

Clinical Notes

Contrast-induced encephalopathy following coronary angioplasty with iopromide

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The administration of contrast agents is usually associated with a few side effects, such as anaphylactoid reaction, heart failure, arrhythmia, renal failure, and encephalopathy. Although non-ionic agents are far less neurotoxic, they have pharmacological side effects, such as seizure, transient cortical blindness, aphasia, and sensory deficit, confusion, short-term memory loss, mental aberrations, hemiparesis and ophthalmoplegia. In the present study, we report a case of a patient who suffered acute encephalopathy after coronary angioplasty was applied with iopromide contrast (Ultravist®, Schering Pharmaceutical Co., Ltd, Guangzhou, China). Similarly to the cases reported by Kocabay et al,¹ the patient also became irritable and suffered mental aberrations, aphasia, short-term memory loss, and paralysis of limbs, and totally recovered except for short-term memory loss 28 hours later without specific medication.

A 64-year-old male patient, 52 kg, who had no history of systemic or neurologic illnesses, was admitted to our hospital, complaining of typical chest pain for 2 years. He had a history of hypertension, diabetes, and coronary artery disease. After the coronary angiography (CAG) (iopromide contrast usage was 50 ml), his heart rate dropped to 57 bpm from 80 bpm, and his blood pressure dropped to 85/60 mm Hg from 110/70 mm Hg. He became irritable, then developed confusion and paralysis of limbs. Atropine 0.5 mg, and diazepam 5 mg was used immediately, and the heart rate and blood pressure rose to normal range 3 minutes later. A 3.0 x 23 mm drug eluting stent was inserted to the left anterior descending branch (LAD). Iopromide, 160ml was used for the entire operation. The patient remained confused and developed paralysis of limbs, mental aberrations, and subsequently aphasia.

His eye movements were not limited, pupils were constricted, reactive, and there was no gaze preference. Positive bilateral Babinski and Chaddock signs were observed. No neck stiffness was observed, and both corneal and oculocephalic reflexes were preserved.

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Muscle powers were as follow: left limbs grade 2, right upper limbs grade 3, right lower limbs grade 2. Both MRI and magnetic resonance angiography (MRA) of the brain were normal. Routine blood tests, including hemoglobin, red blood cells, leukocyte, lymphocyte, and so forth were normal. A CT of the brain showed highlights in the sagittal sinus and middle line immediately after the operation (Figure 1), and only fundamental supportive medications were used.

His signs and symptoms began attenuating gradually 8 hours later, and were fully receded 28 hours after the operation. His neurologic examination returned to normal, and he suffered from a short-term memory loss. The EEG results revealed diffuse slowing in the α range in the occipital region 20 hours after the operation. At 1.5 years after discharge, and after attending monthly clinic follow ups no abnormalities of the nervous system was found by cardiologists and neurologists.

Recently, Kocabay et al¹ reported 2 cases who suffered from encephalopathy after injecting iopromide (Ultravist®). In our case, the contrast used was Ultravist 370. Other contrast agents that could cause encephalopathy included iohexol,² ioxilan,³ iopamidol, and metrizamide. The complications of intravascular administration of contrast agents include idiosyncratic (anaphylactoid) reactions, and non-idiosyncratic reactions such as shock, congestive heart failure, cardiac arrhythmias, acute renal failure, and neurotoxic effects. We speculated that iopromide most likely induced this patient's encephalopathy, as his symptoms started within 1/2 hour after CAG and resolved spontaneously within 28 hours. No other etiologies or contributing causes were suspected. In the absence of other metabolic abnormalities and suspected drugs, spontaneous recovery of the patients' clinical status by only supportive medications also indicates that the encephalopathy is most likely a toxic reaction to the contrast agent, which resolved spontaneously.

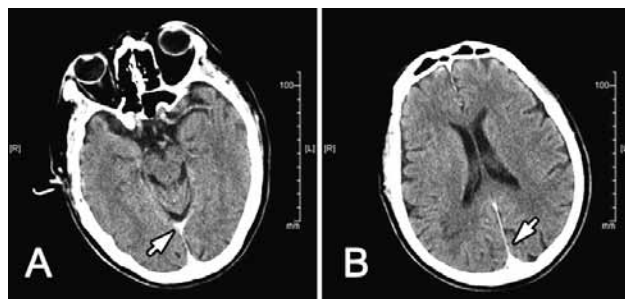


Figure 1 - Brain CT immediately after coronary angioplasty showing highlights in the A) sagittal sinus and B) middle line.

Similar to our study, Kocabay et al¹ reported 2 patients that suffered from nausea, headache, confusion, and agitation one hour after percutaneous transluminal coronary intervention (PCI), which resolved spontaneously within 8-12 hours. Sawaya et al² reported a case whose encephalopathy occurred after coronary angioplasty from iohexol, resolving gradually 12 hours later. Dangas et al⁴ reported a case who developed confusion and left hemiparesis rapidly after carotid artery stenting, and completely recovered in 2 days.

The mechanism of neurotoxicity is controversial. We suppose that the contrast agent had disturbed the blood-brain barrier and entered into the brain in our patient, which maybe the primary mechanism leading to encephalopathy. The following aspects may cause this phenomenon. Firstly, contrast such as hypertonic solutions could draw water out of the endothelial cells of brain capillaries, arterioles, and venules, causing the endothelial cells to shrink and to separate at tight junctions. Secondly, the increase in intraluminal pressure caused by pressure injection of the contrast agent as well as by contrast agent-induced cerebral vasodilatation might contribute to increasing vascular wall tension, further separating tight junctions. Thirdly, vesicular transport maybe a mechanism of osmotic barrier opening. All these mechanisms could lead to the leakage of contrast agent. The leakage of the contrast into the CSF and electrolyte imbalance may cause an encephalopathy and repeated injections of contrast agents within several minutes may result in neurotoxic effects. A patient was found with contrast media located in the sagittal sinus (inferior and superior) and occipital lobe when CT of the brain was performed.¹ Foltys et al⁵ reported a case of cerebral contrast agent extravasation after coronary angioplasty. In our case, a CT of the brain showed only highlights in the sagittal sinus and middle line (Figure 1). No evidence could be found to prove leakage of the contrast into the sagittal sinus or other positions. This might be attributed to the small dose of the contrast, which broke into the blood-brain barrier, and hence, could not be found by CT, MRI, and MRA

immediately after the percutaneous coronary intensive (PCI) showed no hydrocephaly and cerebral infraction, although carotid infusions of hypertonic solutions have been shown to cause focal brain edema with marked swelling of astrocyte foot processes.

In conclusion, contrast-induced encephalopathy following an angiographic procedure should be considered as a new-onset neurological disorder, although its occurrence is rare. Iopromide-induced encephalopathy may occur during angioplasty in patients with no particular predispositions. Isotonic, nonionic contrasts are considered safer, however, more research is needed to confirm this.

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