Contrast-induced encephalopathy following cerebral angiography: A case report

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Abstract. The present study describes the case of patient with contrast-induced encephalopathy following cerebrovascular angiography, and presents the clinical and imaging features, as well as the treatment and prognosis of this patient. Following digital subtraction angiography, cortical blindness and cognitive dysfunction were the main complaints of the patient. The emergency craniocerebral CT scan revealed hyperdense areas in the bilateral cerebellum, thalamus, sulcus and cistern, and a review of the CT scan 24 h following the procedure revealed that the hyperintense lesions were reduced or resolved in these areas. The patient obtained a good prognosis following treatment anti-inflammatory and intracranial pressure reduction treatment. On the whole, the present study demonstrates that cognitive dysfunction may be a clinical manifestation of contrast-induced encephalopathy. Thus, the earlier diagnosis and earlier treatment are crucial for the prognosis of patients.

Introduction

Contrast-induced encephalopathy (CIE) is an acute and reversible transient neurological disorder induced by a contrast agent. This occurs as a rare neurological complication following the intravascular injection of a contrast agent. The incidence of CIE is ~0.3-1%; however, this increases to 4% when hypertonic contrast agents are used (1). The clinical manifestations of CIE vary, such as intracranial hemorrhage, cortical blindness, epileptic seizure, etc.; however, cognitive dysfunction has not been reported to date, at least to the best of our knowledge. The present study describes the case of a patient with cognitive decline as the main clinical manifestation of CIE.

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Case report

The patient described herein was a 57-year-old female, who presented with recurrent headaches for >1 month and was admitted to the Guangdong Second Provincial General Hospital. Although she had a 4-year history of hypertension, she was not taking anti-hypertensive drugs regularly. In 2016, she underwent right internal carotid artery stent implantation and left internal carotid aneurysm embolization in another hospital. Moreover, a left internal carotid artery dissection aneurysm was found without any positive signs upon a hospital physical examination. Subsequently, an auxiliary examination after admission revealed that the creatinine level was 79 μmol/l and the alanine aminotransferase level was 41 U/l. An abdominal ultrasonography revealed fatty liver and slight effusion in the right kidney. Cerebral vascular wall imaging demonstrated the bilateral internal carotid artery with a double lumen in the C1 segment (Fig. 1) and a left internal carotid artery C6 segment aneurysm surgery was performed. A summary of the general information of the patient is presented in Table I.

Nonetheless, bilateral internal carotid artery dissection was not excluded. A cerebral angiography was performed to determine whether there was dissection and whether further interventional treatment was required; the patient then underwent a cerebral angiography with 130 ml iohexol (the iodine concentration of the iohexol was 300 mg/ml) under local anesthesia, which revealed a dissecting aneurysm in the C1 segment of the left internal carotid artery (Fig. 2). The surgery was successfully completed; however, the patient then suffered from headaches, vomiting and visual impairment, with a slight decrease in cognitive function 5 min later. A physical examination revealed that the visual acuity of both eyes had notably decreased, with only light sensation. The emergency craniocerebral CT scan then revealed an increased density in the bilateral cerebellum, thalamus, sulcus and cistern (Fig. 3). The probability of CIE was thus considered, due to the symptoms, signs and CT examination. Thus, the patient was administered anti-emetic (metoclopramide, 10 mg), anti-inflammatory (dexamethasone, 5 mg), vasospasm relief (nimodipine, 10 mg) and intracranial pressure reduction (mannitol, 25 g) treatment. The head CT scan then revealed the resolution of high-density in the aforementioned areas (Fig. 4) at 24 h after the surgery. On the third day after surgery, the visual acuity of the patient had gradually

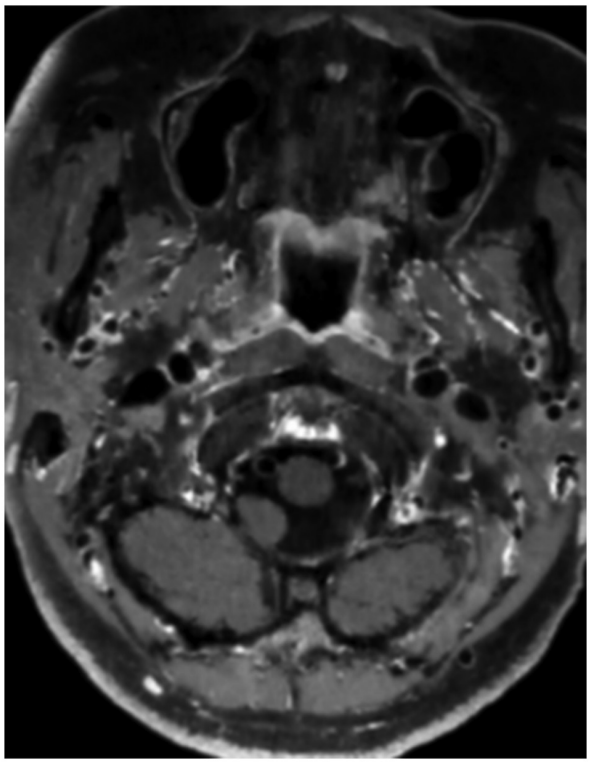

Figure 1. Cerebral vascular wall imaging illustrating a bilateral internal carotid artery with a double lumen in the C1 segment.

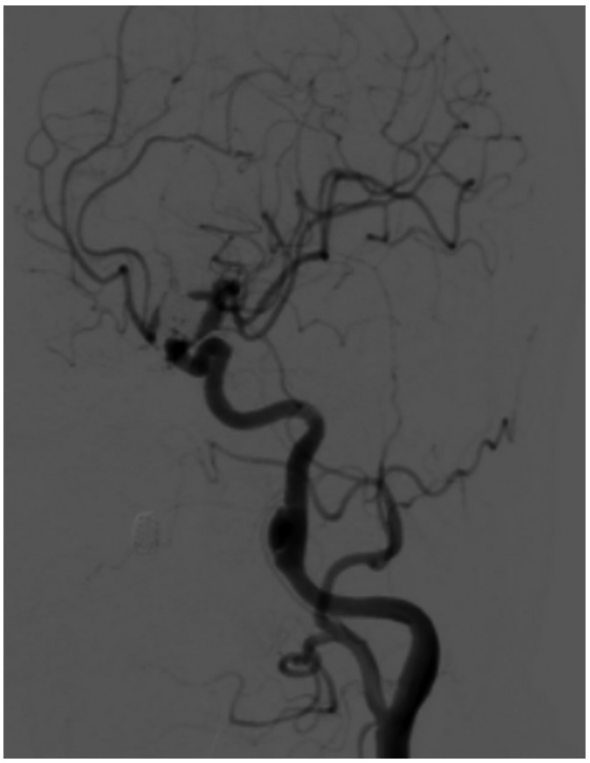

Figure 2. A dissecting aneurysm in the C1 segment of the left internal carotid artery.

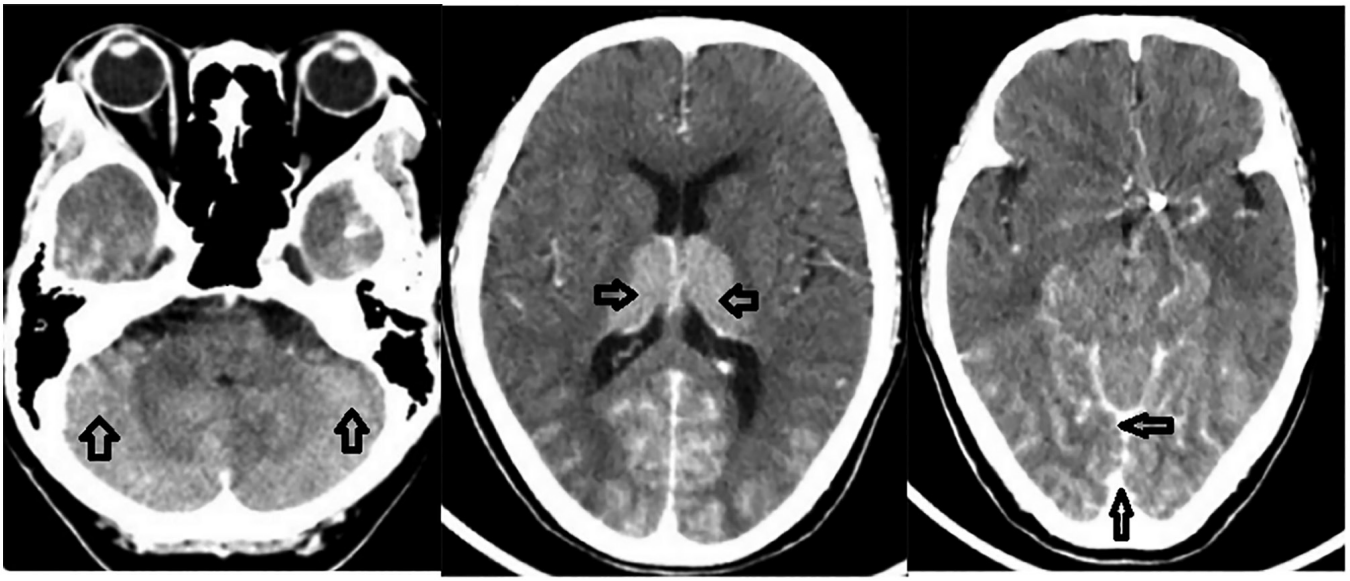

Figure 3. Each of the three images from left to right indicate that the density of the bilateral cerebellum, thalamus and sulcus, cistern increased. Arrows indicate the dense areas.

recovered; however, the development of cognitive function was not evident, which was manifested by occasional incomplete answers. The Mini-Mental State Examination score (2) was 16 points, and combined with the condition of illiteracy, the patient could be diagnosed with mild dementia. The MRI examination of the brain revealed multiple abnormal signal shadows in the bilateral thalamus, occipito-temporal lobe, corpus callosum, brainstem and bilateral cerebellar hemispheres (Fig. 5). Therefore, the administration of an intra-arterial injection of metoclopramide (10 mg) for anti-emetic treatment, dexamethasone (5 mg) for reducing the inflammatory response, nimodipine (10 mg) pump for vasospasm relief, mannitol (25 g) for intracranial pressure reduction and other treatments was continued. At 17 days after the surgery, the cognitive function of the patient gradually recovered and she was discharged from the hospital.

Table I. General information of the patient.

Characteristic	Features/information
Sex	Female
Age	57 years old
Physical examination	No positive signs
Laboratory tests	Creatinine level, 79 μmol/l
	Alanine aminotransferase level, 41 U/l
Imaging	Cerebral vascular wall imaging revealed a bilateral internal carotid artery with a double lumen in the C1 segment
Medical history	
Chief complaint	Recurrent headaches for >1 month
Previous history	She underwent right internal carotid artery stenting and left internal carotid artery aneurysm embolization 4 years prior

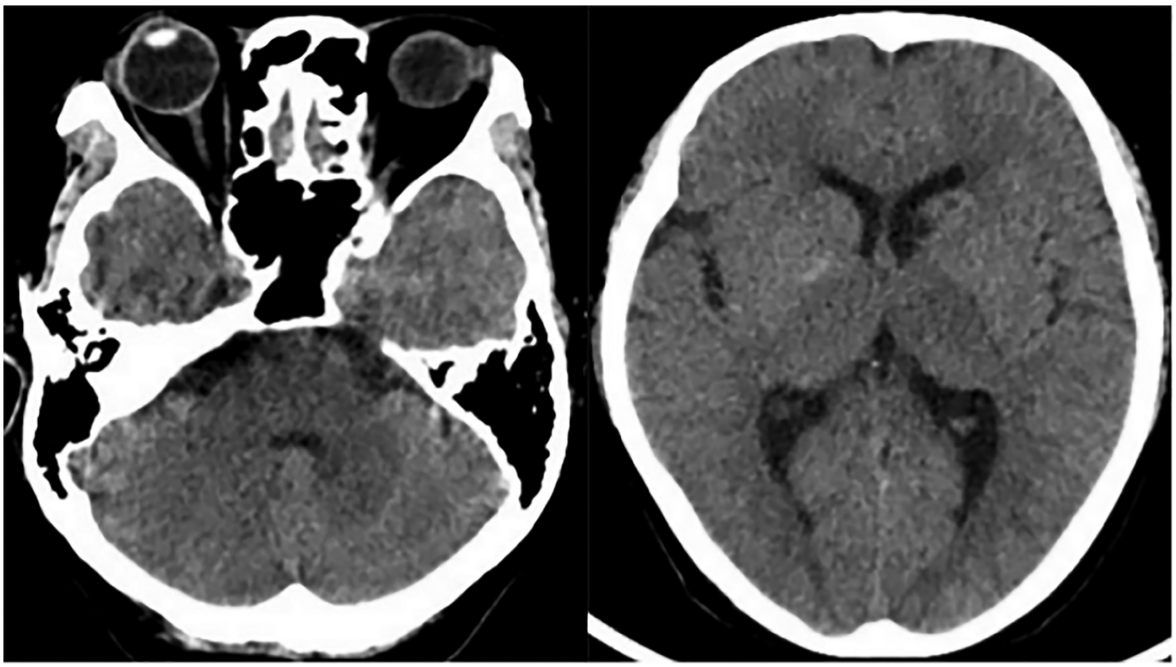

Figure 4. Each of the two images from left to right indicate that the high-density shadow in the bilateral cerebellum, thalamus was attenuated 24 h after the surgery.

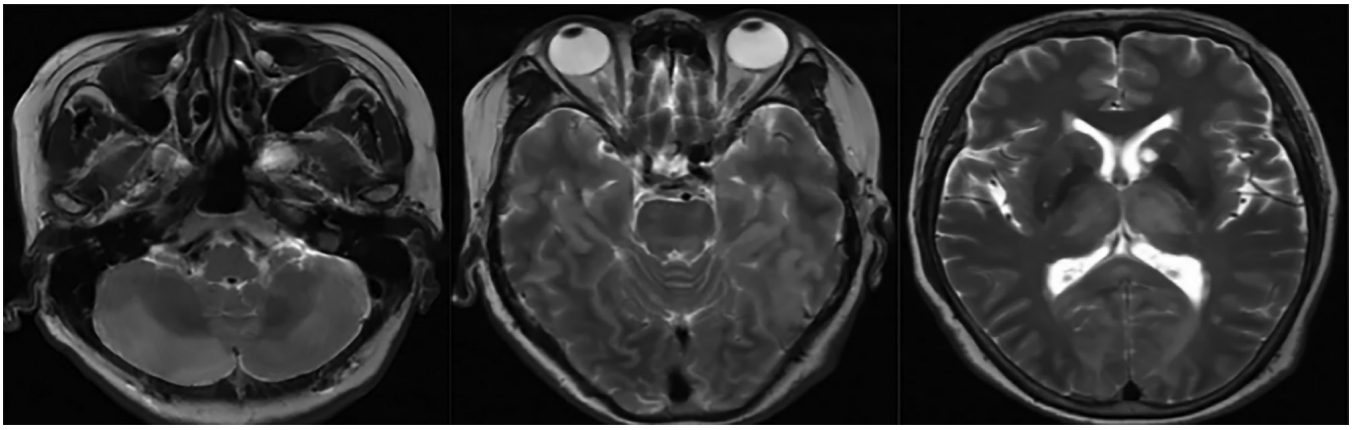

Figure 5. Each of the three images from left to right indicate multiple abnormal signal shadows in the bilateral cerebellar hemispheres, bilateral occipito-temporal lobe and bilateral thalamus.

Discussion

Contrast encephalopathy is an unusual neurological complication and its pathogenesis remains unclear. According to previous studies (3-5) the widely accepted theoretical mechanism is set around the destruction of the blood-brain barrier and the chemical properties of the contrast agents which may be as follows: i) the contrast medium disrupts the blood-brain barrier temporarily and enters the cerebrospinal fluid, increasing the osmotic pressure of the cerebrospinal fluid, and leading to impaired brain function. The majority of contrast agents have an osmolality range of 1.2-1.8 mOsm/l, compared with 0.3 mOsm/l for normal blood, which may open the normal endothelial cell tight junctions of the blood-brain barrier (6,7). ii) Contrast agents exert a direct toxic effect on nerve cells and affect their function. iii) Iodine contrast agents affect the secretion of functional substances regulating vasomotor and causes vasospasm. In this case, the cognitive decline of the patient is considered to be related to the neurological deficits caused by the edema of nerve cells. Moreover, the risk factors of CIE are also not clear. Some studies have suggested that the most common factors related to the occurrence of CIE include the male sex, an advanced age, hypertension, diabetes, renal function impairment, the contrast agent dose, etc. (8,9). CIE can occur at any age; however, patients who are older are more likely to suffer from this condition, although the incidence for each age group has not yet been determined. At the same time, the male sex and hypertension may be the main risk factors for CIE.

CIE may occur within minutes or hours of the application of the contrast agent. Generally, normal conditions will resume within 1-3 days; however, a small portion of patients do not return to a normal state until several weeks after the application of the contrast agent (8). There is as yet no clear explanation available for this phenomenon. In addition, very few fatal CIE cases have been reported (10). The clinical manifestations of CIE are diverse, such as intracranial hemorrhage, meningitis, cortical blindness, epileptic seizure, etc. (11). The typical imaging manifestations of CIE are brain parenchyma, subarachnoid contrast enhancement and brain edema (11); the majority of patients gradually return to a normal state. In the case presented herein, bilateral cortical blindness and cognitive decline occurred within a few minutes following digital subtraction angiography, and cortical blindness gradually recovered within 3 days after the surgery; however, the recovery of cognitive function was not evident. The occurrence of cortical blindness has been demonstrated in previous reports of CIE cases (8), although the occurrence of cognitive decline is very rare. In the present study, combined with the MRI results of the patient, it was considered that this cognitive decline was caused by neurological dysfunction. The head CT examination revealed that the density of the brain parenchyma, sulcus and cistern had increased; however, the head CT re-examination 24 h after the surgery indicated that the high-density shadow in the aforementioned areas was attenuated or had disappeared, which was in line with the typical imaging manifestations of CIE.

Although the majority of patients with CIE have a favorable outcome, often with a complete resolution of symptoms, once a diagnosis of CIE is made, aggressive treatment should be administered immediately to prevent irreversible events. Current treatment options include the promotion of contrast agent excretion, the reduction of cerebral edema, and anti-inflammatory and symptomatic supportive therapy (8). In the case described herein, the clinical and imaging manifestations of the patient gradually improved following the aforementioned combination of supportive treatments.

In conclusion, CIE is a rare neurological complication caused by contrast agents and generally presents as a transient, reversible neurologic disorder. The earlier diagnosis and treatment are crucial for the better prognosis of patients.

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Availability of data and materials

The datasets used and/or analyzed during the current study are available from the corresponding author on reasonable request.

Authors' contributions

YL designed the study, and wrote, edited and reviewed the manuscript, and processed the figures. XL designed the study. HL identified the disease and provided guidance. FW provided the imaging data. ZZ collected the surgical data. SL managed the patient and provided the clinical data. All authors have read and approved the final manuscript. XL and FW confirm the authenticity of all the raw data.

Ethics approval and consent to participate

The institutional Review Board of Guangdong Second Provincial General Hospital approved the study.

Patient consent for publication

Not applicable.

Competing interests

The authors declare that they have no competing interests.

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