Contrast-Induced Encephalopathy Following Diagnostic Coronary Angiography

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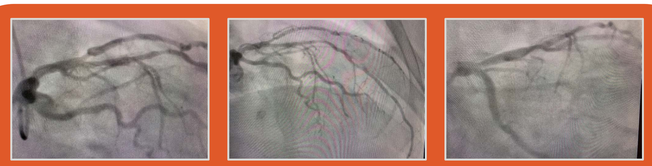
Background

- Contrast-Induced Encephalopathy (CIE) is a rare, reversible complication of coronary angiography that mimics strokes.
- Recent advancements in coronary angiography reduced the complication rate of diagnostic procedures to less than 1%.
- Previous reports of complications focus on arrhythmias, myocardial infarctions, and ischemic strokes increasing anchoring bias.

Figure 1. Diagnostic Coronary Angiography

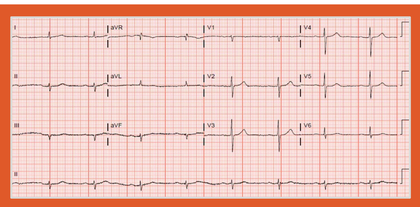


1. Left ventriculography: EG >60%.
2. Left Main: Patent with <20% stenosis
3. LAD: Patent stent <50% in-stent restenosis with diffuse 60-70% mid LAD stenosis. Large first diagonal branch with 50% proximal stenosis.
4. LCx: Nondominant with 1 large OM branch that has diffuse disease with a 70% proximal stenosis.
5. RCA: Dominant with diffuse atherosclerotic disease with <50% stenosis.

Case Presentation

- **HPI:** A 76-year-old Caucasian male with DM2, HTN, HLD, and CAD presented to our ED complaining of acute-onset altered mental status for 3 hours.
- **Cardiac history:** One month ago, he had an outpatient workup for intermittent, substernal chest pain that resolved with rest. Previous PCI w/ DESx1 in the LAD. Nuclear stress testing showed moderate probability reversible apico-lateral ischemia prompting coronary angiography 4 days ago that revealed multivessel CAD (figure 1).
- **Family hx:** Father: rapidly progressive dementia w/ jaundice
- **ROS:** Unable to obtain (refused to answer)
- **Pertinent Physical Exam Findings:**
  - T:98.6°F, HR:82bpm, RR:20bpm, BP:187/123(127)mmHg, SpO2: 96% on room air, WT: 121.561 kg
  - CV: RRR, no murmurs, no carotid bruits, no JVD
  - Neuro: AOx0 and Agitated
- **Pertinent Lab Findings**:
  - Hyperglycemic at 202mg/dL (N:70-110)
  - Magnesium of 1.2mg/dL (N:1.8-2.4)

Figure 2. Admission ECG

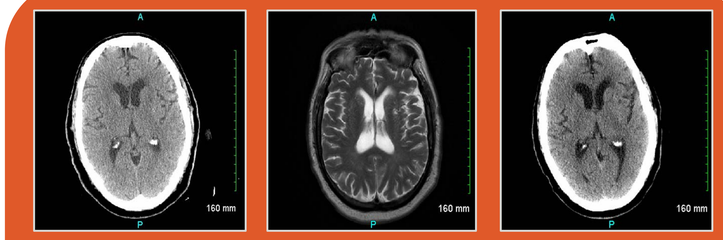


1. Sinus Bradycardia (51bpm)
2. Junctional Rhythm
3. Left Axis Deviation

Hospital Course

- Initial **CTA head and neck** were negative (figure 3a).
- He was admitted to the cardiology service.
- His **HTN resolved** with hydralazine 10mg IVx1, magnesium was repleted, and blood glucose was treated with insulin.
- His cognitive status continued to decline and he **became combative requiring restraints**.
- He developed transient nocturnal **fevers up to 102°F**.
- **All studies of urine, blood and CSF were negative**.
- **MRI Brain** showed white matter changes (figure 3b).
- A **TTE** for concerns of CVA showed normal systolic function with trace mitral, tricuspid, and pulmonic regurgitation.
- **Neurology** and **Infectious Diseases** were consulted to differentiate acute delirium, heavy metal poisoning, viral encephalitis, and rubral tremors.
- On day 6, he developed **dystonic movements** concerning for seizure activity.
- **Repeat CT Head** was negative (figure 3c).
- An **EEG** showed no epileptiform activity.
- Daily lab testing was monitored and revealed no abnormalities throughout his hospital stay.
- His confusion and aggressive behavior persisted until day 10, when he began asking questions and showed signs of being intermittently lucid.
- **By day 16, he was persistently lucid with no recollection of his hospital stay**.
- After excluding all likely alternatives, CIE was diagnosed.
- At a 3-month follow-up, he denied symptom recurrence, reported medication compliance, and decline further evaluation for a possible CABG.

Figure 3. Neurological Imaging



3A; Day 1 CT Head
No acute pathology.

3B; Day 2 MRI Brain
Periventricular white matter change seen on T2/FLAIR.

3C; Day 10 CT Head
No acute pathology

Discussion

- This case highlights the importance of maintaining a wide differential in order to arrive at a diagnosis of exclusion.
- The patient was initially evaluated for a possible stroke with hypoglycemia quickly ruled out and electrolytes repleted. As his symptoms persisted despite optimal medical therapy, rarer etiologies were explored.
- CIE is a rare, self-limiting complication of contrast exposure with variable symptom onset and duration best studied in strokes, where hyperosmolar contrast may disrupt the blood-brain barrier.
- Previous research identifies multiple risk factors for CIE in our patient, including male gender, hypertension, and a large volume injection of contrast used for left ventriculography.
- Symptom onset has been reported to begin within a few hours of exposure, and lasting up to five days.
- Documented radiographic findings in the literature range from none at all to cortical edema with contrast enhancement of parenchyma and subarachnoid tissue in CIE following traditional neurological angiography.
- Additional concerns for etiologies of encephalopathy following coronary angiography include evaluation for aortic atherosclerosis and reducing the overall contrast volume including especially during left ventriculography as our patient received.
- Treatment focuses on adequate hydration, IV steroids and anticonvulsants.
- CIE following coronary angiography warrants additional research regarding disease course, as the prevalence of cardiovascular disease continues to grow globally.

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