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Case Reports

Acute Transient Contrast-Induced Neurologic Deficit as a Complication of Percutaneous Coronary Intervention

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Abstract

Acute transient contrast-induced neurologic deficit is an uncommon condition triggered by the administration of intra-arterial contrast during angiography. It can present with encephalopathy, cortical blindness, seizures, or focal deficits. This report describes a patient who presented with severe neurologic deficits after percutaneous coronary intervention, with complete symptom resolution within 72 hours.

Keywords: Percutaneous coronary intervention; contrast media; neurologic manifestations

Case Report

Presentation and Physical Examination

A 66-year-old woman with a history of coronary artery disease presented at the catheterization laboratory for a staged percutaneous coronary intervention (PCI) of the proximal mid–right coronary artery. Coronary angiography was performed through a transradial approach with a 6F catheter sheath without complications. A total of 130 mL iohexol 350 mg/mL (Omnipaque, low osmolarity, 844 mOsm/kg water, GE HealthCare) was administered throughout the hour-long procedure. Twenty minutes after completing the procedure, the patient presented with left-sided hemiparesis, left facial weakness, left hemineglect with asomatognosia, dysarthria, and altered mental status. Blood pressure was 159/70 mm Hg, heart rate was 88/min, respiratory rate was 18/min, and oxygen saturation was 94% on 2 L/min with nasal cannula. A stroke alert was promptly called, and computed tomography (CT) images of the brain showed a widespread right subarachnoid density with cerebral edema (Fig. 1).

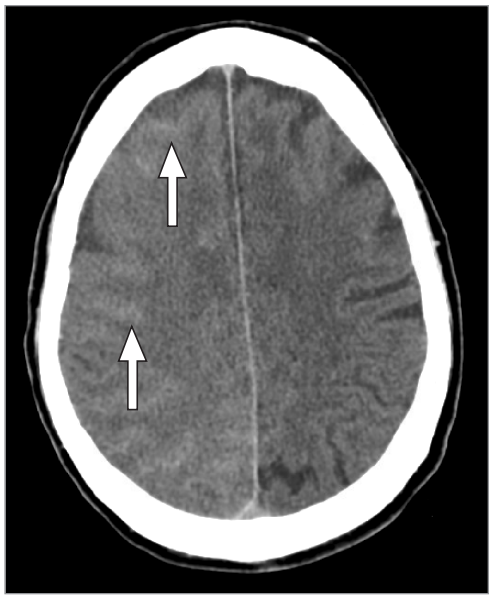

Fig. 1 Computed tomographic stroke imaging in the axial plane. Arrows indicate multiple widespread right subarachnoid densities with cerebral edema.

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Medical History

Medical history was remarkable for triple-vessel coronary artery disease after uncomplicated PCI of the distal left main coronary artery to the proximal left anterior descending and left anterior descending diagonal coronary arteries in 2022, peripheral artery disease after bilateral lower extremity bypass in 2019, hypertension, dyslipidemia, and chronic obstructive pulmonary disease treated with home oxygen.

Differential Diagnosis

Given the new onset of neurologic deficits after PCI, the primary concerns were embolic, ischemic, and hemorrhagic stroke; seizures; sedation side effects; and metabolic disorders, such as hypoglycemia. Given the recent use of iodine contrast and the high possibility of contrast extravasation, acute transient contrast-induced neurologic deficit (ATCIND) was also considered.

Technique

Initial CT with angiography of the brain showed an asymmetrical, widespread subarachnoid space density with sulcal effacement with cerebral swelling, findings suggestive of subarachnoid hemorrhage (SAH). Magnetic resonance images of the brain were obtained 5 hours after symptom onset and showed no acute infarction or hemorrhage, with an interval resolution of the cerebral edema that was seen on the previous CT scan of the brain (Fig. 2).

Given the concern for possible underlying seizures, the patient was placed on continuous electroencephalography monitoring, which showed no seizures or epileptiform discharge after 24 hours.

After the initial CT scan with angiography of the head, thrombolysis and thrombectomy were not pursued because of the concern for SAH. The patient was admitted for telemetry and symptomatic monitoring. Levetiracetam was also administered because of a concern for ongoing seizures, but it was discontinued after electroencephalographs showed the absence of epileptiform activity. Following multidisciplinary discussions among neurology, stroke, neurosurgery, and neurointerventional radiology services, the consensus was that the findings on physical examination and brain imaging studies were overall compatible with a diagnosis of ATCIND.

Key Points

- Clinicians should consider a differential diagnosis of the causes of a new neurologic deficit in a patient who has undergone PCI.
- Clinicians should be aware of ATCIND as a complication of PCI, especially in cases where there is a recent use of iodine contrast.
- A multidisciplinary approach and comprehensive imaging, such as brain CT scans and magnetic resonance imaging, are crucial aspects of early diagnosis and treatment of ATCIND.

Abbreviations and Acronyms

ATCIND	acute transient contrast-induced neurologic deficit
CT	computed tomography
PCI	percutaneous coronary intervention
SAH	subarachnoid hemorrhage

Outcome

The patient was treated with supportive management, and symptoms resolved within 72 hours. A repeat CT scan of the brain on the fifth day after admission showed total resolution of the previously radiologically described findings.

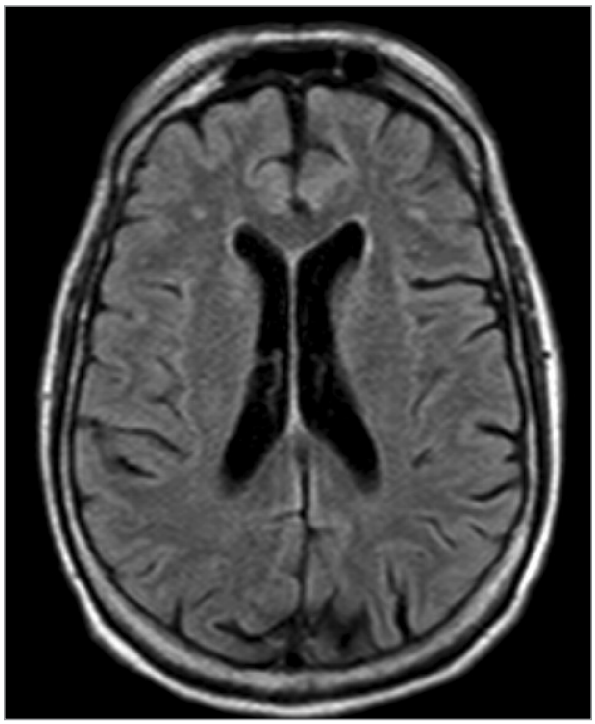

Fig. 2 Magnetic resonance imaging (T2-weighted, fluid-attenuated inversion recovery) of the brain in the axial plane demonstrates interval resolution of subarachnoid lesions and cerebral swelling.

Latest Follow-Up

The patient was seen in the outpatient cardiology clinic 12 days after discharge with documented complete resolution of her neurologic deficits.

Discussion

Acute transient contrast-induced neurologic deficit is a term that encompasses contrast-induced encephalopathy, contrast-induced neuropathy, and angiography-related cortical blindness in both the coronary and cerebral angiographic settings. It was first described in 1970 as contrast-induced neurologic injury secondary to coronary angiography.[1] More recently, these clinical syndromes have been associated with the administration of localized contrast medium injections through coronary, carotid, and cerebral angiography.[1-7] In a recent systematic review, the incidence of this rare entity after PCI was estimated to be 0.15%, even less common than the incidences reported in prior contrast studies.[1] Risk factors for the development of ATCIND include kidney dysfunction, large contrast dose, and contrast agents that have higher osmolality and lower hydrophilicity,[1] which are characteristics of the contrast agents used in PCI. Neurologic symptoms typically present within hours of exposure to contrast and include encephalopathy; motor and sensory disturbances; vision disturbances, including cortical blindness, ophthalmoplegia, and aphasia; and seizures.[1,2,5-7]

Brain imaging—specifically, CT scans without contrast—plays a diagnostic role in the confirmation of ATCIND. Computed tomography may reveal hyperdense lesions in the subarachnoid space, cerebral edema, and cortical enhancement.[1,2,4,5] These findings—notably, the hyperdensities in the subarachnoid space—can be misinterpreted as SAH; however, in the right clinical context and with the appropriate risk factors, ATCIND can be differentiated from SAH. For instance, on CT scan, contrast potentially causing ATCIND can be differentiated from blood observed in SAH by measuring the density: Blood has a density of 40 to 60 Hounsfield units, whereas contrast media have higher attenuation values (100-300 Hounsfield units).[2] Furthermore, magnetic resonance imaging of the brain can be used to identify hyperintense lesions in the cortex on T2-weighted imaging, including both fluid-attenuated inversion recovery and diffusion-weighted imaging, results that are consistent with cerebral edema found in ATCIND.[4]

The pathophysiology of ATCIND is poorly understood, but the suspected mechanism involves disruption of the blood-brain barrier and neurotoxicity induced by contrast. Acute transient contrast-induced neurologic deficit has been associated with decreased expression of claudins, superoxide dismutase, and catalase, leading to increased brain microvascular permeability.[3] Additionally, tissue plasminogen activator administration may increase vascular permeability, which can worsen ATCIND; therefore, it is paramount to differentiate ATCIND from ischemic stroke and avoid the use of tissue plasminogen activator.[8]

The management of ATCIND is supportive, and the prognosis is excellent. In this case, symptoms resolved within 72 hours.[9] In most cases, neurologic deficits resolve within 24 to 48 hours.[1,2] Steroid treatment has been suggested to be beneficial in decreasing cerebral edema but was not used in this patient because its efficacy has been questionable.[5]

Conclusion

Acute transient contrast-induced neurologic deficit is a rare constellation of neurologic symptoms most recognizable after contrast administration for angiography. Diagnosing ATCIND often requires the appropriate context of acute exposure, corresponding neurologic deficits to the appropriately affected cerebrovascular territories, and corroborated imaging findings. Imaging suggestive of progressive infarct or hemorrhage, however, excludes ATCIND as a diagnosis and instigates consideration of other differential diagnoses. Limitations in the diagnosis and understanding of ATCIND lie in the lack of standardized diagnosis criteria, its pathophysiology and heterogeneous presentation, and the absence of prospective studies in the current literature.[1]

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