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Cerebral contrast retention after difficult cardiac catheterization: Case report

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Abstract

Background: We report a diagnostic dilemma in a rare case of cerebral contrast retention after difficult cardiac catheterization in an elderly patient loaded with prasugrel.

Summary: Our case report describes a 77-year-old female with history of hypertension, diabetes, and dyslipidemia who presented to emergency department complaining of chest pain. Patient was found to have an inferior wall ST elevation myocardial infarction. The patient was loaded with aspirin and prasugrel and taken for emergent cardiac catheterization. Cardiac catheterization revealed two-vessel coronary artery disease with unsuccessful attempt of percutaneous intervention. Immediately after procedure, patient developed an episode of seizure. Emergent computed tomography scan of the brain revealed hyperdensity in the right frontoparietal region consistent with intracerebral bleed. Repeat computed tomography (24 h later) revealed substantial interval improvement of hyperdensity. Follow-up magnetic resonance imaging of the head was normal. Given the lack of magnetic resonance imaging changes, the rate of resolution on computed tomography without expected subacute changes, and the lack of neurologic findings, the initial hyperdensity seen on computed tomography of the brain was believed to be secondary to contrast leakage during cardiac catheterization as opposed to intracranial hemorrhage.

Keywords

Coronary angiography, seizure, intracerebral contrast leakage, intracerebral hemorrhage

Learning objective

The learning objective of this case report is to tell the readers that with difficult cardiac catheterization, focal neurological signs can be due to contrast leakage via blood brain barrier and not all hyperdensity focus on computed tomography (CT) scan represents intracranial hemorrhage immediately after cardiac catheterization.

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Introduction

Although the overall rate of stroke associated with cardiac catheterization is low, it remains one of the most devastating complications, and it is associated with significant morbidity and mortality. Acute neurological complication following cardiac catheterization is rare and has been reported to be anywhere between 0.03% and 0.3%[1–3] for diagnostic cases and 0.3% and 0.4% for percutaneous coronary intervention (PCI).[4,5] Given the development and increased uses of newer anti-platelet agents bleeding risk is of increasing concern.[6] Heparin and intravascular iodixanol contrast medium are used for cardiac catheterization. The reported major bleeding after cardiac catheterization is 2.7%.[7] The most commonly associated reversible neurologic complication after cardiac catheterization is global amnesia or cortical blindness for adults,[8,9] and seizure or hemiplegia for children.[10,11] Contrast retention within the brain parenchyma after cardiac catheterization, likely secondary to blood–brain barrier (BBB) disruption, can mimic subarachnoid hemorrhage (SAH) on brain computed tomography (CT) scan[12] and thus present as a diagnostic dilemma.

Case

A 77-year-old female with past medical history significant for hypertension, diabetes, and dyslipidemia without prior history of cerebral vascular accident (CVA) or seizure disorder

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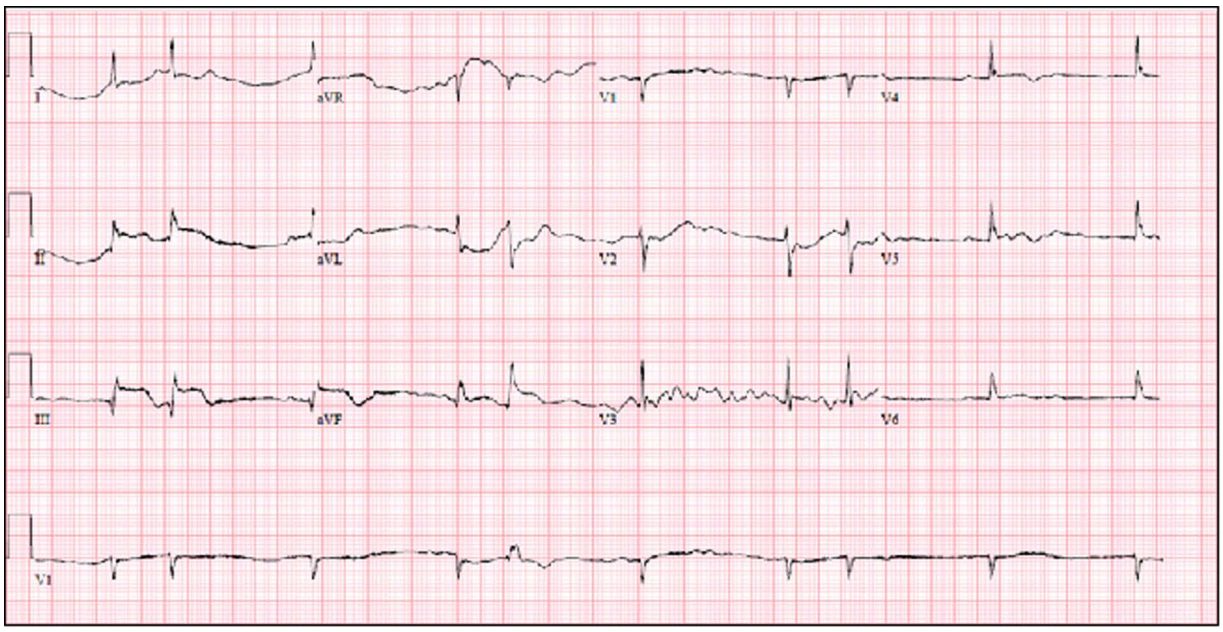

Figure 1. A 12-lead EKG shows atrial fibrillation with ST elevation in leads II, III, and aVF.
EKG: electrocardiogram; aVF: augmented vector foot.

presented to our emergency department complaining of on and off mid sternal non-radiating chest pain of 2 days duration. On presentation the admission electrocardiogram (EKG) showed ST elevation in the II, III, augmented vector foot (aVF) (Figure 1). The patient was treated as an ST elevation myocardial infarction (STEMI) case and was subsequently loaded with 325 mg aspirin and 60 mg prasugrel prior to emergent cardiac catheterization. Cardiac catheterization revealed 100% stenosis within the mid right coronary artery (RCA), and 60%–70% calcified stenosis of left proximal circumflex coronary artery (LCx). PCI of the RCA was attempted and subsequently aborted after multiple unsuccessful attempts at revascularization. Isovue contrast material of 150 mL and 2000 units of heparin were used. Immediately after the cardiac catheterization, patient developed an episode of tonic-clonic seizure and stroke code was activated. Patient's heart rate and blood pressure was 93 bpm (beats per minute) and 156/97 mmHg, respectively. Patient's serum creatinine was 0.99 mg/dL. Neurological exam did not reveal any focal neurological deficit. Patient was seen by neurology and loaded with dilantin. Due to concern for possible acute stroke, CT of the brain without intravenous (IV) contrast was done and it revealed hyperdensity (90 HU (Hounsfield unit)) in the right frontoparietal region with effacement of sulci which was read as SAH by radiologist (Figure 2(a)). CT angiogram of the brain/neck with 90 ml nonionic contrast was performed, which did not reveal any hemodynamically significant stenosis or intracranial aneurysm, and the patient was admitted to the coronary care unit for further observation and management. Antiplatelet therapy was held due to concern for presumed SAH. Early morning of the following day, the patient was found to be in complete heart block associated

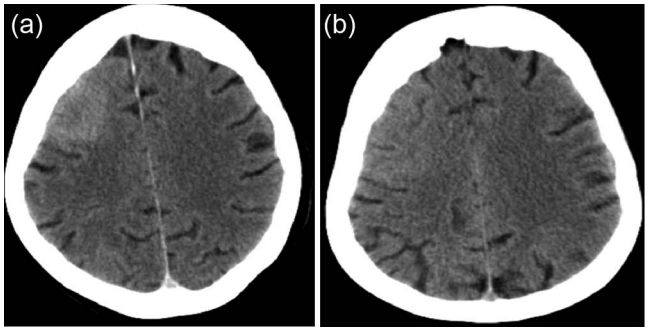

Figure 2. Day of cardiac catheterization: (a) CT scan of brain without IV contrast shows hyperdensity in the right frontoparietal region (90 HU) and (b) 24 h later CT scan of brain without IV contrast shows significant interval improvement of hyperdensity in right frontoparietal region since the first CT scan (30 HU) with more diffuse distribution.
CT: computed tomography; IV: intravenous.

with bradycardia to 40 s and hypotension to the 60 s/40 s necessitating the need for the insertion of temporary transvenous pacer, and the patient was placed on dopamine and levophed drip with subsequent intubation for hypoxemia. A repeat CT of brain without contrast 24 h after the initial CT showed significant interval improvement of the previously seen hyperdensity (30 HU) in the right frontoparietal region with a more diffuse distribution (Figure 2(b)). Her serum troponin level continued to rise and peaked on the second day of hospitalization at 71 ng/mL.

By the third day of her hospitalization, the patient was weaned off of pressors with the return of normal sinus rhythm. Transthoracic echocardiogram at that time showed a

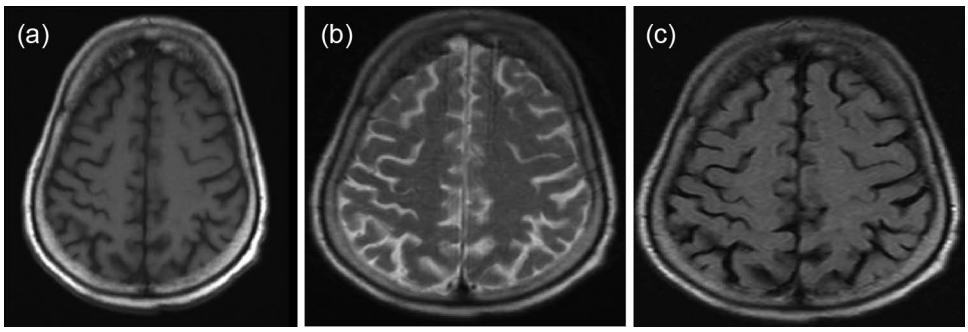

Figure 3. MRI images of brain T1, T2, and FLAIR sequences show no abnormal signal within brain parenchyma. (a) MRI T1 weighted image shows no abnormal signal within brain parenchyma, (b) MRI T2 weighted image shows no abnormal signal within brain parenchyma, and (c) MRI FLAIR image shows no abnormal signal within brain parenchyma.
MRI: magnetic resonance imaging; FLAIR: fluid-attenuated inversion recovery.

reduced left ventricular ejection fraction (LVEF) estimated at 30%–35% with akinesis of the inferior wall and hypokinesis of the lateral wall.

Once it has been established that the patient was able to maintain normal sinus rhythm, the temporary transvenous pacing wire was removed on day 4 of her hospitalization. After extubation and without further seizure episodes, magnetic resonance imaging/magnetic resonance angiogram (MRI/MRA) of brain was done on day 6 of admission. MRI could not performed early on as patient was intubated and was not stable for MRI. The MRI did not show any abnormal focus at T1, T2 or fluid-attenuated inversion recovery (FLAIR) sequences (Figure 3(a)–(c)) to suggest the presence of acute or subacute stroke. There was no aneurysm or vascular malformation noted on the MRI and after discussion with neurology, dilantin was stopped. On the day 8 of admission, the patient developed progressive tachycardia and tachypnea. Due to concern for possible pulmonary embolism (PE), ventilation/perfusion scan was performed, and the results were consistent with low probability for PE. Later on that same evening, the patient had an acute drop in her blood pressure and heart rate; emergent resuscitation and cardiopulmonary resuscitation (CPR) was initiated, the patient subsequently became asystolic and expired. Slow ventricular leak followed by ventricular rupture was the suspected cause of death. Autopsy was offered to but was ultimately refused by the family.

Discussion

Stroke is associated with significant morbidity and mortality and remains one of the most devastating complications of cardiac catheterization and may complicate the decision for the administration of anti-platelet therapy pre- and post-cardiac catheterization. Contrast agent neurotoxicity at times presents radiologically as intracranial hemorrhage.[7] To differentiate blood from contrast medium can be challenging on imaging studies. On CT scan, the Hounsfield measurements increase with increasing amounts of hemorrhage (0% blood, 0 HU; 100% blood, 66 HU).[13] The maximal density (Hounsfield units) of contrast medium is more than 90 HU on brain CT.[14] One way to differentiate blood from contrast material is through the use of MRI. On MRI, T1 relaxation times for blood/cerebrospinal fluid (CSF) mixtures decreases with increasing concentration of blood, ranging from 2200 to 500 ms for 100% CSF and 100% blood, respectively,[13] while T2 findings for CSF/blood mixtures are similar to T1 relaxation times.[13] With small SAH diluted with CSF, the identification is better with FLAIR MRI as opposed to CT scan.[15]

Both osmotic and chemical properties of the contrast media contribute towards contrast-induced neurotoxicity, which is usually transient and rapidly reversible.[7] The contrast agents can cause osmotic disruption of the BBB.[16] Additional risk factor for disruption of BBB and cerebral contrast retention includes renal insufficiency where the blood contrast concentration is increased secondary to reduced renal contrast clearance.[17] Non-ionic contrast media, for example,, iodixanol has a lower incidence of neurotoxic events, including seizures, and motor and speech deficits.[18,19,20]

Conclusion

With stroke being one of the most devastating complications of cardiac catheterization effecting pre- and post-cardiac catheterization's utilization of anti-platelet therapy, extravascular contrast leakage within the brain should be included in the differential diagnosis of focal neurologic deficit/seizure status post-cardiac catheterization. In the case of contrast leakage, the hyperdensity suggestive of bleed should improve rapidly on the subsequent CT images. MRI provides greater sensitivity for SAH and thus allows for discrimination between contrast leakage and SAH. MRI should be utilized for the confirmation of SAH to facilitate the management of post-cardiac catheterization patients. Our recommendation is to perform MRI immediately for suspicious bleed on CT scan status post-cardiac catheterization.

Declaration of conflicting interests

The authors declare that there is no conflict of interest.

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