

Iodinated Contrast Encephalopathy after Coil Embolization of Unruptured Aneurysms

Abstract

Iodinated contrast encephalopathy is known as one of the rare complications of cerebral angiography and neurovascular intervention. The mechanism and causes of contrast encephalopathy are not understood well. In this case, we experienced transient neurological deficit following coil embolization of unruptured aneurysms. A 67-year-old woman with two unruptured aneurysms of internal carotid artery underwent endovascular coil embolization. During the procedure, she presented transient hemiparesis and aphasia. After the treatment, she presented the symptom again, and computed tomography scan showed cortical edema and subarachnoid leakage of contrast agent. Magnetic resonance imaging excluded cerebral infarction. The neurological symptom resolved completely within 24 h after the treatment, and she was diagnosed as the contrast encephalopathy. In our case, we speculated that contrast encephalopathy has occurred subsequently to the transient cerebral ischemia. Iodinated contrast encephalopathy may have induced with neurovascular intervention following cerebral ischemia and increased use of contrast agent. We should consider iodinated contrast encephalopathy when a patient presents hemiparesis and aphasia such as that caused by acute stroke during neurovascular intervention.

Keywords: Coil embolization, contrast encephalopathy, unruptured aneurysm

Introduction

Iodinated contrast encephalopathy is a rare complication of cerebral angiography and neurovascular intervention.^[1] Neurotoxicity from iodinated contrast agents leads to neurological deficits, which resemble acute stroke and transient ischemic attack (TIA).^[2] However, the mechanism of contrast encephalopathy is not clear. In this case, our patient experienced a cerebral ischemia caused by mechanical distortion of tortuous arteries, called the accordion effect, before the contrast encephalopathy. Hence, iodinated contrast encephalopathy may have induced with cerebral ischemia and increased use of contrast agent.

Case Report

A 67-year-old woman with a history of hypertension and two unruptured aneurysms, located at the anterior choroidal artery (4.5 mm) and at the posterior communicating artery (2.7 mm), underwent endovascular coil embolization. She had previously undergone cerebral angiography from the left common carotid artery (CCA)

without an adverse reaction. Angiography showed no collateral circulation from the anterior and posterior circulating arteries.

The coil embolization procedure was started with a femoral puncture. A guidewire was inserted into the left internal carotid artery (ICA) and extended to the tortuous carotid artery. Subsequently, 8 Fr guiding catheters (Asahi Fubuki®; Asahi Intecc Co., Ltd., Aichi, Japan) were placed in the left CCA, and a 6 Fr intermediate catheter (Cerulean DD6®; Medikit Co., Ltd., Tokyo, Japan) was placed at the cervical C1 level of the ICA with some difficulty. The sharp branching angle and tortuosity of the CCA resulted in resistance to moving the distal access catheter to the ICA.

During the procedure, the patient was restless and had right-sided hemiparesis and aphasia. Angiography from the 6 Fr intermediate catheter showed slow anterograde cerebral flow and retrograde pooling of contrast material at the top of the 8 Fr catheter [Figure 1a]. Further down the catheter, an angiographic image showed folds of the left ICA [Figure 1b]. Angiography showed gradual improvement of the kinks of the

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artery. Right-sided hemiparesis and aphasia persisted for 7 min, and the patient then recovered from the symptom. We changed the 8 Fr guiding catheter to the 6 Fr catheter (Asahi Fubuki®) and continued the procedure. The aneurysm was embolized without any neurological deficits [Figure 1c and d].

Thirty minutes after the procedure, she presented with right-sided hemiparesis and aphasia again. Additional cerebral and cervical angiography did not show occlusion or dissection of the arteries. A computed tomography (CT) scan showed cortical edema of the left cerebral hemisphere and contrast agent leakage to the subarachnoid space [Figure 2a]. Diffusion-weighted magnetic resonance imaging (MRI) did not show hyperintensity. Fluctuation of the neurological symptoms continued overnight. We made a diagnosis of contrast encephalopathy and started an antiepileptic drug. On the next day, the neurological symptoms had improved, and a CT scan showed the improvement of cortical edema and contrast washout [Figure 2b]. She was clinically stable and was discharged.

Discussion

In our case, contrast encephalopathy occurred subsequently to transient cerebral ischemic symptoms. Iodinated contrast encephalopathy can resemble acute stroke or TIA.^[1,2] Contrast encephalopathy may have been induced by cerebral ischemia and a short time of using a high concentration of contrast agent.

Iodinated contrast encephalopathy can be induced by cerebral angiography. The most common reported symptom of iodinated contrast encephalopathy is cortical

blindness.^[1] In some cases, transient global aphasia with hemiparesis following angiography has been reported.^[2,3] Our initial diagnosis was acute stroke or dissection of the cervical artery caused by neurovascular intervention. The following CT and MRI did not reveal these findings. In our case, a CT scan of the head showed cortical edema. Subarachnoid leakage of the contrast agent was diminished in a following CT scan, which has been reported in contrast encephalopathy cases.^[1,4] However, a CT scan performed after uneventful endovascular treatment of cerebral aneurysm also showed cortical enhancement and subarachnoid contrast enhancement. Diagnosis of contrast encephalopathy should be made by CT findings and clinical symptoms.^[5]

Iodinated contrast encephalopathy may have been induced by cerebral angiography following cerebral ischemia and subsequent use of a contrast agent in our patient. The mechanisms of contrast encephalopathy are unclear. One possible mechanism is blood-brain barrier (BBB) disruption and leakage of the contrast agent to the cortical cortex and subarachnoid space, which causes neurotoxicity and cortical edema.^[1] The cause of BBB disruption might be cerebral ischemia and the concentration of the contrast agent. Cerebral ischemia and reperfusion result in a fragile BBB, which is attributed to increased permeability of the affected vessel.^[6,7] Another possible mechanism is the short time of use of a high concentration of contrast agent, as suggested in rabbits.^[8]

Cerebral ischemia is caused by mechanical distortion of tortuous arteries, called the accordion effect.^[9] This effect is produced when a blood vessel is compressed during linearization and shortening of a tortuous blood vessel with a mechanical device such as a stiff guidewire or guiding catheter. This effect is well known for complications of percutaneous coronary intervention.^[10] The accordion effect sometimes causes a delay in blood flow and myocardial ischemia. Some cases of the accordion effect during carotid

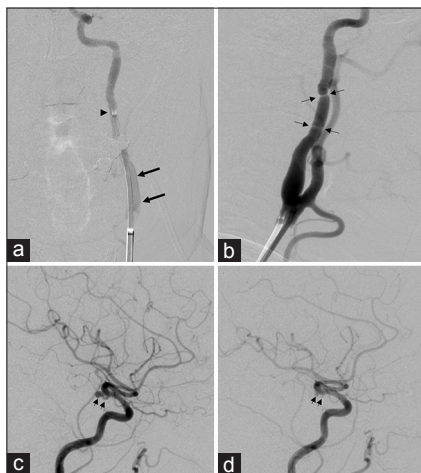


Figure 1: (a) Angiography of the left cervical artery from a 6 Fr intermediate catheter (arrowhead) shows slow anterograde cerebral flow and retrograde pooling of contrast material at the top of the 8 Fr catheter (arrows), (b) lateral subtracted angiography of the left common carotid artery shows folds of the internal carotid artery (arrows), which were formed by linearization of the tortuous vessel during the procedure, (c and d) angiography of the left internal carotid artery before and after the embolization of the aneurysms (arrows)

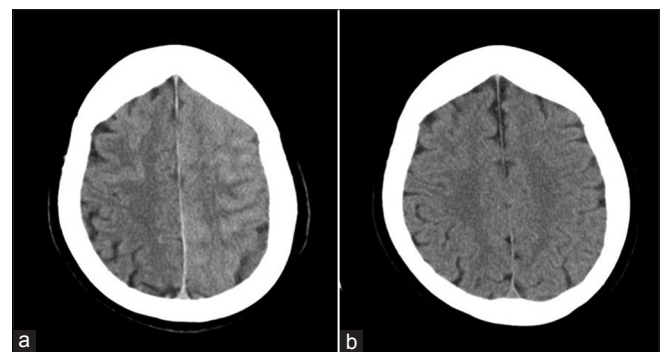


Figure 2: (a) A computed tomography scan after coil embolization shows cortical edema of the left cerebral hemisphere and subarachnoid leakage of the contrast agent, (b) A computed tomography scan on the next day shows that the cortical edema was improved and leakage of the contrast agent to the subarachnoid space was diminished

artery stenting have been reported.^[9] In our case, a tortuous and kinked ICA with poor cerebral collateral circulation from the anterior and posterior communicating arteries caused transient ischemic symptoms early in the procedure, and we continued the procedure. A high concentration of contrast agent was injected from the ICA repeatedly, while angiography performed before this treatment, contrast agent was injected from the CCA, and diluted contrast agent flowed to the ICA without any adverse effects.

This brain ischemia and subsequent repeated use of contrast agent may have led to increased permeability of brain vessels and contrast encephalopathy. Therefore, to prevent iodinated contrast encephalopathy, physicians should avoid cerebral ischemia and additional contrast agent use.

Conclusion

We speculate that iodinated contrast encephalopathy may have been induced by neurovascular intervention following cerebral ischemia in our patient. Iodinated contrast encephalopathy should be considered when a patient presents with hemiparesis and aphasia, such as that caused by acute stroke, during neurovascular intervention.

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Conflicts of interest

There are no conflicts of interest.

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