Contrast-Induced Encephalopathy after Endovascular Treatment: Two Case Reports

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Abstract
Contrast-induced encephalopathy (CIE) is a rare neurological complication that occurs after the use of contrast medium in various angiographic procedures. Symptoms can be different, from headache to severe neurological deficit and coma. In the articles published to date, symptoms appeared immediately after application of contrast agent or within 24 hours. Here we present two cases of patients in whom CIE developed delayed after endovascular treatment.

Keywords
- case
- cerebral angiography
- contrast-induced encephalopathy
- endovascular treatment
- iodinated contrast

Introduction
Contrast-induced encephalopathy (CIE) is characterized by the appearance of a new neurological deficit after intravenous or intra-arterial administration of iodinated contrast agent. The incidence of CIE ranges between 0.3 and 2.4%. The mechanism of CIE was previously explained as the disruption of the blood–brain barrier (BBB), hyperosmolarity, and neurotoxicity from the contrast medium.

Although cortical enhancements and hyperintensities in the subarachnoid space are visible on a computerized tomography (CT), the primary diagnostic tool is magnetic resonance imaging (MRI), where hyperintense areas can be seen on a T2-weighted, fluid-attenuated inversion recovery (FLAIR), and diffusion-weighted imaging (DWI). Diagnostic coronary angiography images of an MRI of the brain can help differentiate CIE from cerebral ischemia.

The symptoms can range from a mild headache to a pronounced neurological deficit, such as aphasia, hemiparesis, and coma. Treatment consists of the use of corticosteroids, mannitol, and hydration. In some patients, the symptoms disappear within 24 to 48 hours, but in rare cases, they can last up to 2 weeks. Although the treatment outcome is generally favorable, fatal cases have also been described in the literature.

Herein, we present two case patients who developed CIE after endovascular treatment.

Case 1
A 64-year-old patient presented to our outpatient clinic with MRI findings. She stated that a few months ago, she had frequent headaches. The patient had a medical history of...
hypertension. There was no known family history of a bleeding disorder. The neurological examination was without focal neurological signs. The MRI and CT angiography of the brain showed an aneurysm of the left internal carotid artery (ICA; Fig. 1). Digital subtraction angiography (DSA) was performed to clarify and assess the aneurysm's anatomy and morphology. During DSA, a total of 60 mL of the nonionic contrast medium iopamidol at an iodine concentration of 370 mgI/mL (Iopamiron 370; Schering, Osaka, Japan) was used.

After interdisciplinary discussion, coil embolization was successfully performed using a transradial approach. The intraoperative and early postoperative course were uneventful. Five days after operation, the patient was discharged from the hospital fully conscious and without any neurological deficit (modified Rankin Scale score: 0).

A follow-up MRI of the brain conducted 2 weeks after intervention showed a hyperintense area in the T2-weighted image in the left occipital lobe (Fig. 2).

Given that the patient had no symptoms, supportive treatment was recommended. Two weeks later, the patient developed weakness and dysesthesia of the right side as well as numbness in the IV and V fingers.

An urgent MRI of the brain showed increased hyperintensity on T2 and FLAIR (Fig. 2). The apparent diffusion coefficient had not changed. A diagnosis of contrast-induced encephalopathy was made.

In addition to adequate hydration, corticosteroid therapy was started, after which the patient's clinical neurological status improved.

Fig. 1 Three-dimensional reconstruction images of computed tomography angiography showing an aneurysm of the left internal cerebral artery.

Fig. 2 Brain magnetic resonance imaging at the 2 weeks (A–C) and 2 months follow-up (D–F). ADC, apparent diffusion coefficient; FLAIR, fluid-attenuated inversion recovery.
Upon discharge from the hospital, the patient completely recovered from sensory dysfunction, but slight weakness of the right-hand grip remained.

**Case 2**

A 75-year-old male patient was admitted to our hospital for endovascular treatment of an ICA aneurysm (Fig. 3). The patient had two aneurysms: a basilar artery-superficial cerebellar artery (BA-SCA) aneurysm and an ICA aneurysm. He underwent stent embolization of the BA-SCA aneurysm 3 years earlier in another hospital.

Upon examination, the patient was alert, cranial nerves were intact, and no sensorimotor neurological deficits were present.

The medical history includes hypertonia and hyperlipidemia. Laboratory findings at the time of admission were in the normal range.

After admission, he underwent cerebral angiography, resulting in successful occlusion of the right ICA aneurysm with endovascular coiling using a transfemoral approach.

During DSA, a total of 60 mL of contrast-medium iopamidol (Iopamiron 370; Schering, Osaka, Japan) was used. The intraoperative and early postoperative clinical courses were uncomplicated.

On the 5th day after endovascular treatment, the patient developed weakness, numbness, and involuntary movements on the left side.

The urgent MRI of the brain showed a hyperintense signal in the right parietal lobe on the DWI (Fig. 4A). The patient was admitted to the hospital with a suspected cerebral infarction. Atrial fibrillation was observed on the electrocardiogram during a routine clinical examination, and Eliquis therapy was initiated after ablation. The patient was discharged home after a few days.

At the follow-up MRI after 3 months, it was observed that the hyperintense area had increased and spread to the right frontal and temporal lobes (Fig. 4B).

Given that the patient had no new neurological symptoms, supportive treatment was continued. After a month, the patient came to the emergency department due to vertigo and was admitted to the cardiology department with a diagnosis of sinus sick syndrome. Given that the hyperdense area in the MRI scan continued to increase, a biopsy of the lesion was performed. Pathohistological findings showed a granuloma, suggesting an allergic reaction to the stent.

After an interdisciplinary consultation, therapy with prednisolone 60 mg per day was started, which was considered effective and was gradually reduced. A follow-up MRI 1 month after the introduction of corticosteroid therapy showed reduced hyperintense areas (Fig. 4C). In addition, the patient’s neurological status slightly improved. The vertigo has completely disappeared, but the patient still has numbness in her lower left leg.

**Discussion**

CIE is a rare transient phenomenon that occurs after the administration of contrast media during endovascular
BBB should be impermeable to contrast agents. Cerebral autoregulatory dysfunction can also cause damage and causes dehydration of the endothelial cells. Disruption of administration of a contrast agent, in some cases, there is a angiography.

CIE occurs 0.5 to 18 hours after contrast agent administration. Diagnosis of CIE can be very challenging, especially when symptoms appear delayed after the procedure. Therefore, it is important to perform a thorough diagnostic workup to rule out differential diagnoses. A differential diagnosis can include posterior reversible encephalopathy syndrome, reperfusion syndrome, recurrent ischemic stroke, or subarachnoid hemorrhage.

Typical radiological CIE findings include cerebral edema and cortical enhancement. MRI is the gold standard for diagnosis and differentiation from other pathologies. The posterior cerebral circulation is the most sensitive to potential damage due to its sympathetic innervation. A previous stroke can disrupt the BBB and affect the leakage of the contrast agent, which can cause tissue reaction and cerebral edema.

Table 1 shows some reported risk factors of CIE:

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<th>Risk factors</th>
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<td>Chronic hypertension</td>
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<td>Diabetes mellitus</td>
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<td>Renal dysfunction</td>
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<td>Administration of large volumes of iodinated contrast</td>
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<td>History of stroke</td>
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<td>Percutaneous coronary intervention</td>
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<td>Selective angiography of internal mammary grafts</td>
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<tr>
<td>Previous adverse reaction of iodinated contrast</td>
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<td>Posterior cerebral circulation</td>
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Abbreviation: CIE, contrast-induced encephalopathy.

Authors’ Contributions
D.J. was involved in collection of the data, analysis of the results, and the writing of the manuscript. R.T., K.S., K.M., S.C., M.N., and T.T. were involved in collection of the data and analysis of the results. F.K., Y.Y., and Y.K. supervised the findings of this work. All authors reviewed the results and approved the final version of the manuscript.

Conflict of Interest
None declared.

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