Contrast-induced encephalopathy (CIE) is a rare complication that presents with transient neurologic deficits and is caused by neurotoxicity of intravascular contrast media. The prognosis can be extremely favorable even in comatose patients. We reported a 76-year-old woman admitted for scheduled coronary angiography. The total amount of Optiray contrast media used was 150 mL. Immediately after the procedure, the patient developed consciousness disturbance, global aphasia, cortical blindness and right-sided weakness. CIE was diagnosed by computed tomography and subsequent magnetic resonance imaging. The patient recovered completely within 48 hours without any neurological deficits.

**Key Words:** Angioplasty • Contrast-induced encephalopathy • Percutaneous coronary intervention

**INTRODUCTION**

Contrast-induced encephalopathy (CIE) after percutaneous coronary intervention (PCI) is an extremely rare complication caused by the administration of intravascular contrast media. One possible underlying mechanism is the neurotoxicity of contrast media, which can cause osmotic disruption of the blood-brain barrier, particularly for the occipital cortex. CIE is characterized by encephalopathy, transient cortical blindness, seizure, or focal neurological deficits. Different kind of contrast media including ionic, non-ionic, hyperosmolar and isosmolar have been reported in the literature to induce CIE. We reported a case of a 76-year-old woman who developed CIE after PCI, after administration of 150 ml of hyper-osmolar and non-ionic contrast media [Optiray 320, Ioversol (Covidien Pharmaceuticals, Hazelwood, MO, USA), with an osmolality of 702 mOsm/kg H2O].

**CASE REPORT**

A 76-year-old woman with a history of hypertension, diabetes mellitus, and hyperlipidemia under medical control for more than ten years was admitted to our hospital for scheduled coronary angiography due to intermittent chest tightness on exertion and positive stress test.

The patient’s vital signs were stable on admission. Further physical examination showed only a grade 3/6 pansystolic murmur loudest at the right upper sternal border. Neurological examination, electrocardiogram and chest X-ray revealed normal and unremarkable patient status. However, laboratory investigation showed that she had dyslipidemia and diabetes mellitus. The serum total cholesterol level was 208 mg/dl and the serum triglyceride level was 230 mg/dl, with a fasting glucose of 198 mg/dl.

Coronary angiogram was performed through a trans-femoral approach with a 6-French short introducer sheath placed into the right common femoral
artery. A hyper-osmolar (Optiray, osmolarity is 702 mOsm/kg H2O) and nonionic contrast medium was used. The result showed that the patient had triple vessel disease. There was a 60% stenosis on the diagonal branch of the left anterior descending artery (LAD), a long-segment lesion with maximum stenosis up to 60% on the left circumflex artery (LCX), and a total occlusion of the middle right coronary artery (RCA) with collateral flow from the left coronary artery.

PCI for RCA was planned and intravenous heparin was given to obtain adequate activated clotting time. The wire (Fielder FC) was successfully advanced to the distal RCA but the balloon catheter could not open due to heavy calcification of the total lesion. Due to the prolonged procedure time (130 minutes), further intervention with other modalities such as rotablator was planned. For the entire procedure, the total volume of Optiray administered was 150 mL. However, consciousness disturbance, global aphasia, cortical blindness, and right-sided weakness developed soon after the patient was transferred to the recovery room. The initial diagnosis was embolic cerebral infarction. However, an emergency contrast computed tomography (CT) showed no intracranial hemorrhage (Figure 1A) or cerebral vascular obstruction (Figure 1B). The patient was transferred to a neurologic intensive care unit. Due to the lack of strong evidence of ischemia stroke, intravenous thrombolytic agents were not given. Intravenous hydration with normal saline was administered. Follow-up magnetic resonance imaging (MRI) 10 hours later on the day after the onset of symptoms showed hyperintensity at the left centrum semiovale and high frontoparietal regions on T2, fluid-attenuated inversion recovery (FLAIR) and diffusion-weighted imaging (DWI). There was no change on the apparent diffusion coefficient (ADC) maps (Figure 2), which differentiated CIE from cerebral ischemia. Patient’s electroencephalography revealed mildly diffuse cortical dysfunction. Ultimately, all neurological deficits recovered gradually within 48 hours, and the patient was discharged on day 9 after admission.

DISCUSSION

The first case of CIE that manifested as transient cortical blindness after coronary angiography was reported in 1970. The incidence of CIE after the administration of contrast media has varied, ranging between 0.3%-1.0%, but reaching 4% in some hyper-osmolar iodinated contrast media. CIE only occurs in subjects receiving localized contrast medium injection (for example, coronary or carotid intervention). Systemic intravenous injection of contrast injection has never been reported to induce CIE.

Patient’s neurological symptoms and signs usually develop within hours of exposure to contrast media. However, spontaneous resolution in neurological status usually occurs over a period of days. The diverse manifestations of CIE are several, and include cortical blindness, hemiparesis, aphasia, loss of coordination, confusion, seizure and coma.
Initially, CIE is difficult to distinguish from other complications, such as subarachnoid hemorrhage (SAH), massive cerebral infarction and hyperperfusion syndrome. Consequently, a brain CT without contrast plays the most important role in diagnosis showing diffuse cortical hyper-attenuation similar to SAH. But normal brain CT did occur in some cases. MRI results have previously shown hyperintensity in the cortex on T2, FLAIR, and DWI, which has been described in the literature. A reliable image modality that can differentiate CIE from cerebral ischemia is ADC, which shows no abnormal intensity in patients with CIE. The ADC provides a quantitative measure of water diffusion. In acute ischemic stroke with cytotoxic edema, decreased water diffusion in infarcted tissue causes a decreased ADC. A significant reduction in ADC lasting for at least 96 hours from stroke onset, which revealed delayed MRI examination in our case, may not affect the final result.

Although the precise mechanism remains unclear, a possible explanation is that the chemical and physical properties of contrast media which could cause osmotic disruption of the blood-brain barrier and the hyperosmolality and direct neurotoxicity of extravasated contrast media could further result in cerebral edema. This hypothesis is supported by animal studies. The hyperosmolality of contrast media and the permeability of blood brain barrier can both cause contrast extravasation to cerebral space, since isosmolar contrast medium could also induce CIE. The occipital cortex is one of the regions with higher permeability of the blood brain barrier. Hence, this can explain why it is the most vulnerable region.

The predisposing factors leading to CIE are chronic hypertension, transient ischemia attack (TIA), impaired cerebral autoregulation, impaired renal function, large contrast volumes, selective vertebral-basilar arteriography (VAG) and male gender. About half of all patients with transient encephalopathy following intra-arterial contrast injection have a history of chronic hypertension. The presence of precedent TIAs with changed permeability of the blood brain barrier could have predisposed the patients to CIE. Impaired renal function leads to decreased clearance and longer exposure to contrast media. The relationship between CIE and contrast medium volume is somewhat complicated. Previous studies showed that the volume of contrast medium inducing CIE ranged from 80 to 400 ml. But one report showed that a local injection of 25 mL contrast medium to the carotid artery can cause CIE. Selective VAG also had a high risk of CIE because of the involvement of the arterial supply to the medulla oblongata, brain stem, cerebellum, and basal parts of the temporal and occipital lobes.

The treatment of CIE is supportive in nature, such as the use of adequate hydration with intravenous crystalloids and anticonvulsants for seizures. In a few cases, patients had been treated with intravenous steroids and anti-edema agents without adverse consequences. The neurologic deficits lasted 15 minutes to 5 days in a majority of cases. Interestingly, re-injection of contrast medium in such patients might not induce CIE again.

CONCLUSION

In conclusion, the correct diagnosis of CIE allowed us to avoid the risks associated with erroneous treatment, such as thrombolytic agents for acute cerebrovascular ischemia or surgery for subarachnoid hemorrhage. In this case, we did not administer thrombolytic agents, and the patient’s symptoms totally resolved within 48 hours.

REFERENCES

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