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Case Report

Posterior Reversible Encephalopathy Syndrome with Cerebellar Localization: A Case Report and Review of the Literature

Amadou HS[1*], Hassane ML[2], Bachar Loukoumi O[1], Alfred Dogbe YZ[1], Mahamane YS[1], Daddy H[3], Gagara M[3], Nanzir SM[1], Moussa DI[1], Chaibou MS[3]

[1]Hopital National de Niamey
[2]Université André Salifou de Zinder
[3]Université Abdou Moumouni de Niamey

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Abstract: Reversible posterior encephalopathy syndrome is a rare acute or subacute clinic-radiological syndrome whose topography most often affects the posterior territories, which are more vulnerable to variations in systemic pressure. Due to the rarity of this syndrome, we report the case of a 31-year-old patient with no known pathological history admitted to the emergency department of Maternité Issaka Gazobi (MIG) in Niamey, where the diagnosis of reversible posterior encephalopathy was made, and her evolution was favorable.

Keywords: Reversible posterior encephalopathy, cerebellar involvement, MIG, Niger.

INTRODUCTION

First described in 1996, posterior reversible encephalopathy syndrome (PRES) is a rare, little-known clinical and radiological entity characterized by reversible central nervous system involvement associated with typical brain imaging with a wide variability in clinical presentation and sometimes atypical imaging features [1-3].

The previously accepted pathophysiological mechanism is that of vasogenic edema linked to systemic hypertension and dysregulation of cerebral perfusion, predominantly in the posterior territories, which are more vulnerable to variations in systemic pressure. This syndrome has been described most often in contexts of arterial hypertension, particularly in eclampsia and renal failure, but also in situations of sepsis, autoimmune diseases, or neurosurgery (20 to 30% of cases occur outside of any context of arterial hypertension) [1-4].

The clinical picture is characterized by neurological manifestations such as headaches, confusion, dizziness, visual disturbances, seizures, and even coma.

Brain imaging (CT and MRI) reveals diffuse edema, more significant in the parietal and occipital lobes bilaterally, but typically reversible [5-7].

We report the case of a patient admitted to the ISSAKA GAZOBI maternity hospital in Niamey for altered state of consciousness in a postpartum context.

CASE REPORT

Ms. FZ, a 31-years-old woman with no known medical history, was a primigravida and delivered at 2 weeks of age to a male infant with an APGAR score of 10 and a weight of 3250g. She was admitted to the MIG emergency department on the 14th day postpartum for altered state of consciousness and agitation. On admission, the patient was agitated, with a blood pressure of 120/70 mmHg, a temperature of 38.5 degrees, a pulse oxygen saturation of 100%, and conjunctiva and mucous membranes discolored and anicteric, without edema or signs of dehydration. The patient presented with a tonic-clonic seizure episode without aura or tongue biting.

On clinical examination, the abdomen was supple with good uterine involution, the vulva was clean, and the vaginal examination was normal.

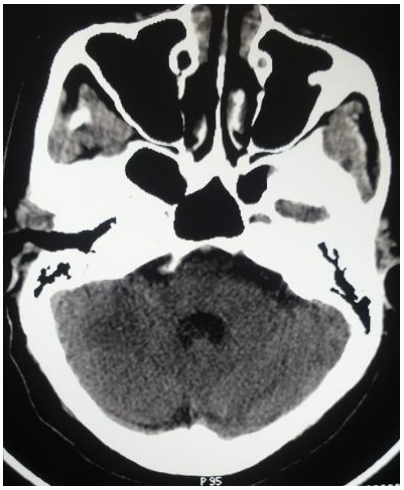
***Corresponding Author:** Amadou HS
Hopital National de Niamey

Cardiovascular and pleuropulmonary examinations were normal.

The assessment performed included a complete blood count (CBC) with a hemoglobin level of 13.3 g/dL, platelets at 271,000 elements/microliter, EG was positive at 80 p/ul, random blood glucose at 1.20 g/L, BUN at 0.16 mg/L, serum creatinine at 9.68 mg/L, AST at 45 IU/L, ALT at 29.4 IU/L, and proteinuria with two positive crosses.

Management consisted of diazepam administration and the initiation of the magnesium sulfate protocol, followed by transfer to the ICU. Upon admission to the ICU, the patient was given oxygen by a single face mask at 6L/min, and ECG, SPO2, and NIBP monitoring were performed. Blood pressure was 140/90mmHg, HR 105bpm, and SPO2 98% by a single face mask at 6L/min. The Glasgow Coma Scale score was 11/15 (E2 V4 M5), pupils were isochoric and reactive, and the ROT was normal, Babinski's sign was negative with no signs of focusing, and the urine output was 1L over 4 hours.

Brain CT was performed on day 2 and revealed spontaneous cerebellar hypodensity of 20 mm in diameter suggesting a right cerebellar ischemic stroke without excluding an ischemic focus of postpartum encephalopathy.

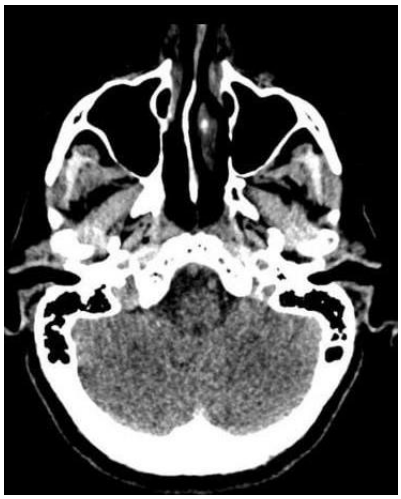


Right cerebellar spontaneous hypodensity of 20 mm in diameter

Figure 1: CT image on day 2

Treatment with enoxaparin 40 mg/12 hours and nicardipine tablets 20 mg twice a day.

The progression was marked by an improvement in neurological status on day 3, from 11/15 (E2 V4 M5) to 15/15. However, there were headaches and rotary vertigo, which eventually improved on day 7. The follow-up brain CT scan performed on day 12 returned normal. Based on these clinical and CT findings, we made the diagnosis of posterior reversible postpartum encephalopathy.



Disappearance of cerebellar hypodensity

Figure 2: Follow-up CT image (day 12)

DISCUSSION

Posterior reversible encephalopathy is an unusual postpartum complication in which vasogenic edema due to disruption of the blood-brain barrier appears to be the main factor [4].

The term PRES was used based on the similarity in imaging appearance, the common location of the parieto-occipital lobe, or the "posterior" location of the lesions. The first clinical description made by Hinchey et al in 1996 was in terms of posterior reversible leukoencephalopathy, and was taken up by Casey, who proposed the name "Posterior Reversible Encephalopathy Syndrome" (PRES) due to the involvement found in the gray matter (4). This is a rare, little-known, and probably underdiagnosed pathology with an unknown worldwide incidence [2,4,8-10].

It is characterized by prolonged but spontaneously reversible vasoconstriction of the cerebral arteries, which occurs either spontaneously or in specific circumstances. On the pathophysiological level, two hypotheses have been put forward [2,4,11,12]. The first hypothesis is based on the sequence hypertension-exceeding cerebral autoregulation-hypoperfusion-vasogenic cerebral edema due to extracapillary fluid leakage. The perivascular sympathetic innervation exerting a protective effect is richer in the anterior circulation and the carotid system, hence the posterior predominance of the lesions.

The second hypothesis supports cerebral vasoconstriction secondary to hypertension or a systemic process. This autoregulatory phenomenon would lead to a decrease in cerebral perfusion and therefore cytotoxic edema. This theory is supported by the absence of blood pressure elevation (or minimal) in some cases. It argues more in favor of a systemic process (infection, preeclampsia, transplantation, cancer chemotherapy) responsible for activation of the immune system and endothelial cells followed by endothelial damage with secondary hypoperfusion (systemic or regional).

Clinically, it manifests as headaches, impaired alertness and behavior, seizures, mental confusion, visual disturbances, and mutism. Convulsions were however the most frequently found sign followed by headaches in 92 and 26 to 53% respectively with a preferential topography located at the level of the posterior white matter of the cerebral hemispheres in 98% of cases. The lesions can also have a non-posterior distribution notably the frontal lobes (68%), the temporal lobes (40%) and the cerebellar hemispheres (30%) [13-15].

The diagnosis is confirmed by imaging (CT, MRI). CT reveals diffuse posterior hypodensities that do not enhance contrast but can be normal in 40% of cases [16]. MRI is the examination of choice for the diagnosis of PRES. They visualize areas of cortico-subcortical hyperintensity.

Signal abnormalities mainly involve the white matter but can also extend to the gray matter. The lesions are often bilateral and located in the occipital and/or frontoparietal regions. MRI may have diagnostic and prognostic value. It can distinguish between the reversible vasogenic edema of PRES and cytotoxic edema with a high risk of progression to irreversible ischemia. Brainstem and cerebellar involvement is common, while frontal lobe involvement is rare and often associated with a poor prognosis. The typical appearance shows diffuse subcortical and deep lesions [2,17].

Numerous treatments have been described for PRES, however, there is no specific treatment. Treatment of hypertension remains the main focus of PRES management, with the use of conventional antihypertensives (beta-blockers, calcium channel blockers, and diuretics), but their use must be cautious to avoid inducing hypotension when cerebral vasospasm already exists, reducing cerebral flow. Corticosteroids are widely used to combat vasospasm and headaches [2,16].

Prompt and appropriate diagnostic and therapeutic management most often prevents the occurrence of irreversible neurological lesions (epilepsy or neurological deficit) and the resolution of clinical and radiological abnormalities (7 to 15 days) [2,16].

CONCLUSION

A rare but reversible pathology, PRES is an unusual neurological condition that appears without any pre-existing pregnancy-related pathology. The syndrome is still poorly understood by clinicians, and the clinical and radiological manifestations are generally reversible. Diagnosis and appropriate and prompt treatment of posterior reversible encephalopathy can prevent the development of irreversible neurological damage and permanent sequelae.

Conflict of Interest: No

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