Cureus
Part of SPRINGER NATURE

Open Access Case Report

Posterior Reversible Encephalopathy Syndrome Associated With Severe Autoimmune Hyperthyroidism: A Case of Cortical Visual Loss Mimicking Posterior Cerebral Artery Infarction

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Review began 10/06/2026
Review ended 10/20/2026
Published 10/25/2026

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DOI: 10.7759/cureus.118573

Abstract

Posterior reversible encephalopathy syndrome (PRES) is a clinico-radiological syndrome characterised by acute encephalopathy, seizures, headache and visual disturbance, with neuroimaging typically demonstrating predominantly posterior vasogenic oedema. Although severe hypertension, renal dysfunction, sepsis and exposure to cytotoxic or immunosuppressive therapies are well-recognised precipitants, PRES associated with autoimmune thyroid disease is uncommon.

We report the case of a 55-year-old woman with no previous history of thyroid disease who presented with acute encephalopathy, fever and hypoxia and was subsequently found to have severe autoimmune hyperthyroidism consistent with Graves' disease. Recurrent focal and generalised seizures and profound bilateral visual loss complicated her clinical course. Initial neuroimaging demonstrated only chronic infarcts. Subsequent MRI revealed right occipital cortical and subcortical abnormalities with diffusion restriction, which were initially interpreted as an acute posterior cerebral artery territory infarction. However, the combination of encephalopathy, seizures, visual loss, and interval radiological improvement prompted tertiary neuroradiology review, and the imaging abnormalities were subsequently considered consistent with PRES. Cerebrospinal fluid analysis and extensive infectious, neuronal and paraneoplastic antibody testing did not identify an alternative inflammatory or infectious cause.

Management included treatment of severe thyrotoxicosis with carbimazole, antiepileptic therapy and treatment of concurrent systemic infection and acute kidney injury. Visual function gradually improved from perception of light only to recognition of colours and objects, although significant visual and cognitive impairment persisted at follow-up.

This case highlights severe autoimmune hyperthyroidism as a potential contributor to PRES in the presence of additional systemic stressors. It demonstrates the diagnostic challenge posed by occipital PRES with diffusion restriction, which can mimic posterior circulation infarction. Recognition of the characteristic clinical-radiological evolution and multidisciplinary neuroradiological review are important to avoid misdiagnosis and guide appropriate management.

Categories: Neurology, Internal Medicine, Endocrinology/Diabetes/Metabolism
Keywords: autoimmune thyroid disease, cortical blindness, graves' disease, hyperthyroidism, posterior reversible encephalopathy syndrome (pres), seizures

Introduction

Posterior reversible encephalopathy syndrome (PRES) is a clinico-radiological syndrome characterised by acute neurological symptoms, most commonly encephalopathy, seizures, headache, and visual disturbance, together with predominantly posterior cerebral vasogenic oedema on MRI [1-3]. Vasogenic oedema refers to the accumulation of fluid within brain tissue due to increased permeability of the blood-brain barrier. Posterior reversible encephalopathy syndrome is thought to result from disruption of cerebral autoregulation, the mechanism by which the brain maintains relatively stable blood flow despite changes in systemic blood pressure, together with endothelial dysfunction and breakdown of the blood-brain barrier [1,3]. Severe or rapidly developing hypertension is a common association, although PRES can occur without marked hypertension, particularly in patients with renal dysfunction, sepsis, autoimmune disease or exposure to immunosuppressive and cytotoxic therapies [1-3].

The radiological appearance of PRES is variable. Although bilateral parieto-occipital vasogenic oedema is characteristic, unilateral involvement, cortical abnormalities, diffusion restriction and haemorrhage may also occur [1,3]. Diffusion restriction refers to reduced movement of water molecules within brain tissue on diffusion-weighted MRI sequences and may indicate cellular swelling or injury. This can create a diagnostic challenge because restricted diffusion is also a characteristic feature of acute cerebral infarction.

How to cite this article
Enver M, Dhamija K, Jabbal H (October 25, 2026) Posterior Reversible Encephalopathy Syndrome Associated With Severe Autoimmune Hyperthyroidism: A Case of Cortical Visual Loss Mimicking Posterior Cerebral Artery Infarction. Cureus 18(10): e118573. DOI 10.7759/cureus.118573

Recognition of the clinical context and demonstration of radiological improvement on follow-up imaging are therefore important when distinguishing PRES from ischaemic stroke [3].

Autoimmune thyroid disease has occasionally been associated with encephalopathy, seizures and reversible cerebral white matter abnormalities. This entity, historically termed Hashimoto encephalopathy or steroid-responsive encephalopathy associated with autoimmune thyroiditis, remains controversial because its clinical and radiological features are heterogeneous and there are no universally accepted diagnostic criteria [4,5]. Although most reported cases involve Hashimoto thyroiditis, neurological manifestations have also been described in patients with Graves' disease, including a reported case with MRI findings compatible with PRES [6]. Reversible posterior white matter abnormalities have also been described in patients with autoimmune thyroid disease and Hashimoto encephalopathy [7,8].

We report a case of severe autoimmune hyperthyroidism associated with PRES, complicated by recurrent seizures and profound cortical visual impairment. The initial occipital MRI abnormality was interpreted as a posterior cerebral artery territory infarction but subsequently demonstrated substantial interval improvement, supporting a diagnosis of PRES. This case illustrates the potential contribution of severe thyrotoxicosis and systemic metabolic stress to PRES and highlights the importance of recognising reversible posterior cerebral abnormalities that mimic acute ischaemic stroke.

Case Presentation

A 55-year-old Caucasian lady with a history of alcohol dependence, anxiety, depression, previous transient ischaemic attack, and a previous intentional drug overdose presented to the emergency department after being found collapsed on the floor of her bedroom. She lived independently with support from her mother and mobilised with a walking aid. She had no previous history of thyroid disease or visual impairment. Her regular medications included duloxetine, quetiapine, clopidogrel, rosuvastatin, lansoprazole, folic acid, thiamine, lactulose, fexofenadine, and nicotine replacement therapy. She had documented allergies to mirtazapine and ibuprofen.

She had experienced one day of progressive drowsiness and cough before admission. On arrival, she was confused but responsive to verbal stimuli, with a Glasgow Coma Scale (GCS) score of 15/15. Initial observations demonstrated sinus tachycardia (118 beats/min), oxygen saturation of 90% on room air, a respiratory rate of 18 breaths/min, and a blood pressure of 92/39 mmHg. She subsequently developed a fever, with a peak temperature of 38.7°C. Arterial blood gas analysis showed mixed respiratory acidosis with hypercapnia (pH 7.31, partial pressure of carbon dioxide (pCO_2) 7.16 kPa) and normal lactate (1.17 mmol/L).

Initial investigations demonstrated acute kidney injury, anaemia and mild biochemical evidence of inflammation. Thyroid function tests revealed severe thyrotoxicosis, with a suppressed thyroid-stimulating hormone (TSH), markedly elevated free thyroxine (FT4) and free triiodothyronine (FT3). Thyroid peroxidase and TSH receptor antibodies were strongly positive, confirming Graves' disease. The principal biochemical results are shown in Table *1*, and thyroid function trends are shown in Table *2*.

Investigation	Admission	Reference range
Haemoglobin (g/L)	93	115-160
White cell count (×10⁹/L)	8.0	4-11
Neutrophils (×10⁹/L)	5.90	2-7.5
Platelets (×10⁹/L)	300	150-400
C-reactive protein (mg/L)	14	<5
Procalcitonin (µg/L)	0.27	<0.5
Sodium (mmol/L)	138	135-145
Potassium (mmol/L)	4.4	3.5-5.0
Urea (mmol/L)	7.8	2.5-7.8
Creatinine (µmol/L)	168	45-90
Estimated glomerular filtration rate (mL/min/1.73 m²)	29	>90
Adjusted calcium (mmol/L)	2.81	2.20-2.60
Phosphate (mmol/L)	2.25	0.8-1.5
Parathyroid hormone (pmol/L)	1.40	1.6-6.9
Bilirubin (µmol/L)	6	<21
Alkaline phosphatase (IU/L)	120	30-130
Alanine aminotransferase (IU/L)	29	<35
Amylase (U/L)	21	25-125

TABLE 1: Blood test investigations on admission

Investigation	Admission	Day six	Day seven	Day 10	Day 20	Reference range
TSH (mIU/L)	<0.008	<0.008	<0.008	<0.008	<0.008	0.3-4.5
FT4 (pmol/L)	34.5	8.5	9.4	8.4	26.3	9-19
FT3 (pmol/L)	29.7	3.0	3.3	2.6	9.3	2.4-6.0
Thyroid peroxidase antibody (kIU/L)	>2000	-	-	-	-	<34

TABLE 2: Thyroid function trends during the inpatient stay

TSH: Thyroid-stimulating hormone, FT4: Free thyroxine, FT3: Free triiodothyronine

The combination of severe biochemical thyrotoxicosis, fever (38.7°C), altered mental status, and acute systemic illness raised concern for a possible thyrotoxic crisis. However, these findings were not specific to thyroid storm, particularly in the presence of suspected systemic infection and subsequent hypercapnic respiratory failure. Although thyroid storm was considered clinically, a definitive diagnosis was not established, and the patient was therefore described as having severe autoimmune thyrotoxicosis.

Given the initial presentation with altered consciousness, hypoxia, and fever, she was initially treated for presumed sepsis with intravenous fluids and broad-spectrum antibiotics. Chest radiography showed no focal consolidation. A CT of the head demonstrated established low attenuation in the right parietal lobe and right basal ganglia consistent with previous infarcts, without acute haemorrhage or mass effect.

Following identification of severe thyrotoxicosis, the working diagnosis was revised to thyrotoxic crisis.

Carbimazole and intravenous corticosteroids were commenced on day one of the admission. Alcohol withdrawal, toxic-metabolic encephalopathy, Wernicke's encephalopathy, postictal states and central nervous system infection remained important differential diagnoses because of her history and evolving neurological features.

Despite initial treatment, her neurological and respiratory status deteriorated. Her GCS fell to 12/15, with increasing oxygen requirements and an inability to maintain her airway independently. She developed severe hypercapnic type 2 respiratory failure, with mixed respiratory and metabolic acidosis. A CT pulmonary angiography excluded pulmonary embolism. She was transferred to the ICU, where non-invasive ventilation, bilevel positive airway pressure (BiPAP), was commenced. Her respiratory acidosis and hypercapnia subsequently improved without the need for vasopressor support.

Possible neck stiffness was noted during the early ICU admission, prompting empirical treatment with intravenous ceftriaxone and aciclovir for suspected meningoencephalitis. Lumbar puncture demonstrated normal CSF protein and glucose, with a negative encephalitis screen. Autoimmune investigations, including antinuclear antibodies, neuronal surface/synaptic antibodies (AMPA1, AMPA2, LGI1, NMDA receptor, GABA-B receptor and CASPR2) and a paraneoplastic antibody panel, were negative. These findings made infectious, autoimmune, and paraneoplastic encephalitis unlikely.

Early MRI of the brain with gadolinium demonstrated chronic parietal white matter changes and bilateral basal ganglia lacunar infarcts, which were unchanged from previous imaging four years ago. No acute infarction or abnormal enhancement was identified on the overall MRI study, and there were no radiological features suggestive of encephalitis. Representative T2-weighted and fluid-attenuated inversion recovery (FLAIR) images are shown in Figures *1*-*2*.

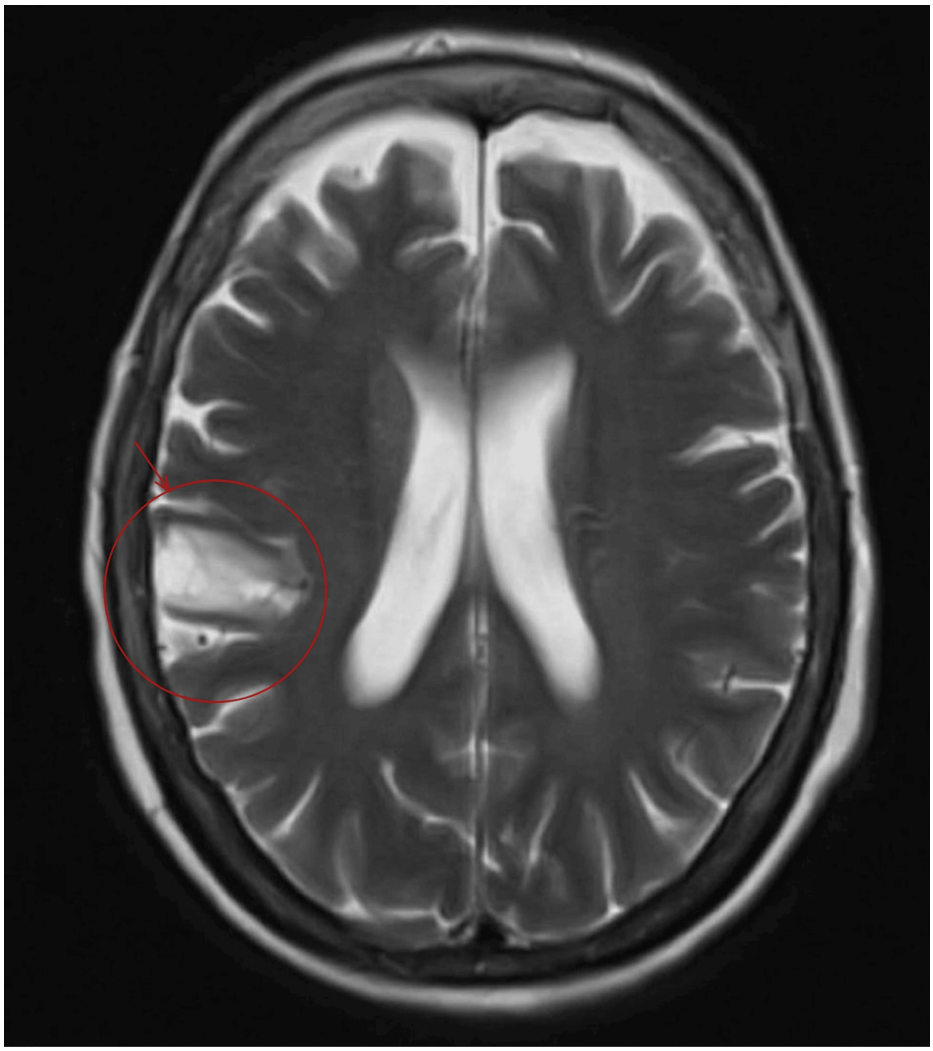


FIGURE 1: Initial MRI of the brain demonstrating chronic parietal infarct-related changes on T2-weighted imaging

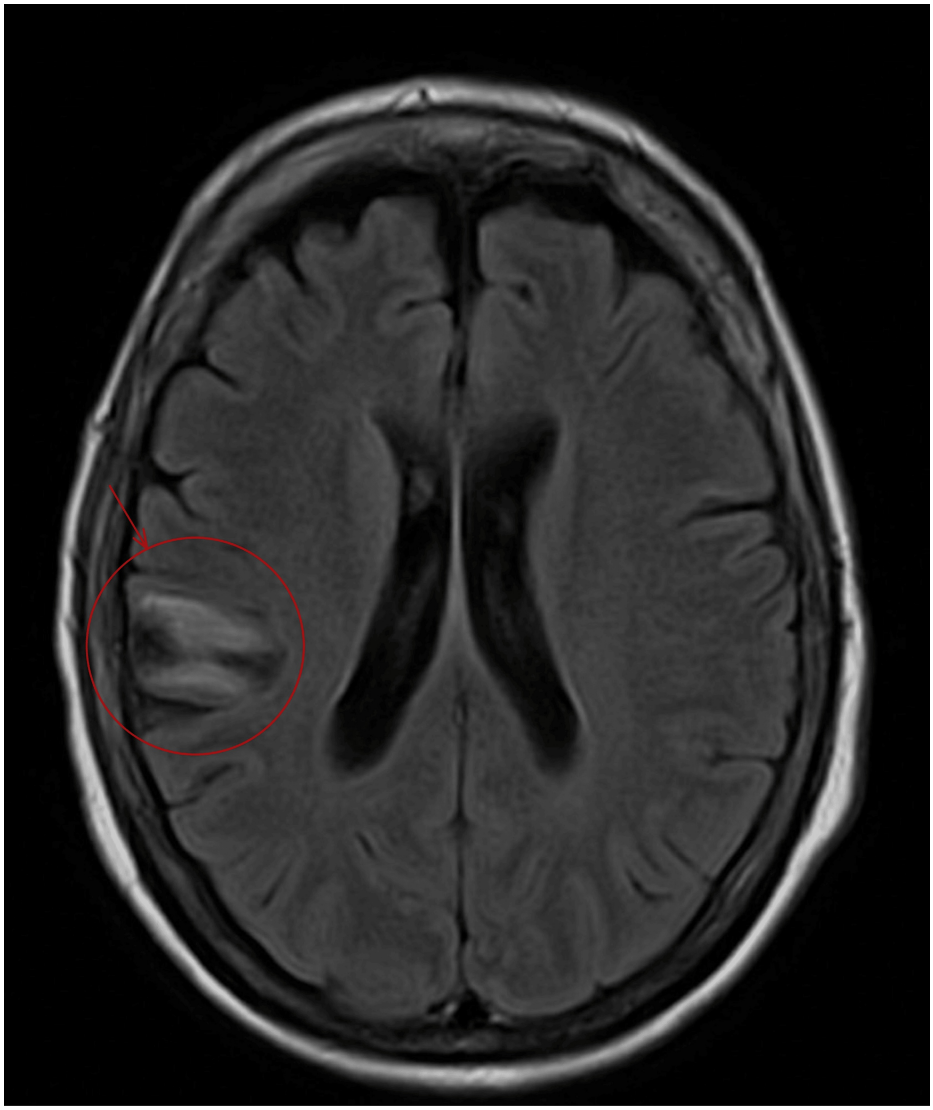


FIGURE 2: Initial MRI of the brain demonstrating chronic infarct-related changes on FLAIR imaging

FLAIR: Fluid-attenuated inversion recovery

During the ICU admission, her neurological state fluctuated, with episodes of confusion, agitation, and abnormal eye movements. She developed an episode of sustained upward deviation of the eyes, initially considered to represent an oculogyric crisis related to metoclopramide, which was prescribed as an antiemetic as part of the management strategy to reduce vomiting and the associated risk of aspiration. This improved following treatment with procyclidine. However, recurrent episodes of abnormal eye movements were subsequently accompanied by altered awareness, transient left-sided neglect, inappropriate speech, failure to follow commands and progressive visual impairment, raising concern for an underlying neurological process. She subsequently developed recurrent focal and generalised seizures. An electroencephalogram (EEG) demonstrated an abnormal slow background consistent with encephalopathy, together with focal epileptiform activity. Serial EEG findings are summarised in Table *3*.

Order	Clinical context	EEG findings	Interpretation
First EEG	Recurrent episodes of abnormal eye movements, altered awareness and suspected focal seizures	Slow background activity consistent with encephalopathy. Intermittent bursts of faster activity occurred predominantly over the left hemisphere and were frequently associated with rightward eye deviation. Occasional epileptiform activity was also observed over the right hemisphere.	Findings are consistent with focal-onset subclinical seizures. No evidence of status epilepticus.
Second EEG (6 days later)	Ongoing episodes of abnormal eye movements and recurrent seizures despite antiepileptic treatment	Persistent sharp and fast epileptiform activity over the right hemisphere, consistent with ongoing focal-onset seizure activity.	Ongoing uncontrolled focal-onset seizures
Following escalation of treatment	Following treatment with levetiracetam, phenytoin, and lacosamide	No further clinical seizures were observed	Clinical seizure control achieved

TABLE 3: Serial EEG findings

EEG: Electroencephalogram

Antiepileptic treatment was sequentially escalated with levetiracetam, intravenous phenytoin and subsequently lacosamide. Following escalation of therapy, clinical seizures resolved, and her GCS improved to 14/15. As her neurological status evolved, her visual function deteriorated significantly. She progressed from visual disturbance to inability to detect hand movements despite preserved pupillary responses. Repeat CT head and CT angiography demonstrated no acute intracranial abnormality. An MRI of the brain demonstrated cortical and subcortical T2/FLAIR hyperintensity involving the right occipital lobe with associated diffusion restriction on diffusion-weighted imaging, together with chronic lacunar infarcts in the right cerebral peduncle and left posterior external capsule. Representative T2-weighted and FLAIR images are shown in Figures *3*-*4*. The occipital abnormality was initially interpreted as an acute infarction within the right posterior cerebral artery territory, and aspirin 300 mg daily was commenced following stroke team advice.

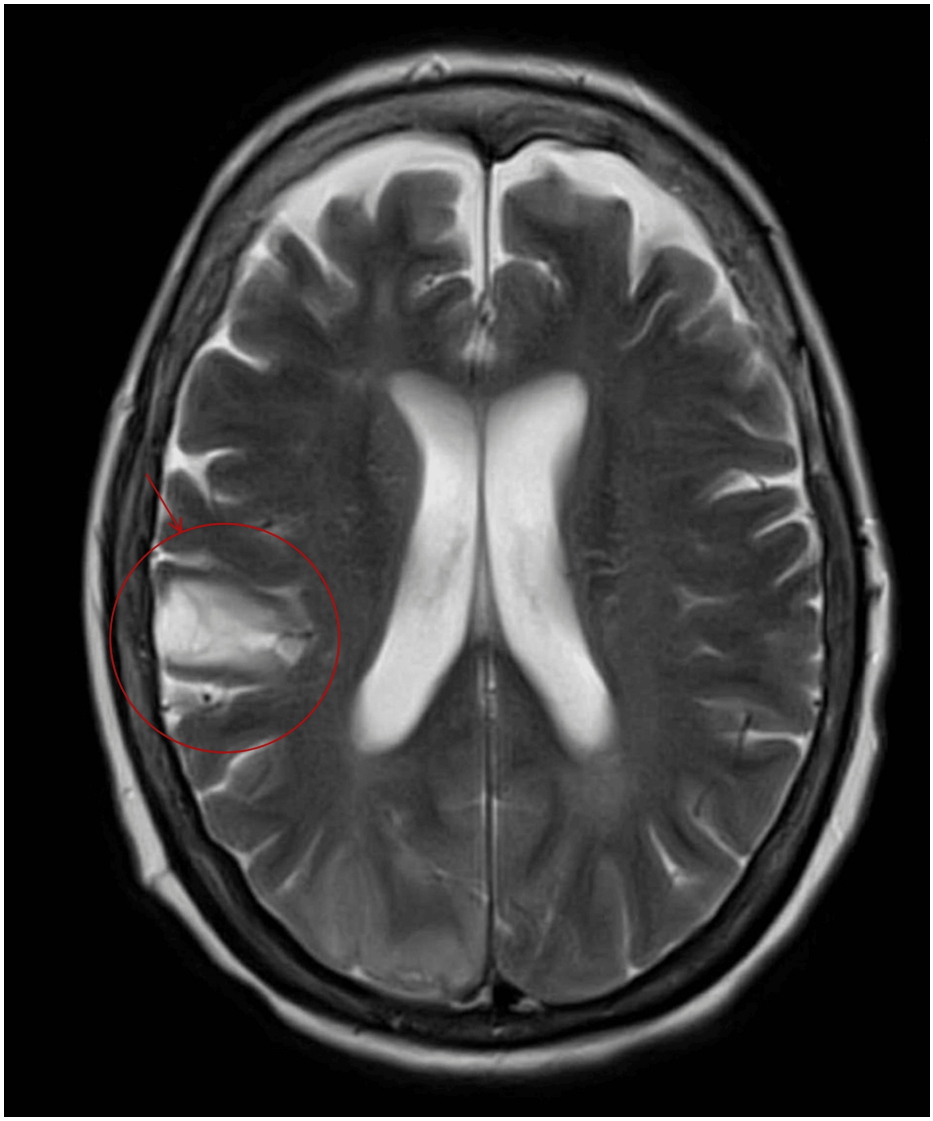


FIGURE 3: MRI of the brain demonstrating right occipital cortical and subcortical T2 hyperintensity.

The abnormality was initially interpreted as an acute right posterior cerebral artery territory infarction.

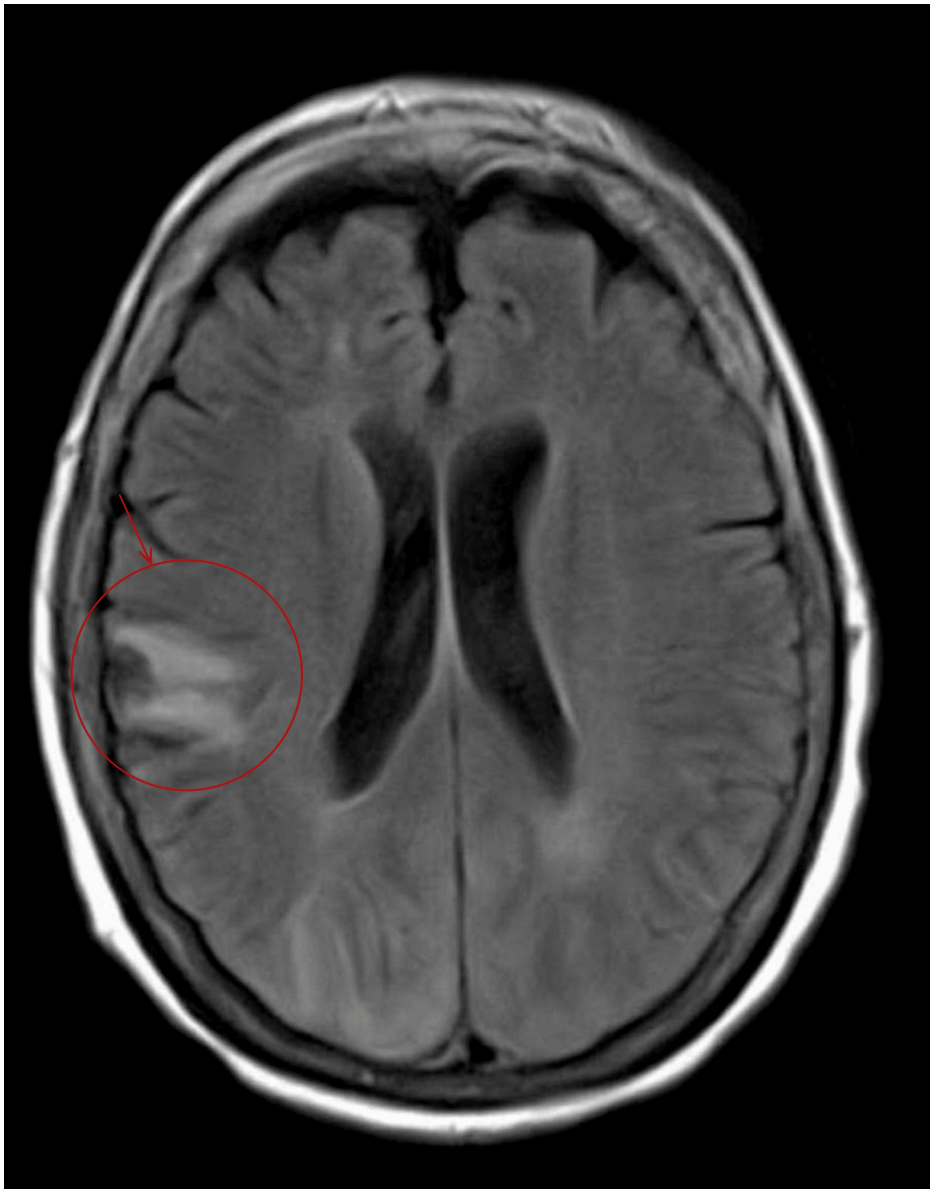


FIGURE 4: MRI of the brain demonstrating right occipital cortical and subcortical FLAIR hyperintensity

The abnormality was initially interpreted as an acute right posterior cerebral artery territory infarction.FLAIR: fluid-attenuated inversion recovery.

The combination of encephalopathy, recurrent seizures, profound bilateral visual loss and the evolving occipital MRI abnormality prompted discussion with neuroradiology specialists at a tertiary centre. Although the diffusion restriction initially raised concern for acute ischaemic stroke, the imaging appearances and clinical context were considered more consistent with PRES, particularly given the subsequent radiological improvement.

Several potential contributing factors for PRES were considered, including severe thyrotoxicosis, acute kidney injury and systemic infection with associated metabolic disturbance. The admission serum sodium concentration was normal at 138 mmol/L. Although blood pressure was elevated during the admission, she had not been hypertensive at initial presentation, and there was no history of chronic hypertension. No recognised offending immunosuppressive or cytotoxic medication was identified.

Ophthalmological assessment demonstrated severe bilateral visual impairment without a relative afferent pupillary defect, supporting a cortical rather than primary ocular cause of visual loss. Although the MRI abnormality predominantly involved the right occipital lobe, the severity and bilateral nature of the visual impairment were not fully explained by this unilateral lesion. The concurrent encephalopathy and recurrent seizures raised the possibility of additional functional disturbance of the visual processing networks that was

not apparent on conventional structural imaging. Toxic optic neuropathy was considered, and outpatient neuro-ophthalmology assessment and electrodiagnostic testing were arranged.

Following the initiation of carbimazole and corticosteroid therapy, thyroid function tests demonstrated an initial biochemical improvement, with FT4 decreasing from 34.5 pmol/L on admission to 8.5 pmol/L by day six and FT3 decreasing from 29.7 to 3.0 pmol/L over the same period. However, repeat testing on Day 20 demonstrated a subsequent increase in FT4 to 26.3 pmol/L and FT3 to 9.3 pmol/L, consistent with recurrent biochemical thyrotoxicosis. The reason for this fluctuation could not be established from the available clinical documentation. Thyroid function subsequently improved by the two-month follow-up. Following a multidisciplinary neuroradiology review, the diagnosis of PRES with cortical visual impairment and secondary seizures was established. Aspirin was discontinued once acute ischaemic stroke was considered unlikely.

In view of her history of alcohol dependence and the risk of thiamine deficiency, the patient received intravenous Pabrinex (high-potency vitamins B and C), administered as two pairs of ampoules three times daily. Each pair contained thiamine hydrochloride 250 mg, riboflavin 4 mg, pyridoxine hydrochloride 50 mg, nicotinamide 160 mg and ascorbic acid 500 mg. This corresponded to a thiamine dose of 500 mg intravenously three times daily. She also completed treatment with intravenous piperacillin-tazobactam (4.5 grams, twice a day) for suspected hospital-acquired pneumonia. She remained seizure-free following escalation of antiepileptic therapy and underwent physiotherapy and occupational therapy during her recovery.

A repeat MRI of the brain demonstrated interval improvement in the previously identified right occipital abnormality, with no convincing persistent diffusion restriction on follow-up apparent diffusion coefficient (ADC) imaging (Figure 5). The radiological improvement further supported a reversible process consistent with PRES.

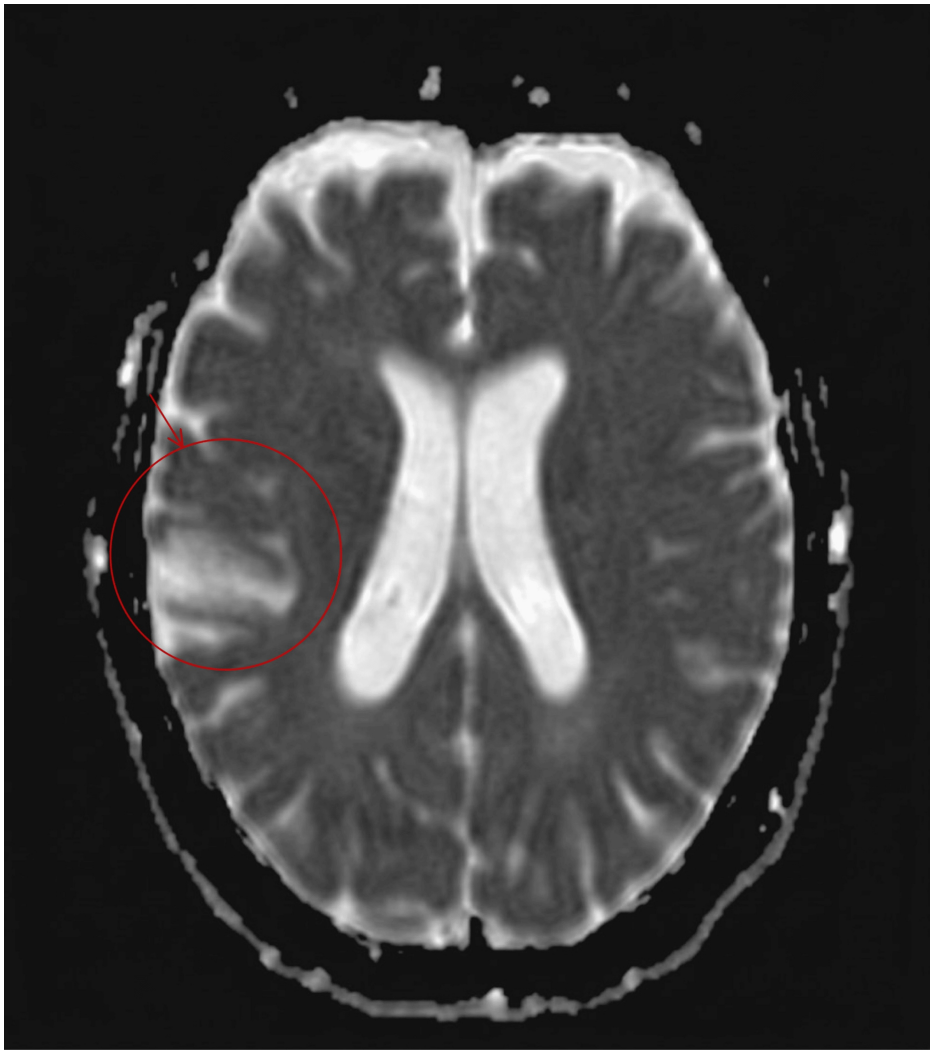


FIGURE 5: Follow-up ADC image demonstrating no convincing persistent diffusion restriction in the previously affected right occipital region, supporting a reversible process consistent with PRES.

ADC: Apparent diffusion coefficient, PRES: posterior reversible encephalopathy syndrome

She was discharged after a four-week hospital stay with diagnoses of Graves' thyrotoxicosis, PRES complicated by focal-onset seizures, and cortical visual impairment secondary to PRES. At discharge, she remained profoundly visually impaired, able to perceive light and vague outlines but unable to count fingers, and required a walking frame and assistance with mobility. Her discharge medications included carbimazole 15 mg once daily, levetiracetam 1.5 g twice daily, phenytoin 100 mg twice daily, lacosamide 50 mg twice daily, duloxetine 60 mg AM and 30 mg PM, quetiapine 50 mg twice daily, clopidogrel 75 mg once daily, rosuvastatin 10 mg once daily, lansoprazole 15 mg once daily, folic acid 5 mg once daily, thiamine 100 mg once daily, zopiclone 3.75 mg once daily and nicotine replacement therapy.

At endocrine follow-up two months post-discharge, thyroid function had improved substantially, with TSH remaining suppressed at <0.008 mIU/L, FT4 9.5 pmol/L and FT3 3.3 pmol/L. Liver function tests and full blood count were normal, and carbimazole was reduced to 10 mg once daily. She reported gradual improvement in visual function and could recognise light, colours, and some objects, although significant visual impairment persisted. She also reported ongoing headaches and memory difficulties during neurological recovery. A timeline of clinical progression can be seen in Table *4* below.

Day no.	Clinical course and investigations	Management/outcome
	Presented after being found collapsed, with one day of drowsiness and cough; GCS 15/15	Initially treated for presumed sepsis with intravenous fluids

Day one	initially. Hypoxic with oxygen saturation of 90% on room air and subsequently febrile. Blood tests demonstrated acute kidney injury and severe thyrotoxicosis. The CT head showed established right parietal and basal ganglia infarcts without acute haemorrhage.	and antibiotics. Severe thyrotoxicosis was identified; carbimazole and intravenous corticosteroids commenced.
Day two	Persistent encephalopathy with worsening respiratory function. Thyroid function confirmed severe autoimmune hyperthyroidism; TSH receptor antibodies were positive, consistent with Graves' disease.	Continued treatment for thyrotoxicosis. Alcohol withdrawal and other toxic-metabolic causes remained differential diagnoses.
Day three	Transferred to ICU; BiPAP commenced with subsequent improvement in hypercapnia and respiratory acidosis. Metoclopramide was prescribed during the ICU admission as an antiemetic to reduce vomiting and the associated risk of aspiration.	Transferred to ICU; BiPAP commenced with subsequent improvement in hypercapnia and respiratory acidosis.
Days four and five	Possible neck stiffness prompted investigation for meningoencephalitis. Neurological status remained fluctuating. The MRI demonstrated chronic infarcts without acute infarction or abnormal enhancement.	Empirical ceftriaxone and acyclovir commenced. A lumbar puncture was performed.
Days six and seven	The CSF analysis and encephalitis screen were negative. The MRI showed chronic left parietal white matter changes and bilateral basal ganglia lacunar infarcts, unchanged from previous imaging. Serial thyroid function tests demonstrated an initial biochemical improvement, with FT4 decreasing from 34.5 pmol/L on admission to 8.5 pmol/L on day six, and FT3 decreasing from 29.7 to 3.0 pmol/L following the initiation of thyroid-directed treatment.	Infectious encephalitis became less likely. Neurological and supportive management continued.
Day eight	Acute episode of confusion with abnormal eye movements, upward gaze deviation, transient neglect, and impaired responsiveness. A CT head and CT angiography showed no acute abnormality.	Stroke team review. Repeat MRI and EEG requested.
Days nine and 10	Recurrent abnormal eye movements and episodes of altered awareness. Progressive visual disturbance developed. The EEG demonstrated encephalopathy with focal-onset subclinical seizures.	Levetiracetam commenced and subsequently escalated.
Days 11 and 12	Recurrent focal and generalised seizures with further neurological deterioration.	Loaded with intravenous phenytoin, and the levetiracetam dose increased. Seizure activity subsequently improved, and GCS increased to 14/15.
Days 13 and 14	Persistent visual impairment despite improvement in consciousness.	Continued antiepileptic and thyroid-directed therapy. An ophthalmology assessment was arranged.
Days 15 and 16	The MRI demonstrated right occipital cortical and subcortical T2/FLAIR hyperintensity with diffusion restriction, initially interpreted as an acute right posterior cerebral artery territory infarction. Visual function deteriorated to the inability to detect hand movements. The EEG demonstrated persistent right hemispheric focal epileptiform activity.	Aspirin commenced following stroke team advice. Lacosamide was added because of ongoing focal seizures.
Days 17 and 18	A multidisciplinary neuroradiology review raised concern that the occipital lesion represented PRES rather than infarction. Contributing factors included severe thyrotoxicosis, acute kidney injury, systemic infection, and metabolic disturbance.	Continued antiepileptic therapy. No further clinical seizures observed.
Days 19 and 20	Severe bilateral visual impairment persisted. Ophthalmological assessment demonstrated anisocoria without a relative afferent pupillary defect, supporting a cortical cause of visual loss. Repeat thyroid function tests on day 20 demonstrated a rise in FT4 to 26.3 pmol/L and FT3 to 9.3 pmol/L, indicating recurrent biochemical thyrotoxicosis following the initial improvement. The cause of this subsequent increase could not be established from the available clinical documentation.	Aspirin was discontinued after the diagnosis of PRES was favoured. Neuro-ophthalmology follow-up was planned. Thyroid-directed treatment continued.
Days 21 and 26	Remained seizure-free with persistent visual impairment. Oculogyric episodes improved with procyclidine.	Continued rehabilitation with physiotherapy and occupational therapy. Antiepileptic therapy was maintained.
		Neurology concluded that no

Day 27	A repeat MRI demonstrated interval improvement in the right occipital abnormalities with no new diffusion restriction, supporting the diagnosis of PRES.	further inpatient neurological intervention was required. Neurorehabilitation and outpatient neurology/neuro-ophthalmology follow-up arranged.
Day 28	Clinically stable and medically optimised for discharge.	Discharged with diagnoses of Graves' disease and PRES complicated by seizures and cortical visual impairment.
Follow-up two months post-discharge	Thyroid function improved (FT4 9.5 pmol/L, FT3 3.3 pmol/L, TSH <0.008 mIU/L). Visual function had improved from light perception only to recognition of light, colours and objects, although significant impairment persisted. Memory difficulties and headaches continued.	Carbimazole was reduced to 10 mg once daily. Continued neurological and ophthalmological follow-up.

TABLE 4: Timeline of clinical progression

GCS: Glasgow Coma Scale, TSH: Thyroid-stimulating hormone, BiPAP: Bilevel positive airway pressure, EEG: Electroencephalogram, FT4: Free thyroxine, FT3: Free triiodothyronine, FLAIR: Fluid-attenuated inversion recovery, PRES: Posterior reversible encephalopathy syndrome

Discussion

PRES and the differential diagnosis

This case demonstrates the diagnostic complexity of PRES when posterior cerebral abnormalities occur in a patient with several potential causes of encephalopathy and seizures. The patient developed acute encephalopathy, recurrent focal and generalised seizures, and profound bilateral visual impairment in the setting of severe autoimmune hyperthyroidism, acute kidney injury, and systemic infection. Although the initial MRI demonstrated no acute intracranial lesion, subsequent imaging showed right occipital cortical and subcortical abnormalities with associated diffusion restriction that were initially interpreted as an acute posterior cerebral artery territory infarction.

Posterior reversible encephalopathy syndrome is believed to arise from a combination of impaired cerebral autoregulation and endothelial dysfunction, resulting in disruption of the blood-brain barrier and predominantly vasogenic cerebral oedema [1,3]. Severe hypertension is an important precipitant, but it is not present in all cases. It has been described in patients with normal or only mildly elevated blood pressure, particularly in association with renal dysfunction, sepsis, autoimmune disease and systemic inflammatory states [1,3,9]. In the present case, blood pressure was not markedly elevated at initial presentation, although higher blood pressures were documented later during admission. The patient also had acute kidney injury, systemic infection and metabolic disturbance, all of which may have contributed to the physiological environment in which PRES developed.

The radiological spectrum of PRES is broader than the term 'posterior' might suggest. Although bilateral parieto-occipital vasogenic oedema is typical, lesions may be unilateral or involve the frontal lobes, cerebellum, brainstem or deep grey nuclei [1,3,9]. Cortical involvement, haemorrhage and restricted diffusion are also recognised atypical features [3,9]. Importantly, diffusion restriction does not necessarily indicate irreversible infarction. In a series of patients with PRES and restricted diffusion, most lesions demonstrated subsequent reversibility, although other studies have associated restricted diffusion with worse clinical outcome and a greater risk of permanent injury [9,10]. This distinction is clinically important when PRES is considered in the differential diagnosis of acute ischaemic stroke.

In this patient, the right occipital abnormality was initially interpreted as an acute posterior cerebral artery territory infarction because of its cortical involvement and diffusion restriction. However, the clinical context was atypical for an isolated territorial infarction. The patient had prominent encephalopathy, recurrent seizures, and profound bilateral visual impairment. Subsequent multidisciplinary neuroradiology review and repeat MRI demonstrated substantial interval improvement of the occipital abnormality without convincing persistent diffusion restriction. This radiological evolution, together with the clinical phenotype, supported PRES over established cerebral infarction. The case therefore highlights the importance of considering PRES when posterior lesions do not conform neatly to a vascular territory or when their subsequent evolution is inconsistent with established irreversible ischaemic injury [3,9,10].

PRES and autoimmune hyperthyroidism

The association between PRES and autoimmune hyperthyroidism appears to be uncommon, although reversible posterior cerebral abnormalities have been reported in patients with Graves’ disease and other

autoimmune thyroid disorders [6-8]. The mechanisms underlying this association remain uncertain. Severe thyrotoxicosis produces marked cardiovascular and metabolic changes, including increased cardiac output and altered systemic vascular resistance [11]. In addition, studies in patients with overt hyperthyroidism, including Graves' disease, have demonstrated changes in circulating markers associated with endothelial dysfunction and inflammation [12]. Endothelial dysfunction is itself an important proposed mechanism in PRES [3,9]. These observations provide biological plausibility for an association, but they do not establish that thyrotoxicosis directly causes PRES.

Previous reports have described reversible posterior cerebral abnormalities in patients with autoimmune thyroid disease. Tateishi et al. reported a patient with autoimmune thyroid disease who developed impaired consciousness and seizures with occipitotemporal MRI abnormalities that subsequently disappeared, with the imaging pattern considered compatible with PRES [6]. Reversible posterior white matter abnormalities have also been described in patients with Hashimoto encephalopathy and autoimmune thyroid disease [7,8]. Taken together, these reports suggest that thyroid autoimmunity or severe thyroid dysfunction may contribute to susceptibility to reversible cerebral oedema in selected patients, although the rarity of reported cases and the presence of other potential precipitants limit conclusions regarding causality.

An important consideration in the present case is whether the neurological syndrome could instead represent encephalopathy associated with autoimmune thyroid disease. This syndrome has been described predominantly in association with Hashimoto thyroiditis and encompasses a heterogeneous range of neuropsychiatric manifestations, including altered consciousness, cognitive impairment and seizures [4,5]. However, its nosology and diagnostic criteria remain controversial [4,5]. The markedly elevated thyroid autoantibodies in this patient demonstrate active autoimmune thyroid disease but do not, in isolation, establish an autoimmune encephalopathy.

Several features in this case support PRES as the principal explanation for the acute neurological syndrome. The MRI abnormality was predominantly posterior and cortical/subcortical and subsequently demonstrated substantial interval improvement. The patient also had prominent seizures and severe visual impairment, both recognised manifestations of PRES [1,3,9]. Cortical visual loss, including profound bilateral visual impairment, is a recognised manifestation of posterior reversible encephalopathy and can initially be mistaken for an irreversible ischaemic event [13]. Cerebrospinal fluid examination did not support an inflammatory encephalopathy, while extensive neuronal surface, synaptic and paraneoplastic antibody testing was negative. Furthermore, the neurological course occurred in the context of multiple systemic stressors, including severe thyrotoxicosis, acute kidney injury and infection.

Nevertheless, severe autoimmune hyperthyroidism may have contributed to the physiological environment in which PRES developed. In this case, it is more appropriate to regard thyrotoxicosis as one of several potential precipitating factors rather than as an established direct cause of PRES. The coexistence of acute kidney injury, systemic infection and metabolic disturbance makes attribution to a single factor inappropriate.

Seizures and visual impairment

Seizures are among the most frequent neurological manifestations of PRES and may be focal, generalised or evolve into status epilepticus [1,3]. In this case, serial EEG demonstrated evolving focal epileptiform activity, initially predominantly over the left hemisphere and subsequently over the right hemisphere. The episodes of abnormal eye deviation and altered awareness therefore represented an important diagnostic clue. Seizure control required escalation from levetiracetam to phenytoin and subsequently lacosamide.

The profound bilateral visual impairment was particularly striking, given that the MRI abnormalities predominantly involved the right occipital lobe. An isolated right occipital lesion would typically produce a left homonymous visual field defect rather than severe bilateral visual loss. Therefore, the extent of the structural abnormality did not fully account for the severity of the clinical presentation. Posterior reversible encephalopathy syndrome can produce visual disturbances ranging from visual field defects to cortical blindness [1,3]. In this patient, concurrent encephalopathy and recurrent focal seizures may have contributed to more widespread functional disruption of cortical visual processing, potentially extending beyond the abnormalities visible on conventional MRI. The absence of a relative afferent pupillary defect was compatible with a retrochiasmal cause of visual impairment, although it did not establish the precise anatomical extent of dysfunction. The subsequent radiological improvement and partial recovery of visual function supported a reversible neurological process. Nevertheless, the precise mechanism underlying the profound bilateral visual impairment could not be definitively established, and the discrepancy between the clinical findings and predominantly unilateral MRI abnormality represents a limitation of this case.

The term 'reversible' in PRES should therefore not be interpreted as synonymous with complete clinical recovery. Although many patients experience substantial neurological and radiological improvement, persistent neurological deficits can occur, particularly in patients with severe disease or atypical imaging features [3,10,14]. In this case, visual function improved from perception of light only to recognition of colours and objects. However, significant functional impairment persisted at follow-up, with ongoing memory difficulties and headaches.

Diagnostic complexity and lessons from the case

The case was further complicated by an oculogyric crisis attributed to metoclopramide. This was clinically important because sustained upward eye deviation could initially be mistaken for focal seizure activity. Oculogyric crisis is a recognised acute dystonic reaction associated with dopamine-blocking drugs, including metoclopramide, and may mimic neurological disorders such as seizures [15]. Recognition of a drug-induced dystonic reaction and response to procyclidine helped distinguish this episode from the patient's subsequent epileptic events.

The principal diagnostic challenge was differentiating PRES from acute posterior circulation infarction. This distinction has important therapeutic implications because treatment pathways differ substantially. The initial use of antiplatelet therapy following the interpretation of the occipital lesion as an acute infarct was subsequently reversed after neuroradiological review and demonstration of interval improvement. This case reinforces the value of integrating clinical features, MRI sequences, and serial imaging rather than relying on a single radiological examination [9,13].

Several potential alternative causes of encephalopathy were actively investigated. Infection was considered because of fever and systemic illness, while alcohol withdrawal and Wernicke's encephalopathy were considered because of her alcohol dependence. Meningoencephalitis was investigated with CSF analysis and empirical antimicrobial therapy, while autoimmune and paraneoplastic encephalitis were assessed using extensive antibody testing. The negative results and subsequent radiological evolution supported PRES as the most coherent explanation for the neurological syndrome.

Conclusions

This case highlights the importance of considering PRES in patients presenting with encephalopathy, seizures and visual impairment, even in the absence of severe hypertension. Multidisciplinary neuroradiological assessment and serial MRI were valuable in establishing the diagnosis and guiding management. Early recognition and treatment of potential precipitating factors are important, although persistent neurological deficits may occur despite radiological improvement.

Additional Information

Author Contributions

All authors have reviewed the final version to be published and agreed to be accountable for all aspects of the work.

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Disclosures

Human subjects: Informed consent for treatment and open access publication was obtained or waived by all participants in this study. **Conflicts of interest:** In compliance with the ICMJE uniform disclosure form, all authors declare the following: **Payment/services info:** All authors have declared that no financial support was received from any organization for the submitted work. **Financial relationships:** All authors have declared that they have no financial relationships at present or within the previous three years with any organizations that might have an interest in the submitted work. **Other relationships:** All authors have declared that there are no other relationships or activities that could appear to have influenced the submitted work.

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