CASE REPORT

Reversible bilateral pyramidal tract lesions after hypertensive crisis and cerebral seizures

Lésions réversibles des faisceaux pyramidaux après crise hypertensive et épileptique

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**KEYWORDS**

Reversible posterior leukoencephalopathy syndrome; Pyramidal tracts; Hypertensive crisis; Seizures

**Abstract** This is a rare case of reversible high signal-intensity changes along the pyramidal tracts in a patient with reversible posterior leukoencephalopathy syndrome (RPLS). A 38-year-old man was admitted to hospital for loss of consciousness and generalized seizures. His systolic blood pressure was 220 mmHg. Neurological examination revealed bilateral pyramidal-tract signs, and paresis of the right arm. Initial MRI showed increased signal intensities on T2-weighted, FLAIR and diffusion-weighted imaging in the following regions: bilateral temporo-occipital white matter and cortex, dorsal parts of the lentiform nuclei, bilateral caudate nuclei and external capsule. High signal intensities were observed in the pyramidal tracts as well. On patient follow-up, MRI signal abnormalities and clinical symptoms were completely resolved after antihypertensive treatment.

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**MOTS CLÉS**

Leucoencéphalopathie postérieure réversible; Faisceaux pyramidaux; Crise hypertensive; Convulsion


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Introduction

Reversible posterior leukoencephalopathy syndrome (RPLS) was first described in 1996 with reference to the reversible signal alterations on MRI [1]. Hypertensive crisis [1], preeclampsia and eclampsia [1,2], and cytotoxic drugs such as gemcitabine [3] are among the known causes of RPLS. Immunosuppressant drugs such as cyclosporin or tacrolimus also are known to trigger RPLS [4,5,6]. Rarely, hypertalencea [7], porphyria [8] and interferon therapy [1] may result in RPLS. The clinical symptoms associated with RPLS include headache, nausea, vomiting, loss of consciousness, seizures and progressive loss of sight leading to associated cortical blindness [1].

We present the clinical and MRI findings with follow-up of a patient with hypertensive crisis and seizures who demonstrated signal abnormalities in the corticospinal tracts in addition to the typical MRI findings seen in patients with RPLS.

Case report

A 38-year-old man was found in a coma. He had experienced gradually increasing severe holocranial headache for 2 days prior to admission. On the way to hospital, he also suffered four episodes of general tonic-clonic seizures. He was intubated upon arrival. His neurological examination showed bilateral pyramidal signs. Brain MRI was obtained at the time of admission; follow-up MRI was done on day 5 after admission and 4 months after the onset of symptoms. Images were obtained with a 1.5-T scanner; all MRI studies included axial T2-TSE (turbo spin echo), FLAIR (fluid-attenuated inversion recovery), T1-SE (spin echo), diffusion-weighted imaging (DWI), postcontrast SE T1-weighted sequences and time-of-flight (TOF) MR angiography (MRA). T2-weighted imaging, FLAIR and DWI of the initial MRI study showed increased signal intensities in the bilateral temporo-occipital white matter and cortex, dorsal parts of the lentiform nuclei (Fig. 1a and c), bilateral caudate nuclei and external capsules. The elevated apparent diffusion coefficient (ADC) value in these anatomical areas was in the range of 0.96–1.26 × 10^{-3} mm²/s. Increased signal abnormality was also noted in the pyramidal tracts, more on the left than on the right (Fig. 1b and d). The increased ADC value was calculated at 1.0 × 10^{-3} mm²/s and 1.1 × 10^{-3} mm²/s in the right and left pyramidal tracts, respectively, in contrast to a value of 0.68 × 10^{-3} mm²/s in normal white matter (Fig. 1e). TOF MRA revealed normal flow related signal in the cerebral arteries.

Continuous blood pressure monitoring showed hyptonensive measurements, reaching systolic pressures of 220 mmHg. There was no evidence of renal artery stenosis or adrenal tumor. Blood pressure was controlled with medication for a period of 6 months, and the patient was also given anticonvulsive therapy.

The initial cerebrospinal fluid (CSF) analysis revealed an elevated protein content of 1050 mg/L with a normal cell count. A lumbar puncture 4 days later ruled out a viral or bacterial infection. Transcranial duplex sonography showed elevated blood flow in the right proximal M1 and left M2 segments, with flow velocities measuring 210 cm/s, which were normal at follow-up.

After being extubated on the second day, the patient was observed to have paresis of the right arm. Then, on day 3, he was reported to be neurologically and psychologically free of any pathological findings. Follow-up MRI performed on day 5 after admission revealed an almost complete resolution of the previously seen signal abnormalities. Electroencephalography (EEG) performed on the same day showed a clear general domination of delta waves without a focus, but this finding also regressed on subsequent EEGs. MRI obtained 4 months later revealed no pathological findings (Fig. 2). The patient was asymptomatic, and his blood pressure stayed within the normal range with medication.

Discussion

In addition to mostly completely reversible white-matter lesions, cortical abnormalities are also noted in over 90% of cases of RPLS (also known as ‘posterior reversible encephalopathy syndrome’) (PRES), ‘reversible leukoencephalopathy syndrome’ (RLS) and ‘posterior encephalopathy’ (PE) [9]. Pathological MRI signal changes in patients with RPLS have been previously described in the parieto-occipital, temporal and fronto-dorsal regions, and basal ganglia, as well as in the pons and cerebellum [9]. There are three pathophysiological aspects. First is cerebral hyperperfusion, caused by systemic hypertension and which leads to inadequate brain autoregulation, especially in the posterior circulation, and secondary dysfunction of the blood–brain barrier (BBB), which results in vasogenic edema [9]. The predisposition of the parieto-occipital area to this condition could be explained by the presence of fewer adrenoreceptors in the posterior vessels than in the anterior vessels, leading to a reduced sympathetic response to an acute onset of systemic hypertension. Another feature of RPLS is vasospasm, thought to be the cause of, in particular, terminal perfusion deficits that subsequently lead to cytotoxic and/or vasogenic changes [10]. RPLS associated with vasospasm has been described in patients with preeclampsia or eclampsia [1,2] and in cases of digoxin intoxication [10]. Finally, in cases of RPLS, cyclosporin A appears to lead to direct damage of the endothelium that, in turn, results in the release of vasoactive substances, such as endothelin-1, with subsequent vasospasm [4]. The lesions caused by cyclosporin A are usually reversible, indicating vasogenic—not cytotoxic—edema.

The completely reversible lesions could be manifestations of RPLS or caused by the seizures that occur later. The locations of the lesions reported in RPLS do not encompass the pyramidal tracts, and signal changes after seizures have been previously reported in the motor cortex and associated underlying white matter [11]. However, MRI findings in our patient failed to demonstrate signal abnormalities in those areas, but instead showed partial involvement along the distal parts of the pyramidal tracts. It may be that these MRI findings are of different origin. The acute hypertensive crisis in our patient may have led to subsequent damage of the BBB, as suggested by lesions in the occipital and temporal white matter and cortex, and in the basal ganglia. The elevated blood flow observed with
Figure 1  (A) Initial fluid-attenuated inversion recovery (FLAIR) imaging reveals high signal intensities in the dorsal basal ganglia, temporo-occipital regions and (B) deep white matter. (C) On diffusion-weighted imaging (DWI), a high signal intensity can be seen in the posterior basal ganglia and (D) along the pyramidal tracts (arrows), with accentuation on the left side. (E) ADC mapping shows elevated values in the pyramidal tracts and caudate nuclei.
transcranial duplex sonography may be related to vasospasm, but was not observed on MRA and, because of the rapid clinical recovery of the patient, digital subtraction angiography was not performed.

Our patient presented with secondary seizures, as previously reported by Hinchey et al. [1], that resulted in signal changes along the pyramidal tracts. These initial MRI signal changes along the pyramidal tracts were well correlated with the neurological findings of a bilateral Babinski sign and paresis of the right arm. ADC mapping showed elevated bilateral ADC values in the temporo-occipital white matter and cortex, dorsal parts of the lentiform nuclei, bilateral caudate nuclei, external capsules and pyramidal tracts. These findings were consistent with vasogenic edema.

In summary, the reversible cerebral signal abnormalities in cases of RPLS, as noted on T2- and diffusion-weighted imaging, can be caused by damage of the BBB by hyperperfusion, vasospasm or direct damage to the endothelium by toxins. The characteristic locations of lesions in RPLS include the occipital, parietal, temporal and frontal lobes, basal ganglia, cerebellum and brainstem. In addition, reversible signal changes can occur along the pyramidal tracts that are probably related to the seizures rather than the initial hypertensive crisis.

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References