CASE REPORT
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ATYPICAL RADIOLOGICAL PRESENTATION OF HYPERTENSIVE ENCEPHALOPATHY

Neurology

Dr. Jyothula Hari Priya Post Graduate Student, Department of Neurology, Dr. Pinnamaneni Siddhartha Institute of Medical Sciences & Research Foundation, Chinnavutapalli.

Dr. Velagapudi Keerthi Bala Intern, Dr. Pinnamaneni Siddhartha Institute of Medical Sciences & Research Foundation, Chinnavutapalli.

Dr. Kotagiri Vamsi Krishna* Associate Professor, Department of Neurology, Dr. Pinnamaneni Siddhartha Institute of Medical Sciences & Research Foundation, Chinnavutapalli. *Corresponding Author

Dr. Gogineni Sujana Associate Professor, Department of Neurology, Dr. Pinnamaneni Siddhartha Institute of Medical Sciences & Research Foundation, Chinnavutapalli.

Dr. Chandra Sekhara Rao Kondragunta Professor, Department of Radiology, Dr. Pinnamaneni Siddhartha Institute of Medical Sciences & Research Foundation, Chinnavutapalli.

ABSTRACT

Hypertensive encephalopathy (HE) is a clinical emergency caused by acute elevations in blood pressure, often presenting with headache, visual disturbances, altered mental status, and seizures. It is classically associated with reversible posterior leukoencephalopathy syndrome (PRES), involving the parieto-occipital white matter. However, atypical variants exist, including Hypertensive Brainstem Encephalopathy (HBE) and rarely, involvement of the optic nerve and chiasm. We report a rare case of HBE with concurrent optic pathway involvement in a middle-aged woman with recent onset hypertension and renal dysfunction. MRI showed hyperintensities in the brainstem and optic nerve sheath extending to the optic chiasm, with no focal neurological deficits.

KEYWORDS

Hypertensive encephalopathy, PRES, Papilledema

INTRODUCTION

Hypertensive encephalopathy (HE) is a life-threatening neurological condition caused by a sudden, severe elevation in blood pressure, resulting in disruption of cerebral autoregulation and subsequent vasogenic edema. It is considered part of the broader spectrum of posterior reversible encephalopathy syndrome (PRES), typically characterized by headache, altered sensorium, seizures, and visual disturbances. Radiologically, HE and PRES are most commonly associated with bilateral symmetric hyperintensities in the parieto-occipital white matter on T2-weighted and FLAIR MRI sequences [1–3].However, in some cases, the distribution of vasogenic edema extends beyond the classical regions. Hypertensive brainstem encephalopathy (HBE) represents an uncommon but recognized variant, where predominant or isolated involvement of the brainstem occurs without the classical posterior cortical changes. Despite dramatic imaging findings, patients with HBE often demonstrate minimal clinical deficits-a phenomenon termed clinical–radiological dissociation[4]. Most patients, like ours, are younger individuals with secondary hypertension, often linked to renal dysfunction [5]. Even more rarely, PRES and HE may involve structures of the visual pathway beyond the occipital lobes. Involvement of the optic nerve sheath, optic chiasm, and optic tracts has been reported in only a handful of cases in the literature[6]. This atypical distribution can complicate diagnosis, particularly in patients presenting with visual symptoms or without overt cortical features of PRES. In this report, we present a unique case of a middle-aged woman with newly diagnosed hypertension and renal dysfunction who developed brainstem-predominant hypertensive encephalopathy with simultaneous optic nerve sheath and chiasmal involvement. The case highlights a rare radiological variant of HE and underscores the importance of considering such atypical presentations in the appropriate clinical context.

CASE PRESENTATION

A 40-year-old female presented to the Neurology Outpatient Department with headache and blurring of vision in the right eye over the past 4 months, associated with shortness of breath on exertion, vomiting, intermittent giddiness, and excessive sleepiness for the past week. Orthopnea was also reported. She had been diagnosed with de novo hypertension (BP 200/100 mmHg) and had started antihypertensive therapy. She had no prior comorbidities.

There was pallor on examination, and neurological examination revealed no focal deficits. Fundus examination showed Grade IV papilledema in both eyes. Investigations revealed microcytic hypochromic anemia with neutrophilic leukocytosis, deranged urea and creatinine levels, and hypokalemia. Ultrasound of the abdomen showed Grade 1 renal parenchymal disease.

Neuroimaging

MRI Brain revealed:

- Diffuse T2 and FLAIR hyperintensities in the brainstem (pons and medulla), with no supratentorial or parieto-occipital involvement.
- Notably, hyperintense signals were seen along the optic nerve sheath bilaterally, extending into the optic chiasm on axial FLAIR images.
- ADC maps showed elevated values without diffusion restriction, consistent with vasogenic edema.

These findings strongly suggested hypertensive brainstem encephalopathy with optic pathway involvement.

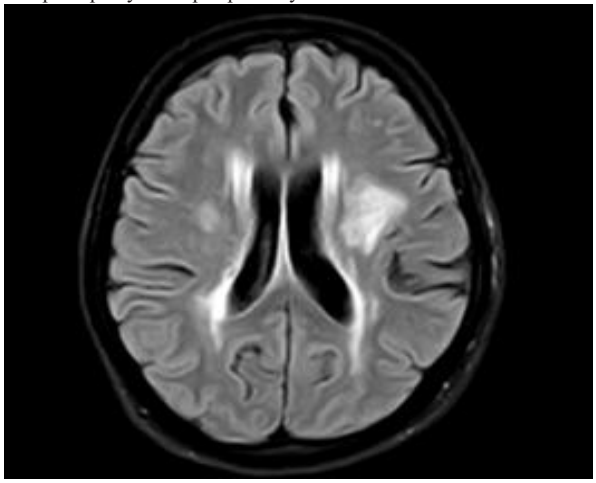

Fig. 1 MRI showing, Axial FLAIR - confluent white matter hyperintensity in left corona radiate

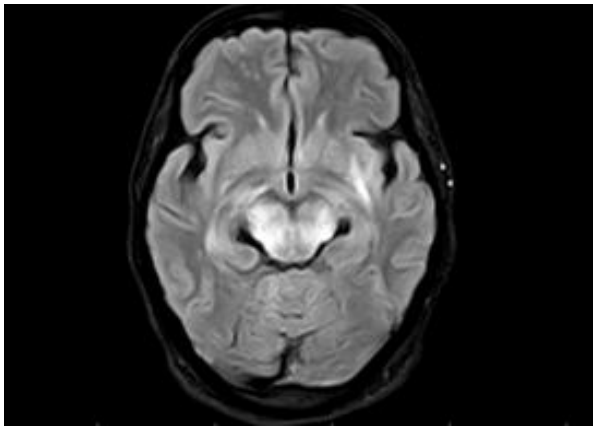

Fig. 2 MRI showing, Axial FLAIR - Diffuse hyperintensity in Mid brain

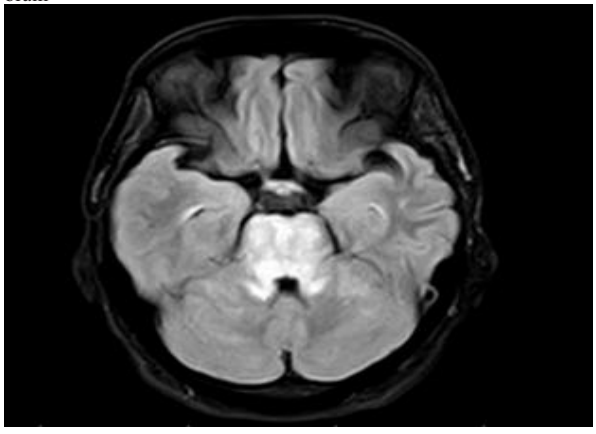

Fig. 3 MRI showing, Axial FLAIR - Diffuse hyperintensity in Pons extending into middle cerebellar peduncle

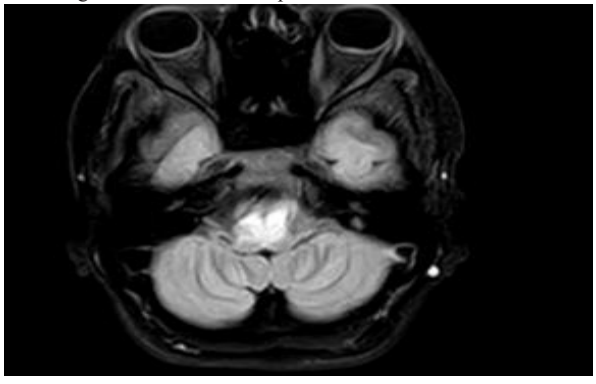

Fig. 4 MRI showing, Axial FLAIR - Diffuse hyperintensity in medulla

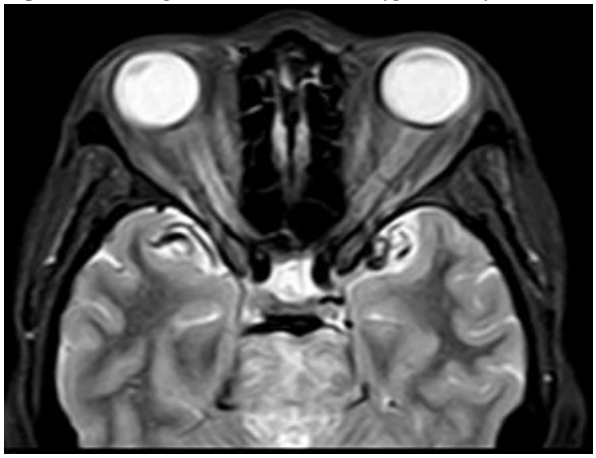

Fig. 5 MRI showing, Axial FLAIR – Hyperintensity of bilateral optic nerve sheath

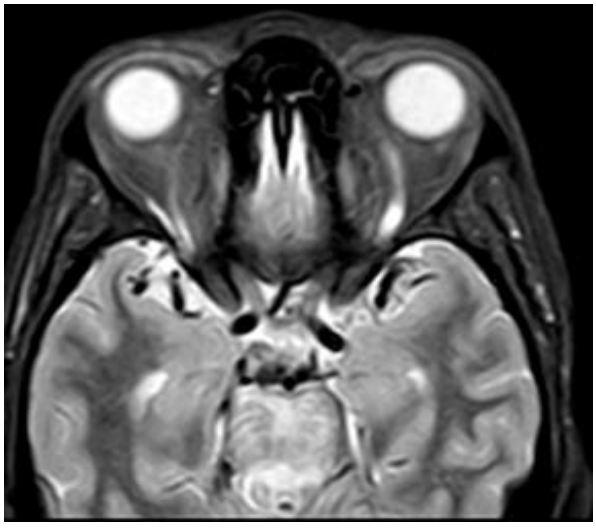

Fig. 6 MRI showing, Axial FLAIR – Hyperintensity of Optic chiasma

DISCUSSION

This case represents a rare and atypical presentation of hypertensive encephalopathy, featuring predominant brainstem involvement along with optic nerve sheath and Optic chiasm. The absence of classical parieto-occipital lesions and the reversibility of findings following antihypertensive therapy point toward hypertensive brainstem encephalopathy (HBE).HBE is an uncommon subtype of hypertensive encephalopathy. Unlike classical PRES, which involves the parieto-occipital regions due to relatively lower sympathetic innervation, HBE primarily affects the brainstem-an atypical site-yet remains clinically milder despite extensive radiological changes, a phenomenon known as clinical–radiologic dissociation [4]. Most patients, like ours, are younger individuals with secondary hypertension, often linked to renal dysfunction [5].What sets this case apart is the rare involvement of the optic nerve sheath and optic chiasm, evident on MRI. While PRES and HBE may involve the brainstem, cerebellum, or basal ganglia, the optic pathway is almost never affected.To our knowledge, literature review reveals only two reported cases of PRES/HE involving the optic chiasm or optic tracts [6]. These cases presented with visual disturbances and showed reversible T2/FLAIR hyperintensities in the optic chiasm and tracts, mirroring our findings. The pathophysiology is presumed to be the same as in PRES-vasogenic edema due to breakdown of the blood–brain barrier, triggered by abrupt elevation in blood pressure. The optic pathway, like posterior brain structures, may be vulnerable due to its vascular architecture. Importantly, reversibility with blood pressure control has been documented. Differential diagnoses for optic pathway lesions include optic neuritis, compressive lesions, ischemic optic neuropathy, demyelination, and central nervous system infections.

CONCLUSION

This case expands the clinical and radiological spectrum of hypertensive encephalopathy. We highlight several important lessons:

1. Unusual MRI findings in hypertensive patients with visual complaints should raise suspicion for optic pathway involvement, especially if paired with brainstem or PRES-like features.
2. HBE can present without any supratentorial or occipital lesions, yet still pose a significant diagnostic challenge.
3. Reversibility of both clinical symptoms and imaging findings reinforces the critical importance of early and aggressive blood pressure control.
4. Clinical–radiologic dissociation remains a hallmark—extensive brainstem and optic abnormalities may exist despite minimal neurological deficits.
5. Our patient's course illustrates that optic pathway involvement is possible in HE and should be considered when visual symptoms are prominent.

In conclusion, this case underscores the need for clinicians and radiologists to be vigilant for atypical PRES/HBE variants, including rare involvement of the optic nerve and chiasm. Early recognition can lead to complete recovery and prevent irreversible damage.

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