

Case Report

Contrast Media-associated Posterior Reversible Encephalopathy Syndrome: A Case Report and Review of the Literature

S ÇELİK, HC TUĞRUL, S ERDOĞAN, G ATAY

Abstract

Posterior reversible encephalopathy syndrome (PRES) is a clinical and radiological condition that is often reversible and is characterised by clinical findings such as headache, visual disturbances, seizures, and altered consciousness. Magnetic resonance imaging of the brain typically reveals vasogenic oedema predominantly in the posterior cerebral hemispheres. Here, we present the case of a 12-year-old girl who developed PRES following a contrast-enhanced computed tomography mesenteric angiogram. We aim to highlight the importance of early recognition and appropriate management of PRES, a rare complication of contrast agents, in significantly impacting patient prognosis.

Key words

Contrast media; Cytotoxic oedema; Posterior reversible encephalopathy syndrome

Introduction

Posterior reversible encephalopathy syndrome (PRES) is a temporary condition characterised by widespread cerebral oedema, more prominent in parietal and occipital regions. It can present with non-specific clinical findings such as headache, nausea, vomiting, visual and mental changes, and generalised or focal seizures. However, if diagnosis and treatment are delayed, serious morbidity and mortality can occur due to complications such as status epilepticus, intracranial haemorrhage, and massive

ischaemic infarction.^{1,2} Contrast-induced encephalopathy is a rare complication of angiography. Its incidence is reported to be 0.06% in patients undergoing coronary angiography and 0.3-1% in those undergoing vertebral angioplasty, and it generally follows a benign course.^{3,4} With the elimination of risk factors and control of blood pressure, complete recovery is usually observed within days to weeks. However, in rare cases, death and severe, permanent neurological sequelae can occur. Severe vasogenic oedema, brain ischaemia, and intracerebral haemorrhage are associated with poor prognosis.

Here, we aim to discuss the pathophysiology of the disease by presenting a paediatric patient who developed PRES following a mesenteric contrast-enhanced computed tomography (CT) angiography.

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Case Report

A patient underwent surgery for a perforated appendix and was subsequently monitored with a colostomy due to recurrent brid ileus 2 months prior to the presentation. The patient was brought to the paediatric emergency department with a complaint of vomiting. A water-soluble contrast follow-through was evaluated as normal, and emergency surgical intervention was not considered. The patient was admitted to the paediatric intensive care unit

for close observation and neostigmine therapy. General condition of the patient was moderate, weak, and lethargic, with a body temperature of 36.8°C, oxygen saturation of 99% on room air, heart rate of 110 bpm, blood pressure of 100/75 mmHg, weight of 32 kg (<5th percentile), and height of 154 cm (10th-25th percentile). Physical examination revealed abdominal distension and absent bowel sounds. Abdominal X-ray showed widespread air-fluid levels.

For the differential diagnosis of paediatric intestinal pseudo-obstruction, a contrast-enhanced CT mesenteric angiogram was performed. Omnipaque (300 mg/mL) 1 ml/kg was administered intravenously for 1 minute as a contrast agent, followed by 10 mL of 0.9% NaCl. Twenty-four hours after the scan, the patient's blood pressure was noted to be elevated, and captopril was initiated as

antihypertensive therapy. However, during follow-up, the patient experienced a seizure lasting approximately 70 seconds, characterised by staring at a fixed point. During this episode, blood pressure was measured at 170/110 mmHg and glucose level was 105 mg/dL. Intravenous metoprolol was administered. A plain brain MRI was performed, which revealed multiple T2/FLAIR hyperintense signal changes (Figure 1) in the frontal, parietal, and occipital lobes, suggestive of cortical-subcortical vasogenic oedema. The diffusion-weighted imaging (DWI) sequences also demonstrated restricted diffusion in the frontal lobes, suggesting cytotoxic oedema (Figure 2), and the ADC values were consistent with this finding. After regaining consciousness and becoming cooperative, the patient was transitioned to oral antihypertensive therapy, and metoprolol treatment was

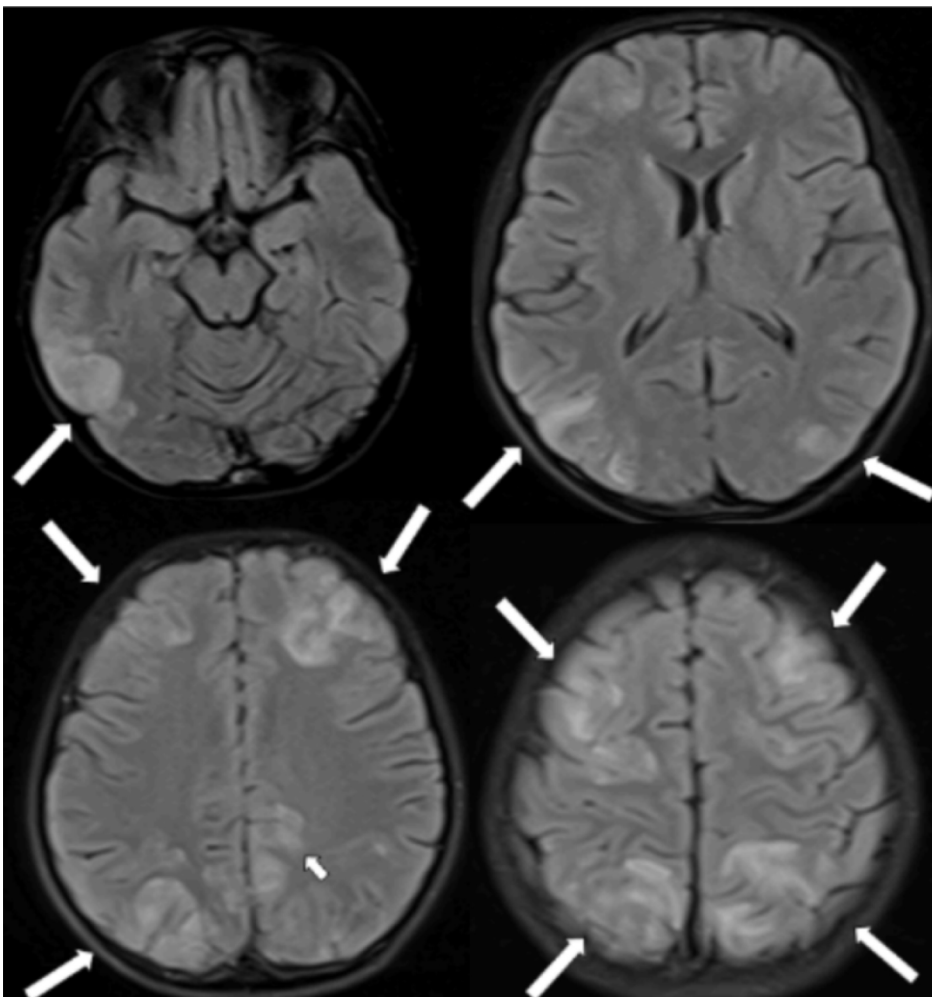


Figure 1. FLAIR images show cortical and subcortical hyperintense signal changes consistent with vasogenic oedema in frontal and occipital regions.

discontinued. The patient's blood pressure remained within normal limits during follow-up, and antihypertensive therapy was gradually discontinued.

The patient achieved a full neurological recovery and was discharged without any residual neurological deficits. A follow-up MRI was not performed. The patient was then transferred to the paediatric surgery department for further care.

Discussion

Posterior reversible encephalopathy syndrome (PRES) is a clinical and radiological condition that often manifests with symptoms such as headache, visual disturbances, seizures, and changes in consciousness. It is frequently associated with vasogenic oedema in the posterior cerebral hemispheres on brain MRI and is usually reversible. PRES can occur at any age, with the youngest reported case in the literature being 2 years old and the oldest 90 years old.^{5,6}

Pathogenesis of PRES is still unclear, but vasospasm and cytotoxic oedema caused by cerebral autoregulation disorders and endothelial dysfunction are considered possible mechanisms. In certain clinical conditions, such as uremia, sepsis, hypomagnesemia, and other metabolic disorders, a disruption of vascular endothelial functions can occur, which may lead to PRES.⁷

Hypertension, which develops alongside autoregulation disorder, is thought to play a significant role in the majority of patients with PRES. Approximately 75%

of patients have moderate to severe hypertension, but PRES can also develop in normotensive patients.

The pathophysiological mechanisms thought to be responsible for the development of PRES include endothelial dysfunction and the direct effects of cytokines associated with sudden changes in blood pressure. This situation leads to disruption of the blood-brain barrier and subsequent brain oedema. In normotensive PRES patients, the neuropeptide theory is more prominent. According to this theory, the release of potent vasoconstrictors such as endothelin-1 (ET-1) can lead to vasospasm, ischemia, and cerebral oedema. It has been reported that ET-1 levels measured after administration of contrast agents in patients undergoing percutaneous coronary intervention show a significant correlation with the amount of contrast agent used.⁸ In this context, contrast agents can temporarily disrupt the blood-brain barrier by directly inducing endothelial cell activity with effects similar to cytokines in PRES. This leads to extravasation of plasma components and the formation of vasogenic oedema. Due to less sympathetic innervation, especially in posterior cerebral circulation, this region is more prone to oedema development. High-osmolality agents can stress endothelial cells by drawing fluid from the plasma into the interstitial space. This type of neurotoxicity appears to be related not only to the ionic properties of the contrast agent but also to its hyperosmolality, lipid solubility, and viscosity. This adverse reaction has been reported for both ionic and non-ionic contrast agents.^{9,10} The toxicity is mostly localised in occipital lobes.

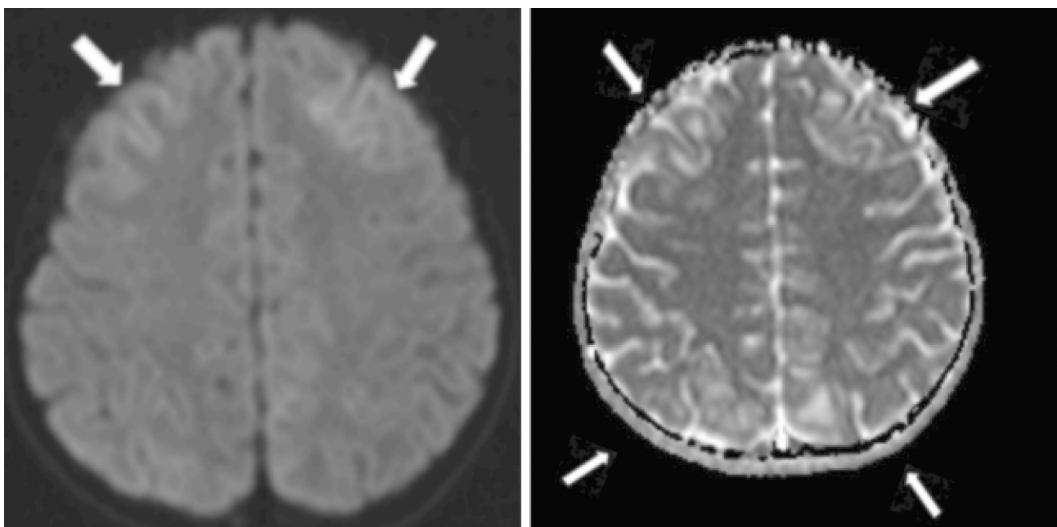


Figure 2. Diffusion-weighted imaging images show diffusion restriction consistent with cytotoxic oedema in frontal lobes, and ADC images.

Under normal circumstances, cerebral circulation is kept constant despite changes in systemic blood pressure. However, autoregulation may be impaired in the presence of hypertension or in cases of cerebral vasodilation due to contrast medium. This impairment increases capillary damage with hyperperfusion and facilitates the development of PRES. Both PRES and contrast-induced encephalopathy (CIE) have similar clinical presentations, reversibility, hyperintense areas on FLAIR sequences of brain MRI, and a common pathophysiology. However, PRES develops due to hypertension, immunosuppression, and cytotoxic agent use, while CIE develops due to the direct toxic effect of contrast medium. While posterior cerebral regions are mostly affected in PRES, CIE usually has hemispheric and sometimes unilateral cortical-subcortical involvement. In PRES, treatment is to correct the underlying cause, but in CIE, symptomatic treatment is sufficient, and no specific treatment is required.

Several factors may have played a role in the development of PRES in our patient during the contrast-enhanced CT mesenteric angiography. Firstly, our patient experienced acute hypertension, which may have increased the osmotic distribution in the blood-brain barrier. A second factor could be the properties of the contrast agent used during the procedure.

Omnipaque (300 mg/mL iohexol, Opakim, Istanbul) is a monomeric, non-ionic contrast agent with an osmolality of 709 mOsm/kg H₂O. Compared to blood osmolality (300 mOsm/kg), it has a relatively higher osmolality, which may have increased the permeability of the blood-brain barrier, leading to brain oedema.

A significant finding in our patient was the presence of restricted diffusion in the frontal lobes on DWI sequences, suggesting cytotoxic oedema. While PRES is typically characterised by vasogenic oedema, the presence of cytotoxic oedema is an atypical and more severe complication. This finding may indicate underlying ischemia and is associated with a higher risk of permanent neurological deficits, though our patient achieved a full neurological recovery.

Early recognition and effective management of PRES, a rare complication of contrast agents, are crucial for improving patient prognosis. Control of hypertension, careful use of medications that may cause endothelial damage, and monitoring of renal functions can

significantly reduce the risk of PRES. Preferring low-osmolality and non-ionic contrast agents in high-risk patients, providing premedication and hydration when necessary, and a multidisciplinary approach play a critical role in preventing this condition.

Written informed consent had been obtained from the patient for the publication of this case report and any accompanying images.

Conflicts of Interest

All authors have disclosed no conflicts of interest.

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