

Using this information, targeted intervention to improve HL in LN, with a focus on language will help to improve HL in LN.

P83 LONG-TERM PROGNOSIS OF LUPUS NEPHRITIS: COMPARISON BETWEEN PEDIATRIC, ADULT, AND ADVANCED AGE ONSET

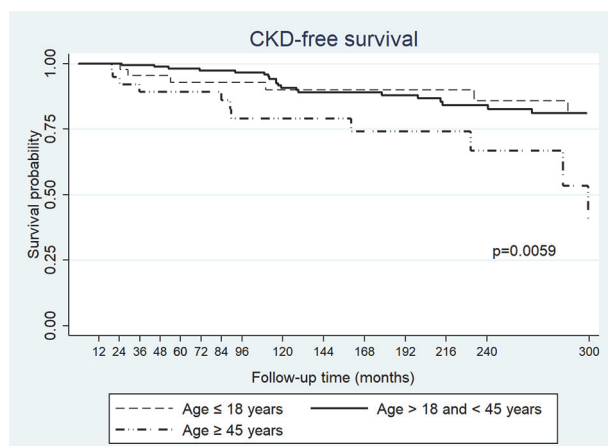
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Objective Lupus nephritis (LN) primarily affects young women, but cases with onset in childhood and advanced age have been reported. The aim of the study is to establish differences in long-term kidney survival among NL onset in childbearing age, in adults, and in older patients.

Methods We included 260 patients, categorized by the age of LN diagnosis (≤ 18 , >18 and <45 , ≥ 45 years). Demographic, clinical, histological and therapeutic data were collected at LN diagnosis. At last observation, we estimated the survival free from chronic kidney disease (CKD: eGFR < 60 ml/min using CKD-EPI/Schwartz) in the three groups using Kaplan-Meier curves, and differences were assessed with the Log-RANK test. Predictors of CKD among the baseline characteristics were analyzed using the Cox proportional hazard model in univariable and multivariable analysis.

Results The diagnosis of LN was performed in patients ≤ 18 years old in 44 cases (16.9%), between 18 and 44 years in 174 (66.9%) and over 45 years in 42 (16.2%). 88% of patients were females, the median serum creatinine of the whole group was 0.9 (0.7–1.3) mg/dl, proteinuria 3.45(2.0–5.5) g/die, and hypertension was present in 47% of patients. At kidney biopsy 76% had proliferative class (III or IV or



Abstract P83 Figure 1 CKD-free survival in LN patients with different classes of age at LN diagnosis: age ≤ 18 years, age > 18 and < 45 years, age ≥ 45 years. CKD, chronic kidney disease

Abstract P83 Table 1 Predictors of CKD development at last observation among the clinical, histological, and therapeutic features at LN diagnosis. eGFR, estimated glomerular filtration rate; CKD, chronic kidney disease

	Univariable analysis			Multivariable analysis		
	OR	CI	P	OR	CI	P
Class of age	1.9218	1.1602–3.1833	0.0115			
Serum creatinine	1.7423	1.372–2.2118	0.0000			
eGFR	0.9745	0.96521–0.9841	0.0001	0.9807	0.9700–0.9914	0.0005
Arterial hypertension	4.1489	2.0746–8.2972	0.0001			
Chronicity index at kidney biopsy	1.3207	1.1814–1.4765	0.00001	1.1988	1.0629–1.3521	0.0033
Serositis	2.1620	1.1465–4.076	0.0248			

Abstract P83 Table 2

	CKD free survival			
	5 years	10 years	20 years	25 years
Age ≤ 18 years	92.87%	90.06%	85.77%	81.01%
Age $> 18 < 45$ years	98.13%	90.77%	82.07%	82.07%
Age ≥ 45 years	89.24%	79.06%	66.71%	40.02%

mixed). Therapeutic approach did not differ among the age groups, but serum creatinine and chronicity index at kidney biopsy were higher, and eGFR was lower in older patients. In pediatric group, proteinuria was higher and serum complement was lower compared to the other groups. After a median follow-up of 174.5 (80.3–294.3) months, CKD was diagnosed in 18% of pediatric, 21% of adults and 33% of elderly ($p=0.18$). The CKD-free survival was better in the pediatric and adult groups than in older patients ($p=0.0059$) (figure 1). Age at LN diagnosis predicted CKD at univariable analysis only (OR:1.9218; CI:1.1602–3.1833; $p=0.0115$). The independent predictors of CKD at multivariable analysis were eGFR (OR:0.9807; CI:0.9700–0.9914, $p=0.0005$) and chronicity index (OR:1.1988; CI:1.0629–1.352, $p=0.0033$) (table 1). **Conclusions** Kidney survival was worst in elderly LN patients and not different between children and adults. Low eGFR and high chronicity index at kidney biopsy were the independent predictors of CKD.

P84 REGULAR MONITORING OF ANTI-C1Q ANTIBODIES CAN BE OF HELP TO PREDICT LUPUS NEPHRITIS (LN) EXACERBATIONS

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Objective This study explores the role of monitoring anti-C1q antibodies to predict lupus nephritis (LN) flares.

Methods In a cohort of LN patients, their clinical/histological/immunological/therapeutic features, at start of study, at 6 and 12 months after beginning therapy (reported in table 1) were tested at univariable and multivariable analysis with Cox proportional hazard models to identify predictors of renal flares occurring after 12-months follow-up. Anti-C1q antibodies were measured by ELISA-test (normal value were <20UA, high value >80UA).

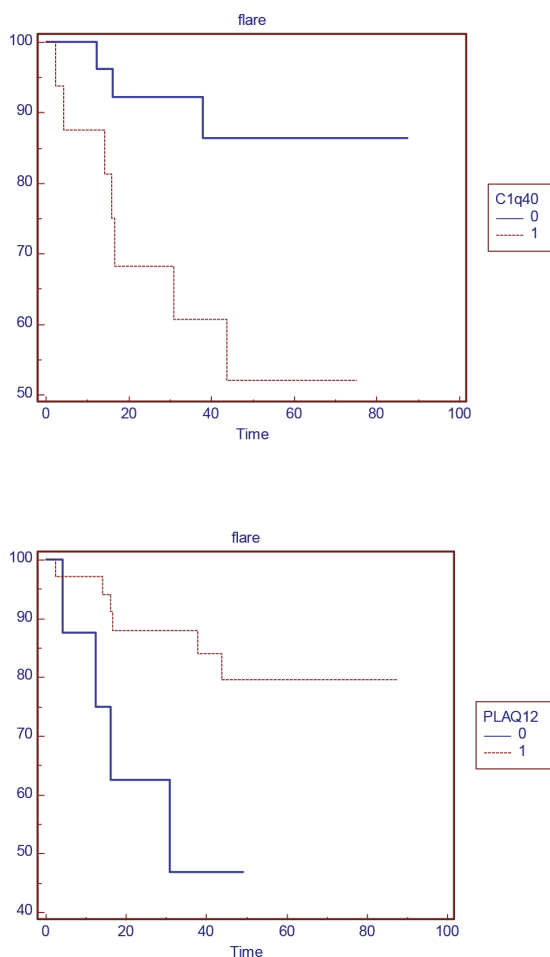
Results Fifty-three LN patients (84.9% were females, with a median age of 39 (29–47) years old, 10 patients with class III, 39 class IV and 4 class V at kidney biopsy categorized by ISN/RPS classification), 36 enrolled at LN diagnosis and 17 at a LN flare, entered this study. At baseline, 17 patients had acute kidney dysfunction (32%), and 17 had nephrotic

Abstract P84 Table 1 Clinical and histological characteristics at Lupus Nephritis diagnosis. Legend: SLE: systemic lupus erythematosus, n.: number

Clinical and histological characteristics at Lupus Nephritis diagnosis	All 53 patients	43 Patients who did not develop renal flare	10 Patients who developed renal flare
Male sex n. (%)	8 (15.09)	6 (13.95)	2 (20)
Age at SLE diagnosis (years)	23 (19–40)	24 (18.5–40.5)	23 (20.75–29)
Age at lupus nephritis diagnosis (years)	32 (24–40)	33 (24–41)	29 (24–37.25)
Duration of SLE at start of the study	101 (13.88–221.38)	84.11 (7.25–226.17)	121.05 (102.41–182–32)
Duration of lupus nephritis at start of the study	6.88 (1.09–94.31)	6.15 (1.15–87.58)	71.92 (12.01–93.01)
Class III n. (%), Class IV n. (%), Class V n. (%), Class V +III n. (%), Class V+IV n. (%)	2 (3.7), 25 (47.2), 4 (7.5), 8 (15), 10 (18.8)	1 (2.32), 21 (48.83), 3 (6.97), 6 (13.95), 8 (18.6)	1 (10), 4 (40), 1 (10), 2 (20), 2 (20)
Activity Index	8 (4.25–13)	8 (4.25–13.75)	9 (5.75–12.25)
Chronicity index	2 (1–3)	2 (1–3)	3 (1.75–3.25)
Follow-up at last observation	62.5 (45.63–78.62)	59.83 (40.51–74.85)	79.24 (64.34–87.77)
Data at start of the study			
At lupus nephritis diagnosis/ at a flare of lupus nephritis	36/17	31/12	5/5
Serum creatinine (mg/dL)	0.9 (0.67–1.15)	0.85 (0.66–1.19)	0.96 (0.88–1.07)
Proteinuria (g/24 h)	2.25 (1.28–4.15)	2.48 (1.37–4.25)	1.65 (1.1–2.95)
Red Blood cells/ul	3940 (3500–4480)	3940 (3565–4480)	4260 (3217.5–4455)
Erythrocyte sedimentation rate (mg/dL)	54 (31.5–76.5)	57 (42–75)	39 (25.25–71.25)
C-reactive protein (mg/dL)	0.1 (0.06–0.81)	0.1 (0.06–0.72)	0.5 (0.05–1.02)
Anti-C1q antibodies (UA)	86 (56.2–140.2)	81.5 (50.5–131.5)	134 (82–205)
C3 (mg/dl)	58 (40.75–72.75)	58 (38.75–72.25)	61.5 (46.25–79)
C4 (mg/dl)	7.5 (3.25–12.75)	6 (3–10.75)	10.5 (6.75–12.75)
Anti-DNA Units	189 (103–450)	248.7 (112–626)	152 (86.5–361)
Hydroxychloroquine n. (%)	35 (66.03%)	29 (67.44)	6 (60)
Methylprednisolone pulses n. (%)	40 (75.47)	33 (76.74)	7 (70)
Prednisone mg/day	30 (25–50)	35 (30–50)	27.5 (25–35.62)
Cyclophosphamide n. (%)	8 (15.09%)	6 (13.95)	2 (20)
Azathioprine n. (%)	3 (5.66)	0 (0)	2 (20)
Cyclosporin n. (%)	7 (13.2)	4 (9.3)	3 (30)
Mycophenolate mofetil n. (%)	36 (67.92)	32 (74.41)	4 (40)

Data at 6 Months			
Serum creatinine (mg/dL)	0.78 (0.66–0.95)	0.76 (0.66–0.88)	1.01 (0.93–1.09)
Proteinuria (g/24 h)	0.39 (0.24–1.15)	0.39 (0.23–1.20)	0.41 (0.33–0.57)
Red blood cells/ul	4470 (4160–4845)	4480 (4157.5–4867.5)	4450 (4200–4545)
Erythrocyte sedimentation rate (mg/dL)	17.5 (10.75–33.50)	18 (11–33)	16 (12.5–35)
C-reactive protein (mg/dL)	0.08 (0.04–0.30)	0.07 (0.04–0.32)	0.14 (0.07–0.24)
Anti-C1q antibodies (UA)	28 (13.25–70.5)	22 (10–41)	79.5 (66.5–91)
C3 (mg/dl)	91 (63.5–100)	92.5 (63.75–102.75)	86 (69.5–91)
C4 (mg/dl)	15 (9–20.75)	16 (8–23)	12 (11–14.5)
Anti-DNA Units, n. (%)	49.3 (29.58–156)	49.2 (22.3–139)	99 (38.75–378.5)
Hydroxychloroquine n. (%)	32 (60.37)	27 (62.79)	5 (50)
Prednisone (mg)	10 (7.5–12.5)	10 (7.5–12.5)	10 (8.75–11.25)
Cyclophosphamide n. (%)	0 (0)	0 (0)	0 (0)
Azathioprine n. (%)	0 (0)	0 (0)	0 (0)
Ciclosporin n. (%)	6 (11.32)	4 (9.3)	2 (20)
Mycophenolate mofetil n. (%)	35 (66.03)	30 (69.76)	5 (50)
Data at 12 Months			
Serum creatinine (mg/dL)	0.81 (0.73–0.95)	0.77 (0.71–0.94)	0.89 (0.83–0.96)
Proteinuria (g/24 h)	0.37 (0.2–0.64)	0.33 (0.17–0.54)	0.56 (0.38–0.83)
Red blood cells/ul	4510 (3945–4800)	4535 (3995–4807.5)	4335 (3895–4715)
Erythrocyte sedimentation rate (mg/dL)	22 (12–36)	19 (11–35)	29 (26–38)
C-reactive protein (mg/dL)	0.13 (0.06–0.36)	0.13 (0.06–0.29)	0.09 (0.05–0.41)
Anti-C1q antibodies (UA)	34 (16–71)	24 (15–45)	80 (45–111.25)
C3 (mg/dl)	87 (70–98)	88.5 (73.5–101.75)	78.5 (67.75–87.75)
C4 (mg/dl)	14 (10–20)	14.5 (9.25–20.75)	11.5 (10.25–14)
Anti-DNA	62 (30–193)	46.8 (25.72–192.25)	133 (79.25–578.1)
Hydroxychloroquine	36 (67.92)	30 (69.76)	6 (60)
Prednisone (mg/day)	5 (5–7.5)	5 (5–7.5)	7.5 (5–9.37)
Cyclophosphamide n. (%)	0	0 (0)	0 (0)
Azathioprine n. (%)	1 (1.88)	1 (2.32)	0 (0)
Cyclosporin n. (%)	7 (13.2)	5 (11.62)	2 (20)
Mycophenolate mofetil n. (%)	40 (75.47)	32 (74.41)	8 (80)

syndrome (32%). Forty patients received methylprednisolone pulses as induction therapy, the others oral prednisone (0.5–1mg/kg/day) associated with an immunosuppressive agent. During a median follow-up of 62.5 (45.63–78.62) months, renal flares occurred in 10 patients (18.86%). All flares occurred after 12-months observation, in median 28.19 (24.84–39.38) months (2 nephritic and 8 proteinuric flares). Among clinical/histological and therapeutic features at start of the study and at 6 months, only anti-C1q antibodies (OR:1.0098; CI:1.0004–1.0193; p=0.04; OR:1.0268; CI:1.0126–1.1041; p=0.001 respectively) predicted renal flares. At 12 months, anti-C1q antibodies (OR:1.0180; CI:1.0064–1.10297; p=0.0020), antiC1q ≥40 UA (OR:4.4345; CI:1.1536–17.0462; p=0.0310), proteinuria (OR:1.8537; CI:1.1237–3.0578; p=0.0160), and no use of hydroxychloroquine (OR:0.2561; CI:0.1009–1.2778; p=0.0370) predicted renal flares at univariable analysis. At multivariable analysis, antiC1q ≥40 UA (OR:5.2421; CI:1.3338–20.6019; p=0.0183) and no use of Hydroxychloroquine (OR: 0.2080; CI:0.0538–0.7573; p=0.0183) were the independent predictors of renal flares (figure 1).



Abstract P84 Figure 1 Survival free of flares. The difference between curves was evaluated by the log-rank test. **1a** Survival free of flares in patients who had at 12 months antiC1q ≥ 40 (1) vs those who had antiC1q < 40 (0) $p=0.031$. **1b** Survival free of flares in patients who were at 12 months in treatment (1) or not (0) with Hydroxychloroquine $p=0.0370$

Conclusion With the limitations of a small study, our results suggest that a high titer of anti-C1q at baseline and failure to normalize (or reducing < 40 UA) during the follow-up are helpful in identifying patients who will develop renal flares. Hydroxychloroquine use reduces the risk of lupus flares.

P85

PREDICTORS OF LLDAS AND REMISSION IN A SINGLE-CENTER COHORT OF PATIENTS WITH LUPUS NEPHRITIS: A RETROSPECTIVE ANALYSIS

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Objective Lupus nephritis (LN), a major involvement of systemic lupus erythematosus (SLE), impacts up to 60% of SLE patients throughout their lives, with a progression rate to end-stage kidney disease ranging from 4.3% to 10.1%. This study aims to 1) assess the attainment of lupus low disease activity state (LLDAS) and remission in SLE patients with LN at a single center and 2) analyze predictors of LLDAS, remission, and stage ≥ 3 chronic kidney disease (CKD).

Methods This retrospective, observational study included SLE patients diagnosed since 1977 with documented renal involvement via kidney biopsy. Demographic, clinical, and laboratory data were collected from the onset of renal disease to the last outpatient visit. CKD was defined as eGFR (calculated using CDK-EPI formula) ≤ 60 mL/min/m², while LLDAS and REM definitions were based on criteria by Franklyn K. et al (Ann. Rheum. Dis., 2016) and van Vollenhoven RF (Lupus Sci. Med., 2021).

Results The study comprised 87 patients, with 42 having proliferative glomerulonephritis (see table 1). During the follow-up, clinical remission was achieved in 63 out of 83 (75.9%) patients, and LLDAS in 73 out of 85 (85.9%) patients, with an average time of 8.46 (± 8.67) and 8.33 (± 8.96) months between biopsy diagnosis and achieving remission and LLDAS, respectively. Univariate analysis indicated a statistically significant association between higher SLEDAI-2K and failure to achieve LLDAS (OR 0.88, p 0.049). The occurrence of at least one LN flare during follow-up was associated with a lower probability of achieving LLDAS (OR 0.17, p 0.01) but not remission (OR 0.29, p 0.05). Predictors of stage ≥ 3 CKD included a longer time between LN diagnosis and LLDAS/

Abstract P85 Table 1

Baseline characteristics (at histological diagnosis)	Total n= 87	Proliferative n = 42	Membranous n= 20
Female, n (%)	82 (94)	38 (90)	20 (100)
Age, (mean \pm SD)	33 (13)	30 (11)	30 (11)
Disease duration (years),(mean \pm SD)	4.6 (8.0)	3.6 (6.7)	3.1 (6.8)
Ethnicity, n (%)			
Caucasian, n (%)	82 (94)	38 (90)	19 (95)
Afroamerican, n (%)	3 (3.4)	2 (4.8)	1 (5)
Other, n (%)	2 (2.3)	2 (4.8)	0
Weight (kg), (mean \pm SD)	67 (16)	70 (18)	67 (14)
Height (cm), (mean \pm SD)	164 (7)	164 (8)	164 (7)
Smoker, n (%)	39 (46)	17 (41)	9 (45)
Creatinine (mean \pm SD)	1.08 (1.07)	1.10 (1.09)	0.74 (0.19)
Nephrosic range proteinuria, n (%)	10 (16)	6 (21)	1 (5)
Anti-dsDNA positivity, n (%)	69 (80)	38 (90)	13 (65)
SLEDAI-2K Score, (mean \pm SD)	9 (4)	10 (5)	8 (2)
SLICC-SDI Score (mean \pm SD)	1(1)	0 (1)	0 (1)
Ongoing therapies at LN diagnosis			
Mycophenolate mofetil			
Ongoing/starting, n (%)	5(6)/22 (27)	2 (5)/18 (46)	0/3 (17)
Hydroxychloroquine			
Ongoing/starting, n (%)	38 (46)/9 (11)	18 (46) / 3 (8)	8 (44)/1 (6)
Azathioprine			
Ongoing/starting, n (%)	7 (8)/9 (10)	3 (8)/1 (2)	3 (16)/3 (16)
Cyclophosphamide			
Ongoing/starting, n (%)	3 (3)/26 (30)	2 (5)/16 (39)	2 (10)/4 (20)
Corticosteroid (including pulses)			
Ongoing/starting, n (%)	52 (61)/25 (29)	26 (63)/16 (38)	11 (58)/8 (42)
Rituximab			
Ongoing/starting, n (%)	0/1 (3)	0/1 (3)	0
ACE-inhibitor			
Ongoing/starting, n (%)	15 (18)/9 (11)	7 (17)/4 (10)	4 (22)/1 (6)
Statins			
Ongoing/starting, n (%)	4 (5)/0	1 (3)/0	1 (6) / 0