American Journal of Case Reports

e-ISSN 1941-5923
© Am J Case Rep, 2019; 20: 776-779
DOI: 10.12659/AJCR.916114

Received: 2018.03.08
Accepted: 2019.03.31
Published: 2019.06.02

Simultaneous Acute Anterior ST-Elevation Myocardial Infarction and Acute Ischemic Stroke of Left Middle Cerebral Artery: A Case Report

Authors' Contribution:
Study Design A
Data Collection B
Statistical Analysis C
Data Interpretation D
Manuscript Preparation E
Literature Search F
Funds Collection G

EF Omar Obaid
EF Hye Rim Smith
EF Daniel Brancheau

Department of Cardiology, Genesys Hospital, Grand Blanc, MI, U.S.A.

Corresponding Author: Omar Obaid, e-mai: omar.obaid@ascension.org
Conflict of interest: None declared

Patient: Female, 41
Final Diagnosis: Acute ischemic stroke and ST elevation myocardial infarction
Symptoms: Chest pain • facial droop
Medication: —
Clinical Procedure: —
Specialty: Cardiology

Objective: Rare co-existance of disease or pathology

Background: Acute ST-elevation myocardial infarction and acute ischemic stroke are both life-threatening conditions with high risk for morbidity and mortality without timely intervention. This simultaneous event has been reported to be as rare as 0.009%. The treatments of both conditions individually have been well documented in the literature and guidelines, but when presenting concomitantly, it poses a unique therapeutic challenge. Immediate treatment of one condition can delay management or even have deleterious effects on the other condition.

Case Report: We present the case of a 41-year-old female with simultaneous acute anterior ST-elevation myocardial infarction and acute left middle cerebral artery ischemic stroke. Due to a low National Institute of Health score she was not a candidate for endovascular treatment and received alteplase per acute ischemic stroke protocol with delayed percutaneous coronary intervention. She was eventually discharged to a long-term acute care facility for continued rehabilitation.

Conclusions: The co-existence of ST elevation myocardial infarction and acute ischemic stroke is rare, but when these 2 common conditions present simultaneously, it provides a unique therapeutic challenge. Although infrequent, this challenging scenario deserves more recognition and a discussion among the medical community.

MeSH Keywords: Anterior Wall Myocardial Infarction • Stroke • Tissue Plasminogen Activator

Full-text PDF: https://www.amjcaserep.com/abstract/index/idArt/916114

Indexed in: [PMC] [PubMed] [Emerging Sources Citation Index (ESCI)] [Web of Science by Clarivate]

Background

ST-elevation myocardial infarction (STEMI) and acute ischemic stroke are both life-threatening conditions. The simultaneous presentation of these, an entity coined by Omar et al. and called acute cardio-cerebral infarction [1], presents a challenge in terms of immediate management. While this is an infrequent occurrence, the physician must be prepared to address both conditions to prevent mortality, avoid irreversible disability, decrease complications (notably bleeding), and minimize delayed interventions [2]. The incidence of acute cardio-cerebral infarction has been estimated to be as low as 0.009% [3]. Although rare, this challenging scenario deserves more recognition and a discussion among members of the medical community.

Case Report

A 41-year-old female presented to the Emergency Department (ED) with a syncopal event and a new facial droop. She had a history of poorly controlled diabetes mellitus, hypertension, hyperlipidemia, nicotine dependence, peripheral arterial disease, and an extensive cardiovascular history which included premature coronary artery disease with previous myocardial infarction at age 31 without any angioplasty due to collateral circulation and chronic total occlusion of the right coronary artery. She also had undergone previous percutaneous coronary intervention with a drug-eluting stent to the mid left anterior descending (LAD) artery 5 years prior for unstable angina, she had 2 drug-eluting stents placed for in-stent stenosis of LAD, and she had new native disease of the left circumflex artery 4 months prior, for treatment of unstable angina. Her history was also significant for an ischemic stroke with residual right-sided hemiparesis. She was maintained on dual antiplatelet therapy with aspirin 81 mg and clopidogrel 75 mg daily in addition to beta blocker, statin therapy, long-acting nitrates, angiotensin-converting enzyme inhibitor, and calcium channel blockers.

Initial vital signs in the ED demonstrated a blood pressure 114/62 mm Hg, heart rate of 83 beats per minute, respiratory rate of 16 breaths per minute, temperature of 36.9°C (98.4°F), and oxygen saturation of 96% on room air. Physical examination was notable for diminished pulses (1+) in the radial, carotid, and dorsalis pedis bilaterally. The patient was lethargic but responsive to noxious stimuli. She was able to move all 4 extremities but would not cooperate with strength testing. Her initial National Institute of Health (NIH) stroke scale score was 6.

A computerized tomography (CT) of the brain with perfusion was obtained and upon return her NIH score was 3. She began complaining of retrosternal chest pain, and the electrocardiogram demonstrated an anterior STEMI (Figure 1). Simultaneously, her CT scan results showed an acute ischemic stroke in the left middle cerebral artery territory (Figure 2). Due to the low NIH score, she was not a candidate for endovascular treatment. A multidisciplinary discussion regarding balancing the risk of bleeding with salvaging brain and myocardial tissue took place. She was subsequently treated with intravenous alteplase as per acute ischemic stroke protocol (0.9 mg/kg, total dose 75 mg). Follow-up CT scan demonstrated no evidence of hemorrhagic conversion.

Upon admission to the intensive care unit, the patient became more hypoxic and hypotensive requiring intubation and mechanical ventilator support along with vasopressor support with norepinephrine. A transthoracic echocardiogram revealed an ejection fraction of 30–35% associated with an akinetic anterior wall and apex without presence of a left ventricular thrombus. Conservative medical treatment was implemented for her STEMI, limited by her continued need for vasopressor support. In addition, all antiplatelet and anticoagulants were held due to the post alteplase state. About 48 hours after admission, she was successfully weaned off the ventilator but shortly after developed a flash pulmonary edema with increasing respiratory distress requiring re-intubation. At this time, an electrocardiogram was obtained showing increased ST-segment elevations in the anterior leads that prompted emergent coronary angiography. An occluded stent in the mid LAD artery was visualized (Figure 3), and percutaneous coronary intervention was performed with 2 drug-eluting stents implanted.

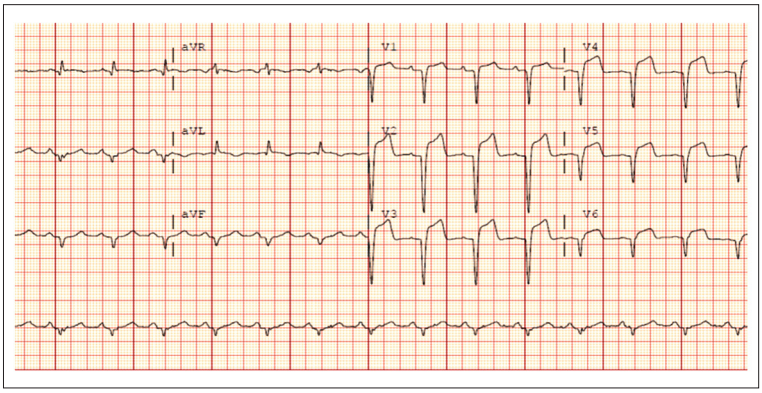

Figure 1. Electrocardiogram (ECG) demonstrating ST elevation in the precordial leads consistent with an anterior myocardial infarction. The patient's ECG demonstrates Q waves in the anterior leads.

Indexed in: [PMC] [PubMed] [Emerging Sources Citation Index (ESCI)] [Web of Science by Clarivate]

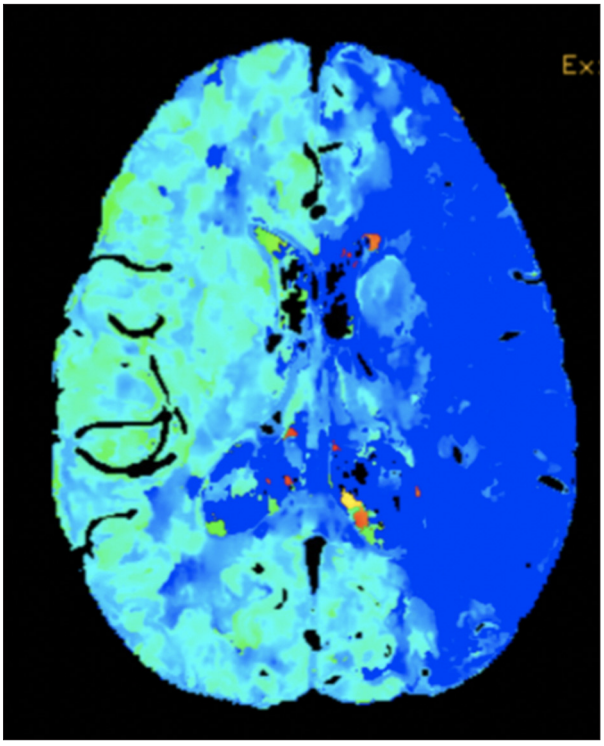

Figure 2. Computed tomography brain with contrast for perfusion demonstrating large left MICA (middle intracerebral artery) distribution penumbra without core infarct.

The patient was restarted on dual antiplatelet therapy with aspirin and clopidogrel. The patient remained in the intensive care unit recovering from ventilator support and her chronic medical conditions.

Ten days later, the patient suffered a pulseless electrical activity and subsequent ventricular fibrillation cardiac arrest. After return of spontaneous circulation, ST-elevations were noted in the anterior leads prompting emergent cardiac catheterization which demonstrated a subacute proximal LAD stent thrombosis and was treated with aspiration thrombectomy and balloon angioplasty. Post-operatively, her clopidogrel was changed to ticagrelor. After a 27-day hospitalization, she was discharged to a long-term acute care facility for tracheostomy care, and she remained aphasic with right hemiplegia but no recurrence of chest pain. The patient's medications upon discharge included dual antiplatelet therapy with aspirin 81 mg daily and ticagrelor 90 mg twice daily, in addition to low dose carvedilol and high intensity atorvastatin.

Discussion

There are multiple theories for how acute cardio-cerebral infarction may occur. One theory is via the formation of a left

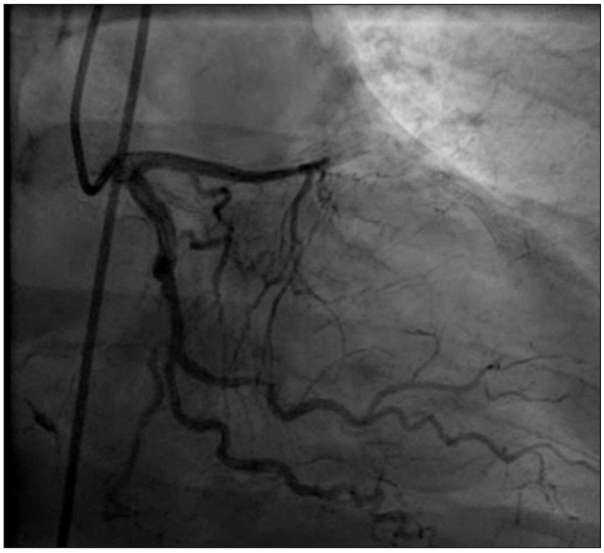

Figure 3. Cardiac catheterization demonstrating an occluded mid left anterior descending stent occluded with faint left to right collaterals.

ventricular mural thrombus [4]. One theory is that acute myocardial infarction, especially anterior and apical wall infarctions and their associated left ventricular systolic dysfunction, provide the substrate for the thrombus and increase the risk of embolization [4]. A similar theory is that the adrenergic surge associated with acute ischemic stroke may result in catecholamine-induced myocardial stunning and intracardiac thrombus formation [4].

The American Heart Association and American Stroke Association in 2018 recommended that in the setting of hyperacute simultaneous cardio-cerebral infarction, treatment with intravenous alteplase at the dose used for cerebral ischemia followed by percutaneous coronary intervention is reasonable (Class IIa; level of evidence C) [5]. However, the different dose requirements and timing of fibrinolytic treatment often hinder the use of alteplase as definitive treatment for both conditions [5–7].

Conclusions

Ultimately the treatment approach for simultaneous STEMI and acute ischemic stroke is a technically challenging scenario that involves high risk of morbidity and mortality for the patient. The diffuse array of treatment options including, but not limited to, endovascular therapy, alteplase at acute ischemic stroke dosing, alteplase at STEMI dosing, and percutaneous coronary intervention requires an individualized treatment approach, and requires further research designed at identifying an optimal treatment algorithm.

Indexed in: [PMC] [PubMed] [Emerging Sources Citation Index (ESCI)] [Web of Science by Clarivate]

References:

1. Omar HR, Fathy A, Rashad R, Helal E: Concomitant acute right ventricular infarction and ischemic cerebrovascular stroke; Possible explanations. Int Arch Med, 2010; 3: 25
2. Akinseye OA, Shahreyar M, Heckle MR, Khouzam RN: Simultaneous acute cardio-cerebral infarction: is there a consensus for management? Ann Transl Med, 2018; 6: 7
3. Yeo L, Andersson T, Yee K et al: Synchronous cardiocerebral infarction in the era of endovascular therapy: Which to treat first? J Thromb Thrombolysis, 2017; 44: 104–11
4. Kijpaisalratana N, Chutinet A, Suwanwela NC: Hyperacute simultaneous cardiocerebral infarction: rescuing the brain or the heart first? Front Neurol, 2017; 8: 664
5. Powers W, Rabinstein A, Ackerson T et al: 2018 Guidelines for the early management of patients with acute ischemic stroke: A guideline for healthcare professionals from the American Heart Association/American Stroke Association. Stroke, 2018; 49(3): e46–99
6. O'Gara P, Kushner F, Ascheim D et al: 2013 guidelines for the management of ST-elevation myocardial infarction: A report from the American College of Cardiology Foundation/American Heart Association Task Force on Practice Guidelines. Circulation, 2013; 127(4): e362–425
7. Yaghi S, Willey J, Cucchiara B: Treatment and outcome of hemorrhagic transformation after intravenous alteplase in acute ischemic stroke: A scientific statement for healthcare professionals from the American Heart Association/American Stroke Association. Stroke, 2017; 48(12): e343–61

Indexed in: [PMC] [PubMed] [Emerging Sources Citation Index (ESCI)] [Web of Science by Clarivate]