CASE REPORT

SLE Patients with Posterior Reversible Encephalopathy Syndrome in Nongkhai Hospital: Case Report

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Background: Posterior reversible encephalopathy syndrome (PRES) is a rare acute or subacute clinicoradiologic disorder. Systemic lupus erythematosus (SLE) is associated with irreversibility, recurrence, and poor prognosis of PRES. Delayed treatment of PRES in SLE can lead to significant neurological deficits and mortality.

Objective: To collect case series of SLE patients with PRES treated in Nongkhai Hospital between 2012 and 2023 and compare with other studies.

Materials and Methods: The medical records of SLE patients with PRES were reviewed and collected. The following information were obtained, gender, occupation, age at SLE diagnosis, age of onset, duration of disease, comorbid or risks, SLE disease activity, glucocorticoids, and immunosuppressants administered before or at diagnosis, clinical presentations, laboratory data, neuroimaging, treatments, complications, length of stay, and outcomes.

Results: Six hundred eleven patients with SLE were identified. Five patients developed PRES. All patients received prednisone with a mean dose of 26.0 (SD 10.4) mg/day before the onset of PRES. Seizures were displayed by all patients. The mean systolic blood pressure was 163.4 (SD 6.3) mmHg. The occipital lobe was the most common brain lesion. Lupus nephritis was observed in all patients. Three of five patients needed intubation and were admitted for closed monitoring in the intensive care unit (ICU). The most common complications during admission were infections, especially sepsis. All patients recovered completely.

Conclusion: The present study suggested that SLE patients with hypertension presenting with seizures and high systolic blood pressure along with a history of moderate to high dose steroids and/or immunosuppressants should always be suspected of PRES. Meanwhile, computerized tomography (CT) and/or magnetic resonance imaging (MRI) brain are the most important tools to establish an early diagnosis. Individualized treatment should be based on clinical presentations, and disease activity evaluation, especially lupus nephritis. Accordingly, sepsis prevention, and ICU admission in severe cases are crucial for a favorable outcome.

Keywords: Posterior reversible encephalopathy syndrome; PRES; SLE; Lupus

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Posterior reversible encephalopathy syndrome (PRES) is a rare acute or subacute clinicoradiologic disorder[1-7] first described in 1996 by Hinchey et al. as Reversible Posterior Leukoencephalopathy Syndrome[8] including headache, mental status alteration, seizures, and visual disturbances[1-8]. Magnetic resonance imaging (MRI) scans of the brain are the recommended neuroimaging modalities for diagnosis. The typical MRI features of PRES show symmetric T2-weighted or fluid-attenuation inversion recovery (FLAIR) hyperintensities located in the occipital and parietal lobes with vasogenic edema in diffusion-weighted imaging (DWI)[1,2,4-8]. PRES may develop in any age group, from age 4 to 90 years, with most common among young and middle-aged adults[2,4,6]. It is associated with several risk factors including severe/uncontrolled hypertension, preeclampsia/eclampsia, renal disease, malignancy, organ transplant, immunosuppressive/cytotoxic therapies, autoimmune diseases such as systemic lupus erythematosus (SLE), thrombotic thrombocytopenic purpura, polyarteritis nodosa, rheumatoid arthritis, scleroderma, Sjogren's syndrome, Crohn's disease, and cryoglobulinemia, substance abuse disorder, and sepsis[1-3,5,6,8]. There have been no randomized controlled trials on the treatment of PRES, and treatment guidelines are by consensus opinion[1,2]. Patients with PRES usually have a good prognosis, but functional impairments or permanent neurological

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Table 1. Baseline characteristics of SLE patients with PRES in Nongkhai Hospital

Characteristics	Case 1	Case 2	Case 3	Case 4	Case 5
Gender	Female	Female	Female	Female	Female
Occupation	Teacher	Employee	Unemployed	Student	Student
Age at SLE diagnosis (year)	17	14	12	22	7
Age of onset (year)	30	17	18	22	15
Duration of disease (year)	13	3	6	3/12	8
ANA	Positive	1:1,280 speckle, 1:80 homogeneous, 1:80 anticytoplasmic	Positive	>1:5,120 coarse speckle	1:320 homogeneous, positive anticytoplasmic
Anti-double strand DNA	Positive	NA	Positive	NA	Positive
Anticardiolipin IgG	Negative	NA	Negative	Negative	Negative
Anticardiolipin IgM	Negative	NA	Negative	Negative	Negative
Lupus anticoagulant	Negative	NA	Positive	Negative	Negative
Beta-2 glycoprotein I	Negative	NA	Negative	Negative	Negative
Comorbid/risks	HT, DLP	CHF	HT, DLP	HT, DLP, AF, Graves' disease	None
Prednisolone (mg/day)	40	10	5	15	60
Immunosuppressive drugs	Cyclophosphamide	No	No	No	No
Hydroxychloroquine	No	Yes	Yes	Yes	No

SLE=systemic lupus erythematosus; ANA=antinuclear antibody; HT=hypertension; DLP=dyslipidemia; CHF=congestive heart failure; AF=atrial fibrillation

deficits can occur in severe or with delayed treatment in 10.0% to 44.0% and the mortality rate is around 3.0% to 6.0%[1-4,6,9,10]. SLE is a chronic systemic autoimmune disease characterized by the production of pathogenic autoantibodies, leading to uncontrolled inflammatory response, heterogeneous signs and symptoms, unpredictable course, and flares[11]. Neuropsychiatric involvement in SLE (NPSLE) is common with an estimated prevalence of up to 50% including syndromes of central, peripheral, and autonomic nervous systems, as well as psychiatric syndromes. However, PRES is not one of the nineteen recognized NPSLE syndromes[12]. The prevalence of PRES in SLE patients is 0.7% to 1.8%[9,13,14], which is the most common autoimmune disease reported. Furthermore, SLE is associated with irreversibility, recurrence in up to 13.0% of cases, and poor prognosis of PRES[13,15]. Delayed treatment of PRES in SLE can lead to significant neurological deficits and mortality. Therefore, PRES should be promptly recognized and monitored closely for disease prevention and treatment[14,16]. For the above reasons, the author collected a case series of SLE patients with PRES in Nongkhai Hospital between 2012 and 2023 and compared it with other studies. Physicians could use the results of the present study for diagnostic and treatment plans in SLE patients.

Materials and Methods

This case report was a retrospective study. The medical records of SLE patients (ICD-10 M320-329) with PRES (ICD-10 I678) treated in Nongkhai Hospital between 2012 and 2023 were reviewed and collected onto a medical record form. The following information were obtained, gender, occupation, age at SLE diagnosis, age of onset, duration of disease, comorbid or risks, SLE disease activity, glucocorticoids, and immunosuppressors administered before or at diagnosis, clinical presentations, laboratory data, imaging (CT brain and/or MRI brain), treatments, complications, length of stay, and outcomes.

Results

Between 2012 and 2023, 611 patients with SLE were identified. Of the 611 patients, five patients developed PRES whose prevalence was 0.8%. All patients were female, with mean age of 14.4 (SD 2.5) years at SLE diagnosis, and 20.4 (SD 2.7) years at the time of PRES, with mean disease duration of 6.1 (SD 2.2) years. Most of them were students. Three patients had hypertension and dyslipidemia. All patients received prednisone with mean dose of 26.0 (SD 10.4) mg/day before the onset of PRES, and there was concomitant use of immunosuppressant in only one patient. Characteristics of SLE patients with PRES are shown in Table 1.

In the present report, three of five patients had the disease active before admission for the past six months. Seizures were displayed by all of the patients, followed by altered mental status, status epilepticus, blurred vision, and headache. The

average time from the initial symptoms to diagnosis was 1.8 (SD 0.5) days. On physical examination, the mean systolic blood pressure was 163.4 (SD 6.3) mmHg. Lymphopenia of less than 1,500 cells/mm³ was observed in two patients. Nevertheless, hypoalbuminemia and proteinuria were observed in all of the patients. Lumbar puncture was done in only two patients, which profile was within normal range. CT brain and/or MRI brain scans were performed in all patients with the occipital lobe being the most common brain lesion involvement, in 80.0%, followed by parietal, and temporal lobe involvement. Active diseases, including lupus nephritis were observed in all patients, followed by the hematological, and cardiovascular systems in 40.0% and 20.0%, respectively. In the current study, all of the patients were treated with high-dose steroids, and only one patient was treated with cyclophosphamide. Three of the five patients needed intubation and were admitted for closed monitoring in the intensive care unit (ICU). All of them received antiepileptic drugs including benzodiazepine and phenytoin, while only two patients needed intravenous antihypertensive drugs. The most common complications during admission were infections, especially sepsis. The mean length of stay was 24.8 (SD 6.4) days. All of the patients recovered completely as shown in Table 2.

Discussion

PRES is a rare acute or subacute clinicoradiologic disorder[1-7]. The prevalence of PRES in SLE patients is 0.7% to 1.8%, affecting women more than men, at a ratio of 4 to 1, and tended to occur in patients with a mean age of 26.3 (SD 8.8) years, which is the most common autoimmune disease reported[9,13,14]. In the present study, of the 611 patients, five patients developed PRES whose prevalence was 0.8%. Of the five cases, all patients were female with a mean age of 20.4 (SD 2.7) years at the time of PRES and three of them had hypertension, which corresponded to the previous studies[1,2,9,13,14,17-24]. All of the patients received prednisone with a mean dose of 26.0 (SD 10.4) mg/day before the onset of PRES, and there was concomitant use of immunosuppressant in only one patient, which was cyclophosphamide. This study is consistent with the previous studies in which PRES in SLE is associated with multiple risk factors including hypertension, renal dysfunction, lymphopenia, dyslipidemia, heart failure, high SLE activity index scores, and younger age[12].

The pathogenesis of PRES is not completely understood[1-6,8,10,13,19]. To date, proposed potential mechanisms consist of 1) the "vasogenic theory" or blood pressure dysregulation proposes a rapid increase in arterial blood pressure to a hypertensive crisis with a sustained mean arterial pressure more than 150 to 160 mmHg, resulting in brain hyperfusion, blood-brain barrier disruption, and vasogenic edema, 2) the "cytotoxic theory" suggests that endogenous chemokines, or exogenous drugs or toxins, lead to endothelial injury, blood flow dysregulation, and immune activation, 3) the "neuropeptide theory" explained by leukocytes activation upregulates the synthesis and release vasoconstrictors leading to vasospasm and ischemia, 4) the "immunogenic theory" associated with immune system and its interaction with the endothelium such as TNF-α, IL-1, and IL-6[2,6,12,13,19]. The pathophysiology of PRES in SLE is also less well understood, which may be related to immunosuppressants, lupus itself, SLE-related hypertension, antiphospholipid antibodies, or renal failure mechanically cytokines, autoantibodies, autoreactive immune cells, endothelial activation, dysfunction, and leukocyte tracking[15,19].

In this context, three of the five patients had the disease active before admission for the past six months including hematological, renal, and cardiovascular systems that were similar to the other studies[12,13,15]. Seizures were displayed by all patients, followed by altered mental status, status epilepticus, blurred vision, and headache. On physical examination, the mean systolic blood pressure was 163.4 (SD 6.3) mmHg like the previous studies[20,23,24,25-30]. All the patients had renal involvement at PRES onset including hypoalbuminemia, proteinuria, and renal insufficiency which is similar to the previous studies[17-20,25-30]. Lumbar puncture was done in only two patients whose profile was within the normal range and did not differ from the other studies[3,6,14]. CT brain and/or MRI brain scans were performed in all patients with the occipital lobe being the most common brain lesion involvement, in 80.0%, followed by parietal, and temporal lobe involvement, which is similar to the previous studies that the posterior circulation is vulnerable to hyperfusion due to less sympathetic innervation to counter reflex parasympathetic vasodilatation[2,17-30] as shown in Table 3.

Currently, there are no established diagnostic criteria for PRES, which is often diagnosed by exclusion of other disorders, and suggested by typical clinical manifestations and neuroimaging[1-6,8,13]. Therefore, the present study suggests that SLE patients with co-morbidity hypertension presenting

Table 2. Clinical symptoms, laboratory data, and treatment outcomes of SLE patients with PRES

Characteristics	Case 1	Case 2	Case 3	Case 4	Case 5
Disease active before admission 6 months	Renal, hematological	No	No	Hematological, cardiovascular	Pulmonary
Onset of symptoms (days)	1	1	1	3	3
Signs & symptoms					
Fever	No	No	No	Yes	No
Headache	No	Yes	No	Yes	No
Nausea/vomiting	No	No	No	No	No
Blurred vision	No	Yes	No	Yes	No
Altered mental status	Yes	Yes	No	Yes	No
Seizure	Yes	Yes	Yes	Yes	Yes
Status epilepticus	Yes	Yes	No	No	No
Systolic blood pressure at presentation (mmHg)	146	184	169	161	157
Blood test at the onset					
Glucose (mg/dL)	92	104	126	146	182
WBCs (cells/mm^3)	24,410	9,200	9,780	17,670	15,390
PMN/L%	84.7/7.1	85.6/10.9	73.3/21.6	80.5/14.3	95.3/3.0
Lymphocyte (cells/mm^3)	1,733.1	1,002.8	2,112.5	2,526.8	461.7
Hb/DCT/ICT	9.8/Neg/Neg	10.0/NA/NA	12.5/Neg/4+	15.5/Neg/2+	13.7/Neg/Neg
PLT	133,000	210,000	251,000	271,000	388,000
Cr (GFR)	1.21 (60.2)	2.07 (35.0)	0.33 (162.5)	0.26 (170.9)	1.68 (41.2)
Albumin (g/dL)	1.9	2.5	2.1	3.4	1.4
AST/ALT (U/L)	462/155	26/10	22/9	27/23	22/10
Urine examination					
Albumin	2+	4+	4+	4+	4+
24 hours protein (mg)	1,635.1	NA	6,670	2,642	5,725
CSF examination	No	No	Yes	No	Yes
Open pressure	-	-	17	-	8
WBCs (cells/mm^3)	-	-	1	-	2
PMN/L %	-	-	0	-	0
Protein (g/L)	-	-	26	-	24
Glucose (mg/dL)	-	-	66	-	91
Neuroimaging					
CT brain	No	Frontal, temporal, parietal lobe	Temporal, occipital lobe	Parietal, occipital lobe	Occipital lobe
MRI brain	Parietal, occipital lobe	No	Temporal, occipital lobe	Parietal, occipital lobe	No
Disease active during admission	Renal	Renal	Renal, hematological	Renal	Renal, hematological, cardiovascular
Treatment					
Intubation	Yes	Yes	No	Yes	No
ICU	Yes	Yes	No	Yes	No
Steroid	Methylprednisolone	Dexamethasone	Dexamethasone	Dexamethasone	Dexamethasone
Immunosuppressive drugs	No	Cyclophosphamide	No	No	No
Antiepileptic drugs	Benzodiazepine, phenytoin	Benzodiazepine, phenytoin, depakin	Benzodiazepine, phenytoin	Benzodiazepine, phenytoin	Benzodiazepine, phenytoin
Antihypertensive drugs	Yes	Yes	No	No	No
Complications	HAP, diarrhea	Diarrhea	Influenza type A	Sepsis	Sepsis
Length of stay (days)	32	45	22	7	18
Outcomes	Full recovery	Full recovery	Full recovery	Full recovery	Full recovery

PMN=polymorphonuclear; L=lymphocyte; WBCs=white blood cells; Hb=hemoglobin; DCT=direct Coomb's test; ICT=indirect Coomb's test; PLT=platelet; Cr=creatinine; GFR=glomerular filtration rate; ALT=alanine aminotransferase; AST=aspartate aminotransferase; CSF=cerebrospinal fluid; ICU=intensive care unit; CT=computerized tomography; MRI=magnetic resonance imaging

Table 3. Case report of PRES in SLE patients[17-30]

First author (year)	No. of cases	Age at PRES onset/ sex	Steroid/ immunosuppression at PRES onset	SBP at PRES onset	Renal involvement at PRES onset	Symptoms	Imaging (CT/MRI brain)	Treatment	Outcomes
Ishimori ML (2007)[25]	4	47/F	Dexamethasone/IVCY	200	No	Headache, focal seizure	Parieto-occipital, cerebellum	-	Full recovery
		25/F	IVMP/none	132	No	Status epilepticus	Parieto-occipital, temporal, frontal	AED	Full recovery
		25/M	Prednisolone/IVCY	176	Yes	Headache, blindness	Parieto-occipital	Anti-HT drugs	Full recovery
		24/F	IVMP/none	169	Yes	Headache, visual change, altered mental status, seizure	Fronto-parietal	Anti-HT drugs	Full recovery
Mak A (2008)[18]	17	29.8/F (16)	IVMP (7), IV steroid (1)/IVCY (6), CSA (2), AZA (1), rituximab (1), NA (2)	187.6	13	Seizure (17), headache (12), cortical blindness (5)	Occipital (19.6%), parietal (21.5%), cerebellar (11.8%), brainstem (2.0%), pons (3.9%)	-	Full recovery (17)
Leroux G (2008)[26]	4	52/F	Prednisolone 50 mg/day/MMF	190	Yes	Headache, visual change, altered mental status, seizure	Occipital, frontal, parietal	Steroids, AED, off MMF	Full recovery
		26/F	IVCY	180	Yes	Seizure, blurred vision	Parieto-occipital, temporal, frontal	AED, off IVCY	Full recovery
		27/F	IVMP	220	Yes	Headache, vomiting, seizures, blindness	Parieto-occipital	AED, anti-HT drugs	Partially improve visual acuity
		43/F	Prednisolone 5 mg/day	210	Yes	Headache, blindness	Parieto-occipital, frontal	IVMP	Full recovery
Baizabal-Carvallo JF (2009)[17]	22	24.9±8.6/F (21)	Prednisolone (9)/AZA (6), IVCY (3)	-	16	Seizure (21), headache (19), amaurosis (10), vomiting (9), delirium (6), focal motor deficit (1)	Occipital (18), parietal (13), temporal (9), frontal (8), cerebellum (12), brainstem (4), basal ganglion (2)	Steroids, immunosuppressants, Anti-HT drugs,	Persistent neurological deficit (2), dead (1)
Chen HA (2010)[27]	1	34/F	Prednisolone 30 mg/day	190	Yes	Headache, seizure, confusion	Cerebellum, pons, midbrain, thalamus, frontal	AED, anti-HT drugs, IVMP	Dead
Sulaiman W (2011)[28]	1	33/F	None	160	Yes	Blurred vision, headache, nausea and vomiting, confusion	Parieto-occipital	None	Against advice
Barber CE (2011)[29]	7	22/F	Prednisolone 15 mg/day/MMF	200	Yes	Headache, visual change, seizure	Parieto-occipital, frontal	AED, anti-HT drug, IVMP, off MMF	Partially improve
		44/F	None	230	Yes	Nausea, vomiting, confusion	Parieto-occipital	IVMP, IVCY	Full recovery
		26/F	IVMP	191	Yes	Seizure	All lobes including cerebellum and midbrain	AED, anti-HT drugs, MMF	Full recovery
		23/F	Prednisolone 20 mg/day	204	Yes	Left side clonus and spasticity	Hematoma at parieto-occipital	IVMP, anti-HT drugs	Full recovery
		31/F	None	244	Yes	Seizure	Occipital	AED, IVMP	Full recovery
		34/F	Prednisolone 50 mg/day/AZA	216	Yes	Headache, blurred vision, nausea and vomiting, confusion	Parietal	Anti-HT drugs, IVCY, high dose prednisolone	Full recovery
		37/F	IVMP	167	Yes	Confusion, agitation, paranoid, acute blindness	Occipital, frontal	Anti-HT drugs, IVMP, IVCY	Full recovery

PRES=posterior reversible encephalopathy syndrome; SBP=systolic blood pressure; CT=computerized tomography; MRI=magnetic resonance imaging; F=female; M=male; IVMP=intravenous methylprednisolone; IVCY=intravenous cyclophosphamide; MMF=mycophenolate mofetil; AZA=azathioprine; IVIG=intravenous immunoglobulin; HT=hypertensive; NA=not available; AED=antiepileptic drug

Table 3. (continued)

First author (year)	No. of cases	Age at PRES onset/ sex	Steroid/ immunosuppression at PRES onset	SBP at PRES onset	Renal involvement at PRES onset	Symptoms	Imaging (CT/MRI brain)	Treatment	Outcomes
Liu B (2012)[19]	10	22.93±2.48/F (10)	NA	HT (8)	Yes (8)	Seizures (8), coma (6), headache (2), cortical blindness (2), stupor (1)	Occipital, parietal and/or cerebellar (10)	Anti-HT drugs, IVMP, IVCY	Full recovery (8), Partially improve (1), dead (1)
Jung SM (2013)[30]	15	31.5	Prednisolone 88 mg/day/IVCY (7), MMF (2), mizoribine (2), IVIG (1), none (3)	172	14	Seizure (14), headache (9), blurred vision (4), cortical blindness (1), psychosis (1)	Occipital (14), frontal (10), parietal (12), temporal (9), cerebellum (4)	Anti-HT drug (15), AED (13), steroid (15)	Full recovery (13), dead (1), NA (1)
Lai CC (2013)[20]	23	26.4±7.6/F (22)	Steroids (NA)/ immunosuppressive drugs (16)	187±21	23	Seizure (17), altered mental status (15), visual impairment (15), headache (13), vomiting (8)	Occipital (23), parietal (17), temporal (15), frontal (12), cerebellum (7), intracranial hemorrhage (3)	Anti-HT drugs (22), IVMP (4), immuno-suppressive drugs (12)	Full recovery (15), partially improve (1), dead (7)
Merayo-Chalico J (2014)[21]	48	27.9±1.05/F (43)	Prednisolone 31.5±3.0 mg/day/ AZA 17.5±5.3 mg/day, CY 1,385±475 mg/day, MMF 180.8±78.1 mg/day	HT (68.0%)	NA	Seizure (81%)	Occipital, parietal, frontal, cerebellum	Anti-HT drug, AED	NA
Budhoo A (2015)[22]	10	24.9/F (10)	Prednisolone (8), IVMP (3)/ MMF (5), AZA (1), none (1)	HT (8)	8	Seizure (8), altered mental status (5), focal weakness (4), visual disturbances (3), headache (2)	Occipital (9), parietal (9), frontal (4), cerebellum (3), brainstem (2), temporal (1)	Anti-HT drug (8), AED (6), IVMP (3), IVCY (4), MMF (3)	Full recovery (7), dead (3)
Damrongpipatkul U (2018)[23]	24	27.25±12.35/F (24)	IVMP (4)/IVCY (6)	186.46±29.21	86.67%	Seizure (93.33%), status epilepticus (10.71%), headache (56.67%), alteration of consciousness (56.67%), blurred vision (36.67%),	Occipital (86.67%), parietal (80.00%), frontal (76.67%), temporal (46.67%), cerebellum (43.33%)	Anti-HT drug, AED, IVMP, IVCY, rituximab, plasma exchange	Dead (7)
Cui HW (2019)[24]	30	22.83±7.64/F (28)	NA	164.83±18.42	90.00%	Seizure (80.00%), headache (43.33%), visual impairment (13.33%), coma (23.33%)	Occipital, parietal (100%), frontal (93.33%), temporal (76.67%), cerebellum (36.67%)	Anti-HT drug, AED, IVMP, IVCY, MMF	Dead (8)
Present study	5	30	Prednisolone 40 mg/day/ cyclophosphamide	146	Yes	Status epilepticus, altered mental status	Occipital, parietal	AED, IVMP	Full recovery
		17	Prednisolone 10 mg/day	184	No	Status epilepticus, altered mental status, headache, blurred vision	Frontal, temporal, parietal	AED, IVCY, dexamethasone	Full recovery
		18	Prednisolone 5 mg/day	169	No	Seizure	Temporal, occipital	AED, dexamethasone	Full recovery
		22	Prednisolone 15 mg/day	161	No	Seizure, altered mental status, headache, blurred vision	Parietal, occipital	AED, dexamethasone	Full recovery
		15	Prednisolone 60 mg/day	157	No	Seizure	Occipital	AED, dexamethasone	Full recovery

PRES=posterior reversible encephalopathy syndrome; SBP=systolic blood pressure; CT=computerized tomography; MRI=magnetic resonance imaging; F=female; M=male; IVMP=intravenous methylprednisolone; IVCY=intravenous cyclophosphamide; MMF=mycophenolate mofetil; AZA=azathioprine; IVIG=intravenous immunoglobulin; HT=hypertensive; NA=not available; AED=antiepileptic drug

with seizures and high systolic blood pressure along with a history of moderate to high dose steroids and/or immunosuppressants administration should always be suspected of PRES. Meanwhile, CT brain and/or MRI brain scans are the most important tools to establish an early diagnosis.

As well as the diagnosis of PRES, there have been no randomized trials on the various interventions used to treat PRES in SLE and treatment guidelines depend on consensus opinions. Acute management of PRES includes removing or reversing the precipitating factors and controlling blood pressure, seizure, and disease activity. Nevertheless, the discontinuation or initiation of immunosuppressive drugs should be individualized according to the whole clinical manifestations due to steroids, cyclophosphamide, and plasma exchange have proven to be effective[1-5,8-10,13]. In the current study, all the patients were treated with high-dose steroids, and only one patient was treated with cyclophosphamide. Three of five patients needed intubation and were admitted to closed monitoring in the ICU. All of them received antiepileptic drugs including benzodiazepine and phenytoin, while only two patients needed intravenous antihypertensive drugs. The most common complications during admission were infections, especially sepsis. All the patients recovered completely. Thereby, the present study suggests that treatment of PRES in SLE should be individualized based on clinical presentations, and disease activity especially lupus nephritis should be evaluated. Accordingly, sepsis prevention, and ICU admission in severe cases are crucial for a favorable outcome.

Conclusion

The present study suggests that SLE patients with co-morbidity hypertension presenting with seizures and high systolic blood pressure along with a history of moderate to high dose steroids and/or immunosuppressants administration should always be suspected of PRES. Meanwhile, CT brain and/or MRI brain scans are the most important tools to establish an early diagnosis. Individualized treatment should be based on clinical presentations, and disease activity evaluation, especially lupus nephritis. Accordingly, sepsis prevention, and ICU admission in severe cases are crucial for a favorable outcome.

What is already known on this topic?

Currently, there are no established diagnostic criteria for PRES, consequently, PRES is often diagnosed by exclusion of other disorders, and suggested by typical clinical manifestations and neuroimaging. As well as the diagnosis of PRES, there have been no randomized trials on the various interventions used to treat PRES in SLE and treatment guidelines depend on consensus opinions.

What does this study add?

SLE patients with co-morbidity hypertension presenting with seizures and high systolic blood pressure along with a history of moderate to high dose steroids and/or immunosuppressants administration should always be suspected of PRES. Meanwhile, CT brain and/or MRI brain scans are the most important tools to establish an early diagnosis. Individualized treatment should be based on clinical presentations, and disease activity evaluation, especially lupus nephritis. Accordingly, sepsis prevention, and ICU admission in severe cases are crucial for a favorable outcome.

Ethical approval

The present case reports were approved by The Research Ethics Committee (REC) at Nongkhai Hospital (No. 41/2566). The consent form was waived due to the retrospective nature of the presentation.

Conflicts of interest

The authors declare no conflict of interest.

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