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Contrast-Induced Encephalopathy Post Cardiac Catheterization, A Rare Mimicry of Acute Stroke - Case Presentation and Review of the Literature

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Abstract Contrast-induced encephalopathy (CIE) or contrast-induced neurotoxicity is a rare, elusive, and puzzling diagnostic entity, even for most astute clinicians. Only a handful of publications have been written describing this rare phenomenon. A recent systematic review has only identified a total of 52 cases between 1970 - 2017 [1]. The most common neurological complication associated with cardiac catheterization is an atheroembolic stroke. CIE is not a well-known complication. Clinicians involved in administering high volumes of contrast solutions, as seen in coronary catheterizations, should be aware of this complication as it may be misdiagnosed as an acute stroke, leading to unnecessary additional contrast administration for imaging, as well as invasive and non-invasive interventions. In this report we present a case of an 87-year-old woman known coronary artery disease (CAD) who presented with acute coronary syndrome (ACS) and underwent a successful cardiac catheterization with stent placement followed by left-sided weakness a few hours later due to CIE. We also provide review of the literature and discuss management strategy of this rather rarely encountered diagnosis.

Keywords: *contrast-induced encephalopathy, contrast-induced neurotoxicity, stroke, cardiac catheterization*

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1. Introduction

Coronary catheterization has revolutionized cardiac care. It allows clinicians to physically observe coronary pathology as well as treat patients simultaneously. Advancements in medical technology come with the inevitability of associated risks and complications. The most common neurological complications associated with cardiac catheterization are acute thromboembolic stroke, air emboli, vasospasms, and intimal dissections. The incidence of these complications range from 0.05% to 0.10%. CIE, however, is not a well-known complication that is associated with cardiac catheterization. To date, only 52 total cases have been published regarding CIE from cardiac catheterization in the past 50 years [1].

In the relatively few cases that have been published, CIE appears to have a broad spectrum of symptoms, which include cortical blindness, headache, memory loss, confusion, seizures, hemiparesis, aphasia, or even coma [2]. Symptoms may occur while the patient is still on the cardiac catheterization table, or up to 12-24 hours after the procedure has ended [3]. The disease course of CIE is typically benign, though there have been reported cases of permanent neurological deficits and fatalities [4]. The pathophysiology of CIE is poorly understood, however, it is likely related to a transient breakdown of the blood-brain barrier (BBB) resulting in direct neurotoxicity from the contrast itself [5]. We review the literature and discuss a case of CIE in an 87-year-old female who developed symptoms of acute stroke shortly after urgent cardiac catheterization.

2. Case Presentation

An 87-year-old black female with a past medical history of diabetes type II, hypertension, hyperlipidemia and coronary artery disease with previous myocardial infarction (MI) requiring percutaneous coronary interventions in mid- left anterior descending artery, mid-right coronary artery, obtuse marginal one and three arteries. She presented to the emergency room with an episode of typical chest pain. The chest pain had started a

day prior to presentation and was over the left chest, "pressure like" sensation, 6/10 in intensity without any radiation. The chest pain got relieved with sublingual nitroglycerine. The patient had difficulty breathing with diaphoresis and nausea with no emesis. She endorsed that this presentation was similar to her previous episode of myocardial infarction.

Patient was afebrile at presentation, heart rate was 74 beats per min, blood pressure was 138/76 mm Hg, respiratory rate was 16 cycles per minute and saturating at 98% on room air. Patient was given aspirin 375 mg. Electrocardiogram (EKG) showed T-wave inversion in leads I and aVL. Initial troponin at was 0.14 ng/ml and a repeat measurement showed 6.1 ng/ml. Chest X-ray showed small bilateral pleural effusions and an enlarged cardiac silhouette. The patient's pain resolved, however, she was deemed high risk and was admitted to cardiac telemetry service with a diagnosis of non-ST segment elevation myocardial infarction (NSTEMI).

Upon further chart review, the patient was noted to have a follow-up coronary angiogram two years prior to presentation showing 50-60% restenosis of the left anterior descending artery.

The patient remained stable and asymptomatic with no telemetry events recorded. A decision was made to proceed with cardiac catheterization. The patient underwent coronary angiography with successful PCI with rotational atherectomy and drug-eluting stent (DES) placement in mid-left anterior descending (LAD) coronary and distal-LAD. The patient tolerated the procedure well. A few hours after the procedure, in the coronary care unit (CCU), the patient was noted to have left-sided weakness and a stroke code was called. On examination, the patient had 3/5 left upper extremity (LUE) and 4/5 left lower extremity (LLE) power and decreased sensation to the left side with no abnormal mentation or cranial nerve palsies. National Institute of Health Stroke Scale of 9 was noted and an emergent non-contrast computer tomography (CT) was obtained, thus immediate stroke workup was recommended.

Non-contrast CT-brain with CT angiogram head and neck rule out intracranial hemorrhage. CT brain showed poor differentiation of the right frontal lobe sulci secondary to edema and subtle subarachnoid contrast/hemorrhage suggestive of contrast-induced neurotoxicity. Repeat non-contrast CT-brain one day later redemonstrated subtle loss of right frontal gray-white differentiation with mild mass effect consistent with edema (Figure 1) and bilateral subarachnoid contrast uptake (Figure 2). While in the CCU, the patient exhibited left-sided power as low as 0/5 and 2/5 for LUE and LLE respectively before improving to 3/5, 3/5 two days post-event and being downgraded to the general medical floor. With supportive care and daily physical and occupational therapy, she subsequently returned to baseline four days post-event and plans were made for discharge to subacute rehab.

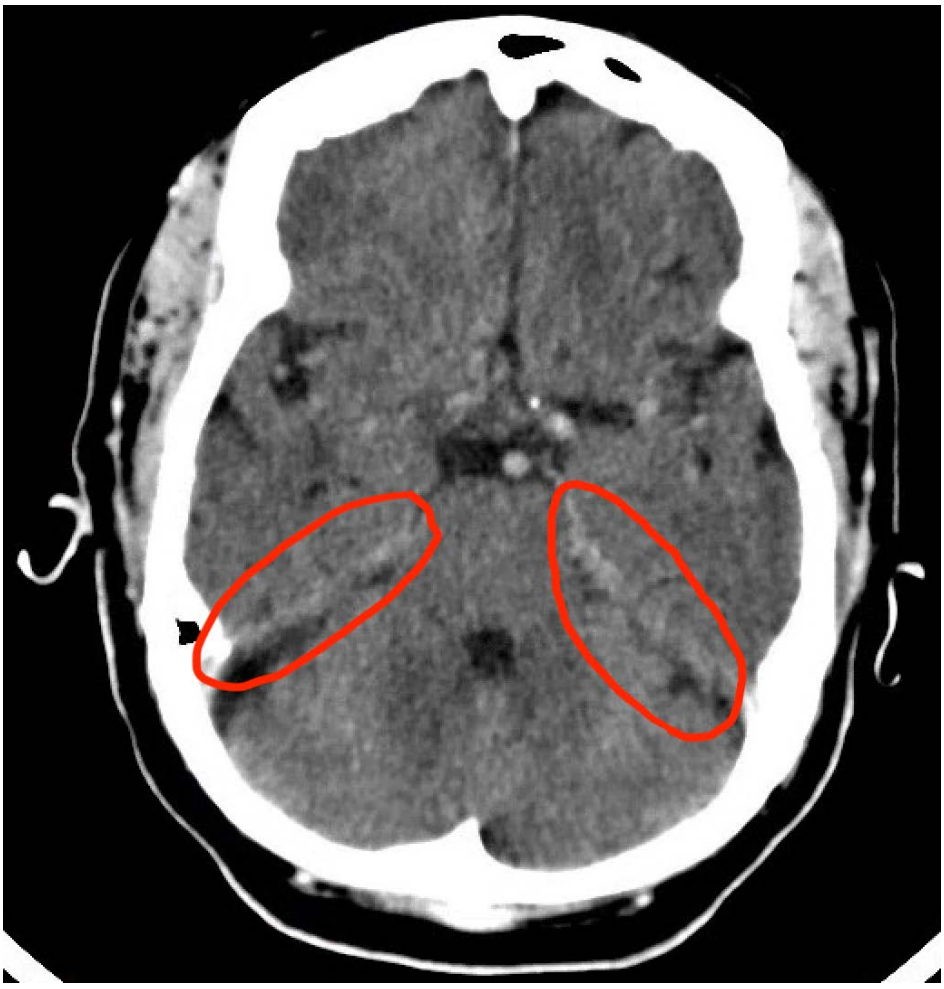

Figure 1. CT Brain demonstrating bilateral subtle subarachnoid contrast uptake (circled)

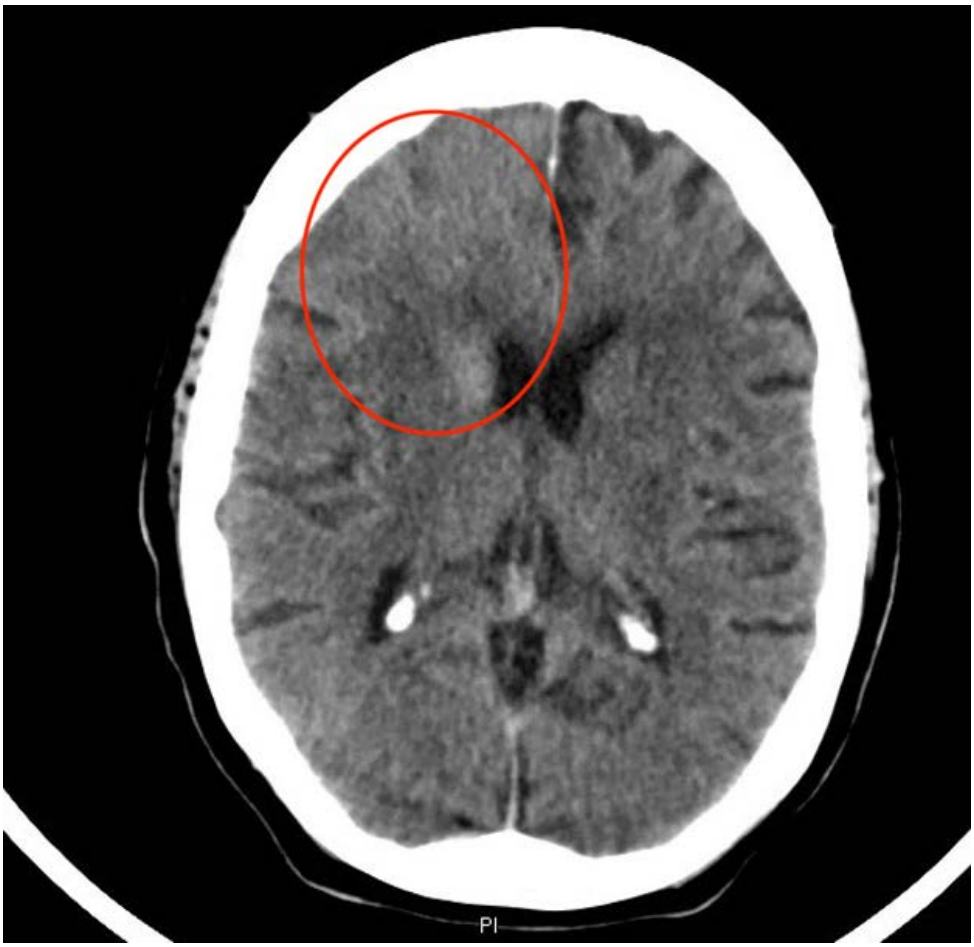

Figure 2. CT Brain demonstrating poor differentiation of the right frontal lobe sulci secondary to edema

3. Discussion

CIE is a rare condition occurring subsequent to the intravascular administration of iodinated contrast for diagnostic procedures.[6] CIE is more likely to occur following procedures involving high doses of contrast injected directly into the aortic arch or cranial vessels such as coronary (in this case) and cerebral angiography respectively. [7] The exact pathophysiology of this condition remains undetermined. It is likely related to osmotic disruption of the blood-brain barrier by hyperosmolar ionic contrast, resulting in cerebral edema, decreased neuronal excitability, and subsequent neurologic dysfunction. [8]

The typical presentation involves transient neurological deficits 2-12 hours following contrast administration which varies depending on the affected region of the brain. [6] This is congruent with this patient's clinical course wherein she developed a left-sided hemiparesis as her right frontal lobe (harboring the precentral gyrus/primary motor cortex) was affected by the contrast. Other common signs and symptoms which were not present in this case include headaches, seizures, cortical blindness, ophthalmoplegia, and global amnesia. [9]

If CT imaging is performed immediately after symptom onset, hyperdensity in the cortical or subarachnoid regions can be visualized and is due to residual contrast, as seen here (Figure 1). Dual energy/spectral CT (not done for this patient) is required to accurately distinguish between contrast and hemorrhage in the subarachnoid space however, given the clinical presentation, it is reasonable to attribute the subtle subarachnoid hyperdensity to contrast leakage. [10,11] Otherwise, mass effect secondary to edema at the affected site is a common feature, as seen in this case (Figure 2).

CIE is a disorder with a great prognosis. Most cases involve transient self-limiting neurological deficits as seen in the featured patient. Some patients unfortunately suffer from permanent deficits and very rarely, death. [8,12] A retrospective analysis of nine patients with CIE demonstrated a mean neurological recovery time of 14.2 ± 6.7 h (range, 8-30 h). [13] In the featured patient, the hemiparesis failed to resolve until 4 days post-event, which appears to be atypical. However, in the aforementioned study, the mean age was 64.6 ± 7.8 years (range, 47-72 years) versus our patient who was 87 years old. This begs the question of whether increased age contributes to prolonged recovery time from CIE. It is also worth noting that of nine patients included in the study, only one was female and she happened to be the only case of persistent neurological deficits. This also prompts a question of whether the female gender has any correlation to prolonged recovery time and/or worse prognosis of CIE.

A controversial point in the management of this case is that the patient received an additional dose of intravenous contrast for CT angiography of the head and neck as part of her stroke workup. There is justification in the fact that CIE is a very rare diagnosis that had not yet been established in this patient and these investigations were ordered per protocol in the evaluation of possible stroke

which is a far more common diagnosis. Administration of contrast in a patient with known previous CIE should be done with extreme caution as there is potential for recurrence despite documented uneventful 're-challenge' with iodinated contrast. [14,15]

4. Conclusion

Because of its low prevalence and unpredictable course CIE has no formal diagnostic criteria or treatment regimen. In large part, treatment is based on anecdotal evidence or the treatment of the resulting side effects rather than the disorder itself. When patients ultimately develop symptoms of stroke a thorough workup is warranted, however, the clinician should be aware of judicious use of further contrast-based imaging. CT head without contrast may be utilized in suspected cases of CIE, as in some instances the contrast itself can be visualized intracranially further confirming the diagnosis of CIE. Additionally, the gadolinium contrast used in MRIs has not been correlated with CIE thus providing another possible imaging modality to rule out a stroke. [16] No formal treatment protocol exists for CIE; however, the disorder is self-limiting, and most patients have a recovery within 24-72 hours. [5] If patients have renal dysfunction this could prolong the time of resolution as the contrast is primarily renally excreted. In patients with renal dysfunction such as end-stage renal disease, urgent hemodialysis may be warranted. Overall, very little is known about this rare complication, treatment is focused on complications of CIE rather than directed therapy, and to prevent further damage with additional contrast load or inadvertently treating for stroke.

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