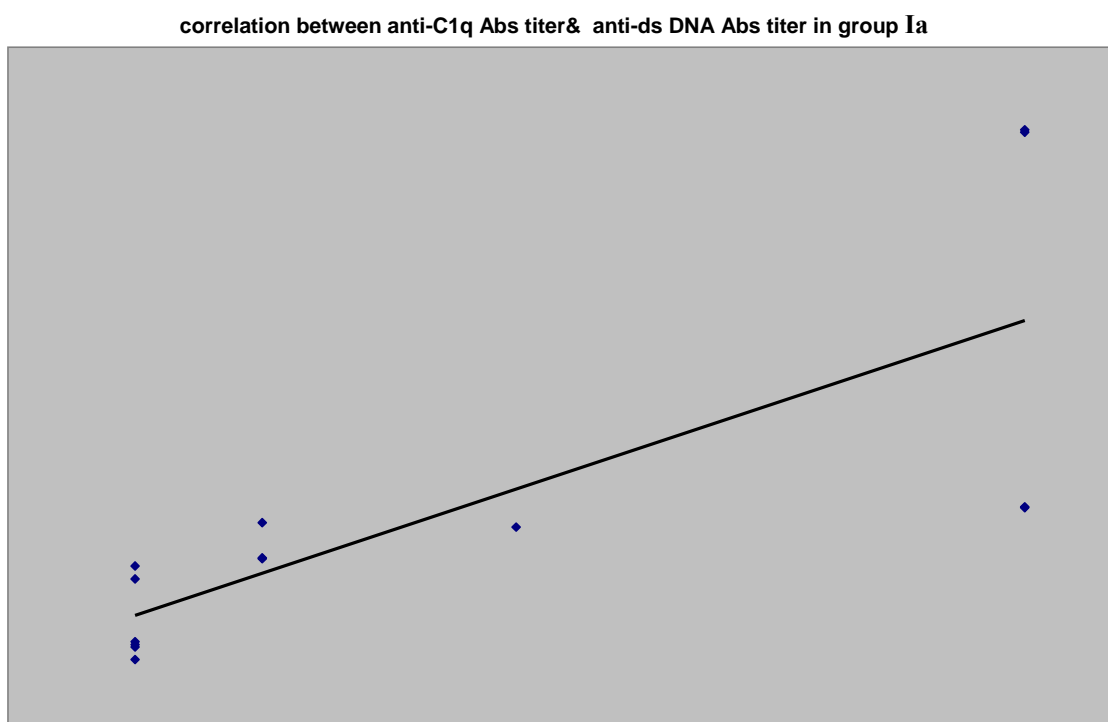


Figure (20):correlation between anti-C1q Abs titer& anti-ds DNA Abs titer in group Ia

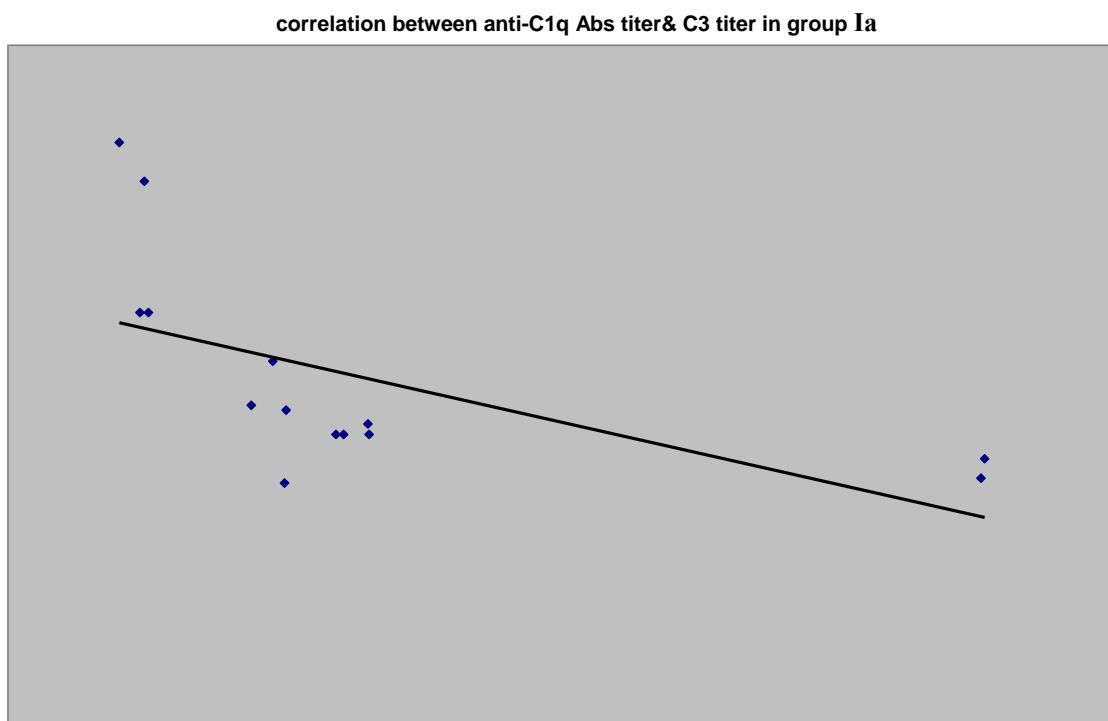
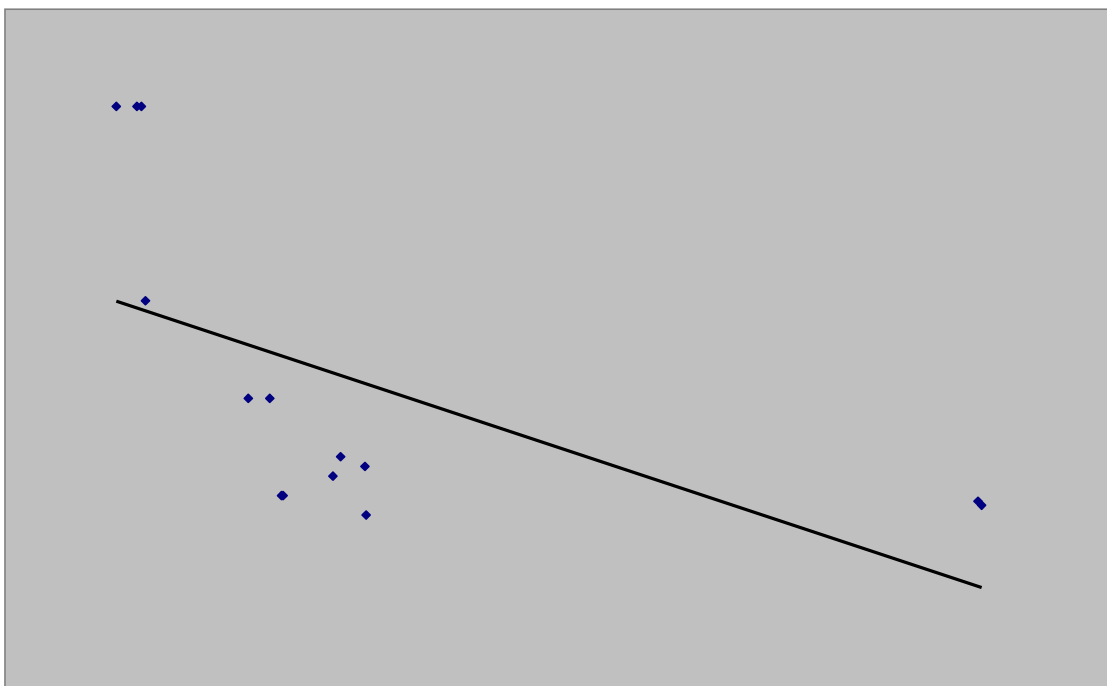
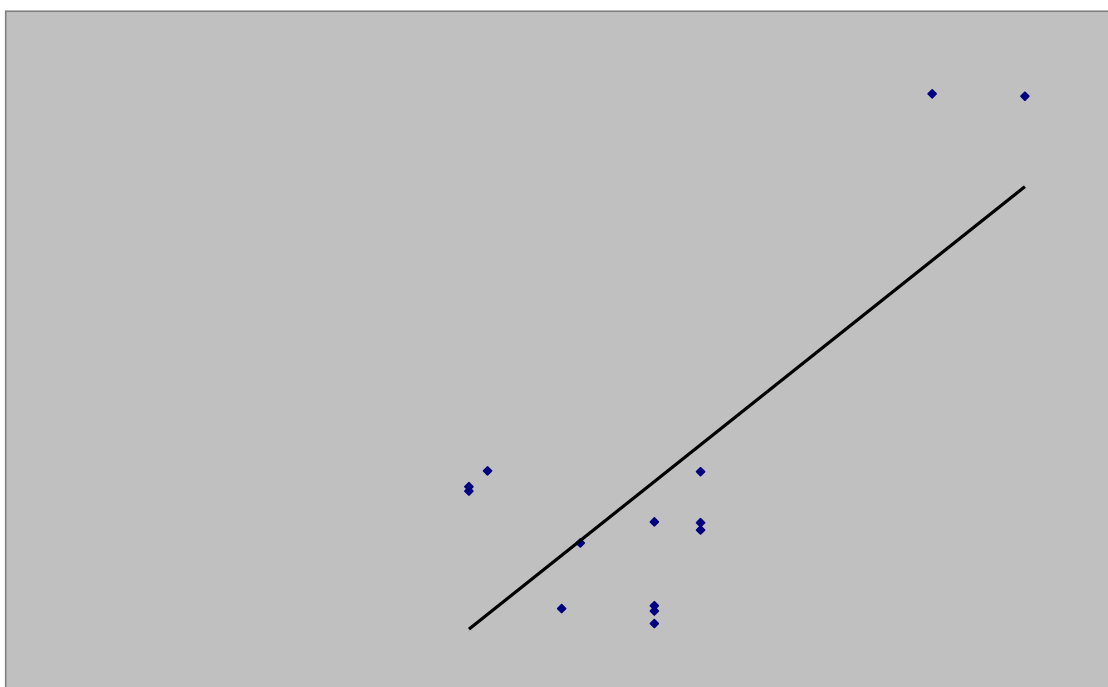


Figure (21):correlation between anti-C1q Abs titer& C3 titer in group Ia

correlation between antiC-1q Abs titer& C4 titer in group Ia

*Figure (22):correlation between antiC-1q Abs titer& C4 titer in group Ia*

correlation between anti-C1q Abs titer& ESR in group Ia

*Figure (23):correlation between anti-C1q Abs titer & ESR in group Ia*

♦Table (29): correlation between antiC1q Abs titer& anti-ds DNA, C3, C4, ESR in group Ib1:-

Laboratory data	r	p
anti-ds DNA	0.74	*<0.05
C3	-0.66	*<0.05
C4	-0.65	*<0.05
ESR	0.69	*<0.05

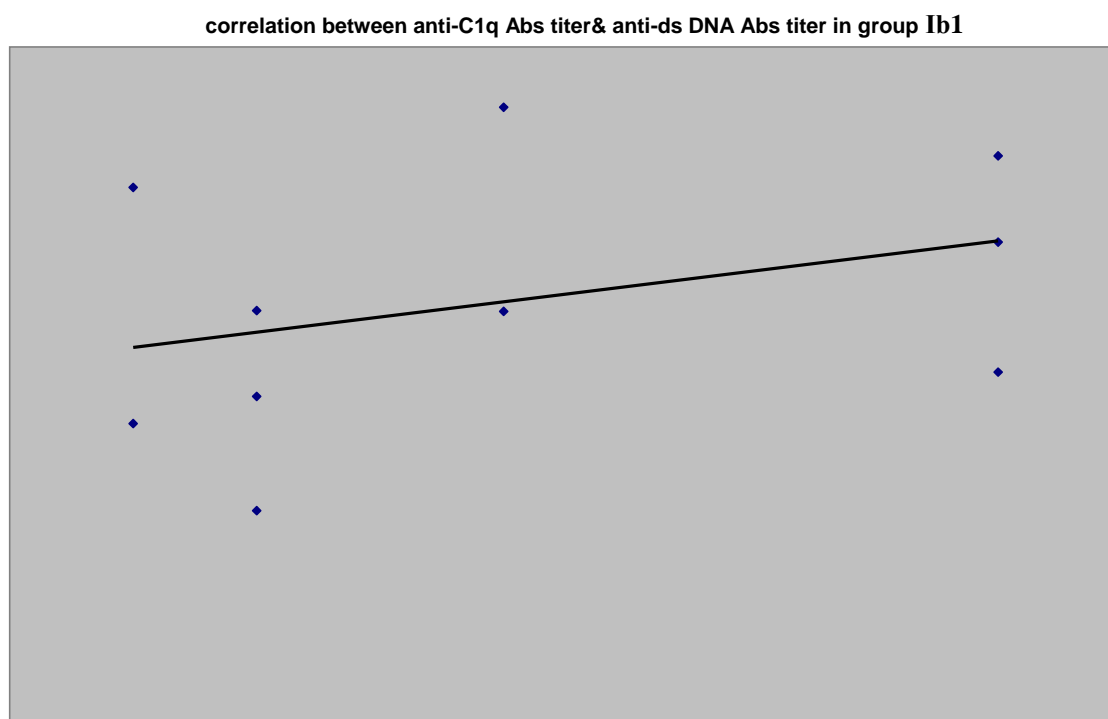
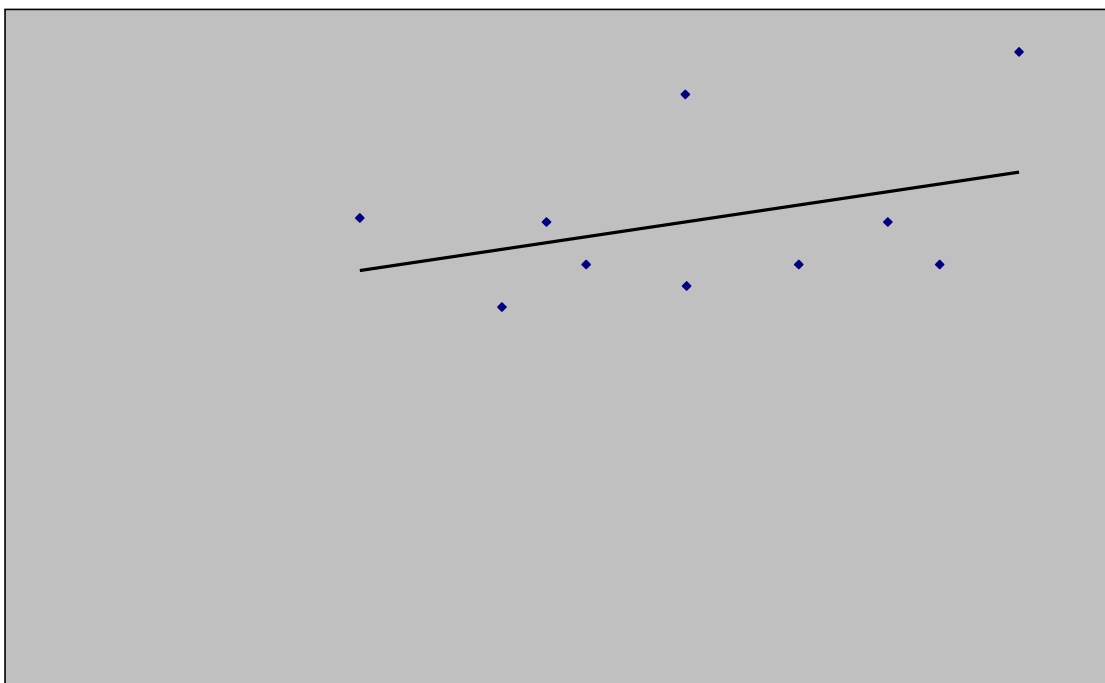
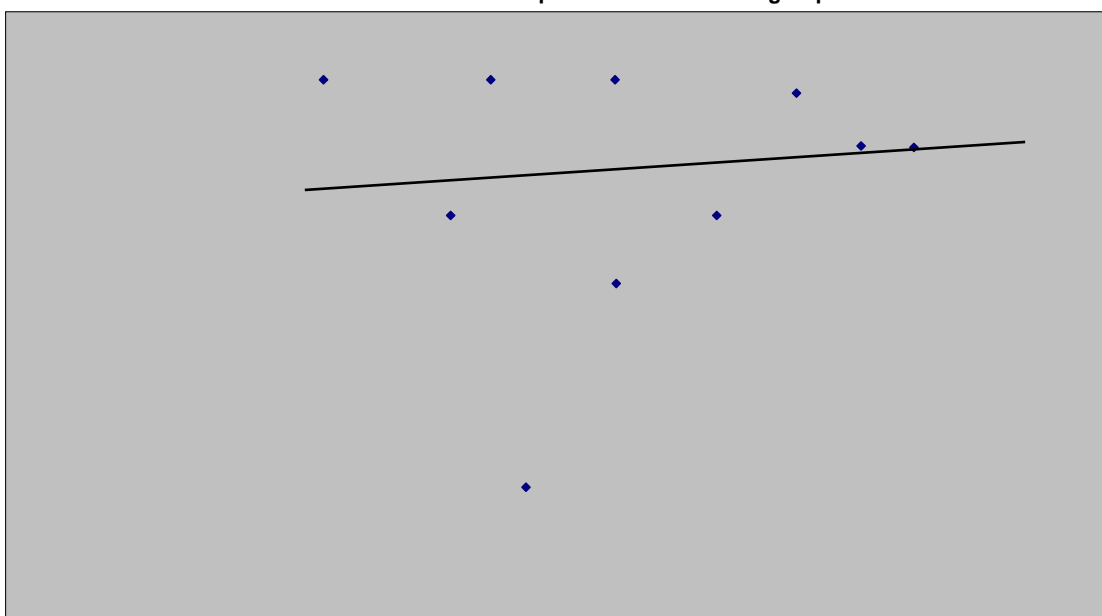


Figure (24):correlation between anti-C1q Abs titer& anti-ds DNA Abs titer in group Ib1

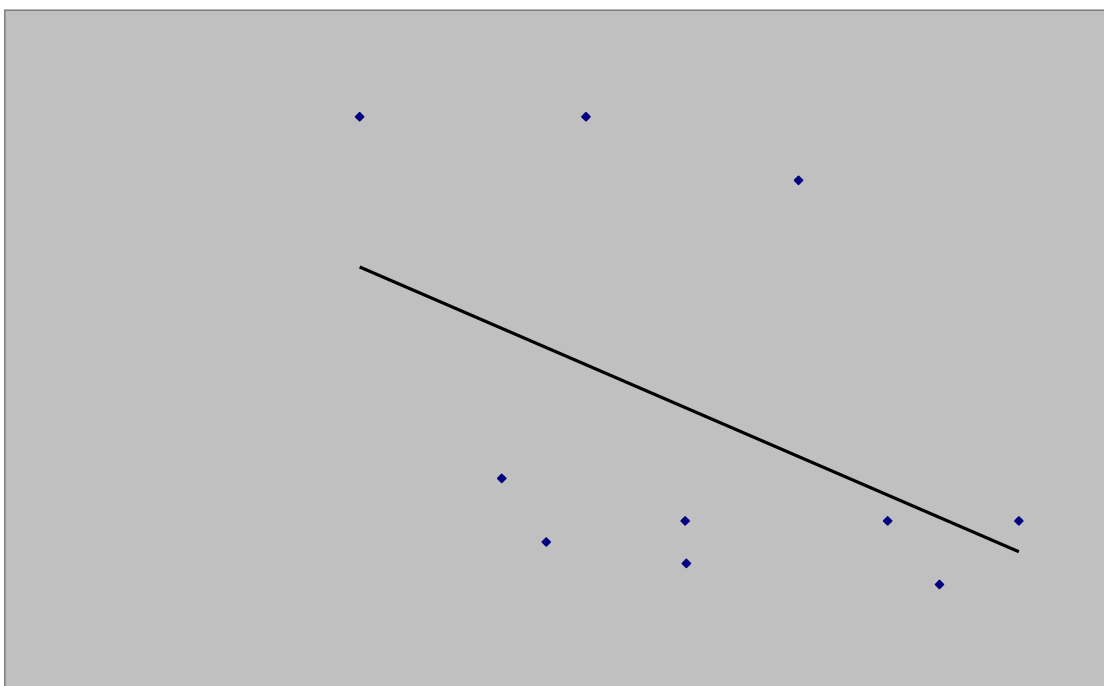
correlation between anti-C1q Abs titer & C3 titer in group Ib1

*Figure (25): correlation between anti-C1q Abs titer & C3 titer in group Ib1*

correlation between anti-C1q Abs titer & C4 titer in group Ib1

*Figure(26): correlation between anti-C1q Abs titer & C4 titer in group Ib1*

correlation between anti- C1q Abs titer &ESR in group Ib1

*Figure (27) : correlation between anti- C1q Abs titer &ESR in group Ib1*

♦Table (30): correlation between antiC1q Abs titer& anti-ds DNA, C3, C4, ESR in group Ib2:-

Laboratory data	r	P
anti-ds DNA	0.58	>0.05
C3	0.03	>0.05
C4	-0.09	>0.05
ESR	-0.89	>0.05

♦Table (31):correlation between antiC1q Abs titer& Activity Index and Chronicity Index, in group Ia:-

Index	r	p
AI	0.7	*<0.05
CI	- 0.57	*<0.05

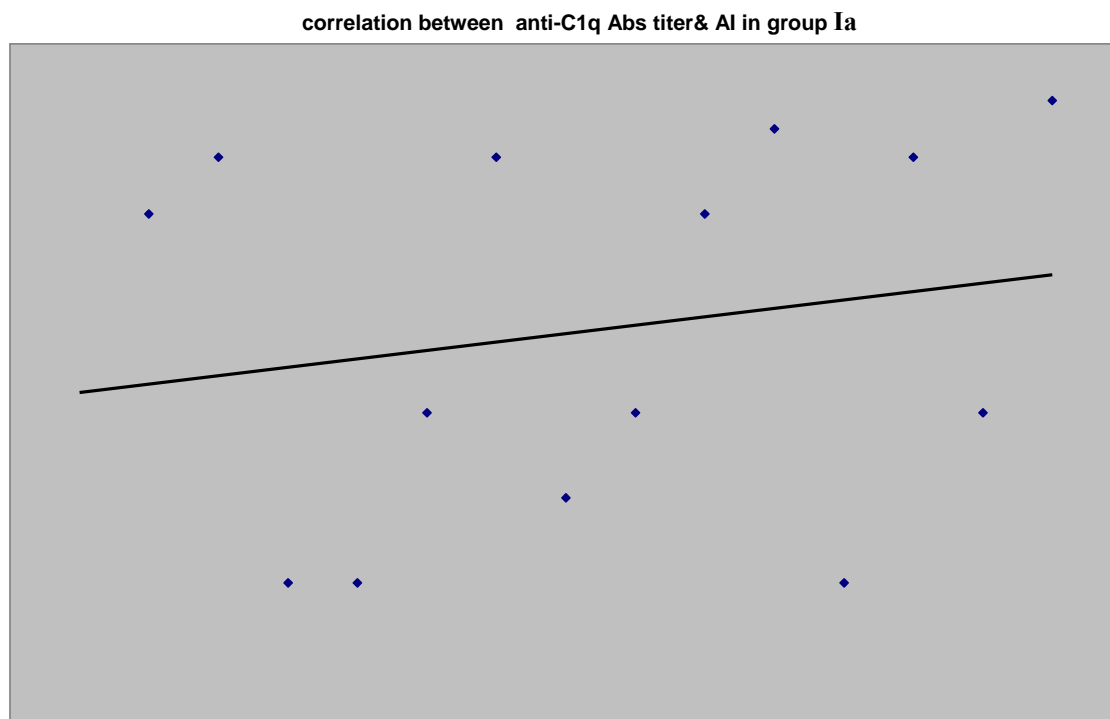
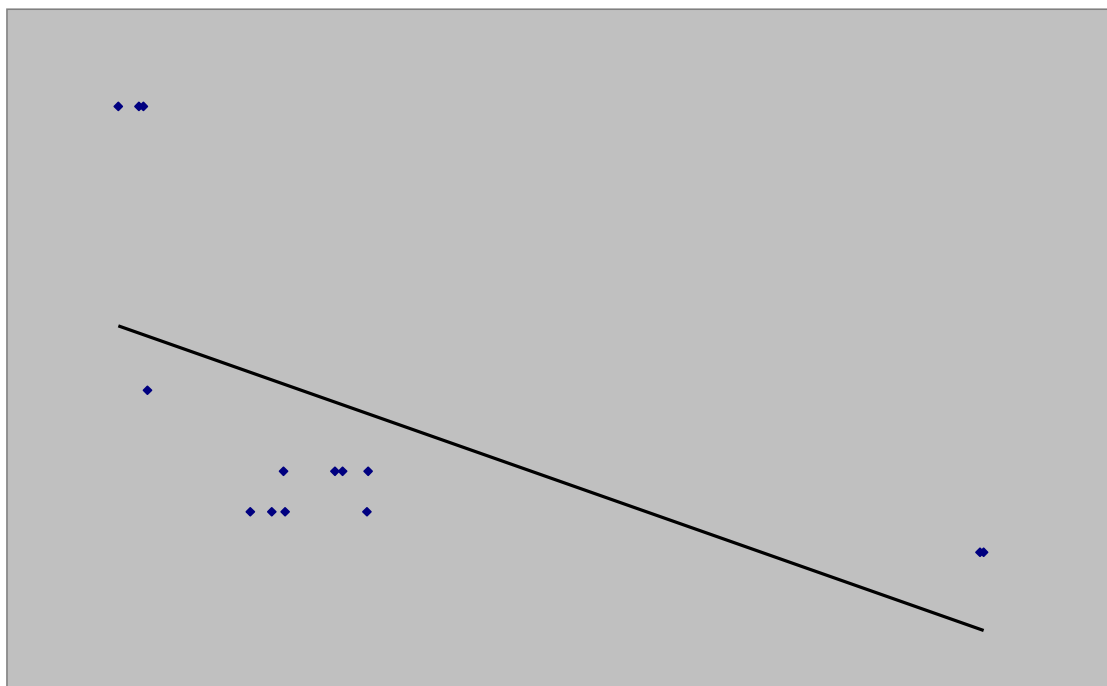


Figure (28):correlation between anti-C1q Abs titer& AI in group Ia

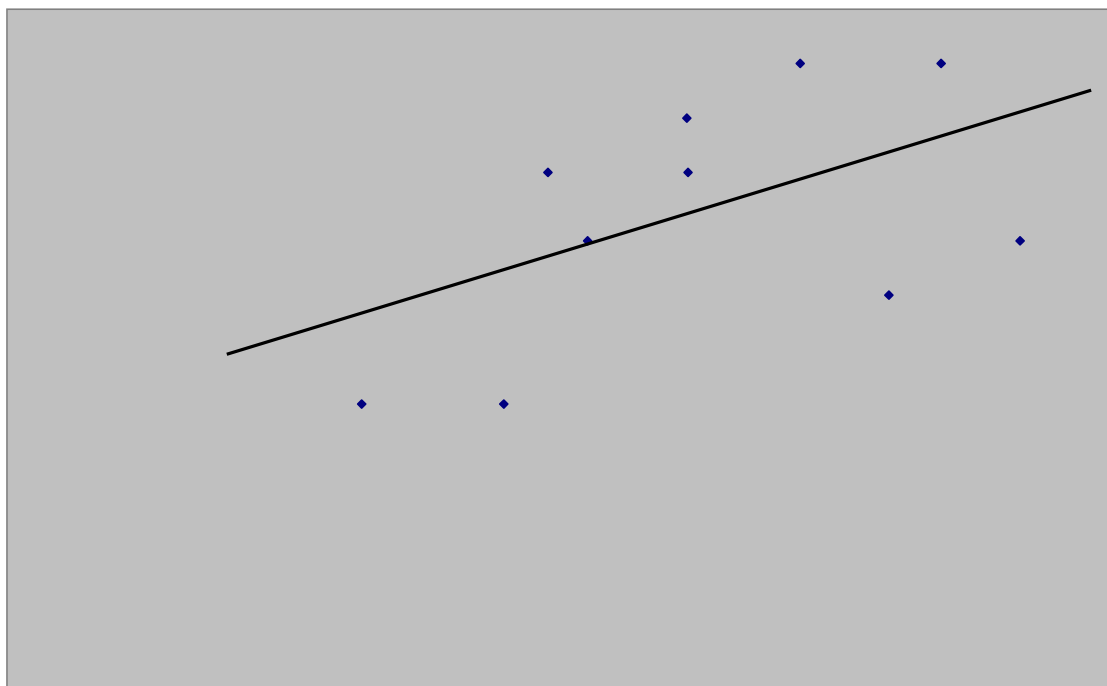
correlation between anti-C1q Abs titer&CI in group Ia

*Figure (29):correlation between anti-C1q Abs titer&CI in group Ia*

♦**Table (32):correlation between antiC1q Abs titer& Activity Index and Chronicity Index, in group Ib1:-**

Index	R	p
AI	0.7	*<0.05
CI	- 0.12	>0.05

correlation between antiC1qAbs tite&AI in group Ib1

*Figure (30):correlation between anti-C1q Abs titer & CI in group Ib1*

♦**Table (33): Sensitivity, specificity, positive predictive value (PPV) and negative predictive value (NPP) of anti-C1q Abs for lupus nephritis:-**

Anti-C1q Abs	Sensitivity	Specificity	PPV	NPP
Percentage	95.8%	80.76%	82.1%	95.4%

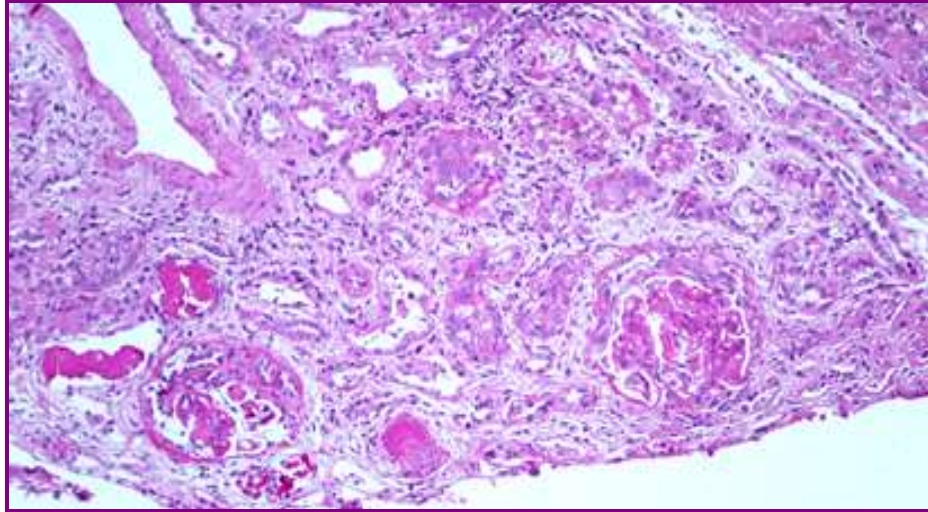


Figure (31): Diffuse proliferative lupus nephritis with moderate chronicity(WHO Class IV LN) (hematoxylin and eosin stain, original magnification x200).

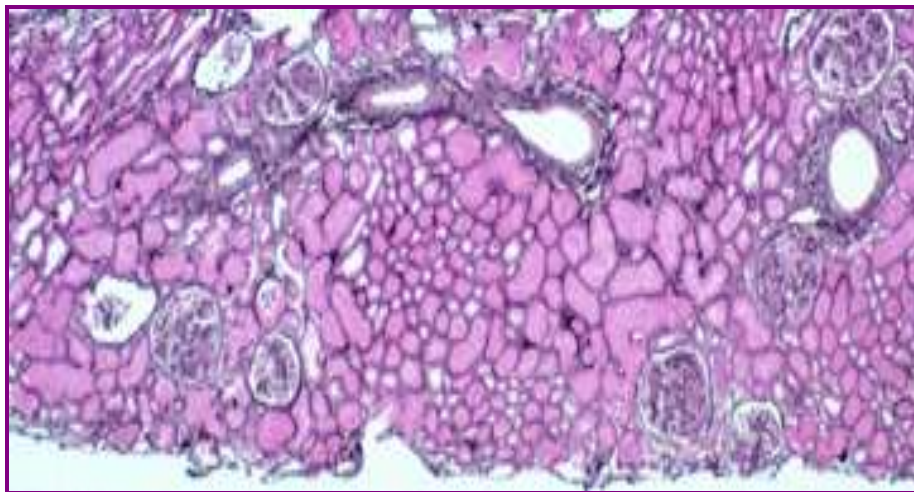


Figure (32): The focal segmental nature of proliferative lupus nephritis with moderate activity (WHO Class III LN) (hematoxylin and eosin stain, original magnification x200).

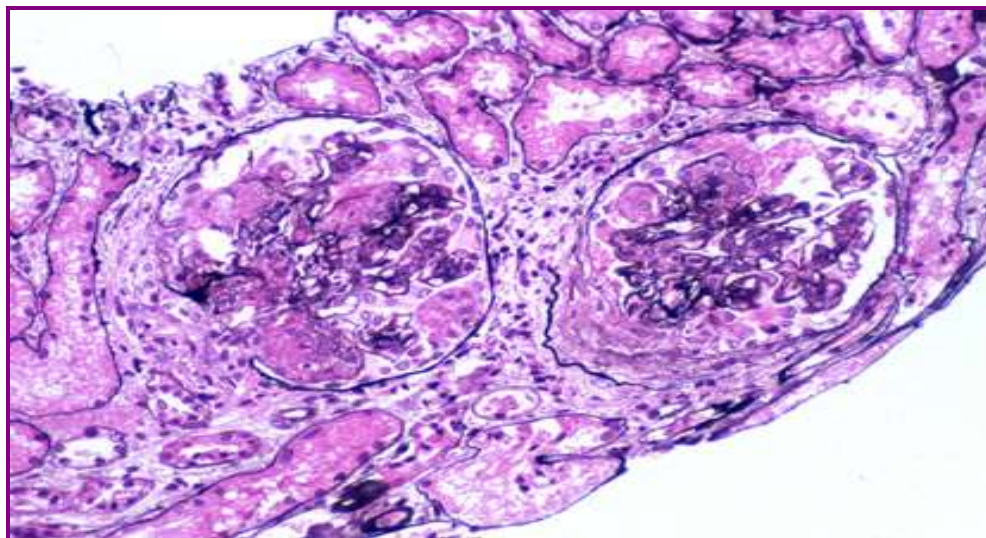


Figure (33): The focal segmental nature of proliferative lupus nephritis (WHO Class III LN) (hematoxylin and eosin stain, original magnification x200).



Figure (34): Technique of ultrasound-guided biopsy procedure in one of our patients.