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Lupus Nephropathy

without affecting autoantibody profile, splenic lymphocytes and macrophages.

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Clinical Characteristics and Outcomes of Lupus Podocytopathy with Different Glomerular Lesions

<u>W. X. H. Hu</u>, S. F. W. Wang, H. C. Chen, Y. H. C. Chen, Z. Z. L. Liu, H. T. Z. Hai, Z. H. L. Liu

National Clinical Research Center of Kidney Disease, Research Institute of Nephrology, Jinling Hospital, Nanjing University School of Medicine, Nanjing, Jiangsu, China

Objectives: To investigate the clinical characteristics and outcomes of lupus podocytopathy with different glomerular lesions.

Methods: Lupus podocytopathy (defined as foot process effacement 50% of the glomerular capillary loop area without deposits in capillary wall under electron microscopy) was screened from 3750 lupus nephritis patients in Nanjing Jinling hospital between January 2000 and December 2013 and were divided into 3 groups including minimal change disease (MCD), mesangial proliferation (MP) and focal segmental glomerulosclerosis (FSGS) by light microscopy. The clinical characteristics, treatment response and outcomes were retrospectively studied and compared among 3 groups.

Results: 53 patients (1.41%) were re-classified as lupus podocytopathy. including 13 with MCD, 31 with MP and 9 with FSGS. 50 (94.3%) patients presented with nephrotic syndrome, 3 (5.7%) patients showed non-nephrotic proteinuria, 17 (32.1%) patients had concomitant AKI. The incidence of AKI was significantly higher in FSGS group (77.8%) than in MCD (23.1%) and MP (22.6%) groups (P < 0.01). Low serum C3 was more frequently seen in FSGS (88.9%) and MP groups (83.9%) than in MCD group (23.1%, P < 0.05). No difference was observed in foot process effacement among the 3 groups. More patients had severe acute tubulointerstitial lesions in FSGS group (77.8%) than in MCD (7.7%) and MP (22.6%) groups (P < 0.01). 50 (94.3%) patients achieved remission after immunosuppressive induction treatment. among whom 41 (77.4%) had complete remission (CR). The CR rate in FSGS group (22.2%) was significantly lower than in MCD (92.3%) and MP (87.1%) groups (P < 0.01). During follow-up for 44 months (9-125 months), 29 (54.7%) patients had renal relapse, no patient died or developed end stage renal disease.

Conclusion: Lupus podocytopathy with different glomerular lesions showed variable clinical characteristics with benign outcomes. Patients with MCD and MP shared similar clinical features, which were different from patients with FSGS in terms of AKI incidence, tubular injury severity and response to immunosuppressive treatment.

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Renal Pathological Classification in Lupus Nephritis: What Else Can We Tell?

J. Y. Yang, D. D. L. Liang, H. T. Z. Zhang, Z. Z. L. Liu, W. B. L. Le, M. L. Z. Zhou, W. X. H. Hu, C. H. Z. Zeng, Z. H. L. Liu National Clinical Research Center of Kidney Diseases, Jinling Hospital,

Nanjing University School of Medicine, Nanjing, Jiangsu, China

Objective: To observe the discrepancy between class III and class III+V, between class IV and class IV+V, and between subclass IV-S and IV-G on clinicopathological features and renal outcomes and explore the pathological lesions associated with poor renal outcomes in patients with different classes.

Methods: The records of all adult patients with biopsy-proven proliferative lupus nephritis followed for at least 1 year were reviewed. All patients were pathologically classified according to the 2003 ISN/RPS classification of lupus nephritis and each pathological lesion was semiquantatively scored. **Results:** Patients with class III+V (class IV+V) presented with more severe proteinuria and chronic pathological lesions and milder acute pathological lesions than patients with class III (class IV) (Tables 1–2, 4–5); patients with subclass IV-G presented with more severe hypertension, proteinuria

and hypocomplementemia, lower ANCA positivity rate, more severe glomerular cell proliferation and hyaline deposit, and milder fibrinoid necrosis and crescent than patients with subclass IV-S (Tables 3, 6). The renal outcomes between patients with class III and class III+V, between class IV and class IV+V, and between subclass IV-S and subclass IV-G were not different respectively (Figures 1–3). Global glomerulosclerosis, cellular crescent, fibrous crescent, glomerular cell proliferation, tubular acute injury, interstitial inflammation and TMA were predictors for ESRD.

	III	III + V	P value
Patients (n)	207	169	
Female (n, %)	186 (89.9%)	146 (86.4%)	0.335
LN onset age (years)	30.9±8.3	30.1±8.1	0.371
Mean arterial pressure (mmHg)	95.5±13.2	99.2±13.7	0.008
Haemoglobin (g/dl) ^a	10.5±2.1	10.9±2.3	0.053
Haematuria (10 ⁴ cells/ml)	36.0 (3.0-150.0)	11.0 (2.0-50.0)	0.397
24-h urinary protein (g/24h)	1.66±1.99	3.16±2.72	<0.001
Serum creatinine (mg/dl) ^a	0.91±0.50	0.90±0.81	0.890
Serum complement C3 (g/L)	$0.598{\pm}0.260$	0.637±0.287	0.182
Serum complement C4 (g/L)	0.156±0.126	0.167±0.132	0.401

^a Conversion factors for units: serum creatinine in mg/dl to μ mol/l, \times 88.4; haemoglobin in g/dl to g/l, \times 0.1.

Table 2	Baseline clinical	features of LN	natients with	class IV and IV+V.
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	IV	IV + V	P value
Patients (n, %)	559	233	
Female (n, %)	493 (88.2%)	202 (86.7%)	0.554
LN onset age (years)	31.0±8.4	30.2±8.9	0.220
Mean arterial pressure (mmHg)	101.2±14.5	100.2±12.5	0.350
Haemoglobin (g/dl) ^a	8.9±2.0	9.3±2.1 [#]	0.018
Haematuria (10⁴cells/ml)	90.0	60.0	0.012
	(15.0-265.0)	(12.0-187.5)	
24-h urinary protein (g/24h)	3.41±2.92	4.19±3.10	<0.001
Serum creatinine (mg/dl) ^a	1.48±1.33	1.34±1.21	0.077
Serum complement C3 (g/L)	$0.505{\pm}0.269$	0.486±0.230	0.382
Serum complement C4 (g/L)	0.145±0.124	0.134±0.104	0.278

 a Conversion factors for units: serum creatinine in mg/dl to $\mu mol/l,$ $\times 88.4;$ haemoglobin in g/dl to g/l, $\times 0.1.$

Table 3. Baseline clinical features of LN patients with subclass IV-S and IV-G.

	IV-S	IV-G	P value
Patients (n, %)	117 (20.9%)	442 (79.1%)	
Female (n, %)	104 (88.9%)	389 (88.0%)	0.873
LN onset age (years)	32.2±8.2	30.8±8.4	0.108
Mean arterial pressure (mmHg)	98.0±13.0	102.1±14.8	0.004
Haemoglobin (g/dl) ^a	9.2±1.9	8.8±2.0	0.124
Haematuria (10 ⁴ cells/ml)	60.0	100.0	0.419
	(10.5-244.0)	(15.0-271.3)	
24-h urinary protein (g/24h)	2.75±1.76	3.59±3.13	<0.001
Serum creatinine (mg/dl) ^a	1.36±1.30	1.51±1.33	0.254
Serum complement C3 (g/L)	0.597±0.297	0.481±0.256	<0.001
Serum complement C4 (g/L)	0.163±0.123	0.140±0.123	0.071
ANCA positive rate (%)	9.5%	5.2%	0.130

 a Conversion factors for units: serum creatinine in mg/dl to $\mu mol/l,$ $\times 88.4;$ haemoglobin in g/dl to g/l, $\times 0.1.$