

this finding. Since most of SLE patients are female, and they have been mostly diagnosed as SLE in young adult age, thalassemia carrier screening is of great importance for future family planning.

## 9. Lupus nephritis

### LP-124 CHILDHOOD-ONSET LUPUS NEPHRITIS: REAL WORLD LONG-TERM OUTCOME DATA FROM A SINGLE CENTER

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**Background** While lupus nephritis (LN) is more common with higher severity and mortality in children than in adult-onset disease, data regarding long-term outcome of childhood-onset LN is rare.

**Methods** Long-term renal outcome and their treatment were assessed in 57 Korean childhood-onset LN patients diagnosed between 1999 and 2020 from a tertiary single center in Korea.

**Results** Median age at diagnosis of LN was 14.5 (range 7.8–17.8) years. Median follow-up period was 135 (30–266) months. 26.3% (15/57) of patients progressed to chronic kidney disease (CKD) stage 3–5 (CKD stage 3: 6 patients, stage 4: 1 patient, stage 5: 8 patients). 10-year renal survival was 100% in patients diagnosed after 2011, compared to 80.3% in patients diagnosed before 2011 (p=0.049). Comparing the two eras, there were no clear difference between the laboratory finding at diagnosis, the drug used, and the cumulative

dose of cyclophosphamide or hydroxychloroquine, except that primary renal response rate was higher at the latter era (55.2% vs 82.1%, p=0.029), and the diagnosis age was slightly younger in the earlier era (median 13.5 vs 14.6, p=0.056). Hydroxychloroquine use was significantly associated with maintaining renal estimated glomerular filtration rate (eGFR) higher than 60ml/min/m<sup>2</sup> at 10 years (95.2% vs 77.2%), and 20 years (68.6% vs 16.1%), respectively (p=0.033).

**Conclusions** Early diagnosis and timely adequate treatment, and the use of hydroxychloroquine are important in the long-term kidney prognosis of childhood-onset LN.

### LP-125 GLOMERULONEPHRITIS IN SYSTEMIC LUPUS ERYTHEMATOSUS: 'NOT EVERYTHING IS LUPUS' A CASE SERIES

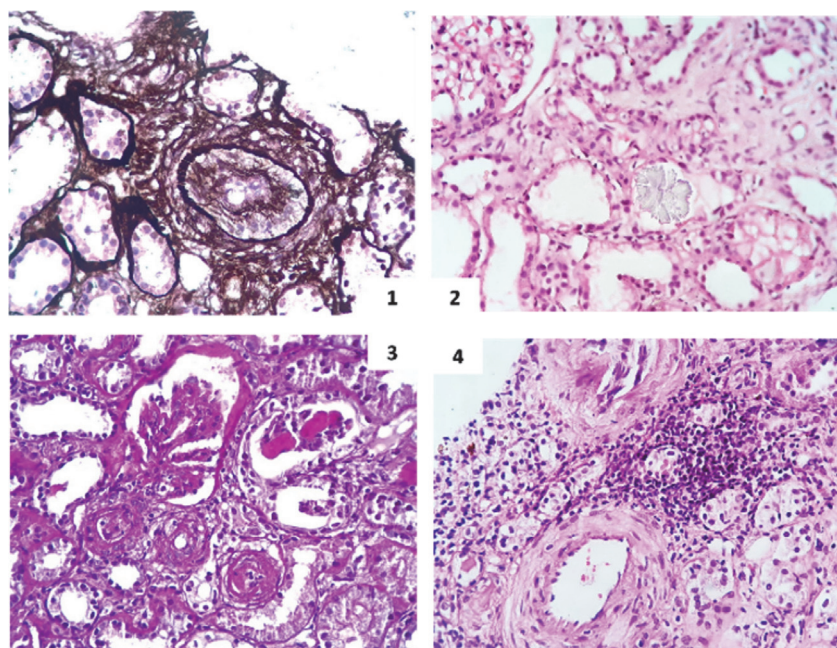
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**Description** 4 CASES with clinical manifestations of systemic lupus erythematosus (SLE) and proteinuria in nephrotic range. Case 1, a 30-year-old woman with multiple sclerosis history, using β1 interferon for 16 years, present dyspnea, delirium, edema in the lower limbs, hypertension, lymphopenia, thrombocytopenia, active sediment, 2.8 g/24 hrs. proteinuria, hypocomplementemia, ANA and anti-DNA+. Case 2, a 40-year-old woman with no medical history, arthritis, non-scarring alopecia, oliguria, and emergency hemodialysis criteria, anemia, hypocomplementemia, 0.5 g/24 hrs. proteinuria, ANA, and anti-DNA +. Case 3, a 46-year-old woman with no previous

Abstract LP-125 Table 1 Clinical manifestations according to SLE EULAR/ACR 2019 classification criteria

Characteristic/ Case No.	1	2	3	4	N
ANA 1:80 (%)	x	x	x	x	100
<b>Clinical domains and Criteria</b>					
<b>Hematologic</b>					
Leukopenia (≥)	2.18	4.2	2.07	3.9	3.09
Thrombocytopenia (≥)	58		58		58
Autoimmune Hemolysis (≥)		6.8	7.8		9.1
<b>Neuropsychiatric</b>					
Delirium (%)	x				25
<b>Mucocutaneous</b>					
Non-scarring alopecia (%)		x		x	50
Acute cutaneous lupus (%)			x	x	50
<b>Serosal</b>					
<b>Musculoskeletal</b>					
Joint involvement (%)		x			25
<b>Renal</b>					
Proteinuria ≥ 0.5g/24 horas (≥)	2.8	0.5	0.6	4.1	2
<b>Immunology domains and Criteria</b>					
<b>Antiphospholipid antibodies</b>					
Lupus anticoagulant (%)			x		25
<b>Complement proteins</b>					
Low C3 and low C4 (%)	x	x		x	75
<b>SLE-specific antibodies</b>					
Anti-dsDNA antibody (≥)	55.41	104		68.6	76
Total score (≥)	23	29	23	29	26
<b>kidney biopsy</b>					
thrombotic microangiopathy/mucoid fibrosis		Oxalate nephropathy	thrombotic microangiopathy	Crescentic glomerulonephritis	
<b>Final diagnosis</b>					
Interferon toxicity		Star fruit glomerulonephritis	Antiphospholipid syndrome	Anca-Associated Vasculitis	
<b>Treatment</b>					
Methylprednisolone/Rituximab 375 mg/m <sup>2</sup> / 4 weeks		Hydration/hemodialysis Prednisone/HCO/AZA	Prednisone/ anticoagulation	Methylprednisolone/CYC	
<b>Response/8 weeks</b>					
Other clinical domains (%)	x	x	x	x	100
Renal Function (%)	x	CKD	x	CKD	50
<b>Proteinuria</b>					
Partial response (%)	x		x		50
Complete response (%)					
No Response (%)		x		x	50



Abstract LP-125 Figure 1

history, high blood pressure, leukocytoclastic vasculitis, edema in the lower limbs, anemia, thrombocytopenia, active sediment, 0.6 g/24 hrs. proteinuria, ANA, lupus anticoagulant, and SSA +. Case 4, a 39-year-old woman with no medical history, non-scarring alopecia, malar erythema, emergency hemodialysis criteria, lymphopenia, anemia, hypocomplementemia, 4.1 g/24 hrs. proteinuria, ANA, and anti-DNA +. The clinical manifestations are summarized in (table 1) according to the SLE EULAR/ACR 2019 classification criteria. Renal biopsy was performed in 4 cases (table 1), the first being associated with the long-standing use of Interferon  $\beta$ 1, the second with star fruit consumption, the third with antiphospholipid syndrome, and the fourth ANCA-Associated Vasculitis (figure 1). The treatment and evolution of the patients 8 weeks after identification are shown in (table 1).

**Conclusions** Glomerulopathy in SLE is a frequent and severe manifestation, so timely treatment is necessary. However, the role of renal biopsy becomes important despite the clinical characteristics, where its performance shows us that in the clinical spectrum of glomerulopathies not everything is Lupus.

LP-126

#### THE INFECTIOUS COMPLICATION IN MULTITARGET THERAPY OF CLASS V LUPUS NEPHRITIS: A CASE REPORT

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**Description** Agents such as cyclosporine A(CSA) and tacrolimus(TAC) have long been used in SLEpatients. A new therapeutic approach of lupus nephritis(LN) is a multitarget therapy: calcineurin inhibitors with mycophenolate mofetil (MMF).

Here is a case report of lupus nephritis (class V) and infectious complication in a SLE patient treated with low-dose combination of CSA and MMF.

A 40-year-old woman(caucasoid), the disease debut at the age of 25, duration 16 years(since 2006), the diagnosis of SLE was established in 10.2011(full picture after childbirth). History: LN (class IV, with nephrotic syndrome, azotemia – 2011), nervous system (migraine with aura, sensorimotor polyneuropathy of the lower extremities, dysuria – 2011), arthritis and Raynaud's phenomenon (2006, 2010), thrombocytopenia (2011), positive anti-ds-DNA, anti-Sm, ANA, hypocomplementemia (2011). In 2011, therapy was carried out with high doses of prednisolone(max 40mg/day), cyclophosphamide (total 5000mg, 2011–2012years), rituximab (1000 mg No. 2, 2012–2013years), MMF 2.5–1 g/day (2012 -2017years), hydroxychloroquine(HCQ). Low disease activity was achieved in 2016–12.2020years: therapy with prednisolone 5mg/day and HCQ 200mg/day.

In 12.2020 there was a disease relapse – isolated persistent proteinuria 1.3g/day. Repeated nephrobiopsy was performed: membranous glomerulonephritis(class V) was revealed. The dose of prednisolone was increased from 5 to 30mg/day, MMF 2 g/day was added, HCQ. After 5 months of this therapy, proteinuria did not decrease – 1.2g/day. A decision was made to switch to multitarget therapy: a combination of MMF 1g/day and CSA 150mg/day (2 mg/kg/day) from 06/14/2021, but on 07/16/2021 panaritium of the 2nd toe of the foot developed. Resumption of multitarget therapy 08/12/2021. By September 2021 proteinuria decreased to 0.6g/day, but on 09/28/2021, purulent bursitis of the right elbow joint developed. The patient was transferred to monotherapy of MMF 1–2 g/day, prednisone10–7.5mg/day, HCQ 200mg/day, proteinuria 0.18 g/day from 03.2022

**Conclusions** Multitarget therapy with CSAandMMF is effective in treating LN(classV), but can lead to purulent infectious complications.