A Case of Unilateral Posterior Reversible Encephalopathy Syndrome Occurring after Carotid Artery Stenting

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Objective: A case that developed unilateral posterior reversible encephalopathy syndrome (PRES) after carotid artery stenting (CAS) is reported.

Case Presentation: The patient was a 79-year-old man who underwent CAS for symptomatic right internal carotid artery stenosis. Left hemiparesis and unilateral spatial neglect appeared on the day after the procedure. MRI presented findings characteristic of PRES although unilaterally. Temporary disruption of the blood–brain barrier due to blood pressure fluctuations in chronic unilateral hypoperfusion caused by stenosis of the right internal carotid artery or toxicity of the contrast medium was suspected.

Conclusion: PRES is considered to be a complication worth attention in CAS.

Keywords: posterior reversible encephalopathy syndrome, carotid artery stenting, endovascular treatment

Introduction

Posterior reversible encephalopathy syndrome (PRES) is a condition that exhibits clinical symptoms such as headache, convulsion, disturbance of consciousness, and visual abnormalities and characteristic imaging findings, which are primarily posterior white matter lesions, and takes a reversible course. We encountered a patient who developed unilateral PRES after carotid artery stenting (CAS). This rare case, in which PRES appeared unilaterally and exhibited relatively mild symptoms, is reported.

Case Presentation

The patient was a 79-year-old male who complained of weakness of the left upper and lower extremities. He suffered cerebral infarction with weakness of the left upper and lower extremities as a presenting symptom, was found by a local physician to have narrowing at the origin of the right internal carotid artery, and was referred to us.

On arrival, MRI diffusion-weighted imaging (DWI) and FLAIR showed fresh foci of infarction diffusely distributed in the right watershed area and fronto-parieto-occipital cortex (Fig. 1). Vulnerable plaque was suspected on black blood T1-weighted imaging of carotid artery MRI (Fig. 2A), and CT perfusion images provided by the physician who had examined him showed reduced cerebral blood flow (CBF) of the right hemisphere. On intracranial angiography, 86% stenosis by the North American Symptomatic Carotid Endarterectomy Trial method was observed (Fig. 2B).

Oral administration of aspirin at 100 mg/day and cilostazol at 200 mg/day was initiated on the day of admission, and CAS was performed 6 weeks after the onset of cerebral infarction by avoiding the acute period. Since ischemic tolerance was expected to be low, the lesion was approached via the right femoral artery under general anesthesia and lower extremities as a presenting symptom, was found by a local physician to have narrowing at the origin of the right internal carotid artery, and was referred to us.

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Blood flow was arrested over a total of 10 minutes at predilation and stent deployment and for 7 minutes at postdilation, and 80 mL of blood was aspirated at each time, but the amount of debris contained in the aspirated blood was very small. As a contrast medium, 80 mL of Iopamiron 300 (Bayer, Osaka, Japan) was used, and the systolic blood pressure, which decreased to 84 mmHg immediately after stent deployment, temporarily increased to a maximum of 162 mmHg immediately after the procedure.

The state of consciousness immediately after the procedure was satisfactory, there was no exacerbation of headache or motor paralysis, but somewhat euphoric speech and behavior were noted. While the systolic blood pressure was maintained at 120–140 mmHg, mild left hemiparesis and left unilateral spatial neglect were observed the next morning. MRI DWI showed no clear areas of infarction (Fig. 3A), FLAIR imaging revealed diffuse hyperintense lesions from the right fronto-parieto-occipital cortex to the subcortical white matter (Fig. 3B). On gadolinium (Gd) contrast-enhanced T1-weighted imaging, contrast enhancement was observed in limited areas of the right frontoparietal cortex, but no tumor was detected (Fig. 3C), and MRI perfusion images were negative for hyperperfusion (Fig. 3D). From these clinical symptoms and imaging findings, a diagnosis of PRES was made despite the unilaterality of the lesions, and conservative treatment including the blood pressure control, anticonvulsant medication...
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2,3) Our patient showed no abnormal signals on DWI images, hyperintensities on FLAIR images, and reversible clinical symptoms as findings consistent with PRES but was non-typical in that the lesions were unilateral and also involved the anterior circulation. PRES is considered likely to occur in the posterior circulation because the vertebrobasilar system is more prone to the effects of blood pressure fluctuations and is more likely to develop disruption of the blood–brain barrier as it is less densely innervated by the sympathetic nervous system than the anterior circulation. 3) However, in addition to the classic pattern, in which the bilateral parieto-occipital cortex and white matter are damaged, Bartynski et al. 4) suggested the presence of a type of PRES with lesions distributed along the vasculature and marked impairment of the superior frontal gyrus, and lesions may also occur in the anterior circulation or unilaterally if there are factors that contribute to disruption of the blood–brain barrier. 3) A small number of cases of unilateral PRES have been reported, including those in which the disease was induced by hypertension 5–7) or ventriculoperitoneal shunt 8) in the presence of unilateral cerebral vasospasm or brain tissue injury after subarachnoid hemorrhage as a background factor and one in which

(levetiracetam at 1000 mg/day), and rehabilitation was continued. As the symptoms were gradually alleviated on a weekly basis, the patient was discharged 3 weeks after the procedure, being capable of unassisted ambulation. On a review of serial changes in FLAIR images, the condition recovered nearly to the preprocedural state 5 weeks after the procedure (Fig. 4).

Discussion

PRES is a syndrome diagnosed based on characteristic imaging findings and clinical symptoms including headache, convulsion, disturbance of consciousness, and visual abnormalities and a reversible course in the presence of a number of background factors such as hypertensive encephalopathy, use of immunosuppressants, blood diseases, and drugs. Its primary pathology is considered to be impairment of CBF autoregulation or vascular edema induced by disruption of the blood–brain barrier due to vascular endothelial injury. 1) The lesions are mostly symmetric, most often affect the parietal and occipital lobes, and exhibit characteristics such as hyperintensity on FLAIR images but no abnormal signals on DWI images with an elevation of the apparent diffusion coefficient (ADC) value. 2) However, there are often non-typical findings such as microbleedings and partial contrast enhancements with occasional hyperintensity on DWI images and a decrease in the ADC value. 2,3) Our patient showed no abnormal signals on DWI images, hyperintensities on FLAIR images, and reversible clinical symptoms as findings consistent with PRES but was non-typical in that the lesions were unilateral and also involved the anterior circulation. PRES is considered likely to occur in the posterior circulation because the vertebrobasilar system is more prone to the effects of blood pressure fluctuations and is more likely to develop disruption of the blood–brain barrier as it is less densely innervated by the sympathetic nervous system than the anterior circulation. 2,3) However, in addition to the classic pattern, in which the bilateral parieto-occipital cortex and white matter are damaged, Bartynski et al. 4) suggested the presence of a type of PRES with lesions distributed along the vasculature and marked impairment of the superior frontal gyrus, and lesions may also occur in the anterior circulation or unilaterally if there are factors that contribute to disruption of the blood–brain barrier. 3) A small number of cases of unilateral PRES have been reported, including those in which the disease was induced by hypertension 5–7) or ventriculoperitoneal shunt 8) in the presence of unilateral cerebral vasospasm or brain tissue injury after subarachnoid hemorrhage as a background factor and one in which

| Fig. 2 | (A) A black blood T1-weighted MRI reveals hyperintense plaque in the right internal carotid artery. (B) A pre-operative right carotid angiogram shows severe stenosis of the right internal carotid artery. (C) A post-procedural angiogram shows resolution of the stenosis. |
the disease was induced by the use of immunosuppressants associated with organ transplantation in a patient with a history of chronic occlusion of the left middle cerebral artery. In these reports, new vascular endothelial injury and blood pressure fluctuations in chronic unilateral hypoperfusion were considered to be factors of temporary disruption of the blood–brain barrier and the development of PRES. Our patient also had hypoperfusion of the right hemisphere due to marked stenosis of the internal carotid artery or vascular endothelial injury caused by cerebral infarction in the background, and disruption of the blood–brain barrier was considered likely to be induced even in the anterior circulation by rapid intraprocedural changes in the blood pressure in a state of reduced autoregulation of the cerebral circulation.

There have been a number of reports that transient neurological symptoms and abnormal images were observed after cerebral angiography or intracranial endovascular surgery similar to our patient, with some having unilateral lesions. Adverse reaction to the contrast material is considered to be the primary cause of these events because there was a tendency such as that the contrast agent was used in a large amount, generally 200–300 mL or more or was used without dilution. Contrast-induced encephalopathy can occur when a large amount of a contrast agent is administered in a short period into the same vessel for cerebral angiography or intracranial endovascular treatment. Neurological symptoms, such as headache, convulsion, and cortical blindness, often appear during or immediately after the procedure but are resolved within a few days. These symptoms are considered to be ascribed to localized brain edema induced by disruption of the blood–brain barrier due to the toxicity of the contrast agent and its leakage into the subarachnoid space. The diagnosis is based on confirmation of high density areas indicating leakage of the contrast agent in the subarachnoid space by CT or detection of the iodine-based contrast agent in the cerebrospinal fluid. In our patient, no finding suggestive of leakage of the contrast agent could be obtained because no high density area was observed on CT on the day after the procedure, and the cerebrospinal fluid examination was not performed. CT findings indicating leakage of the contrast

![Fig. 3 MRIs 1 day post-CAS. (A) DWI shows almost no abnormalities. (B) FLAIR image shows extensive hyperintense lesions in the right front-parieto-occipital region predominantly involving cortical and subcortical white matter. (C) Post-contrast T1-weighted image shows infinitesimal enhancement lesions (white arrows). (D) Perfusion MRI image shows mild hypoperfusion in the right cerebral hemisphere. CAS: carotid artery stenting; DWI: diffusion-weighted image]
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Contrast medium is suspected to be involved as in our patient from contrast-induced encephalopathy. Other diseases that should be differentiated from PRES include encephalitis, malignant brain tumors, hyperperfusion syndrome, transient cerebral ischemia due to intraprocedural devascularization, and cerebral infarction due to scattering of thromboembolus. In our patient, we considered encephalitis unlikely as there were no fever or blood test results suggestive of inflammation. Hyperperfusion syndrome often occurs within several hours to a few days after the procedure and is diagnosed on the basis of an increased CBF by techniques capable of evaluation of the cerebral perfusion such as SPECT and positron emission tomography (PET). In our patient, the symptoms may have been induced by toxicity of the contrast medium because we repeatedly performed angiography to check if there was plaque shift after stent placement, but the dose of the contrast medium (80 mL) was not very large. We diagnosed the condition as PRES, considering that the symptoms triggered by intraprocedural blood pressure changes in the presence of impaired autoregulation of the cerebral circulation and vascular endothelial injury as background factors rather than were caused by the contrast medium alone. However, the pathologies of PRES and contrast-induced encephalopathy have temporary impairment of the brain parenchyma due to disruption of the blood–brain barrier in common, and it is difficult to clearly distinguish PRES in which the contrast medium is suspected to be involved as in our patient from contrast-induced encephalopathy.

Fig. 4 Sequential changes on MRIs in this case. (A) FLAIR images on admission show infarction in the right front-parieto-occipital region. (B) MRIs 1 day post-CAS show PRES in the ipsilateral hemisphere. Follow-up MRIs at 3 weeks (C) and 5 weeks (D) post-CAS show gradually improvement in abnormalities. PRES: posterior reversible encephalopathy syndrome; CAS: carotid artery stenting.
were inconsistent with hyperperfusion. In addition, as the symptoms appeared about half a day after the procedure, the possibility of transient cerebral ischemia due to intraprocedural flow arrest was considered negative, and subsequent changes in imaging findings also excluded de novo cerebral infarction or brain tumor.

Conservative treatment such as the blood pressure control is a common approach to PRES, but as convolution might occur in not only PRES but also encephalitis, hyperperfusion syndrome, and cerebral infarction, which are its differential diagnoses, and exacerbate the condition, we treated our patient by the concomitant use of anticonvulsants with a favorable outcome.

Conclusion

We encountered a possible case of unilateral PRES that occurred after CAS. In CAS for symptomatic internal carotid artery stenosis, the condition is often complicated by chronic hemispheric hyperperfusion or vascular endothelial cell injury following cerebral infarction, and the risk of induction of PRES even by the administration of a smaller dose of a contrast medium must be remembered.

Disclosure Statement

There are no conflicts of interest to disclose regarding this paper.

References