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Case Report

Posterior reversible encephalopathy syndrome: A case report ☆

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ABSTRACT

Posterior Reversible Encephalopathy Syndrome (PRES) is a radio-clinical entity associating reversible damage of the central nervous system and typical brain imaging. The clinical context is often suggestive with, in half of cases, the use of vasoactive substances (cannabis, antidepressants, nasal decongestants) and/or postpartum. The etiologies are dominated by hypertensive encephalopathy, preeclampsia, eclampsia, immunosuppressive therapies, and systemic diseases. We report a case of posterior encephalopathy syndrome occurring in a young female without hypertension. It was about a 40-year-old female without hypertension underlying condition, received at the emergency department for headaches and generalized tonic-clonic seizures. The physical examination was unremarkable, and her blood pressure was 130/70 mm Hg. CT scan revealed bilateral white matter hypodensity in the posterior occipital regions and a right frontal subarachnoid hemorrhage. There was no aneurysmal malformation of the polygon of Willis and no cerebral thrombophlebitis. Brain MRI showed T2 and FLAIR hypersignal areas in the occipital and frontal cortico-subcortical regions, with no diffusion signal abnormalities or contrast enhancement, and a right frontal subarachnoid hemorrhagic lesion with no other impairment. The diagnosis of reversible posterior encephalopathy syndrome was made up, and the outcome was favorable under treatment. Posterior reversible encephalopathy syndrome is an uncommon but probably underdiagnosed condition. Hypertensive encephalopathy is the most common etiology. However, there would be cases of PRES without hypertension as shown in this observation.

☆ Competing Interests: The authors declare that they have no conflict of interest.

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Introduction

Posterior reversible encephalopathy syndrome (PRES) is a clinico-radiological entity that was first described in 1996 in a series of 15 patients presenting with neurological symptoms including headache, seizures, visual disturbances, and other focal neurological deficits [1]. This is an uncommon pathology whose epidemiological data must be interpreted cautiously, as it is under-diagnosed and sometimes difficult to confirm [2]. Etiologies are dominated by hypertensive encephalopathy, pre-eclampsia, eclampsia, immunosuppressive therapy, and systemic diseases [3]. Imaging, in particular cerebral MRI, allows to diagnose the posterior reversible encephalopathy syndrome. Although CT scans can still rule out a hemorrhagic accident, they can evoke this syndrome in 50% of cases [4]. We report a case of posterior encephalopathy syndrome in a young, non-hypertensive patient.

Observation

This was a 40-year-old female patient with no history of hypertension and a history of papillary thyroid carcinoma operated on in 2016 and adjuvant chemotherapy. She was admitted as an emergency patient with headache and tonic-clonic generalized seizures.

Clinical examination was unremarkable, with BP at 130/70 mm Hg.

A non-contrast CT scan revealed a right frontal subarachnoid hemorrhage, and bilateral white matter hypodensity in the posterior occipital regions (Fig. 1). There was no aneurysmal malformation of the polygon of Willis or cerebral thrombophlebitis.

Cerebral MRI showed T2 and FLAIR hypersignal areas in the occipital and frontal cortico-subcortical regions, with no diffusion signal abnormalities or contrast enhancement, and a right frontal subarachnoid hemorrhagic lesion with no other abnormalities (Fig. 2).

This led to the diagnosis of reversible posterior encephalopathy syndrome.

The patient was admitted to the intensive care unit and placed on magnesium sulfate, clonazepam, and dexamethasone.

The outcome was favorable under treatment, with the disappearance of the abnormalities on the follow-up MRI at 3 weeks (Fig. 3).

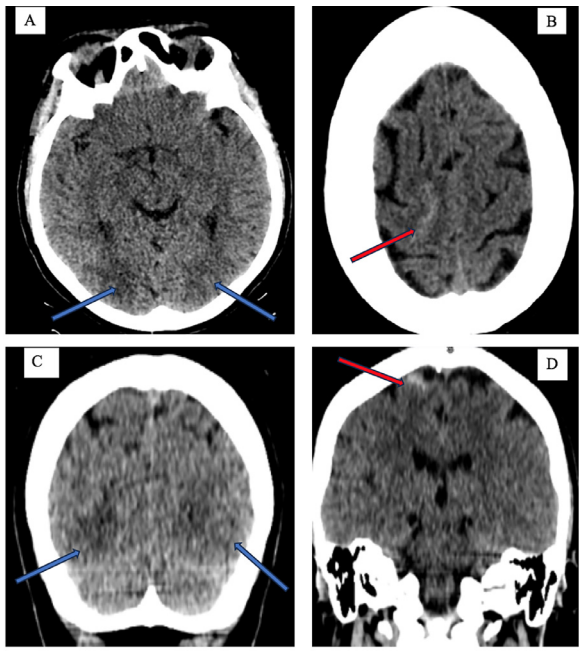

Fig. 1 – Cerebral CT scan without injection in axial section (A, B) and coronal reconstruction (C, D) showing hypodensity of the white matter of the posterior occipital regions (blue arrows) and a right frontal subarachnoid hemorrhage (red arrows).

Discussion

Posterior reversible encephalopathy syndrome (PRES) is a radio-clinical entity characterized by a series of neurological symptoms and neuroradiological signs reflecting vasogenic and cytotoxic edema [2]. The pathophysiology of this edema is based on two theories. The first theory is that of cerebral hyperperfusion: arterial hypertension exceeds the brain's capacity for self-regulation, leading to vascular alteration and arteriolar vasodilatation. The subsequent rupture of the blood-brain barrier causes fluid leakage from the vessels into the brain parenchyma, resulting in reversible vasogenic edema [5]. The second theory is that of cerebral hypoperfusion secondary to arterial hypertension or a systemic process. It can be observed in pre-eclampsia, infections, and chemotherapy. Indeed, when the immune system is activated, endothelial cells are subsequently damaged, resulting in cytotoxic edema [6].

The second theory appears to be applicable in our patient since she underwent chemotherapy.

The most frequent neurological symptoms are headaches. These are generally diffuse and progressive in onset. If left untreated, symptoms progressively worsen over several days to weeks. They can progress to encephalopathy, confusion, convulsions, and even coma [7].

Diagnosis is made on the basis of imaging, with MRI being the gold standard, although CT scans can also be used for orientation.

This pathology appears on CT as hypodensity in the posterior fossa and occipital lobes. MRI shows T2 and Flair hyperdensity and T1 hyposignal. Sometimes the T2 sequence cannot distinguish between vasogenic edema, which is reversible, and cytotoxic edema, which is irreversible, hence the importance of diffusion to distinguish between the 2 [4,8]. The lesions of PRES syndrome appear as hyperintense on diffusion-weighted imaging without restriction of the ADC coefficient, unlike cytotoxic edema.

These lesions may be associated with intracerebral hemorrhage [9] as was the case in our patient.

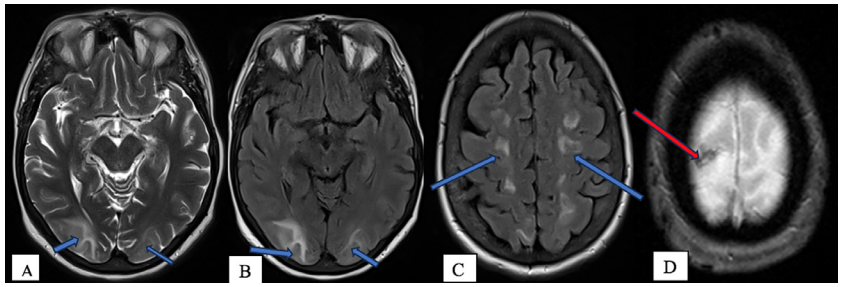

Fig. 2 – Cerebral MRI showing areas of T2 hypersignal (A) and T2 FLAIR (B, C) in the occipital and frontal regions, cortico-subcortical, with no abnormal diffusion signal or contrast, and a subarachnoid hemorrhagic lesion on the right frontal T2* (D).

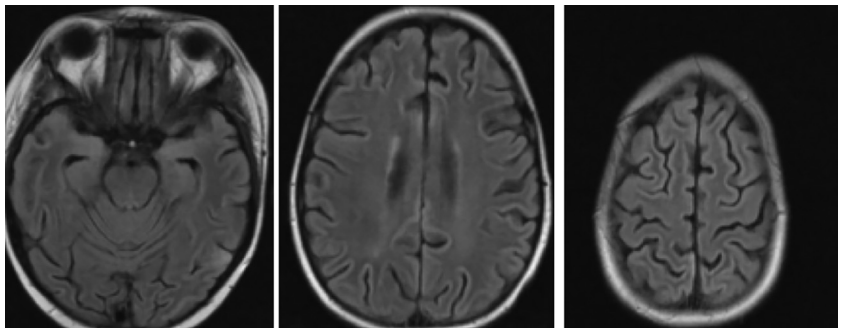

Fig. 3 – Brain MRI at D22 showing almost complete disappearance of abnormalities.

The outcome under treatment is favorable within a week, with regression of clinical signs, but can be unfavorable, with persistent neurological sequelae or death [2,10].

Conclusion

Reversible posterior encephalopathy syndrome is considered uncommon but is probably underdiagnosed. Hypertensive encephalopathy is the most common etiology. However, there are cases of PRES without elevated blood pressure, as shown in this observation.

Patient consent

Written informed consent for the publication of this case report was obtained from the patient.

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