Contrast-Induced Transient Neurological Symptoms Following Percutaneous Coronary Intervention: A Case Report

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Patient: Male, 78-year-old
Final Diagnosis: Contrast-induced neurological injury
Symptoms: Transient hemiparesis and dysarthria
Medication: —
Clinical Procedure: Percutaneous coronary intervention
Specialty: Neurology

Objective: Rare disease
Background: Transient neurological symptoms after a percutaneous coronary intervention (PCI) are not uncommon manifestations. In clinical practice, the development of these symptoms might be a warning sign for PCI-related ischemic or hemorrhagic stroke. However, there is a reported risk of contrast-induced neurological injury (CINI) after PCI, which results in a broad spectrum of transient and benign neurological symptoms. Advanced age, renal disease, diabetes, hypertension, and brain parenchymal lesions are risk factors for CINI.

Case Report: A 78-year-old man with diabetes and impaired renal function developed left-sided hemiparesis and dysarthria within one hour of PCI. Non-contrast CT head showed hyperdense lesions in both frontal lobes, while the susceptibility-weighted sequence of magnetic resonance imaging (SWI-MRI) excludes hemorrhage. Hemodialysis had to be started for fast contrast clearance, and he had recovered completely within 24 hours.

Conclusions: This case demonstrates that CINI is an important differential diagnosis that cardiologists and neurologists must be familiar with, especially for high-risk patients. The prognosis is good; whether an appropriate contrast’s dose or type for PCI or a need for early hemodialysis to avoid CINI in those patients is unclear.

MeSH Keywords: Cerebral Hemorrhage • Contrast Media • Coronary Angiography • Extravasation of Diagnostic and Therapeutic Materials • Stroke

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Background

Cardiac catheterization is currently performed frequently, leading to increased risk of associated neurologic complications [1,2]. The frequency of neurologic complications after cardiac catheterization has been reported to range from 0.03% to 0.3% for diagnostic procedures and from 0.3% to 0.4% for therapeutic procedures [1–3]. CINI is a rare complication of coronary angiography, with few reported cases [4]. Clinical symptoms may vary from an altered level of consciousness and cortical blindness to seizures and focal neurological symptoms [4]. Neuroimaging is an important tool that has helped physicians to diagnose contrast retention and to exclude ischemic or hemorrhagic complications of PCI [5]. However, in some cases, it can pose a diagnostic dilemma and thus complicate the decision about the use of antithrombotics following PCI. For example, post-procedural intracerebral hemorrhage (ICH) may necessitate holding anti-platelets, with increased risk for stent thrombosis [2], while post-PCI ischemic stroke may require thrombolysis use with increased risk of hemorrhages, especially in high-risk patients [2]. As CINI can resemble stroke clinically or radiologically, further neuroimaging studies, including dual-energy CT scan or brain MRI, may be necessary to differentiate lesions as being related to contrast vs. stroke [6–9]. We present the case of highly possible CINI with radiological findings of contrast enhancement and edema. Brain MRI ruled out hemorrhage, and he had a complete recovery with hemodialysis and supportive care.

Case Report

We present the case of a 78-year-old right-handed man with a history of uncontrolled diabetes, hypertension, and end-stage renal disease ESRD, who was on peritoneal dialysis and a history of right middle cerebral artery (MCA) stroke 6 months ago, who presented to the Emergency Department with a complaint of chest pain. His electrocardiogram demonstrated a non-ST elevation myocardial infarction. The coronary angiogram was performed via right radial artery access, followed by PCI with a drug-eluting stent deployed into the circumflex and right coronary artery (RCA). Around 190 mL of iohexol (Omnipaque 350) was used during the procedure. He also had received 7500 IU heparin, 300 mg clopidogrel, and 100 mg aspirin. The procedure lasted for 55 min, with stable blood pressure and excellent angiographic results. One hour later, the patient developed left-sided weakness and dysarthria. The stroke code was activated by the Cardiology team. On examination, his vital signs were stable. He had moderate dysarthria, mild left upper motor neuron facial palsy, and the motor power in the left arm and left leg was 3 out of 5 on the Medical Research Council scale, along with decreased sensation on the left side of the body. Plantar response was extensor bilaterally. The National Institute of Health stroke scale score was 7. His blood glucose was 197 mg/dl. A head CT showed hyperdense lesions in both frontal lobes (Figure 1). A diagnosis of ICH was considered; therefore, an MRI head was done immediately to differentiate contrast extravasation from bleeding. SWI-MRI did not show any signal drop-out in areas that showed hyperdense lesions on head CT (Figures 2, 3), and DWI-MRI did not show restricted diffusion. Aspirin and clopidogrel were resumed. Hemodialysis

Figure 1. Coronal views of CT head showing hyperdense lesions in the left frontal lobe (A) (yellow arrow) and right frontal lobe (B) (red arrow) with surrounding areas of hypodensity.
had to be started for quick contrast removal, and his symptoms had improved completely within 24 h. Laboratory investigations excluded infection but showed a raised hemoglobin A1c of 10.3 and creatinine of 5.6 mg/dl. His echocardiogram demonstrated moderate global hypokinesia, with moderate left ventricular systolic dysfunction (EF 33%). Supportive care was maintained for 4 days, and he was discharged with complete recovery. His CT head was repeated after 72 h, showing complete resolution of the hyperdense lesions.

Discussion

This case demonstrates that CINI can be a possible cause of new neurological symptoms following PCI, especially in an elderly diabetic person with impaired renal function. This may have an impact on the clinical decision-making process in situations in which differentiating contrast retention from stroke is crucial and might be even a life-saving. In our patient, the brain MRI was performed to exclude ICH, and antiplatelet was thus

Figure 2. Axial view of CT head (A) showing hyperdense lesion in the left frontal lobe (yellow arrow) and SWI-MRI (B) of the corresponding section not showing any signal drop-out in areas corresponding to the hyperdensity.

Figure 3. Axial view of CT head (A) showing hyperdensity (yellow arrow) and SWI-MRI (B) of the corresponding section not showing any signal drop-out in areas corresponding to the hyperdensity.
resumed to keep stent patency. Consistent with the previous report, this patient experienced complete recovery within 24 h.

The mechanisms by which contrast enhancement occurs following coronary angiography remain largely obscure. Many hypotheses have been proposed, including direct disruption of the blood-brain barrier (BBB) induced by the chemical or ionic properties of the contrast material [9,10]. The hyper-osmolarity changes, hyperviscosity, and vasodilator characters of the contrast agents might create high intraluminal pressure and thus a greater BBB disruption [9,10]. In the present case, the contrast enhancement in the right frontal lobe

| Study                        | Age/sex | Risk factors          | Type and dose of the contrast | Neurological findings                                      | The onset of the neurological symptoms after PCI | Neuroimaging findings | Treatment                              | Outcome                               |
|------------------------------|---------|-----------------------|-------------------------------|------------------------------------------------------------|-------------------------------------------------|-----------------------|----------------------------------------|---------------------------------------|
| Law S, et al. (2012) [12]    | 69 y/F  | HTN, DM, impaired renal function | Iodixanol, 320 mg/mL          | Seizures, left-sided weakness, homonymous hemianopia, hemisensory loss, and hemineglect | 12 h                                            | Initial CT: frontal and parietal lobes edema | Intravenous thrombolysis           | Complete recovery within 24 h          |
| Heemlar J, et al. (2018) [13]| 67 y/F  | HTN/DM                | Iodixanol, 100 ml             | Altered level of consciousness, right-sided hypertonia and bilateral upgoing plantar | 0–4 h                                          | CT: right-sided hypodensities in the watershed regions. Repeated scan at 4h showed diffuse bilateral cerebral edema | Intubation, dexamethasone, and levetiracetam | 4 days: marked improvement. 3 weeks: complete resolution of the radiological findings on MRI. 6 weeks follow up: complete neurological recovery |
| Raju NS, et al. (2015) [14]  | 44 y/F  | HTN/DM/ESRD on peritoneal dialysis | Iohexol (Omnipaque 350), 190 mL | Headache, seizure, and left-sided weakness                | A few hours                      | CT: contrast retention in the right cerebral hemisphere and left cerebral hemisphere watershed territories | Lorazepam and levetiracetam for seizures, peritoneal dialysis | Complete recovery within 3 days        |
| Yildiz A, et al. (2003) [15] | 63 y/M  | NA                    | Iomeprol (imeron), 450 ml     | Amnesia, headache and right upper extremity numbness      | NA                                      | CT: contrast retention at the right frontal, occipital, and parietal lobes | Dexamethasone                 | Complete neurological and radiological recovery within 25 h |
| Velden I, et al. (2003) [16] | 82 y/F  | HTN, impaired renal function | Iomeprol (imeron), 500 ml     | Altered LOC, Malignant HTN, aphasia, and right-side hemiparesis | 6 h                                          | CT: contrast retention the sulci of both brain hemispheres | Close observation, supportive care | Complete recovery at 40 h               |

Table 1. Case reports of patients who have undergone PCI and developed focal neurological symptoms.
may be partially explained by the presence of chronic infarction in areas of extravasation, causing BBB rupture. However, the patient also had contrast retention of the left frontal lobe. Certain well-recognized clinical factors [11] have been attributed to this finding in our patient. First, clearance of contrast material was probably delayed because of ESRD. Second, uncontrolled diabetes may contribute to a higher vascular permeability and BBB disruption. Third, advanced age and poor cardiac function may be an additional factor. Hypertension is a well-known factor for CINI as it can also affect BBB [11]; however, as the patient’s blood pressure readings were well-controlled, it does not seem to play a large contributory role here.

A recent systematic review of the literature involving 75 cases of CINI in the context of coronary angiography showed that reversible cortical blindness is the most commonly reported symptom, followed by changes in mental status. Focal neurological symptoms are uncommon and have been reported in only 5 cases [12–16]. Neurological events usually develop within 2 to 12 h of coronary angiography and mostly improve in 24 to 72 h [4]. Table 1 summarizes the reported cases in the literature of transient focal neurological symptoms following PCI. The clinical and radiological findings in our case seem to be similar to the previous reports [12–16]. There is no existing recommendation for contrast type or dose used for a coronary angiogram to avoid CINI. In our case, the patient had ESRD and 120 ml of lohexol was administered. It is hypothetically correct that lohexol has a low risk for CINI as it is a non-ionic contrast agent with a relatively low or iso-osmolarity feature. Kariyanna et al. stated that cerebral contrast retention has been reported with all types of contrast agents, regardless of their osmolality or ionic features, there is no clear association between the contrast dose and CINI, and most of the events occur with administration of 100 ml or more of the contrast agent, similar to our case [4].

Given the well-recognized neurological complications of PCI [2], it is crucial to differentiate between stroke and contrast extravasation. IV thrombolysis (IVT), which is the standard acute therapy [8]. In our patient, MRI of the brain with SWI was chosen because it is fast, reliable, and easily accessible.

Overall, complete clinical and radiological recovery of CINI have been reported with supportive management, including adequate hydration and close observation. In most of the reported cases [4], as in our patient, rapid recovery occurs within days. Considering the impaired renal function and CINI, we considered hemodialysis for rapid contrast clearance. In our case, complete neurological recovery, as well as the resolving radiological finding, on follow-up CT led to the diagnosis of CINI. Further studies are required to determine the optimal contrast dose in ESRD patients, as well to determine the timing and need for hemodialysis following PCI to avoid this rare but important condition.

Conclusions

CINI is an essential differential diagnosis that must be considered in high-risk patients following PCI. This condition is benign but can pose a diagnostic dilemma. Once the diagnosis has been confirmed, supportive care is often adequate. It is unclear whether hemodialysis is essential or has an impact on outcomes that need to be investigated in future studies.

Conflict of interest

None.
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