

uptake pattern was significantly lower in HAVS than in primary RP.

Conclusions There were significant differences in hand perfusion scintigraphic features between primary RP and HAVS. These results suggest that the underlying pathophysiology of the two diseases differs; thus, different criteria should be applied for their evaluation.

372 CLINICAL FEATURES, OUTCOMES AND RISK FACTORS FOR THE DEVELOPMENT OF POSTERIOR REVERSIBLE ENCEPHALOPATHY SYNDROME (PRES) IN THAI PATIENTS WITH SYSTEMIC LUPUS ERYTHEMATOSUS (SLE)

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Background and aims PRES in SLE is increasingly recognised. This study aimed at determining the prevalence, clinical features, brain imaging findings, risk factors, and outcomes of PRES in Thai SLE patients.

Methods SLE patients with PRES seen between 1 January 1986 and 31 August 2013 were identified. Controls were matched with hospital number and disease duration to cases (ratio, 1:4). Clinical features, brain imaging patterns, risk factors, treatment and outcome of PRES were determined.

Results Of 1141 SLE patients, 26 PRES episodes occurred 20 in females (prevalence 1.8%). Mean±SD age at diagnosis and disease duration was 29.3±13.1 and 2.8±3.4 years, respectively. Among the 26 episodes, 24 (92.3%) had seizure, 14 (53.8%) headache, 9 (34.6%) fever and vomiting and 8 (30.8%) visual disturbance. All of them had acutely elevated blood pressure. 20 and 23 patients had active lupus nephritis (LN) within 3 months prior to and at PRES onset, respectively. Dominant parietal-occipital pattern was the most common brain imaging abnormality. 22 episodes improved with blood pressure control. Immunosuppressive therapy was given for active disease in 8 episodes. Anti-convulsive therapy could be discontinued in 21 of 22 episodes (median duration 3 months). Auto-immune hemolytic anaemia (AIHA) and LN were PRES risk factors (OR 6.55, 95% CI 1.09–39.39, p=0.04 and OR 3.06, 95% CI 1.12–8.39, p=0.03, respectively). 6 patients (23.1%) died during PRES episodes. The mortality rate in SLE patients with PRES was significantly higher than those without (30% vs. 10%, p<0.001).

Conclusions The mortality rate was high in Thai SLE with PRES. AIHA and LN were risk factors for PRES.

373 ASSOCIATION BETWEEN DISEASE ACTIVITY, QUALITY OF LIFE AND QUALITY OF SLEEP IN PATIENTS WITH SYSTEMIC LUPUS ERYTHEMATOSUS (SLE)

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Background and aims The aim of this study was to examine the association of SLE disease activity, quality of life and quality of sleep in patients diagnosed with SLE.

Methods 132 SLE patients with a confirmed diagnosis of SLE, according ACR classification criteria, were enrolled in this study. The patients completed the following questionnaires: the Pittsburg Sleep Quality Index (PSQI), the 12 item Short Form Health survey (SF-12), the Lupus Patient-Reported Outcome tool (LupusPRO), SLE Quality of Life Questionnaire (L-QoL). Clinical information, including the SLE Disease Activity Index (SLEDAI), was obtained from medical records. Student's t-test, ANOVA, Pearson correlation measured were used in statistical analysis.

Results The majority of the participants (84,4%) had sleep disturbances (PSQI >5). Total PSQI score was weakly associated with all of the SF-12 subcategories and showed weak to moderate associations with LupusPRO subcategories (r<0,05), except for medication (r<0,2). "Sleep efficiency" was weakly associated with "physical health", "physical function", and "pain" in the SF-12 and LupusPRO. "Sleep quality" and "sleep disturbances" were weakly associated with "pain" and the "emotional" and "mental" subcategories in the SF-12 and LupusPRO. SLE QoL was significantly higher in patients with good sleep.

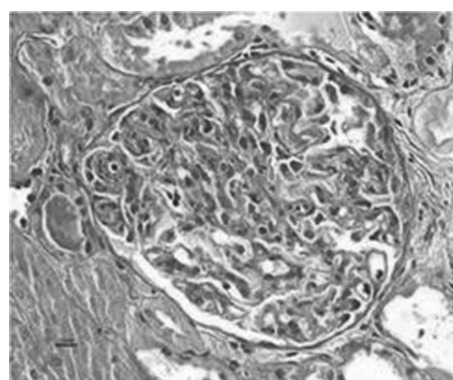
Conclusions We found that quality of sleep, especially "sleep efficiency", was poor for the majority of patients with SLE. Quality of sleep was associated with various aspects of quality of life, especially pain, vitality, and emotional health. Management of pain and emotional health may be important for improving quality of sleep in SLE patients.

374 ANA NEGATIVE RENAL LIMITED LUPUS NEPHRITIS –A RARE ENTITY

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Background and aims Antinuclear antibodies (ANA) in serum is considered a decisive diagnostic test for SLE. ANA negative SLE is a subgroup of SLE that is infrequently recognised. We report an unusual case of seronegative SLE which presented



Abstract 374 Figure 1

Abstract 374 Table 1

Clinical findings	
Fever	38°C
Blood pressure	150/90 mmHg
Facial puffiness and pedal edema	++
Hepato-splenomegaly	No
Laboratory findings	
Hemoglobin (g%)	10.1
Total WBC counts	9800/cumm
Platelets	167000/ cumm
Blood urea (mg/dl)	136
Serum creatinine(max) (mg/dl)	5.4
Serum albumin(g/dl)	2.7
24 hour urine protein(g/day)	6.3
Urine RBCs(/hpf)	50/hpf
C3(mg/dl)	59
C4(mg/dl)	15
ANA	Negative
Anti – ds DNA	Negative
Anti-neutrophil cytoplasm antibody (ANCA)	Negative
Anti cardiolipin antibody	Negative

as rapidly progressive renal failure with no other systemic manifestations.

Methods 34 year old female presented with fever, nephrotic range proteinuria and rapidly progressive renal failure. She did not have any other systemic features of SLE. Her clinical, biochemical and serological findings are as shown in table 1. She had low complementemia, but her ANA, ANA profile including anti double stranded DNA (anti- dsDNA) antibodies and anti cardiolipin antibody was negative. Renal biopsy on light microscopy showed diffuse proliferative glomerulonephritis with a full house on immunofluorescence including C1q consistent with class 4 lupus nephritis (Figure 1). A diagnosis of ANA negative renal limited lupus nephritis was made.

Results She was treated with pulse methyl prednisolone followed by oral steroids 1mg/kg/day and pulse cyclophosphamide 500–750 mg/m² body surface area as per NIH protocol. She recovered completely and is on follow-up for two years. She has remained persistently negative for all ANA antibodies including anti-dsDNA antibodies.

Conclusions Ours is an unusual case of ANA negative renal limited lupus nephritis. The low complement levels, full house nephropathy in immunofluorescence and response to therapy were important clues in diagnosing the case. We report this patient to highlight the possibility of SLE in seronegative patients as well in order to avoid delay in the management.