A Study of Lupus Nephritis

Pages with reference to book, From 167 To 170

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Abstract

Twenty-seven patients of Systemic Lupus Erythematosus were seen over a perion or Syears. Female to male ratio was 8:1. Most of the cases were young and presented with proteinuria with or without nephrotic syndrome. Active disease was associated with depression of C3, positive ANA and LE cell and raised titre of Anti DNA antibodies. Twenty patients underwent renal biopsy. Patients were treated with steroids and or other immunosuppressive drugs. Causes of death were different from those in western series. (JPMA 35 : 167, 1985).

Introduction

The incidence of Lupus Nephritis, a major cause of mortality and morbidity in patients with Systemic Lupus Erythematosus (SLE)[1,2,3] varies between 50-89 percent[4]. Renal involvement in SLE is suspected by the presence of protein, red cells and casts in the urine and confirmed by renal biopsy. This report describes the experience with 27 patients of lupus nephritis.

Material and Methods

All patients fulfilled four or more of the clinical criteria of the American Rheumatism Association[5] and renal biopsy was done in 20 cases.

BUN was estimated by Diacetyl method using thiosemicarbazide[6] Creatinine and urinary creatinine by Alkaline Picrate method;[7] S. Cholesterol by the method of Ferro and Ham;[8] and Urine protein was measured by using 3% trichioracetic acid.[9]

LE cell was demonstrated by making smears from buffy coat of defibrinated blood.' 10C3 was estimated by Immunodiffusion method; using Biomerix kit ANA by Immunoflourescence technique and Anti DNA antibodies by Radio- Immunoassay.

Renel biopsy was taken by Tru-Cut neele with the patint in prone positon.Sections were cut at 3-4 microns form paraffin material and stained with haemoteoxylin and eosin.

Results

Twenty-seven patients (24 females and 3 males) met four or more of the clinical criteria of Cohen et al.[5] Their ages at presentation ranged from 10 to 54 years with a mean age of 24.4 years (Fig).

Fig. 1 Age at presentation in SLE with nephritis:

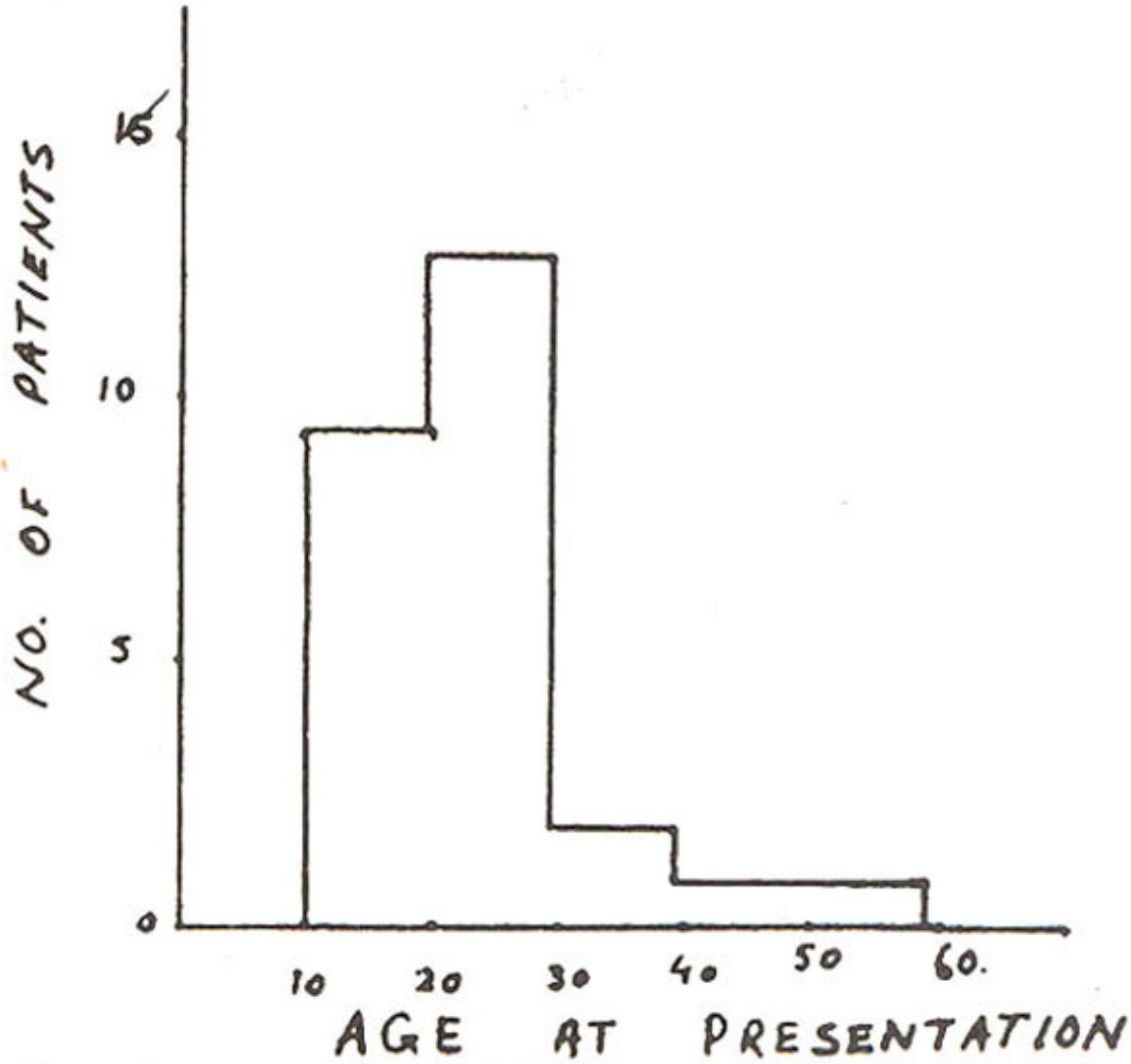

Fig. Age at presentation in SLE with nephritis.

Nine patients presented with nephrotic syndrome, 7 had proteinuria, 6 with acute nephritis and 5 with chronic renal failure.

The main clinical features: oedema, arthralgia, haematuria and anaemia were seen in more than 50% of patients (Table I).

Table I
Presenting Clinical Features.

Clinical Features	No. of Patients	(%)
Oedema	24	(89)
Arthritis	23	(85)
Haematuria	21	(78)
Anaemia	15	(59)
Fever	13	(47)
Skin rash	8	(32)
Hypertension	7	(29)
Liver involvement	7	(29)
Heart involvement	6	(26)
Lung involvement	5	(19)
Oral ulcers	4	(16)
Alopecia	1	(3.7)

Ninety-two percent had proteinuria, 89% positive ANA, 74% positive LE cell and anti DNA antibodies were raised in all the patients (15/15). Forty-eight percent had azotaemia (Table II).

Table II
Biochemical and Immunological Investigations.

Investigation	No. Tested	No. Positive (%)
Proteinuria	27	25 (92)
Raised BUN & Creatinine	27	13 (48)
Raised cholesterol	11	6 (66)
Positive LE cell	27	20 (74)
Positive ANA	27	24 (89)
Raised titre of anti-DNA antibodies	15	15 (100)
Low C_3	11	8 (73)

Renal biopsy was done in 20 patients; in others it was not done either because of raised BUN or refusal of the patients. In one case biopsy could not be done because of pregnancy. The histological findings are shown in Table III.

Table III
Histological Pattern (ON Light Microscopy).

Histological Diagnosis	No. of Patients (%)
Minimal change	4 (20%)
Focal Proliferative	4 (20%)
Diffuse proliferative	4 (20%)
Chronic nephritis	4 (20%)
Membranous nephropathy	3 (15%)
Interstitial nephritis	1 (5%)

Follow-Up.
Patients have been followed from one month to six years. Twenty were followed from 1 to 9 months and 7 from 15 months to 6 years. More than one-third of the patients (11/27) died. Two died of chronic renal failure (CRF), 5 of septicaemia, 2 of hepatic coma and one each due to pulmonary oedema and neurological involvement.
The patients in remission (4) on biopsy showed either focal proliferative, focal segmental glomerulitis or membranous nephropathy. Four patients had active disease i.e. positive ANA, LE cell or raised titre or anti DNA antibodies. One had chronic nephritis, one showed minimal change and in two others biopsy could not be done one due to pregnancy and the other refused. Two patients relapsed, one with focal nephritis after 3 years and the other with minimal change after 4 years.
Six patients were lost to follow-up. One was from Saudi Arabia and had chronic nephritis and an other from Sri Lanka, who had membranous nephropathy. One was a docter who didn't believe that she has SLE. She had normal renal functions and did not agree for renal biopsy, in one it was not done because of a high BUN. One patient with chronic nephritis was lost to follow-up after one year and one with chronic nephritis never returned after biopsy.
Treatment
Most of the patients (18) were treated with combined prednisolone and cyclophospha mide. Seven patients were given prednisolone (40-60mg) alone and only one patient was treated with cyclophosphamide only. One patient was on haemodialysis also.
Side effects were those usually observed with corticosteroids or immunosupressive drugs. Patients treated with steroids were temporarily cushingoid. One patient developed avascular necrosis of hip and another collapse of a lumbar vertebra. Few patients developed hypertension and psychosis developed in two cases. One patient suffered an attack of Herpez zoster during treatment with cyclophospharnide. Cyclophosphamide also caused alopecia, marked blackening of knuckles, nails and dark complexion generally. In few cases transient leukopaenia was also observed.

Discussion

Lupus nephritis in the series was more common in females below the age of 30 years and the clinical features were similar to those of lupus asawhole.[11,12]
Most of the patients presented with nephrotic syndrome or proteinuria. The frequency was less than that reported by cameron et al.[13] but similar to other reported series.[14,15]
A correlation between the presence of both complement fixing antibodies to DNA, low levels of serum compliment (C_3) and active renal disease has also been observed[16] in this series C_3 levels returned to normal as the disease activity subsided.
The treatment of SLE remains one of the controversial topics[17,22] W Most of the patients were given combined prednisolone and cyclophosphamide and only 7 patients were treated with large dose of prednisolone. One patient was given cyclophosphamide only. It cannot be said that combination is better than prednisolone alone or vice versa, as two of each group were in remission and also one each of the two with relapse were on prednisolone or combination therapy.
No serious side effects of cyclophosphamide i.e. haemorrhagic cystitis or development of malignancy were seen.
The pattern of death in this series was quite different.[13-23] The causes of death being renal failure/septicaemia, and myocardial infarction, whereas, in this series sepsis was the most common cause. It is not clear whether this relates to the disease itself, or the use of steroids and immunosupressive drugs. Only one patient took a fulminant course and died within one month and

others from 6 months to 4 years after diagnosis. Renal failure in an infrequent cause of death in SLE[11,24,26]. Only two patients in the series died of renal failure.
One patient was pregnant at presentation, who delivered a live baby at full term though an increased foetal loss occurs in lupus[27] Patients may have an exacerbation of the disease, during pregnancy, a spontaneous abortion or may suffer from pre-eclampsia.

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