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Case Report

Contrast-induced posterior reversible encephalopathy syndrome following diagnostic angiography of vertebral artery

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ABSTRACT

Background: Posterior reversible encephalopathy syndrome (PRES) is characterized by transient vasogenic edema, predominantly affecting the white matter in the posterior cerebral hemispheres. It presents with acute neurological symptoms such as headaches, visual disturbances, and seizures. The pathophysiology of PRES, including its overlap with contrast-induced encephalopathy and transient cortical blindness, remains unclear.

Case Description: A 76-year-old woman with a basilar artery aneurysm underwent diagnostic angiography. During the procedure, she experienced a hypertensive spike following the injection of contrast medium. Four hours post-angiography, she developed disorientation and bilateral light perception, which progressed to complete blindness. Magnetic resonance imaging revealed bilateral occipital hemisphere edema, confirming a diagnosis of PRES. All neurological symptoms resolved within 48 h. Subsequently, she successfully underwent coil embolization of the aneurysm. With careful blood pressure management and a switch to a different type of contrast medium, PRES did not recur despite the use of a larger volume of contrast medium.

Conclusion: This case of contrast-induced PRES underscores the potential overlap in pathogenesis between PRES and contrast-induced encephalopathy. It emphasizes the need for careful blood pressure management and consideration of contrast medium type in patients undergoing angiography, especially those with a history of PRES. The successful management of this case provides valuable insights into the prevention and treatment of PRES in similar clinical scenarios.

Keywords: Contrast-induced encephalopathy, Posterior reversible encephalopathy syndrome, Transient cortical blindness

INTRODUCTION

Posterior reversible encephalopathy syndrome (PRES) is characterized by transient vasogenic edema, predominantly affecting the white matter in the posterior cerebral hemispheres, especially the parieto-occipital regions.[8,12] Patients typically present with acute neurological symptoms such as headaches, visual disturbances, and seizures, which are generally reversible.[9] Although various pathophysiological hypotheses for PRES have been proposed, including hypertension, the exact underlying mechanisms remain elusive. There is a suggested overlap in the

pathophysiology of PRES, contrast-induced encephalopathy, and trans-cortical blindness.[6,23] This report presents a case of contrast-induced PRES following diagnostic angiography of the vertebral artery, where a basilar artery aneurysm was diagnosed. Despite the initial occurrence of PRES during diagnostic angiography, the patient subsequently underwent successful coil embolization of the aneurysm without any recurrence of PRES.

CASE DESCRIPTION

A 76-year-old woman was referred to our department for a basilar artery aneurysm. She had no neurological symptoms or significant past medical history, including hypertension, immunosuppressive therapy, or renal disease. The aneurysm had enlarged to 4 mm over 5 years, prompting a diagnostic angiography of the basilar artery. Upon admission, her systolic blood pressure rose to 160 mmHg, higher than her usual range of 120–140 mmHg at home. She was then diagnosed with possible white-coat hypertension.

During the diagnostic cerebral angiography of the bilateral vertebral artery, a 2.7 × 5.6 mm basilar artery aneurysm was detected [Figure 1a]. A total of 30 mL of iopamidol (Iopamidol, 300 mg I/mL, Nichi-Iko, Japan) were used. Her systolic blood pressure spiked to 240 mmHg after the iopamidol injection, and intravenous nicardipine was administered. Post-angiography, her blood pressure stabilized between 130 and160 mmHg, and she showed no neurological symptoms.

Four hours after the angiography, she became disoriented and experienced bilateral light perception. Computed tomography (CT) and magnetic resonance imaging (MRI) scans showed no signs of intracranial hemorrhage, extravasation of contrast medium, or stroke [Figures 1b-d]. However, she then developed complete bilateral blindness, and her blood pressure rose to 180 mmHg. Given the hypertension during angiography and sudden neurological deficits, including bilateral blindness, PRES was suspected. Intravenous corticosteroids and nicardipine were administered.

Twenty hours post-angiography, her orientation improved, but she remained disoriented to time. Her vision improved to hand motion. MRI revealed bilateral occipital hemisphere edema, especially in the white matter [Figure 2], confirming a diagnosis of PRES. Forty hours after angiography, she fully recovered from her neurological and visual disturbances. She was scheduled for coil embolization of the basilar artery aneurysm and was discharged with a modified Rankin Scale (mRS) score of 0. She was initiated on amlodipine and olmesartan at discharge for blood pressure control.

One month later, she underwent coil embolization under general anesthesia. Her blood pressure was controlled under

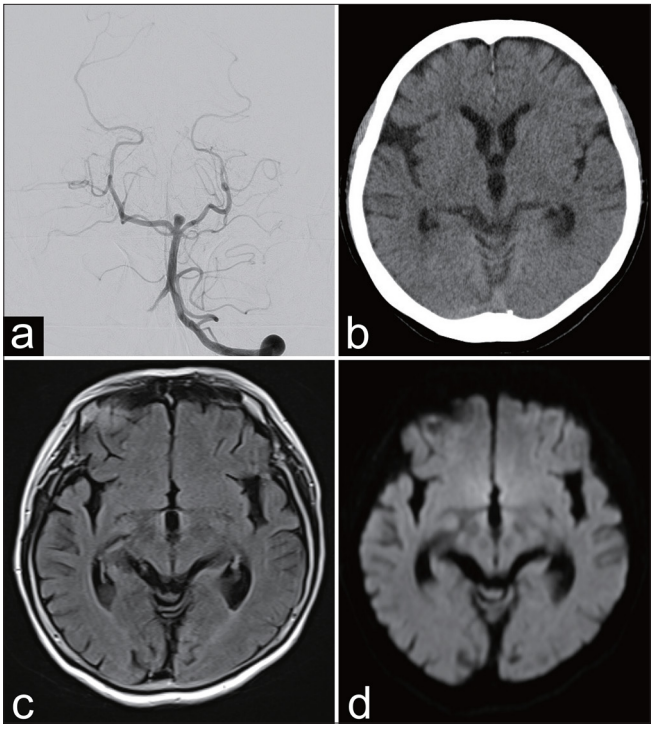

Figure 1: (a) Left vertebral angiography shows a 2.7 × 5.6 mm basilar artery aneurysm. (b) Computed tomography 4 h post-angiography reveals no hyperdense areas in the posterior hemisphere. (c) Fluid-attenuated inversion recovery imaging scan shows no hyperintense regions. (d) Diffusion-weighted imaging scan show no hyperintense regions.

120 mmHg during the procedure. A total of 93 mL of iohexol (Omnipaque, 300 mg I/mL, GE Healthcare, Japan) were used. The aneurysm was successfully embolized [Figures 3a and b] without any blood pressure spikes. Post-anesthesia, she had no neurological deficits, including visual disturbances. Post-embolization MRI showed no parieto-occipital edema [Figures 3c and d]. She was discharged 4 days after treatment with an mRS score of 0.

DISCUSSION

We present a case of PRES triggered by a spike in blood pressure following contrast medium injection during diagnostic angiography. Notably, even though a larger volume of contrast medium was used during subsequent coil embolization of the aneurysm, PRES did not recur.

PRES is a neurological disorder characterized by specific clinical and radiological features. The typical clinical symptoms of PRES include headaches, altered consciousness, visual disturbances, and seizures.[11,26] Radiologically, PRES is commonly associated with vasogenic edema, predominantly affecting the parieto-occipital white matter.[2,12] MRI is particularly useful for diagnosing PRES, with T2-weighted imaging and fluid-attenuated inversion recovery images

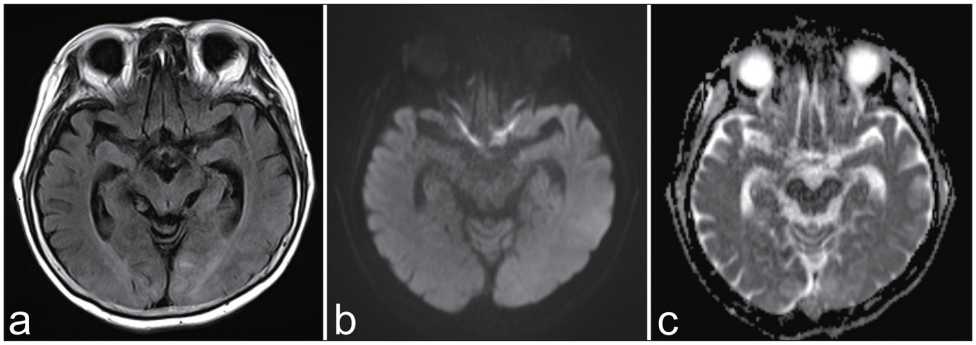

Figure 2: (a) Fluid-attenuated inversion recovery (FLAIR) scan 20 h post-diagnostic angiography, displaying hyperintense areas in the white matter of the parieto-occipital region. (b) Diffusion-weighted imaging reveals no parieto-occipital changes. (c) Apparent diffusion coefficient map revealing hyperintensity in the same area as seen on the FLAIR scan.

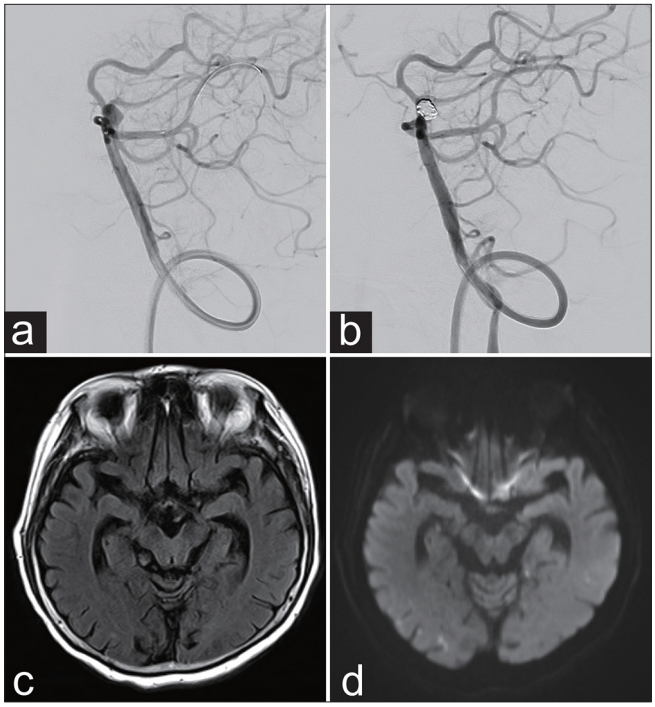

Figure 3: (a) Pre-embolization and (b) post-embolization angiography confirming successful embolization of the basilar aneurysm. (c) Post-embolization fluid-attenuated inversion recovery image showing no abnormalities in the posterior hemisphere. (d) Diffusion-weighted imaging scan revealed a spot of hyperintensity in the posterior lobe.

typically showing hyperintense regions in the posterior hemisphere. These regions exhibit low or isointense signals on diffusion-weighted imaging and high signal intensity on apparent diffusion coefficient maps.[2] These features are indicative of vasogenic edema, which is distinct from the patterns seen in acute cerebral infarction.[2] These clinical and radiological symptoms are usually fully reversible within days to weeks after removing the inciting factor and controlling blood pressure.[11,13] These clinical and radiologic features are consistent with our presented cases, then this patient was diagnosed with PRES.

A wide range of medical conditions have been implicated as causes of PRES, with common risk factors including hypertension, immunosuppressive therapy, renal disease, and eclampsia.[8,25] Hypertension is particularly closely related to the pathogenesis of PRES. The proposed mechanism suggests that hypertension can lead to disordered cerebral autoregulation and endothelial dysfunction. This, in turn, results in brain hyperperfusion and the extravasation of fluid and blood components.[4] It is hypothesized that rapid rises or fluctuations in blood pressure may be more significant than the absolute blood pressure levels.[10,22] In some cases, a hypertensive crisis may precede the neurological syndrome by several hours or more.[4] In our case, hypertension exceeding 240 mmHg immediately after the injection of contrast medium preceded the neurological symptoms by 4 h. This clinical course suggests that the contrast medium injection triggered the hypertensive spike, leading to neurological symptoms consistent with PRES.

Diseases that present symptoms similar to PRES include contrast-induced encephalopathy and transient cortical blindness. Contrast encephalopathy typically shows characteristic CT findings, such as abnormal cortical contrast enhancement and edema, subarachnoid contrast enhancement, and striatal contrast enhancement, especially if the CT scan is performed early.[21] The prevailing hypothesis is that these symptoms result from contrast medium extravasation due to increased vessel permeability.[8,9] The most commonly reported clinical presentation of contrast-induced encephalopathy is transient cortical blindness, occurring in 0.3–1% of patients undergoing vertebral angiography[28] and in 3% of patients receiving endovascular treatment for posterior circulation aneurysms.[21] Other neurological symptoms can mimic stroke, such as hemiplegia, aphasia, and seizures.[15] Contrast-induced encephalopathy is

known to have a benign clinical course, with neurological recovery typically occurring within several hours to 2–3 days.[14]

Therefore, the clinical course of contrast-induced encephalopathy with transient cortical blindness can sometimes resemble that of PRES, and recent reports indicate that MRI findings of transient cortical blindness and PRES can also be similar.[23] Some case reports have suggested a potential overlap between contrast-induced encephalopathy with transient cortical blindness and PRES.[5,6] In our case, the hypertensive spike and subsequent neurological symptoms occurred following the injection of contrast medium. This suggests that the case also had characteristics of contrast-induced encephalopathy. However, there were no findings of contrast medium extravasation on early CT scans. Therefore, we diagnosed this case as contrast-induced PRES. This case strongly supports the hypothesis that there is an overlapping pathogenesis between contrast-induced encephalopathy and PRES. The pathological relationships between PRES, contrast-induced encephalopathy, transient cortical blindness, and this case are depicted in Figure 4.[5,6,9,12,14] We searched for possible cases of contrast-induced PRES following cerebral angiography that met the following criteria: Patients presenting with visual impairment or altered consciousness after angiography, no signs of contrast medium extravasation identified on early CT scans, and lesions in the occipital lobe evident on MRI [Table 1].[3,5,17,21,23,24,27] These cases suggest that contrast-induced PRES can occur even with a small volume of contrast agents. Symptoms typically start within 12 h after the injection of the contrast agent. Resolution of symptoms may take up to a month, but nearly complete recovery has been reported in all cases. The underlying mechanism of contrast-induced PRES remains unclear. However, we can infer potential mechanisms based on similarities to related conditions such as transient cortical blindness and contrast-induced encephalopathy. Both conditions are considered to involve disruption of the blood–brain barrier (BBB) through endothelial injury and osmotic changes caused by contrast agents.[18,23] In contrast, PRES is typically attributed to mechanical damage and subsequent breakdown of the BBB due to hypertension, particularly in the occipital region.[20,30] The common underlying feature of these conditions is disruption of the BBB. Therefore, we hypothesize that contrast-induced PRES may involve both osmotic and endothelial disruption caused by contrast media as well as mechanical damage resulting from elevated blood pressure.

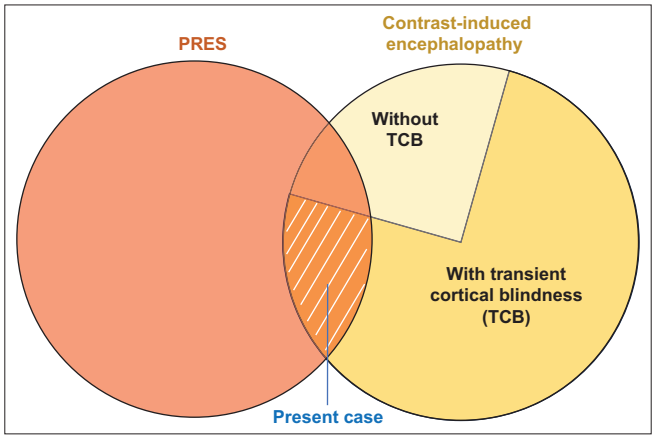

Figure 4: The pathological relationships between PRES, contrast-induced encephalopathy, transient cortical blindness, and this case. CT: Computed tomography, MRI: Magnetic resonance imaging, PRES: Posterior reversible encephalopathy syndrome.

In our case, the initial diagnostic angiography triggered PRES, but subsequent endovascular therapy was completed without a recurrence of PRES. PRES recurs in approximately 5–10% of cases, more frequently in patients with uncontrolled hypertension than in those with other triggers such as immunosuppressive therapy or renal failure.[16] In our case, hypertension and contrast medium use were the likely triggers for PRES during the initial angiography. To manage this, we maintained normotension under general anesthesia during subsequent endovascular therapy and switched to a different contrast medium to mitigate the risk of recurrent PRES. There is currently no clear evidence suggesting that changing the contrast medium reduces the risk of recurrent PRES. However, recent evidence indicates that adverse effects and allergic reactions associated with contrast media can be more effectively prevented by switching the contrast medium rather than using premedication such as steroids.[1,19,29] In addition, some reports have demonstrated successful prevention of recurrent contrast-induced encephalopathy by changing the contrast agent.[7] Therefore, we decided to switch the contrast medium in this patient. A total of 30 mL of contrast medium was used during the first diagnostic angiography, while 93 mL was used during the endovascular treatment. Larger doses of contrast medium were considered to be a risk factor for contrast-induced encephalopathy.[31] Considering the pathological overlap between contrast-induced encephalopathy and PRES, higher doses of contrast medium may trigger PRES. In cases where contrast-induced PRES occurred during diagnostic angiography, subsequent endovascular treatments have sometimes been canceled.[27] However, our case successfully underwent endovascular treatment without recurrence of PRES. This fact demonstrates that even in patients who develop contrast-induced PRES with a small amount of contrast medium, appropriate management can enable the successful completion of more extensive endovascular treatments.

Table 1: Possible cases of contrast-induced PRES after cerebral angiography.

	Age/ gender	Arteriography	Procedure	Contrast agent	Volume	Onset time	Presentation	MRI findings	Clinical resolution
Saigal *et al.* (2004)[23]	45/F	VA	Coiling for the right SCA An	iohexol	NA	12 h	Confusion, cortical blindness	Bilateral occipital	7 days
Saigal *et al.* (2004)[23]	73/F	CA	Diagnosis for left ICA An	iohexol	NA	20 min	Cortical blindness	Bilateral occipital	5 days
Niimi *et al.* (2008)[21]	54/F	VA	Coiling for basilar An	Non-ionic	62 mL	3 h	Cortical blindness	Bilateral occipital	1 month
Lo *et al.* (2015)[17]	57/M	VA	Post-embolization check for left PICA An	iohexol	20 mL	immediately	Cortical blindness	Bilateral occipital	1 day
Baguma *et al.* (2017)[3]	58/F	VA	Coiling for Right VA An	iodixanol	98 mL	immediately	Cortical blindness	Right occipital	6 days
Das *et al.* (2018)[5]	54/F	VA	Post-embolization check for vertebrobasilar An	iohexol	NA	immediately	Confusion, seizure, cortical blindness	Bilateral occipital	NA (improved completely)
Tong *et al.* (2018)[27]	61/F	CA, VA	Diagnosis for left ICA An	iohexol	NA	Immediately after contrast agent injection into the VA	Left hemiparesis, cortical blindness	Right occipital	3 months
Spiriev *et al.* (2022)[24]	70/F	VA	Coiling for basilar An	Non-ionic, iso-osmolar	200 mL	immediately	Cortical blindness	Bilateral occipital	3 days
Present case	76/F	VA	Diagnosis for basilar An	Iopamidol	30 mL	4 h	Confusion, cortical blindness	Bilateral occipital	2 days

An: Aneurysm, CA: Carotid artery, ICA: Internal carotid artery, MRI: Magnetic resonance imaging, NA: Not available, PICA: Posterior inferior cerebellar artery, PRES: Posterior reversible encephalopathy syndrome, SCA: Superior cerebellar artery, VA: Vertebral artery

CONCLUSION

We presented a case of contrast-induced PRES. The clinical history strongly suggests a pathological overlap between contrast-induced encephalopathy, transient cortical blindness, and PRES. Despite the initial PRES being induced by a small volume of contrast medium during diagnostic angiography, the patient successfully underwent endovascular therapy with a larger volume of contrast medium. This was achieved through careful blood pressure management and switching the type of contrast medium used.

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