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Original Article

Nephritis and Its Outcome in Systemic Lupus Erythematosus

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Abstract

Background: Nephritis is usually the most serious manifestation of systemic lupus erythematosus (SLE). Nephritis is asymptomatic in most lupus patients and small proportion of SLE patients have nephrotic syndrome. The patient can develop end-stage renal disease within 2 years if diffuse proliferative glomerulonephritis is untreated, hence aggressive immunosuppression is required.

Materials and Methods: A total of 25 patients who were admitted and fulfilled the inclusion/exclusion criteria were evaluated by history, physical examination and lab tests.

Results: Out of 25 patients, 23 were females and only 2 were male, i.e., 92% were female which shows that this disease is more common in females than in males. The age distribution showed that the disease is more common in younger age group. 13 patients were of age group 31-40 (52%), 10 were 21-30 (40%), 1 patient <20 years (4%), and between 41 and 50, there were 2 patients (8%). Mostly the females were of child bearing age group.

Conclusion: Out of 25 patients of SLE studied, 22 developed nephritis and 3 did not. Among the 22 patients of nephritis, 12 (48%) had complete remission, 2 (8%) had multiple relapses, 1 (4%) had late onset lupus nephritis (LN), 1 (4%) had resistant LN 1 (4%) lost follow-up, 2 (8%) expired, and 1 (4%) developed end-stage renal disease.

Key words: Clinical profile, End-stage renal disease, Females, Nephritis, Systemic lupus erythematosus

INTRODUCTION

Systemic lupus erythematosus (SLE) is a disease in which organs and cells are damaged due to tissue binding autoantibodies and immune complexes. 90% of patients are women of child bearing years; people of both sexes, all ages and all ethnic groups are susceptible. In India, its prevalence is 3.2/100000 populations.

Pathogenesis and Etiology

Interactions between susceptible genes and environmental factors result in abnormal immune response. Those responses include (1) activation of innate immunity (dendritic cells) by DNA, DNA in immune complexes and RNA in RNA/protein self-antigens, (2) lowered activation thresholds of adaptive immunity cells (antigen-specific T- and B-lymphocytes), (3) ineffective regulatory and inhibitory CD4+ and CD8+ T-cells, and (4) reduced clearance of apoptotic cells and of immune complexes. Self-antigens (nucleosomal DNA protein; RNA protein in Sm, Ro and La, and phospholipids) are available for recognition by the immune system in surface blebs of apoptotic cells, thus antigens, autoantibodies and immune complexes persist for prolonged period of time allowing inflammation and disease to develop. Immune activation of circulating and tissue bound cells accompanied by increased secretion of proinflammatory tumor necrosis factor and Type 1 and 2 interferons (INF) and the B cell-driving cytokines B-lymphocyte stimulator and interleukin-10 (IL). Upregulation of genes induced by INF is a genetic "Signature" of SLE. However, lupus T and natural killer cells fail to produce enough IL-2 and transforming growth factor to induce regulatory CD4+ and inhibitory CD8 + T-cells. The result of these abnormalities is sustained

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production of pathogenic autoantibodies and immune complexes, which bind to target tissues, with activation of complement and phagocytic cells that recognize Ig-coated circulating blood cells. Activation of complement and immune cells leads to release of chemotaxins, cytokines, chemokines, vasoactive peptides, and destructive enzymes. In the setting of chronic inflammation, accumulation of growth factors and products of chronic oxidation contribute to irreversible tissue damage in glomeruli, arteries, lungs, and other tissues.

In lupus nephritis (LN), most of the time there is deposition of glomerular immunoglobulin and complement component. The basic immunologic abnormality in the kidney is the accumulation or deposition of Ig and complement components in the mesangium. Such depositions depend on a variety of factors, including mesangial and mononuclear phagocyte function, size, composition, and perhaps, charge of the immune complexes or antigen, as well as other poorly defined considerations. This pattern of immune deposition represents the background on which the more severe glomerular lesions are imposed. Depending on the composition and circulatory load of immune complexes, capillary wall localization may also occur. The local glomerular reaction and cellular constituents form the circulating blood may result in varying pattern of glomerular injury seen by light microscopy. Alternatively, immune complexes may form *in-situ* as a consequence of circulating antibody reacting with a planted non glomerular antigen such as ssDNA or an intrinsic glomerular antigen. In addition to classification of the glomerular lesions according to general appearance on light, election, and immunofluorescence microscopy, they are also classified into active and chronic lesion.

Classification of LN

International society of nephrology and renal pathology society.

Class I: Minimal mesangial LN
Class II: Mesangial proliferative LN
Class III: Focal LN
Class IV: Diffuse LN
Class V: Membranous LN
Class VI: Advanced sclerotic LN.

Nephritis is usually the most serious manifestation of SLE. Nephritis is asymptomatic in most lupus patients and small proportion of SLE patients have nephrotic syndrome. The patient can develop end-stage renal disease within 2 years if diffuse proliferative glomerulonephritis is untreated,

Table 1: Gender wise distribution of 25 patients having systemic lupus erythematosus

Gender	Count (%)
Male	2 (92)
Female	2 (38)
Total	25 (100)

Table 2: Age distribution

Age (years)	Count (%)
<20	1 (4)
21-30	10 (40)
31-40	13 (52)
41-50	2 (8)
Total	25 (100)

Table 3: Presenting symptoms

Symptoms	Count (%)
Nephritis	
Swelling if face legs	7 (28)
Headache	1 (4)
Decreased micturation	1 (4)
SLE	
Other symptoms	16 (64)
Total	25 (100)

SLE: Systemic lupus erythematosus

Table 4: Pulse

Pulse	V1	V2	V3	V4	V5	V6	V7
N	25.00	24.00	24.00	23.00	23.00	23.00	23.00
Mean	84.32	82.33	82.08	81.57	81.74	82.87	83.04
Median	82.00	83.00	82.00	82.00	82.00	84.00	84.00
Min	64.00	68.00	66.00	64.00	66.00	62.00	64.00
Max	116.00	92.00	94.00	90.00	90.00	90.00	100.00
Siddev	11.21	6.61	6.71	6.74	6.42	7.16	7.74
P-value		0.233	0.160	0.084	0.0954	0.331	0.575

Table 5: SBP

SBP	V1	V2	V3	V4	V5	V6	V7
N	25.00	24.00	24.00	23.00	23.00	23.00	23.00
Mean	129.04	127.50	124.75	122.65	121.65	123.39	124.43
Median	120.00	130.00	120.00	120.00	120.00	120.00	120.00
Min	110.00	120.00	120.00	110.00	90.00	120.00	120.00
Max	210.00	140.00	140.00	149.00	140.00	140.00	150.00
SIDDEV	19.36	6.08	6.46	7.31	9.18	5.51	7.84
P-value		0.812	0.270	0.104	0.052	0.148	0.301

SBP: Systolic blood pressure

Table 6: Diastolic BP

SBP	V1	V2	V3	V4	V5	V6	V7
N	25.00	24.00	24.00	23.00	23.00	23.00	23.00
Mean	83.84	82.75	81.25	81.65	79.91	80.43	82.61
Median	80.00	80.00	80.00	80.00	80.00	80.00	80.00
Min	78.00	80.00	80.00	70.00	70.00	70.00	80.00
Max	110.00	90.00	90.00	90.00	90.00	90.00	90.00
Siddev	7.57	4.45	3.05	4.77	4.07	3.67	4.49
P-value		0.553	0.088	0.1687	0.015	0.012	0.425

BP: Blood pressure

hence aggressive immunosuppression is required. Drugs of choice in nephritis patients are mycophenolate mofetil (MMF), cyclophosphamide, and azathioprine. With all these agents glucocorticoids are used, LN tends to be an ongoing disease, with flares requiring retreatment for many years. For more people of LN, accelerated atherosclerosis becomes important after. Several years of disease, attention must be given to control of blood pressure, hyperlipidemia, and hypertension.[1-6]

Aims and Objectives

- To study the incidence of LN in 25 patients of SLE
- To study the outcome in these cases of LN
- To study the efficacy of different immunosuppressive agents.

MATERIALS AND METHODS

This is a hospital based prospective observational study.

Diagnosis of systemic erythematosus on the following American College of Rheumatology (ACR) criteria is made:

1. Malar rash
2. Discoid rash
3. Photosensitivity
4. Oral ulcer
5. Arthritis
6. Serositis
7. Renal disorder
8. Neurological
9. Hematological: Anemia/leukopoenia/thrombocytopenia/erythrocyte sedimentation rate (ESR)
10. Immunological: Anti-dsDNA/Anti-SM/Anti-phospholipids
11. Antinuclear antibody (ANA)

Out of above 11 criteria at least 4 are required to fulfill. All 4 may not be present at beginning but may develop sequentially.

The patients are than clinically evaluated in detail for renal involvement, urine routine and microscopy, renal function test (RFT), C3-C4 levels, 24 h urine proteins, and renal biopsy.

Sample Size

A total number of 25 patients attending tertiary care hospital were considered for this study.

Inclusion Criteria

- Patient fulfilling the ACR criteria for diagnosis of SLE are enrolled
- Newly diagnosed case of SLE.

Exclusion Criteria

1. Pregnancy
2. Planning for pregnancy
3. Patients on oral contraceptive pills
4. Known case of acute or chronic renal failure
5. Known case of hypertension and diabetic mellitus
6. Known case of cardiac diseases.

Methods

This study is conducted in a tertiary care hospital in Navi Mumbai. All patients who fulfill the ACR criteria were enrolled.

This study is prospective study of 18 months.

Evaluation Visit Schedule

Patients are evaluated at 3 months intervals:

- Clinically and routine laboratory investigations - 3 monthly
- Serology if required - 6 months.

Table 7: CBC result

CBC result	Count (%)
Improved	20 (80)
Stable	2 (8)
Leukocytosis and thrombocytosis	1 (4)
Lost to follow-up	1 (4)
Death	1 (4)
Total	25 (100)

CBC: Complete blood count

Table 8: ESR result

ESR result	Count (%)
Active disease	3 (12)
Disease under control	13 (52)
No disease activity	6 (24)
Lost to follow-up	1 (4)
Death	1 (4)
End-stage renal disease	1 (4)
Total	25 (100)

ESR: Erythrocyte sedimentation rate

Table 9: Urine R/M (proteinuria)

Urine R/M (proteinuria)	V1	V2	V3	V4	V5	V6	V7
Absent	11	6	8	17	15	17	15
Mild proteinuria	2	7	10	1	6	2	2
Moderate proteinuria	4	4	3	3	1	2	4
Severe proteinuria	0	6	3	2	0	0	0
Very severe proteinuria	8	1	0	0	1	2	2
LUP	0	0	0	1	1	1	1
Death	0	1	1	1	1	1	1
Total	25	25	25	25	25	25	25

P=0.906, Not-significant (V1-7)

Laboratory Investigations

Complete blood count (CBC), ESR, RFT, urine routine and microscopy, 24 h urine proteins - 3 monthly.

Blood sugar, ANA by IF, anti dsDNA, C3-C4 levels, lipid profile-6 monthly, if required.

RESULTS

This study studied 25 patients with systemic lupus erythematosus for LN (Tables 1-20).

Table 10 shows that 24 urine protein is more important than simple urine routine and microscopy.

Table 11 shows that even though patient has heavy proteinuria RFT can be normal.

Table 12 shows that dyslipidemia is common association with lupus nephritis

Table 13 shows that ANA is positive in all the patients of SLE.

Table 14 shows that anti-dsDNA is commonly positive in lupus nephritis.

Table 10: Urine 24 h protein level

Protein level	V1	V2	V3	V4	V5	V6	V7
N	13.00	18.00	19.00	19.00	19.00	18.00	19.00
Mean	4.30	5.00	1.30	0.88	0.49	1.02	0.84
Median	3.92	2.00	1.00	0.55	0.42	0.39	0.39
Min	0.10	0.50	0.46	0.13	0.02	0.12	0.13
Max	14.70	4.88	4.29	4.12	1.20	6.10	4.73
Stddev	4.25	1.09	1.08	0.91	0.35	1.58	1.29
P-value		0.006	0.005	0.019	0.006	0.020	0.015
Protein level	**V1**	**V2**	**V3**	**V4**	**V5**	**V6**	**V7**
Nephrotic	8	7	2	1	0	2	2
Nephrotic	5	11	15	10	7	3	3
Normal	1	0	2	8	12	13	14
Not done	12	7	6	6	6	7	6
Total	25	25	25	25	25	25	25

P=0.008, Significant

Table 11: RFT

RFT	V1	V2	V3	V4	V5	V6	V7
Normal	20	22	23	22	21	21	21
Mildly deranged RFT	3	2	0	1	2	2	2
Moderately deranged RFT	1	0	1	0	0	0	0
Severely deranged RFT	1	0	0	0	0	0	0
LUP	0	0	0	1	1	1	1
Death	0	1	1	1	1	1	1
Total	25	25	25	25	25	25	25

P=0.636, Not-significant (V1-7). RFT: Renal function test

Table 15 shows that complement levels are significantly reduced in lupus nephritis.

DISCUSSION

SLE is a multisystem autoimmune disease. The wide range of organ system involved in the disease includes musculoskeletal, cutaneous, hematological, neurological, cardiac, pulmonary, renal, gastrointestinal, vascular, and ocular systems. LN is one of the most serious manifestations of SLE and it usually arises within 5 years of diagnosis.[7-12]

This study is done in a tertiary center at Navi Mumbai. The patient fulfilling the ACR criteria for SLE were included

Table 12: Dyslipidemia development status for nephritis patients

Dyslipidemia status	V1	V2	V3	V4	V5	V6	V7
Positive	6	7	0	0	0	1	0
Negative	16	14	21	20	20	19	20
LUP	0	0	0	1	1	1	1
Death	0	1	1	1	1	1	1
Not done	3	3	3	3	3	3	3
Total	25	25	25	25	25	25	25

P=0.012, not-significant (V1-7)

Table 13: ANA

ANA	V1 (%)
Homogenous	23 (92)
Speckled	2 (8)
Total	25 (100)

ANA: Antinuclear antibody

Table 14: ANTI-dsDNA

ANTI-dsDNA	V1	V2	V3	V4	V5	V6	V7
Positive	15	6	0	0	0	0	1
Negative	2	0	0	1	0	0	0
Not done	8	18	24	22	23	23	22
LUP	0	0	0	1	1	1	1
Death	0	1	1	1	1	1	1
Total	25	25	25	25	25	25	25

P=0.003, Significant (V1-7)

Table 15: C3-C4

C3-C4	V1	V2	V3	V4	V5	V6	V7
C3-C4 mild	12	Not done	5	4	1	1	3
C3-C4 moderate	2		0	0	0	0	0
C3-C4 normal	1		10	9	12	14	11
Note done	10		9	10	10	8	9
LUP	0		0	1	1	1	1
Death	0		1	1	1	1	1
Total	25	25	25	25	25	25	25

P=0.003, Significant (V1-7)

in the study. 25 patients were included in the study. Out of 25 patients, 23 were females and only 2 were male, i.e., 92% were female which shows that this disease is more common in females than in males. The age distribution showed that the disease is more common in younger age group. 13 patients were of age group 31-40 (52%), 10 were 21-30 (40%), 1 patient <20 years (4%), and between 41 and 50; there were 2 patients (8%). Mostly the females were of child bearing age group. These findings are as per with other studies that it is more common in female in child bearing age group.

Most of the LN patients present with symptoms of hypertension, proteinuria, and renal failure. Among our 25 patients, 9 patients (36%) presented with signs and symptoms related to these conditions. Common symptoms were swelling of the face and legs, decreased micturation and headache. Rest 16 patients (64%) presented with other symptoms of SLE such as on and off fever, joint pain, malar rash, photosensitivity, and alopecia. Active LN can present with such other symptoms of active SLE.

One patient had almost all the systems involved namely musculoskeletal, cutaneous, renal, central nervous system, pulmonary, hematologic, and cardiovascular. Such presentation is rare.

In the study, pallor was significant finding ($P = 0.002$) when compared to other findings of nephritis, i.e., edema ($P = 0.308$) which was not significant finding. Even sororities was not significant ($P = 0.143$). $P = 0.308$.

The patients were subjected to different tests at each follow-up. The ESR results showed significant improvement at the end of the study. 52% patients had disease activity under control, 12% had active disease, and 24% were stable. Many patients also had improved CBC results at the end of the study. 80% of patients had improved, 8% had no change, 4% leukocytosis and thrombocytosis, 4% lost follow-up, and 4% expired.

Urine routine and microscopy, and 24 h urine protein excretion are very important to diagnose LN early in the disease. The urine routine and microscopy (when compared between first and last visits) did not come out as an indicator for good prognosis. As it was hypothesized ($P = 0.905$), but 34 h urine protein was a significant prognostic indicator during each visit ($P = 0.008$). Bastian *et al.*, concluded in their study that LN can be diagnosed on following dependable variables:

1. Renal biopsy
2. Proteinuria >0.5 g/24 h, or
3. One of the following features such as proteinuria >2+, serum creatinine >1.4, creatinine clearance< or = to 79 ml/min, 10 RBC or WBC per high power field.

However, in this study only renal biopsy and 24 h urine proteins were found to be significant during the follow-up.

Low C3-C4 levels are associated with active nephritis, especially focal proliferative and diffuse proliferative LN. C3 and C4 levels turned out to be significant predicators LN in this study ($P = 0.003$).

In LN complement levels are reduced due to deposition in the renal tissue at various sites. Dolley *et al.* had shown that low C3. C4 levels in associated with focal and diffuse proliferative LN, renal biopsy should be considered in the disease course as early as possible. Out of 25 diagnosed cases of systemic LN, 20 patients undergone renal biopsy and 5 did not because of poor socioeconomic condition. In country like India where renal biopsy study is costly investigation all patients could not be advised renal biopsy.

Table 16: Renal biopsy

Renal biopsy	Count (%)
Performed	20 (80)
Not performed	5 (20)

Table 17: LN classification

Renal biopsy	V1	V2	V3	V4	V5	V6	V7	Total
Class I	0	0	0	0	0	0	0	0
Class II	0	1	0	0	0	0	1	2
Class III	1	4	1	0	0	0	0	6
Class IV	8	2	0	1	1	0	0	12
Class V	0	0	0	0	0	0	0	0
Class VI	0	0	0	0	0	0	0	0
Note done	16	17	23	22	22	23	22	-
LUP	0	0	0	1	1	1	1	-
Death	0	1	1	1	1	1	1	-
Total	25	25	25	25	25	25	25	20

LN: Lupus nephritis

Table 18: Medication

Medication	*n*	Result
Cyclophosphamide	12	Improved-11 LUP-1
Cyclophosphamide+MMF	2	Not improved-1 Relapsed-1
Cyclophosphamide+methylprednisolone	2	Improved-1 Death-1
Dialysis	1	Improved-1
MMF	3	Improved-1 Relapse-1 Death-1
Steroids	4	Late LN-1 Not developed nephritis-3
Dialysis+steroids	1	Improved-1

LN: Lupus nephritis, MMF: Mycophenolate mofetil

Out of five, three patients did not have proteinuria, hence renal biopsy was not advised and 2 patients, who had proteinuria, were not affording. Out of 20 biopsied patients, 12 (60%) had Class IV LN, 6 (30%) had Class III, and 2 (10%) had Class II LN. None of the patients had Class I or Class VI. Das *et al.*, also had similar incidence, twenty-too (75.9%) patients had diffuse proliferative glomerulonephritis (Class IV), 6 (20.7%) focal proliferative glomerulonephritis (Class III), and one (3.4%) Class V. Dyslipidemia is a common finding in LN. Nephritic syndrome leads to dyslipidemia. Which is a consistent finding in this study ($P = 0.012$), when compared between visits 1 and visit 7.

When LN is properly treated, it has a good prognosis. The first-line treatment given, for most patients, was injection cyclophosphamide 12 patients received cyclophosphamide 500 mg/m^2 only as the treatment. After 6 cycles of cyclophosphamide therapy, patients were given maintenance dose of cyclophosphamide every 3 months for 6 cycles. Oral azathioprine as maintenance therapy was given later. 11 out of 12 patients improved and 1 patient the follow-up. Thus at per with other studies cyclophosphamide had good response in LN. 2 patients did not respond to cyclophosphamide in these patients MMF was given as second line therapy. 1 of these 2 patients had resistant LN not controlled by MMF. We had planned to give biologics (i.e., rituximab) for this patient. Melander *et al.* had shown in their study of 20 patients that rituximab was effective in Class IV LN. The other patient had good response to MMF but relapses are common with LN, this patient also had relapses and the dose of MMF had to be increased.

2 patients had presented with severe flare, hence they were given cyclophosphamide and methylprednisolone as the first-line therapy. 1 patients had improved and put on azathioprine maintenance therapy:

1 patient succumbed to death due to complications.

1 patient end-stage renal disease and she was being treated with dialysis.

3 patient received MMF as the first line therapy. 1 of these patients was not compliant for the treatment and expired. This proved that patient education and compliance are very important in this disease. 1 patient had relapses and the dose had to be adjusted and 1 patient improved without any relapses.

4 patients were given only steroids for treatment of SLE. 1 among them developed nephritis later in the disease course and 3 patients did not develop nephritis.

One case needs to be emphasized. This patient presented with urinary tract infection and her RFTs were severely deranged. She was properly worked up and she and ANA positive. Tentative diagnosis of LN was made and she given dialysis and injectable steroids. Then, renal biopsy was done showing Class IV LN. When she renal functions were settled, she was put on deflazacort and now she is doing well on steroids itself.

All patient received hydroxychloroquine and steroids. Most of the patients received supportive therapy such as enalapril for proteinuria and statins dyslipidemia. Those who had hypertension were given antihypertensives and hypertension was strictly controlled.

Table 19: SLEDAI score

Result	Count (%)
Mid/moderate flare (>3 and≤12)	18 (72)
Complete remission	8 (32)
Multiple relapses	2 (8)
Death	1 (4)
Partial remission	2 (8)
Not developed nephritis	3 (12)
Resistant LN	1 (4)
Late LN	1 (4)
Severe flare (>12)	7 (28)
Complete remission	4 (16)
Lost to follow-up	1 (4)
Death	1 (4)
End-stage renal disease	1 (4)
Total	25 (100)

LN: Lupus nephritis, SLEDAI: Systemic lupus erythematosus disease activity index

Table 20: Final result

Result	Count(%)
Complete remission	12 (48)
Partial remission	2 (8)
Multiple relapses	2 (8)
Not developed nephritis	3 (12)
Resistant LN	1 (4)
Late LN	1 (4)
Lost to follow-up	1 (4)
Death	2 (8)
End-stage renal disease	1 (4)
Total	25 (100)

LN: Lupus nephritis

Incidence of LN

During this study, 22 patients developed nephritis and 3 did not develop. Thus, the incidence of nephritis is 88%.

SLE disease activity index (SLEDAI) is used to see the flares. If it is >3 and <12, it is mild to moderate flare; if it is >12, it is severe flare. SLEDAI is calculated at the presentation and at the end of the study. 18 patients (72%) had mild to moderate flare. Among these 8 had complete

remission, 2 had partial remission, 2 had multiple relapses, 1 had resistant nephritis, 1 developed nephritis later, 1 died, and 3 did not develop nephritis.

Out of 25 patients had severe flare, 4 had complete remission, 1 expired, 1 developed end-stage renal disease, and 1 patient lost the follow-up.

Result

Out of 25 patients of SLE studied, 22 developed nephritis and 3 did not. Among the 22 patients of nephritis, 12 (48%) had complete remission, 2 (8%) had multiple relapses, 1(4%) had late onset LN, 1 (4%) had resistant LN 1 (4%) lost follow-up, 2 (8%) expired, and 1 (4%) developed end-stage renal disease.

CONCLUSION

The following the findings from this study:

- 23 patients were females and 2 were males.
- Maximum female patients were of child bearing age group.
- Out of 25 patients, 9 patients presented with symptoms associated with nephritis, and 16 patients presented with other symptoms of SLE.
- The incidence of LN is 88%. Out of 25 patients, 22 patients developed nephritis and 3 did not.
- Pallor was significant finding in the study on general physical examination, but not edema and serositis.
- Good response in terms of CBC is seen with treatment.
- ESR is determinant of active disease. ESR improved in 52% patients.
- ANA and anti-dsDNA are positive in SLE.
- Determination of 24 h urine proteins is the best method for early diagnosis of LN.
- Renal biopsy is indicated early in the disease. 12 patients had Class IV LN, 6 had Class III, and 2 patients Class II LN. None of the patients had Class I or Class VI LN.
- Renal biopsy and 24 h urine proteins are the important determinants in the outcome of LN.
- C3-C4 levels are significantly decreased in severe LN dyslipidemia is present in LN.
- Cyclophosphamide is cheap and effective treatment of LN. 11 out of 12 patients improved with cyclophosphamide only.
- Out of 25 patients of SLE studied, 22 developed nephritis and 3 did not. Among the 22 patients of nephritis, 12 (48%) had complete remission, 2 (8%) had partial remission, 2 (8%) had multiple relapses, 1 (4%) had late onset LN, 1 (4%) had resistant LN, 1 (4%) lost follow-up, 2 (8%) expired, and 1 (4%) developed end-stage renal disease.

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