Posterior Reversible Encephalopathy Syndrome (PRES) Associated with Cerebral Microbleeds in a Child with Severe Acute Asthma. Medical Case Report / Case Report.

ABSTRACT

Background: Posterior Reversible Encephalopathy Syndrome (PRES) is a rare clinico-radiological entity characterized by signal abnormalities predominantly involving the posterior cortico-subcortical regions. Its association with severe asthma exacerbation and cerebral microbleeds represents an exceptionally rare clinical presentation, especially in pediatric patients.

Case Presentation: We report the case of a 14-year-old girl with a history of asthma admitted to the pediatric intensive care unit for severe acute asthma exacerbation complicated by refractory status epilepticus. Brain MRI revealed bilateral fronto-parieto-occipital FLAIR hyperintensities consistent with PRES, associated with multiple cerebral microbleeds involving the corpus callosum and white matter, as well as a small subacute hematoma of the left cerebral peduncle. The patient improved under anticonvulsant therapy and intensive management of severe acute asthma.

Conclusion: This case highlights the importance of considering PRES in any asthmatic patient presenting neurological manifestations in the intensive care setting. Cerebral microbleeds represent a serious complication requiring close neuroradiological follow-up.

Keywords: PRES; cerebral microbleeds; severe acute asthma; pediatrics; pediatric intensive care; status epilepticus.

1. Introduction

Posterior Reversible Encephalopathy Syndrome (PRES) is a clinico-radiological syndrome first described by Hinchey et al. in 1996. It is characterized by acute neurological manifestations including headaches, seizures, altered consciousness, and visual disturbances associated with vasogenic cerebral edema predominantly involving the posterior regions of the brain on magnetic resonance imaging (MRI).

Although PRES is classically associated with arterial hypertension, eclampsia, drug toxicity such as immunosuppressive agents and chemotherapy, or renal diseases, its etiology remains multifactorial. Cases occurring in the context of severe acute asthma and respiratory distress remain extremely rare in the literature, and the association with cerebral microhemorrhages is even more exceptional.

We report the case of a 14-year-old adolescent hospitalized in the pediatric intensive care unit for severe acute asthma exacerbation complicated by refractory status epilepticus, whose brain imaging revealed posterior reversible encephalopathy syndrome associated with multiple cerebral microbleeds.

2. Case Presentation

2.1 Patient Information and Reason for Admission

The patient was a 14-year-old female adolescent with no parental consanguinity and no notable family history. Her past medical history included personal atopy, recurrent episodes of tonsillitis, hospitalization at the age of one year for wheezing dyspnea, and known asthma under regular follow-up. There was no recent history of corticosteroid therapy. She was initially admitted to the general pediatric department on 11/12/2025 for severe acute asthma exacerbation and was subsequently transferred to the pediatric intensive care unit because of clinical deterioration.

2.2 History of Present Illness

Two days before admission, the patient developed wheezing dyspnea associated with dry cough. Initial treatment at home consisted of inhaled salbutamol (Ventolin). Due to progressive worsening and lack of clinical improvement, she was hospitalized in the general pediatric department. Her respiratory and neurological condition progressively deteriorated, leading to transfer to the pediatric intensive care unit for management of severe acute asthma complicated by refractory status epilepticus.

The patient had been followed for asthma for several years and was receiving maintenance treatment. However, she had not attended specialized follow-up consultations for approximately eight months, during which she experienced weekly dyspneic episodes that were inadequately treated.

2.3 Clinical Examination on Admission to the Pediatric Intensive Care Unit

Neurological examination: Glasgow Coma Scale = 15/15, conscious patient with recurrent seizures.

Respiratory examination: Oxygen saturation = 99% under high-concentration mask, respiratory rate = 37 cycles/minute, diffuse bilateral wheezing on auscultation, bilateral intercostal retractions, and use of accessory respiratory muscles.

Hemodynamic examination: Heart rate = 169 beats/minute, capillary refill time < 3 seconds, body temperature = 37.1°C.

Continuous monitoring was initiated with continuous nebulized salbutamol, intravenous corticosteroid therapy (2 mg/kg/6h), magnesium sulfate, and amoxicillin-clavulanic acid. Due to neurological worsening, admission to intensive care became necessary.

2.4 Evolution in the Pediatric Intensive Care Unit

The patient was intubated and mechanically ventilated because of worsening neurological and respiratory status. Ventilatory parameters were adjusted accordingly.

Arterial blood gas analysis showed severe respiratory acidosis:
- pH = 7.016
- $PaCO_2$ = 91 mmHg
- PaO_2 = 150 mmHg (FiO_2 = 42%)
- Hematocrit = 42%
- Total hemoglobin = 14.3 g/dL
- Oxygen saturation = 88%

Laboratory investigations revealed:
- Hemoglobin = 8.5 g/dL
- White blood cells = 19,000/mm^3
- Normal platelet count
- Elevated C-reactive protein
- Sodium = 133 mEq/L
- Potassium = 4.3 mEq/L
- Elevated total bilirubin

Blood cultures were positive for Serratia marcescens sensitive to ceftriaxone, gentamicin, imipenem, and ciprofloxacin, with resistance to several cephalosporins, as well as Burkholderia cepacia sensitive to selected antibiotics. Urine culture was sterile.

The infectious management included ceftriaxone and trimethoprim-sulfamethoxazole. Renal ultrasound showed minimal left hydronephrosis.

2.5 Neurological Assessment

Because of recurrent seizures refractory to midazolam, a complete neurological workup was performed.

Electroencephalography (EEG): Periodic right occipital spike-wave discharges of lesional origin.

Brain CT scan (04/01/2026): Bilateral fronto-parietal cortical-subcortical hypodense lesions predominantly on the left side and right frontal region, with no enhancement after contrast injection. Minimal biventricular dilatation without evidence of active hydrocephalus.

Brain MRI (05/01/2026): Findings consistent with posterior reversible encephalopathy syndrome involving the posterior cerebral regions. Multiple cerebral microbleeds involving particularly the corpus callosum, possibly related to acute respiratory distress syndrome. Small subacute hematoma of the left cerebral peduncle. Radiological signs suggestive of intracranial hypertension.

2.6 Treatment

Therapeutic management in intensive care included:

- Anticonvulsants:
  - Phenobarbital (Gardenal) 250 mg/day
  - Carbamazepine (Tegretol) 200 mg twice daily

- Antibiotics:
  - Ceftriaxone
  - Trimethoprim-sulfamethoxazole

- Anti-asthmatic treatment:
  - Continuous salbutamol nebulizations
  - Corticosteroid therapy

- Gastric protection:
  - Proton pump inhibitor 40 mg/day

- Nutrition:
  - Enteral feeding through nasogastric tube

- Additional supportive therapy:
  - Smecta if diarrhea
  - Multivitamin supplementation
  - Nicardipine (Loxen)
  - Repeated ocular care

2.7 Outcome

The patient progressively improved under treatment. Neurologically, seizures were controlled with anticonvulsant therapy. The patient was successfully extubated on day 38 of hospitalization. Respiratory status gradually stabilized with control of severe asthma. Close neurological monitoring was maintained throughout hospitalization.

3. Discussion

3.1 PRES: General Considerations

Posterior Reversible Encephalopathy Syndrome is an anatomo-clinical entity characterized by acute neurological symptoms associated with imaging abnormalities. Its pathophysiology is believed to involve dysregulation of cerebral vascular autoregulation leading to vasogenic edema predominantly affecting occipital and parietal regions, which are less richly innervated by the sympathetic nervous system.

The most commonly identified precipitating factors include severe hypertension, eclampsia, immunosuppressive therapy, chemotherapy, severe infections, and hemolytic uremic

syndrome. In pediatrics, PRES represents approximately 3–5% of reported cases and occurs predominantly in nephrological and oncological settings.

3.2 PRES and Severe Acute Asthma: Potential Mechanisms

The association between PRES and severe acute asthma is extremely rare. In our case, several mechanisms may explain the development of PRES:

• Severe hypercapnia: arterial blood gas analysis showed marked respiratory acidosis (pH = 7.016, $PaCO_2$ = 91 mmHg), which may induce cerebral arterial vasodilation and disruption of autoregulation.

• Cerebral hypoxia: despite acceptable oxygen saturation under oxygen therapy, severe asthma may have caused fluctuations in cerebral oxygenation.

• Hypertensive surges: blood pressure fluctuations frequently observed during severe asthma exacerbations may exceed cerebral autoregulatory capacity.

• Associated bacterial sepsis: positive blood cultures for Serratia marcescens may have contributed to blood-brain barrier dysfunction.

• Adverse drug effects: high doses of bronchodilators and corticosteroids may participate in cerebral vascular instability.

3.3 Cerebral Microbleeds Associated with PRES

Cerebral microbleeds are a recognized but uncommon complication of PRES, reported in approximately 9–15% of cases. These lesions, visualized on susceptibility-weighted imaging sequences, reflect blood-brain barrier disruption with erythrocyte extravasation into the cerebral parenchyma.

In our patient, microbleeds were diffuse and involved white matter, basal ganglia, and the corpus callosum. Corpus callosum involvement has been particularly reported in PRES associated with acute respiratory distress syndrome. This unusual radiological presentation highlights the severity of neurovascular injury in this setting.

3.4 Status Epilepticus and PRES

Status epilepticus is a common manifestation of PRES, reported in 60–75% of cases. In our observation, seizures were refractory to initial treatment and required dual anticonvulsant therapy with phenobarbital and carbamazepine. EEG findings of periodic right occipital spike-wave discharges were consistent with MRI lesion topography.

The refractory nature of seizures may be explained by the coexistence of vasogenic edema, cerebral microbleeds, and cerebral peduncular hematoma, creating multiple irritative foci.

3.5 Management and Prognosis

Management of PRES relies primarily on treatment of the triggering cause and correction of aggravating factors. In our patient, management included:

- Intensive treatment of severe acute asthma with protective mechanical ventilation, bronchodilators, and corticosteroids.
- Appropriate anticonvulsant therapy.
- Targeted antibiotic therapy adapted to microbiological results.
- Close neurological monitoring with clinical and neuroradiological follow-up.

The prognosis of PRES is generally favorable with resolution of MRI abnormalities within weeks after treatment. However, the presence of cerebral microbleeds may be associated with a higher risk of neurological sequelae, justifying prolonged neuroradiological follow-up. In our patient, evolution was favorable with successful extubation and progressive neurological stabilization.

4. Conclusion

We report an exceptional case of posterior reversible encephalopathy syndrome associated with multiple cerebral microbleeds and cerebral peduncular hematoma occurring in the context of severe acute asthma in a 14-year-old adolescent.

This case illustrates the necessity of maintaining a high level of vigilance regarding rare neurological complications during management of severe acute asthma in pediatric intensive care.

Multidisciplinary management integrating early brain MRI, neurological expertise, and targeted therapy allowed a favorable outcome in this complex case. Long-term neurological and neuroradiological follow-up remains essential to assess lesion reversibility and detect potential sequelae.

This observation contributes to the literature regarding atypical and severe pediatric forms of PRES and supports improved awareness of this clinical entity among pediatric intensive care teams.

References

1. Hinchey J, Chaves C, Appignani B, et al. A reversible posterior leukoencephalopathy syndrome. N Engl J Med. 1996;334(8):494-500.

2. Fugate JE, Rabinstein AA. Posterior reversible encephalopathy syndrome: clinical and radiological manifestations, pathophysiology, and outstanding questions. Lancet Neurol. 2015;14(9):914-925.

3. Bartynski WS. Posterior reversible encephalopathy syndrome, part 1: fundamental imaging and clinical features. AJNR Am J Neuroradiol. 2008;29(6):1036-1042.

4. Liman TG, Bohner G, Heuschmann PU, et al. The clinical and radiological spectrum of posterior reversible encephalopathy syndrome: the retrospective Berlin PRES study. J Neurol. 2012;259(1):155-164.

5. Tan MA, DeGuzman GE. Posterior reversible encephalopathy syndrome in children. Pediatr Neurol. 2010;42(4):289-293.

6. Roth C, Ferbert A. The posterior reversible encephalopathy syndrome: what's certain, what's new? Pract Neurol. 2011;11(3):136-144.

7. McKinney AM, Short J, Truwit CL, et al. Posterior reversible encephalopathy syndrome: incidence of atypical regions of involvement and imaging findings. AJR Am J Roentgenol. 2007;189(4):904-912.

8. Global Initiative for Asthma (GINA). Global Strategy for Asthma Management and Prevention, 2024.

9. Hefzy HM, Bartynski WS, Boardman JF, et al. Hemorrhage in posterior reversible encephalopathy syndrome: imaging and clinical factors. AJNR Am J Neuroradiol. 2009;30(7):1371-1379.

10. Covarrubias DJ, Luetmer PH, Campeau NG. Posterior reversible encephalopathy syndrome: prognostic utility of quantitative diffusion-weighted MR images. AJNR Am J Neuroradiol. 2002;23(6):1038-1048.