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# Long-Term Follow-Up of Patients with a Diagnosis of Posterior Reversible Encephalopathy Syndrome

Mehmet Canpolat[1], Gül Demet Kaya Özçora[1], Hakan Poyrazoğlu[1], Huseyin Per[1], Abdulhakim Çoşkun[2], Hakan Gümüş[1], Duran Arslan[3], Ekrem Ünal[4], Musa Karakükçü[4], Türkan Patıroğlu[4], Sefer Kumandaş[1]

[1]Department of Pediatric Neurology, Erciyes University School of Medicine, Kayseri, Turkey
[2]Department of Pediatric Radiology, Erciyes University School of Medicine, Kayseri, Turkey
[3]Department of Pediatric Gastroenterology, Erciyes University School of Medicine, Kayseri, Turkey
[4]Department of Pediatric Hematology-Oncology, Erciyes University School of Medicine, Kayseri, Turkey

## What is already known on this topic?

- *PRES is often reversible, but the development of epilepsy is known to be a neurologic complication.*

## What this study adds on this topic?

- *Information on the long-term neurologic prognosis due to childhood PRES and duration of antiepileptic treatment.*

## ABSTRACT

**Objective:** The essential characteristics of posterior reversible encephalopathy syndrome (PRES) are the presence of acute onset neurologic symptoms, focal vasogenic edema at neuroimaging, and reversible clinical and/or radiologic findings. This study aimed to evaluate the clinical findings, causes, radiologic findings, and prognoses of patients with PRES.

**Methods:** Patients with PRES confirmed with clinical and radiologic findings by a pediatric neurologist were evaluated retrospectively.

**Results:** Seventeen patients with PRES were evaluated (mean age at onset, 10.23 ± 4.65 years; range, 2-17 years; girls, 29.4% [n = 5]). The mean length of follow-up was 6 ± 2.3 years (range, 3.4-10 years). Mortality due to primary disease occurred in 4 patients (23.5%) during follow-up. PRES was derived from renal diseases in 10 patients (58.8%), hematologic diseases in 6 patients (35.3%), and liver disease in one patient (5.9%). Hypertension was present in 16 patients (94.1%) at onset of PRES (>99th percentile). Seizure, the most frequent initial symptom, was observed in 82.4% (n = 14). Blurred vision and headache were the initial symptoms in 3 patients (17.6%). Sequelae were observed at magnetic resonance imaging (MRI) in 6 patients. Development of epilepsy was determined as a sequela in 4 patients (23.5%) and mental motor retardation in 2 patients (11.8%).

**Conclusion:** Epilepsy is uncommon in patients who have recovered from PRES. The presence of gliosis on MRI and interictal epileptic discharges on electroencephalograms are major risk factors for the development of epilepsy. Antiepileptic treatment can be stopped in the early period in patients with normal MRI and electroencephalogram by eliminating the factors that trigger the seizures.

**Keywords:** Child, hypertension, posterior reversible encephalopathy syndrome, prognosis

## INTRODUCTION

Posterior reversible leukoencephalopathy syndrome (PRES) was first reported by Hinchey et al.[1] in 1996. Its clinical manifestation is characterized by altered mental function, loss of vision, altered consciousness, headache, and seizures, frequently associated with reversible vasogenic edema in posterior cerebral white matter.[1-4] Analysis of the general characteristics of patients with PRES shows that the main causes include pre-eclampsia, eclampsia, hypertensive encephalopathy, rheumatologic diseases, renal diseases, solid organ or bone marrow transplantation, blood transfusion, hypomagnesemia, sepsis, malignancy, hematologic diseases such as sickle cell anemia, immune failure, and use of immunosuppressive drugs.[1-11]

**Corresponding author:**
Gül Demet Kaya Özçora
✉guldemetkaya@hotmail.com
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The main underlying diseases in children and adults are hematological or neoplastic disorders and kidney diseases.[7] The radiologic findings of PRES are similar to hypertensive encephalopathy.[5] Although the radiologic findings generally indicate involvement of the posterior regions of the brain, involvement of frontal and parietal lobes, basal ganglia, brainstem, and spinal cord may also be seen.[1-11] The underlying pathophysiology of PRES is vasogenic edema due to cerebral autoregulation (hypertension and cerebral hyperperfusion) and endothelial dysfunction.[3,4]

The number of studies evaluating the long-term neurologic prognosis and sequelae, particularly in children, is limited.[12-19] The purpose of this study was to contribute to the current literature by evaluating the clinical findings, causes, radiologic findings, and long-term neurologic prognoses of patients with a diagnosis of PRES followed up in our clinic.

## METHODS

This study was approved by the Erciyes University Scientific Research Committee (nos.2016/436,2017/211 and 2017/574). Patients followed up with the diagnosis of PRES by the Department of Child Health and Diseases at Medical Faculty between 2008 and 2016 were included in the study. Clinical and radiologic findings were evaluated retrospectively by a pediatric neurologist and pediatric radiologist. Informed consent was obtained from all participants.

The demographic data, symptoms on presentation, cerebral magnetic resonance imaging (MRI) findings, causative risk factors, electroencephalographic findings, and neurologic prognoses (such as epilepsy and mental motor retardation) were assessed. Motor impairment was evaluated by neurological examination, and mental impairment was evaluated with age-appropriate development and intelligence tests.

Mean arterial pressure was evaluated for all cases. Hypertension was defined as a value exceeding the 95th percentile for height based on the criteria of the National High Blood Pressure Education Program Working Group on High Blood Pressure in Children and Adolescents.[20] All patients underwent MRI, and examination included axial T1-weighted images (WI), T2-WI, fluid-attenuated inversion recovery (FLAIR), and diffusion-weighted sequences for the diagnosis of PRES. Cranial MRI performed using a 1.5 T scanner within 48 hours from the onset of disease were regarded as acute MRI; those conducted after 2 weeks and in the third month were defined as follow-up MRI. All MRI findings were evaluated by an experienced pediatric radiologist. The patients' ictal encephalography (EEG) records were not obtained. Interictal sleep and awake EEG were performed in the acute period (within 48 hours) and at routine follow-up (1 and 3 months) for at least 30 minutes using the 10-20 system in the interictal period. The records were evaluated by a pediatric neurologist. The patients' neurologic prognoses (mental motor delay, epilepsy, etc.) were based on routine follow-up findings at 1, 6, and 12 months and then during annual routine follow-ups.

## RESULTS

Seventeen patients diagnosed with PRES were investigated. The mean age at onset of PRES was 10.23 ± 4.65 years (range, 2-17 years), and 29.4% (n = 5) of patients were girls. The mean duration of follow-up was 6 ± 2.3 years (range, 3.4-10 years). Four patients (23.5%) died from primary diseases during the follow-up period (nos. 1, 4, 10, and 11). Patients 4 and 11 died due to primary disease in the acute period, and patients 1 and 10 died during the follow-up. PRES resulted from renal diseases in 10 patients (58.8%), hematologic diseases in 6 patients (35.3%), and liver disease in one patient (5.9%). The demographic and causative characteristics of the patients in this study are shown in Table 1.

Four patients (23.5%) were receiving immunosuppressive therapy at onset of PRES, 5 (29.4%) were receiving therapy in accordance with the specific protocols of induction phase chemotherapy, 2 (11.8%) were receiving corticosteroid therapy, 2 (11.8%) were receiving corticosteroid and immunosuppressive therapy, and 4 (23.5%) were receiving no treatment (Table 1).

The most frequent initial symptom in the present study was seizure, observed in 82.4% (n = 14) of patients. Initial symptoms were blurred vision and headache in 3 cases (17.6%) (nos. 2, 9, and 14) (Table 1). Seizure was also observed in 3 patients (nos. 2, 9, and 14) during follow-up.

Clinical status epilepticus was not observed in any patient. The most frequent symptoms after seizure were altered mental status in 47% (n = 8), headache, nausea, and vomiting in 35.3% (n = 6) and visual disturbance in 29.4% (n = 5).

Generalized tonic–clonic seizures were observed in 58.8% (n = 10) of patients and focal seizures with or without secondary generalization in 7 patients (41.2%) (nos. 3, 9, 10, 11, 12, 14, and 17). Convulsions were drug-resistant during the acute period in 2 patients (11.8%) (nos. 4 and 17) and were brought under control with secondary antiepileptic treatments. Seizures recurred during the first 24 hours in 6 patients (35.3%) (nos. 4, 9, 10, 11, 12, and 17), and acute recurrence after 24 hours was determined in 2 patients (11.8%) (nos. 4 and 17).

Acute EEG recordings revealed diffuse delta slowing in 3 patients (17.7%) (nos. 1, 3, and 4), intermittent focal or diffuse delta slowing in 4 patients (23.5%) (nos. 2, 9, 10, and 13), background slowing in 8 patients (47%) (nos. 5, 6, 7, 8, 11, 12, 15, and 16), diffuse delta slowing plus sharp wave discharges in one patient (5.9%) (no. 17), and intermittent generalized slowing plus sharp waves in one patient (5.9%) (no. 14). Non-convulsive status epilepticus was not observed in any patient (Table 2). Fourteen patients underwent first control EEGs within a mean of 32 ± 7 days (range, 21-45 days). EEG could not be performed due to exitus in 2 patients (nos. 4 and 11) and in one patient due to illness. EEGs were normalized in 6 patients (6 of 14, 42.8%) (nos. 2, 3, 9, 12, 15, and 16), and exhibited background slowing in 6 patients (6 of 14, 42.8%) (nos. 5, 6, 7, 8, 10, and 13), background slowing plus interictal epileptiform discharge (IED) activity in patient 14 (1 of 14, 7.1%), and intermittent generalized delta slowing plus IED in patient 17 (1 of 14, 7.1%) (Table 2). Thirteen patients underwent second control EEGs (76.5%) (3 patients died [nos. 1, 4, and 11] and one patient failed to attend for the control EEG [no. 16]) within a mean 169 ± 36 days (range, 86-210 days). EEG findings were normal in 76.9% of patients (10 of 13). Background slowing plus bitemporal, centroparietal sharp waves were observed in patient 14, background slowing

**Table 1.** Clinical and Demographic Data of the Patients With PRES

| Case | Sex/Age (Years) | Primary Diagnosis | Underlying Cause of Primary Diagnosis | Drugs | Initial PRES Symptom | Acute Seizure Recurrence | Blood Pressure | Neurologic Prognosis |
|---|---|---|---|---|---|---|---|---|
| 1 | M/9 | Renal Tx | VUR | AZA, CsA | Seizure | None | 210/110 | Exitus (second month) |
| 2 | M/9 | APSGN | APSGN | – | Blurred vision and headache | None | 190/140 | No sequelae |
| 3 | M/10 | CRF | Nephrotic syndrome | Methylprednisolone | Seizure | None | 210/120 | Epilepsy |
| 4 | F/8 | CRF | Henoch-Schonlein purpura | Methylprednisolone, eculizumab | Seizure | + | 220/110 | Exitus acute period |
| 5 | M/17 | HSCT/aGVHD | CML | Tacrolimus, intravenous immunoglobulin | Seizure | None | 120/70 | No sequelae |
| 6 | M/6 | Burkitt lymphoma | Burkitt lymphoma | Vincristine, cyclophosphamide, methylprednisolone, methotrexate, adriamycin, cytosine arabinoside | Seizure | None | 110/80 | No sequelae |
| 7 | F/2 | ALL | ALL | Vincristine | Seizure | None | 210/110 | Epilepsy |
| 8 | M/7 | Wilms tumor | Wilms tumor | Vincristine | Seizure | None | 230/120 | No sequelae |
| 9 | F/17 | CRF | Systemic lupus erythematosus | Methylprednisolone | Blurred vision and headache | + | 170/100 | No sequelae |
| 10 | F/5 | Relapse ALL | ALL | Methotrexate, vincristine, L-asparaginase, cytarabine | Seizure | + | 200/110 | Exitus (eighth month) |
| 11 | M/11 | ALL | ALL | Methotrexate, vincristine, L-asparaginase, cytarabine | Seizure | + | 220/110 | Exitus acute period |
| 12 | M/13 | APSGN | APSGN | – | Seizure | + | 180/110 | No sequelae |
| 13 | M/14 | CRF | FSGS | Methylprednisolone | Seizure | None | 180/130 | No sequelae |
| 14 | M/4 | Liver Tx | Biliary atresia | Tacrolimus | Blurred vision and headache | None | 110/90 | Epilepsy+mental motor retardation |
| 15 | M/16 | CRF, peritoneal dialysis | Bardet–Biedl syndrome | | Seizure | None | 180/130 | No sequelae |
| 16 | M/11 | APSGN | APSGN | – | Seizure | None | 150/100 | No sequelae |
| 17 | F/14 | CRF, renal Tx | VUR | Tacrolimus, mycophenolate mofetil | Seizure | + | 160/100 | Epilepsy+mental motor retardation |

ALL, acute lymphoblastic leukemia; APSGN, acute post-streptococcal glomerulonephritis; AZA, azathioprine; CML, chronic myeloid leukemia; CRF, chronic renal failure; CsA, cyclosporine A; F, female; FSGS, focal segmental glomerulosclerosis; HSCT/aGVHD, hematopoietic stem cell transplantation/acute graft-versus-host disease; M, male; Tx, transplant; VUR, vesicoureteral reflux.

**Table 2.** Demographic, Radiologic, and EEG Data of the Patients with PRES

| Patient No. | Sex/Age (Years) | Primary Diagnosis | Acute MRI Findings | Control MRI | Acute EEG | First Control EEG | Second Control EEG | Prognosis |
|---|---|---|---|---|---|---|---|---|
| 1 | M/9 | Renal Tx | Bilateral FPO, thalamus, basal ganglion, brainstem, cerebellum | Cerebral cerebellar atrophy | Continued generalized delta slowing | None | None | Exitus (second month) |
| 2 | M/9 | APSGN | Bilateral PO | Normal | Intermittent focal delta slowing | Normal | Normal | No sequelae |
| 3 | M/10 | CRF | Bilateral PO | Normal | Continued generalized delta slowing | Normal | Normal | Epilepsy |
| 4 | F/8 | CRF | Bilateral PO, thalamus, brainstem, cerebellar infarction | None | Continued generalized delta slowing | None | None | Exitus acute period |
| 5 | M/17 | HSCT/aGVHD | Bilateral FPO | Normal | Background slowing | Background slowing | Normal | No sequelae |
| 6 | M/6 | Burkitt lymphoma | Bilateral PO, frontal | Normal | Background slowing | Background slowing | Normal | No sequelae |
| 7 | F/2 | ALL | Bilateral PO | PO gliosis | Background slowing | Background slowing | Normal | Epilepsy |
| 8 | M/7 | Wilms tumor | Bilateral PO | Normal | Background slowing | Background slowing | Normal | No sequelae |
| 9 | F/17 | CRF | Bilateral PO | Normal | Intermittent focal delta slowing | Normal | Normal | No sequelae |
| 10 | F/5 | ALL | Bilateral PO, basal ganglion | PO gliosis, putaminal necrosis, cerebral atrophy | Intermittent diffuse delta slowing | Background slowing | Background slowing | Exitus (eighth month) |
| 11 | M/11 | ALL | Bilateral FPO | None | Background slowing | None | None | Exitus acute period |
| 12 | M/13 | APSGN | Bilateral FPO | Normal | Background slowing | Normal | Normal | No sequelae |
| 13 | M/14 | CRF | Bilateral PO | Normal | Intermittent focal delta slowing | Background slowing | Normal | No sequelae |
| 14 | M/4 | Liver Tx | Bilateral PO, frontal | PO gliosis, cerebral atrophy | Intermittent generalized slowing+sharp waves | Background slowing+ sharp wave | Background slowing+ sharp wave | Epilepsy+mental motor retardation |
| 15 | M/16 | CRF | Bilateral PO | Cerebral atrophy | Background slowing | Normal | Normal | No sequelae |
| 16 | M/11 | APSGN | Bilateral FPO | Normal | Slow background activity | Normal | None | No sequelae |
| 17 | F/14 | CRF, renal Tx | Bilateral FPO basal ganglion, cerebellar infarction | PO gliosis, cerebral atrophy, cerebellar volume loss | Continued generalized delta slowing+plus sharp wave | Intermittent generalized delta slowing+sharp wave | Background slowing+sharp waves | Epilepsy+mental motor retardation |

ALL, acute lymphoblastic leukemia; APSGN, acute post-streptococcal glomerulonephritis; CRF, chronic renal failure; F, female; FPO, fronto-parieto-occipital; HSCT/aGVHD, hematopoietic stem cell transplantation/acute graft-versus-host disease; M, male; PO, parieto-occipital; Tx, transplant.

plus parieto-occipital sporadic sharp waves in patient 17, and background slowing in patient 10 (Table 2).

Antiepileptic therapy was tapered and stopped within 1 year in patients 2, 3, 5, 6, 8, 9, 12, 13, 15, and 16. Patient 16 discontinued antiepileptic therapy of his own volition. However, patients 7, 10, 14, and 17 continued to receive antiepileptic therapy. Patient 10 died during follow-up. Treatment was maintained due to sequelae at MRI for patient 7. Only patients 14 and 17 were still receiving antiepileptic therapy at the end of 2 years.

IED activity persisted in patient 14 at a 5-year follow-up with no seizure recurrence. At the end of 5 years, seizure recurrence was observed when antiepileptic therapy was tapered. In patient 17, seizures and epileptic activity on EEGs persisted after 6 years and follow-ups are continuing. This patient has received 2 antiepileptic treatments. Non-provoked seizures were seen 27 months after antiepileptic treatment cessation in patient 3 and after 23 months in patient 7. Development of epilepsy as a sequela was detected in 4 patients (23.5%) (nos. 3, 7, 14, and 17) and mental motor retardation in 2 patients (11.8%) (nos. 14 and 17).

MRI T2-WI and FLAIR sequences in the acute period revealed bilateral involvement of the occipital and parietal lobes in 100% of patients, frontal lobes in 41.2%, basal ganglia and cerebellum in 17.6%, thalamus and brainstem in 11.7%, and temporal lobe involvement in 5.9%. Cerebellar infarction was detected in 2 patients (11.8%) (nos. 4 and 17) (Table 2).

Sequelae at MRI were observed in 6 patients at 52.5 ± 24.6 days (nos. 1, 7, 10, 14, 15, and 17) (excluding the patients who died in the acute period). Cerebral and cerebellar atrophy was observed in patient 1, parieto-occipital gliosis in patient 7, putaminal necrosis, cerebral atrophy, and parieto-occipital gliosis in patient 10, cerebral atrophy and bilateral parieto-occipital gliosis-volume loss in patient 14, moderate cerebral atrophy in patient 15, and cerebral atrophy, cerebellar volume loss, and parieto-occipital gliosis in patient 17 (Table 2).

## DISCUSSION

The 3 basic features of PRES are the presence of acute onset neurologic symptoms, findings of (focal) vasogenic edema on neuroimaging, and reversible radiologic and/or clinical findings. Children are more vulnerable to PRES than adults because cerebral autoregulation is accustomed to lower blood pressure.[3] A greater prevalence of this syndrome has been suggested in kidney transplant recipients and patients with kidney disease.[21] In our cases, PRES resulted from renal diseases in 10 patients (58.8%).

Although encephalopathy and altered mental status are the most common initial symptoms in adulthood, as in our patients seizure is often the most common initial symptom in children with PRES due to delayed recognition of altered mental status or visual changes.[3] Clinical status epilepticus was not observed in any patient. The occipital and parietal lobes were most commonly involved. In 7 cases ( nos. 2, 3, 7, 8, 9, 13, and 15) only parieto-occipital lobe involvement was detected. However frontal lobe, temporal deep white matter, thalamus, basal ganglia, brainstem, and cerebellum involvement may also be seen atypically.[13] MRI involvement was not affected by etiological factors but basal ganglia involvement was more common in cases with preeclampsia-eclampsia and cerebellar involvement was more common with autoimmunity.[22,23] All of our cases with cerebellar involvement were receiving immunosuppressive therapy.

Lesions are often reversible but irreversibility can be seen in atypical involvement. A large study that followed up MRI imaging of 364 patients revealed no significant change in 4 patients, but lesions were reversible in 360 patients (partial, 87 patients; mediantime, 18 days [range, 0.5-300 days]; complete, 273 patients; median time, 21 days [range, 1-720 days]).[24] Similarly, in the present study, sequelae at MRI were observed at first control in 6 cases (40%) (median time, 52.5 ± 24.6 days; excluding 2 patients who died in the acute period). Half of the patients who had sequelae had undergone transplantation and half of the patients had renal diseases. Two of the patients with sequelae died during follow-up due to their primary disease. Atypical and widespread involvement was due to the primary disease and increased the likelihood of sequelae. While there was no immunosuppressive therapy in cases with parieto-occipital involvement, it was present in 5 cases with atypical involvement.

Generalized and focal epileptic seizures are seen in approximately one-third of all patients; seizures in 3-13% of these cases can result in status epilepticus, one of the most severe and potentially life-threatening complications of PRES.[3] In this study, generalized tonic–clonic seizures were seen in 58.8% (n = 10) of patients and focal seizures with or without secondary generalization in 41.2% (n = 7)). Refractory clinical seizures not occur, it was determined that altered consciousness or vision changes could indicate focal status epilepticus, and focal rhythmic activities were observed in ictal EEGs.[16,17] Convulsions were drug-resistant in the acute period in 2 patients (11.8%) and were controlled with a second antiepileptic therapy. We observed that these 2 cases had widespread involvement, sequelae, and exitus. Perhaps these cases also had non-convulsive seizures. We believe that EEG is as important as MRI and can provide data that may affect the prognosis. EEG is described as a useful examination for the diagnosis and follow-up of PRES.[25]

Although focal rhythmic activities are observed in the acute period, the principal EEG findings in patients with PRES are diffuse theta slowing, delta slowing, rhythmic delta activity, diffuse or focal (symmetric) slowing interfering with background activity, epileptiform discharges, and periodic lateralizing epileptiform discharges.[3] The most common EEG finding in other studies was generalized or focal delta slowing.[18] We did not have normal EEGs, and our EEG findings did not follow a specific pattern consistent with the literature. Fourteen cases underwent first control EEGs in a mean of 32 ± 7 days (range, 21-45 days) and normalized in 6 patients (42.8%); second control EEGs were performed on 13 patients in a mean of 169 ± 36 days (range, 86-210 days) and were normal in 76.9% of patients. IED discharges detected at the first EEG did not disappear.

Outcomes in cases of pediatric PRES are good, although long-term neurologic sequelae may develop, particularly epilepsy and residual MRI abnormalities. The patients did not have any other PRES attacks during the follow-up period. Development of epilepsy as a sequela was observed in other studies: Datar et al.,[16] 2.4%; Sha et al.,[18] 2.6%; Heo et al.,[19] 3.9%; Darwish et al.,[26] 8;3%; Endo et al.,[15] 25%. The longest median follow-up in these studies was 3.2 years.[16] In the present study, the mean duration of follow-up was 6 ± 2.3 years (range, 3.4-10 years). Thus, our study is the longest pediatric study to date.

Development of epilepsy was detected as a sequela in 2 patients (11.7%) at the end of 2 years and in 4 patients (23.5%) at the end of 5 years. The difference in the rate of development of epilepsy in studies may be due to variation in the follow-up period, the number of patients, and the cause.

Changes in the MRI findings were present in 3 (75%) of the 4 patients with a diagnosis of epilepsy as a sequela (Table 2). Four patients followed up with epilepsy, only patient 17 had a history of afebrile seizure after a ventriculoperitoneal shunt operation in the infantile period before PRES. Two epilepsy diagnosed patients had a transplantation backgrounds, one due to chronic renal failure and the another due to acute lymphocytic leukemia. Two of the 6 patients who had sequelae on MRI died during the follow-up and 3 of the remaining 4 patients had seizures. Patient 15 had cerebral atrophy on MRI but no gliosis. We believe that cerebral atrophy has a cumulative effect due to the underlying chronic disease rather than the course of PRES or to the immunosuppressive or immunomodulatory therapies used. For example, patient 15 has Bardet–Biedl syndrome and cerebral and cerebellar atrophy is associated with that.

Anticonvulsive therapy is frequently required.[27,28] However, the optimal duration of therapy is uncertain. Generally, it is reported that anticonvulsive drugs can be reduced and discontinue when the patient is asymptomatic and the lesions are entirely reversed at imaging. In our study, antiepileptic treatment was continued for at least 6 months and stopped when EEG and MRI findings were normal. At the end of 2 years, antiepileptic treatment was continued in only 2 patients who had abnormal findings on MRI. At the end of 5 years, seizure recurrence was observed in 4 patients (nos. 3, 7, 14, and 17). MRI pathology continued in 3 patients and EEG abnormality in 2 patients, but patient 3 did not have any predictive factors and had no family history of epilepsy. We found that long-term use of antiepileptics did not prevent the development of epilepsy. Patient 7 received antiepileptic therapy for 2 years due to MRI pathology. We found that the prognosis was better in those with acute diseases and the antiepileptic treatment can be terminated in a short time. Chronic diseases (especially renal diseases and transplantation) increase the PRES attack severity and depending on this the sequelae lesions and development of epilepsy. Two cases who has mental impairment after PRES were transplanted patients and those who had severe attacks. They had no mental impairment before PRES attack. We think that mental impairment sekonder due to a severe PRES attack. Depending on the presence of the underlying chronic disease and the severity of the disease increases the severity of the attack and the attack severity causes the development of sequela and epilepsy. Mortality was higher in atypical involvement (brainstem, basal ganglia ve cerebellum). We observed these risks more in transplanted patients with renal diseases and in the use of immunosuppressive therapy.

## CONCLUSION

We believe that the underlying disease was a facilitating factor for the neurologic sequelae, also the development of epilepsy is associated with the presence of sequelae changes on MRI and IEDs in EEG. The limiting factors of our study are the different causes and the inability to record ictal EEG. PRES is a reversible condition in patients without chronic disease and in those who are not receiving extreme immunosuppressive therapy. Antiepileptic treatment can be stopped in an early period in patients with normal findings on MRI and EEG by eliminating the factors that trigger the seizures.

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**Informed Consent:** Written informed consent was obtained from all participants who participated in this study.

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