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Research Article

A PROSPECTIVE OBSERVATIONAL STUDY ON THE CLINICAL PROFILE AND OUTCOMES OF LUPUS NEPHRITIS IN PATIENTS OF SYSTEMIC LUPUS ERYTHEMATOSUS

Netaji Garad

Department of Gastroenterology & Hepatology, Shri Balaji Institute of Medical Science, Raipur, Chhattisgarh 492001

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ABSTRACT

Background &Objective: Systemic lupus erythematosus (SLE) is a systemic autoimmune disease, with a wide range of clinical presentations resulting from its effect on multiple organ systems. This research was conductedto elucidate clinical profile and the outcomes of lupus nephritis (LN) in patients of SLE.
Methods: This prospective observational study of forty patients was conducted in Jawaharlal Nehru Hospital and Research Centre, Bhilai, Chhattisgarh, India in 2011-12. Patients follow-up was done on quarterly basis during the period of one year. The clinical manifestations, laboratory parameters and other imaging findings were analyzed.
Results: Out of 40 patients studied, 37 were females and 3 males with sex ratio 13:1. The mean age at presentation of lupus nephritis was 22.67 ± 3.51 years for male and 29.89 ± 8.40 years for female. Most common symptoms were myalgia (80%), arthralgia (80.30%), weight loss (75%) and oral ulcer (63%). 50% of patients had class IV LN with increased Erythrocyte Sedimentation Rate (ESR)($p > 0.05$) followed by significant decrease in ESR, serum creatinine, 24 hours urinary protein level ($p < 0.05$) and increased in the level of C3 and C4 post-treatment. Two patients (5%) in the study died within 6 month of diagnosis and belonged to class IV LN.
Interpretation & Conclusion: Most of LN are often asymptomatic and can be diagnosed by simple laboratory investigation and urinary examination. Immunosupressive and cytotoxic drugs have major role in treating serious form of disease and decreasing morbidity and mortality. At the end of 6 month, complete response was found in 16.66%with CYC and at 12 months 43.75%. The complications during follow-up period were gastrointestinal side effects, IGT, osteoporosis and infections.

INTRODUCTION

Systemic Lupus Erythematosus (SLE) is a chronic inflammatory disease characterized by multiple organ involvement, production of autoantibodies to nuclear components and local formation or deposition of immune complexes in different organs. Lupus Nephritis (LN) is a common and serious manifestation of SLE and more commonly seen in adolescent female. Lupus is a multisystemic autoimmune disease of unknown origin.[1] The incidence and prevalence of Lupus nephritis are influenced by age, gender, ethnicity, geographic region and environmental factor. The symptoms of lupus nephritis are generally related to hypertension, proteinuria, microscopic hematuria and renal failure.[2] Since nephritis is asymptomatic in most of lupus patients, urinalysis should be done in patients suspected of having SLE.[3]

The principal goal of therapy in lupus nephritis is to normalize renal function or, at least, to prevent the progressive loss of renal function.[3] Over the ensuing 35-40 years, advances in clinical medicine including cytotoxic therapy, immunosuppressive, antihypertensive drugs and new antibiotics, as well as the introduction of dialysis and renal transplantation have resulted in a drop in 5-year mortality from LN to less than 10%.[4] In the early 1950s lupus nephritis was the main contributor to early death in SLE patients, with an estimated 5-year survival rate of 25-40% following the diagnosis of nephritis. [5]

Based on the introduction mentioned above and retrospective and prospective observational study done below, the goal of this study is to evaluate the clinical profile, histopathological features, complications, renal involvement and the outcome in patients with SLE; considering Indian demographics. Since, there is a lacunae in this type of study especially in the Indian setup, this work may provide a more comprehensive view of illness an insight into potential etiology and patient management which will ultimately guide the development of policies designed to reduce the burden of mortality and morbidity due to SLE in India.

Corresponding author:* **Netaji Garad
Department of Gastroenterology & Hepatology, Shri Balaji Institute of Medical Science, Raipur, Chhattisgarh 492001

MATERIALS & METHODS

Site & Source of data: The present study was done in the Department of General Medicine, Jawaharlal Nehru Hospital and Research Centre Bhilai, Chhattisgarh; over a period from 30th November, 2011 to 29th October 2012.

Sample Size: The sample size for the study was calculated using the following sampling method:

$$n = \frac{Z^2PQN}{e^2 (N - 1) + Z^2PQ}$$

Z=1.96
N=50 (Number of SLE patients enrolled in the study).
P= Prevalence of Lupus[1] = 0.67
Q=1-P = 0.33
e=0.05

Therefore, n= 43.70 i.e. 44 Lupus Nephritis patients were enrolled but study was concluded with 40 patients as remaining four patients didn't satisfied the following inclusion and exclusion criteria.

Inclusion Criteria

1. Patients diagnosed as SLE based on the ACR clinical criteria.
2. SLE Patients came for follow up in medical OPD.
3. SLE patients admitted for cytotoxic drugs therapy.
4. Patients with age 12 years or more.

Exclusion Criteria

1. Congenital renal disease.
2. Diabetic nephropathy.
3. Pregnant female.
4. HIV positive individual.
5. Patients less than 12 years of age.

Procedure used

In this study, 40 subjects that fulfilled the inclusion and exclusion criteria were recruited over a period of one year. RENAL function assessment was in terms of:

1. Clinical symptoms.
2. General and systemic examination findings.
3. USG abdomen and pelvis.
4. Renal function test & Urine routine microscopy.
5. 24 hrs urinary protein, spot protein / creatinine ratio.
6. Renal biopsy by automated renal biopsy instrument.
7. Lipid profile.
8. Markers of disease activity- ESR, double stranded DNA (ds DNA).
9. Other relevant investigations depending on other organ involvement.

Histologic class of nephritis was classified according to the original World Health Organization classification: class I: normal, class II: mesangial proliferation, class III: focal proliferative lupus nephritis (<50% glomeruli involved), class IV: diffuse proliferative lupus nephritis (≥50% glomeruli involved), and class V: pure membranous lupus nephritis.[6,7] In addition, class VI: was used to denote advanced glomerulosclerosis.

Data collection & Statistical methods

A pre-designed, self-administered proforma was designed keeping the objectives of the study at the centre point. The purpose of the study was explained to the patient and informed consent was obtained. Patients were selected for study which satisfies all criteria. In the construction of the proforma, utmost care was taken to make it broad based, so that all the aspects desired to be studied could be incorporated in its body.

Patients were monitored during the period of hospital stay to note their outcome. All patients were followed up to discharge or other outcomes, whether in the department or after being transferred to other wards. Follow up data were retrieved from digital and written patient records, including discharge letters and any other relevant documentation. The data collected through the questionnaire was entered on pre-designed proforma. It was then tabulated in master chart with the help of Microsoft excel spread sheet. The categorical variables were presented as frequencies and percentage.

The data was analyzed with the help of SPSS Trial Version 22 statistical package. Quantitative variables were analysed and compared using parametric tests (student's t-test), whereas qualitative data was analyzed with the help of non-parametric tests (Chi-square test). P-values were derived. P-values lower than 0.05 were considered as significant.

Standard treatment protocol was followed for the treatment of lupus nephritis as well as other manifestation of SLE. Other co-existing diseases were also treated as per respective protocols.

Renal Biopsy

A Renal Biopsy is the "Gold Standard" for diagnosis of lupus nephritis and the basis for treatment strategies.[8] Indications of Renal Biopsy in SLE were as follows:

1. A patient with glomerular disease in whom the diagnosis was not certain.
2. Mild proteinuria and hematuria.
3. Nephrotic Syndrome with bland sediment.
4. A repeat renal biopsy may be performed for late progression of the disease to distinguish between active lupus (which may require immunosuppressive therapy) and scarring of previous inflammatory injury.

Procedure of Renal Biopsy

In all patients in whom USG guided precutaneous renal biopsy was to be performed, it was established that the patient has a normal haemoglobin, platelet count, bleeding time, clotting time, prothrombin time and thromboplastin time before the procedure is undertaken. Renal biopsy was not done in the scheduled patients with hypertension (BP > 140/90 mm Hg) till the blood pressure was brought down to the normotensive range. Informed consent was obtained from all the patients.

The use of ultrasonography has made renal biopsy safer and easier. Both the ultrasound and the renal biopsy were performed on all patients by using Philips HD 11 and GE Logic P5 Ultrasound machines. Automated biopsy guns (gauze size and needle length-18g; 16cm) were used. The patient's skin surface was cleansed and draped. Then, 3.5 MHz transducer was used to localize the lower pole of the kidney. The distance to the biopsy point from the skin surface was

assessed and the skin surface was marked at the expected needle entry point. The skin, subcutaneous, and peri-renal tissues were infiltrated with local anesthetic using ultrasonic guidance, ensuring adequate local anesthesia along the intended biopsy pathway. A small incision was made through the weal to facilitate passage of the biopsy needle. The biopsy needle was then directed through the skin incision, and then under real-time ultrasonic guidance toward the lower pole of the kidney. Patients were asked to hold their breath when the needle approached the kidney. Advancement of the needle was halted when the tip of the needle was seen to penetrate the renal capsule. The gun was then fired, instantaneously advancing the cannula over the stylet and obtaining a core of renal parenchyma. The sampling time was less than 1 second. Repeat passes were performed to obtain two or three adequately sized biopsy specimens are obtained. After completion of the biopsy, patient was instructed to remain at bed rest for 24 hours. Blood pressure and pulse were monitored every 15 minutes for 1 hour, every 30 minutes for 1 hour, then hourly for 4 hours, and finally every 4 hours for the 24-hour period. A sample of each voided urine in a separate clear plastic specimen jar labeled with the date and time, which was kept at patient's bedside, was saved for inspection. The kidney was scanned to assess for the presence of hematoma or active bleeding. All the patients were observed in intensive care unit by trained renal nurses for a period of 24 hours post procedure. The patients were returned to the hospital ward for overnight observation if there were no complications. A second check ultrasonogram was done at 24 hours just before discharge to watch for any peri-renal bleed or hematoma which would have developed later.

RESULTS

Of 40 patients studied, there were 37 females and 3 males with sex ratio 13:1. The mean age at presentation was 26.28 ± 5.95years, with maximum were age group 20 -29 years. The mean age at presentation of LN was 22.67 ± 3.51 years for male and 29.89 ± 8.40 years for female. In patients with diagnosed LN, patients had mucocutaneous involvement (60%), musculoskeletal involvement (92.5%), gastrointestinal involvement (20%), cardiovascular involvement (17.5%), hematological involvement (60%), thyroid involvement (10%), respiratory involvement (17.5%) and neuropsychiatric involvements (12.5%). Most common symptoms were myalgia (80%), alopecia or significant hair loss (58%), arthralgia (80.30%), oral ulcer (63%), photosensitivity (58%) and weight loss (75%). 50% of our subjects fall under class IV LN.

There was significant increase in the level of Haemoglobin and platelet level but the change in WBC was not significant following standard treatment. All patients of LN were with increased ESR ($p>0.05$) and also found that there was a significant decrease in ESR following treatment. There was significant decrease in serum creatinine, 24 hours urinary protein level ($p<0.05$) and increased in the level of C3 and C4 following standard treatment. All 40 patients were ANA positive, most of them had homogeneous pattern and 70 % were Anti ds DNA positive ($p<0.05$). Most of other antibodies to ENA like Anti Sm, Anti Ro, Anti La, Anti RNP and Antinuclesome were found in class IV LN.

DISCUSSION

Demographic profile

In our study, out of 40 diagnosed cases of LN (n=37) were female and (n=3) were male. The sex ratio was 13:1. The difference in mean age of female and males was not statistically significant ($p>0.05$). Maximum patients i.e. 16 (40%) were in 20-29 years age group, only 5 (12.5%) patients were in the age group of more than 40 years. Mean duration of symptoms before diagnosis of LN was 3-6 months. M:F ratio was similar to other Indian study,[9] suggestive of female predominance but somewhat different ratio was documented in Western study group[10] which can be attributed to different geographic area, genetic and environmental factor and also most of the previous studies were retrospective and our study group involved prospective study of 40 diagnosed LN.

Symptoms

The retrospective Indian study by Dhir V *et al*[9] had fever (91.3%), malar rash (83%) and arthralgia (80.30%) as most common symptoms. Prospective follow up study by Satirapoj *et al*[9] had quite different baseline clinical characteristics. The renal symptoms such as edema feet (82.5%) and puffiness of face (62.5%) and decreased urine output (12.5%) were presentations in present study and these symptoms were comparable with Satirapoj *et al*.[11]

As different studies showed different baseline clinical characteristics one should not depend on clinical parameters to diagnose LN and hence routine urinary examination is must in case of SLE for early diagnosis of LN and to improve the outcome of LN

Table I Comparison of baseline clinical characteristics

Symptoms	Dhir *et al*[9]	Satirapoj *et al*[11]	Present study
Fever	91.30%	50%	60%
Oral ulcer	45.90%	45%	63%
Arthralgia	80.30%	65%	85%
Alopecia or significant hair loss	52.90%	30%	57.5%
Photosensitivity	34.60%	60%	58%
Discoid rash	NA	25%	52.5%
Malar rash	83.2%	65%	65%
Raynauld	<1%	20%	2.5%
Edema feet	NA	75%	90%
Puffiness of face	NA	NA	70%
Dyspnea	NA	NA	42.5%
Palpitaion	NA	NA	2.5%
Lymphadenopathy	NA	10%	5%
Myopathy	<1%	NA	2.5%

Table II Comparison of systemic involvement

System involved	Vaidya[12]	Satirapoj *et al*[11]	Present study
Cardiovacsular	5.3%	2.2%	17.5%
Respiratory	8%	10%	17.5%
Gastrointestinal	NA	15%	20%
Neuropsychiatric	13.3%	25%	12.5%
Hematologic	NA	55%	60%
Mucocutaneous	64%	85%	60%
Musculoskeletal	89.3%	65%	92.5%
Thyroid	NA	NA	10%

Class of Lupus Nephritis

In our study LN is defined according to SLEDAI[13,14] except for the criteria pyuria because this often turned out to be due to sample contamination. The histological finding of renal biopsy according to ISN/RPS[13] were class II 20% (n=8), class III 25% (n=10), class IV 50% (n=20) and class V 5% (n=2) which is somewhat relatable to some Indian and Western studies. Renal biopsy was done to diagnose class and severity of LN.

Table III Comparison of class of LN

Class of LN	Dhir *et al*[9]	Faurschou *et al*[16]	Present study
I	0	0%	0%
II	16.2%	11%	20%
III	26.5%	22%	25%
IV	44.9%	58%	50%
V	11.8%	8%	5%
VI	0.7%	1%	0

Renal Function Test

In present study, patients with increased serum urea (≥40 mg/dl,p>0.05) and serum creatinine (≥1.5 mg/dl,p>0.05) was 77.5% (n=31). All these patients had low GFR, calculated by Cockcroft-Gault equation.[1] There was significant decrease in serum creatinine level, decrease in 24hr urinary proteinuria and improvement in urinary sediment following standard treatment; in corelation with other Indian studies.[17]

ANA, Anti ds DNA, C3, C4 (Marker of disease activities)

The ANA positivity was present in all patients detected by immunoflurenscence method and most of them had homogenous pattern. ANA was best screening test of present study .The Anti ds DNA was positive in 70% (n= 28, p<0.05) patients. Low C3 and low C4 was present in 55 % (n=22, p>0.05) and 52.5% (n=21, p>0.05) respectively in our study at baseline. Low complement level was seen mainly in class IV lupus nephritis.

In study by Satirapoj *et al*,[11] almost all patients were ANA positive (94.4%) with varying patterns such as speckled, nucleolar, homogeneous and peripheral (41.2%, 23.2%, 29.4% and 0% respectively). Anti ds DNA was positive in 87.5%. In Indian study by Dhir *et al*,[7] ANA positivity was 95.3%, anti ds-DNA positivity was 52.8%.In present study, ANA positivity was similar to the other study,[10] while Anti ds DNA was positive in 70% in our study which is quite high as compared to Dhir V *et al*[9] as shown in table below which could be due to earlier diagnosis of LN by physician at our centre and initiation of treatments.

The difference in the level of C3 and C4 alongside other studies[9,18] was attributed to different methodology used for measuring C3 and C4 respectively.

Table IV Comparison of antibody markers

Markers	Korbet *et al*[18]	Dhir *et al*[9]	Present study
ANA positivity	93.3%	95.3%	100%
Anti dsDNA positivity	76%	52.5%	70%
Low C3(< 90 mg/dl)	60.8%	69.5%	52.5%
Low C4(<15 mg/ml)	70.7%	73.5%	55%

Antibodies to extractable nuclear antigen (Ab to ENA), RA Factor and other antibodies

In our study, RA was strongly positive in 22.5 % (n=9, p<0.05) patients, majority of which were in class III and class IV LN; findings similar to Vaidya[12] study in which 20% (n=15) had RA positive and explained the musculoskeletal manifestation of lupus nephritis. Either DCT or ICT was positive in 20% (n=8) patients (75% (n=6) in class IV and remaining 25% (n=2) in class III). Anti Sm was positive in 50% (n=15, p<0.05) patients belonging to class II (n=4), class III (n=6) and class IV (n= 4).

24 hours urinary proteins

In present study, nephrotic range proteinuria was present in 30% (n=12, p<0.05) patients belonged to class IV LN, 7.5% (n=3, p<0.05) patients belonged to class III LN and 5% (n=2, p<0.05) patients belonged to class V LN. In our study, total 42.5% patients (n=17, p<0.05) had nephrotic range proteinuria, comparable to Indian study.[9]The 24 Hours urinary protein was decreased post immunosuppressive treatment at the end of study.

In study by C Chrysochou *et al*,[19] nephrotic range proteinuria was found in 12 % patients. Indian studies have shown higher percentage of nephrotic range proteinuria. In study by Dhir *et al*,[9] 34% patients had nephrotic range proteinuria. While in study by Raphael V,[20] 66% had nephrotic range proteinuria. Both of above Indian study were retrospective. In our study, percentage of nephrotic range proteiuria was 42.5 % which could be due to the fact that our study was prospective which helps to detect proteinuria earlier and helped in early initiation of appropriate treatments.

Response to treatments

We analyzed the treatment response mainly in patients with class III, class IV and class V LN who were either on cyclophosphamide and glucocorticoids for induction of remission at 6 months and 12 months and only glucocorticoid for patients with class II LN. After 6 month of treatment, complete response with cyclophosphamide was seen in 16.66% (n=6) patients, partial response was seen in 41.66% (n=15) patients and 30.55% (n=11) patients did not respond to therapy. Complete remission was assessed by significant improvement in urinary sediment, decreased 24hr urinary protein level and increased in fractional value of complement C-3 and C-4. At the end of 12 months, the complete response with cyclophosphamide was seen in 43.75% (n=14) patients, partial response in 25% (n=8) patients and 31.25% (n=10) did not respond to therapy.

In study by Dhir *et al*[9] in 188 patients, at the end of 12 months, 38% patients (n=71) achieved partial remission. In study by Raphael V,[20]patients of severe lupus nephritis were treated with single monthly cyclophosphamide (0.75-1 gm/m^2) with oral prednisolone (0.5 mg/kg per day) and appropriate hydration. After a mean of followed-up of 15.8 months, out of 29 patients, 13 (44.8%) had achieved complete remission, 7(24.1%) partial remission and 9 (31%) cases did not respond to the therapy.

Mortality

In our study, two patients with class IV LN died. The cause of deathin one patient was severe disease activity (nephrotic range proteinuria) and in another patient, septicemia with pneumonia. In study by Dhir *et al*,[9] 16 patients died. The causes of death were :- infections (sepsis in 4, disseminated tuberculosis in 3 and pneumonia in 1), subdural hematoma in 1, severe bone marrow aplasia with pulmonary hemorrhage in 1, post surgery sudden death in 1, diabetic ketoacidosis in 1, suicide in 1, and unknown in 2. In study by Cevera R *et al*,[21] "Morbidity and mortality in systemic lupus erythematosus during a 10-year period: a comparison of early and late manifestations in a cohort of 1,000patients", the most frequent causes of death were active SLE (5%), thrombosis (26 %) and infections (25%) during initial 5 years period while thrombosis became most common cause of death during last 5 years. The morbidity and mortality data was lower in our study as compared to other Indian study,[9] because of small sample size and only one year of study period. Although most patients with lupus nephritis in our country are referred to tertiary care, a

referral bias toward more severe patients in our cohort cannot be ruled out. We excluded patients biopsied elsewhere due to a lack of details of initial investigations and initial treatment given outside. To conclude, Indian data on prospective study of Lupus Nephritis and their response to various treatment modalities is lacking. Most of LN are often asymptomatic and can be diagnosed by simple laboratory investigation and urinary examination. Our study showed that females are more prone to LN with a ratio of 13:1. Most common system involvement was musculoskeletal followed by mucocutaneous, gastrointestinal and haematological system. At the end of 6 month, complete response was found in 16.66% with CYC and in 43.75% at 12 months. The complications at the end of one year were gastrointestinal side effects, hypertension, infections and osteoporosis.

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