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Simultaneous cardiac and cerebral infarction – a case report

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ABSTRACT

Introduction and aim. Concurrent cardiocerebral infarction (CCI) is a rare condition defined by the simultaneous presentation of acute myocardial infarction (AMI) and acute ischemic stroke (AIS). This case report aims to illustrate the clinical presentation, diagnostic challenges, and treatment considerations in a patient with CCI.

Description of the case. We describe the case of a 61-year-old Asian patient with symptoms of AMI, which was successfully treated with primary percutaneous coronary intervention (PCI). Six hours after the initial presentation, the patient developed symptoms of AIS. Imaging revealed an acute infarct in the left globus pallidus and small lacunar infarcts in the left thalamus region. Due to the location of the cerebral infarct, the patient was managed conservatively for AIS. The patient showed a positive response to the treatment, with no recurrence of chest pain or neurological symptoms observed at the six-month follow-up.

Conclusion. This case emphasizes the importance of prompt brain imaging to distinguish between different types of stroke and highlights the challenges in managing CCI, a condition linked to high mortality and morbidity. Early recognition and tailored therapy are crucial for improving prognosis.

Keywords. acute ischemic stroke, acute myocardial infarction, cardio-cerebral infarction, percutaneous coronary intervention

Introduction

Acute myocardial infarction (AMI) is diagnosed based on the presence of elevated cardiac enzymes, ischemic symptoms, ECG changes, loss of viable myocardium on non-invasive testing, or the presence of a coronary artery thrombus on angiography. Acute ischemic stroke (AIS) is characterized by the sudden onset of a focal neurological deficit caused by an acute focal injury to the central nervous system caused by arterial occlusion or thromboembolism.[1] Cardio-cerebral infarction (CCI) is a rare condition that occurs

when AMI and AIS happen simultaneously. The overall prevalence of CCI is low, with few cases having been reported in the literature.[2-4]

Description of the case

A 61-year-old patient with a history of hypertension, diabetes mellitus, and stage 3A chronic kidney disease (CKD) presented to our emergency department with two hours of left-sided constricting chest pain and profuse sweating. He had no previous history of stroke or ischemic heart disease. Upon admission, vital signs were stable, with a blood pressure of 160/100 mmHg and a heart rate of 50 beats per minute. ECG showed ST elevation in leads II, III, and aVF, with reciprocal ST depression in V1–V3 (Fig. 1).

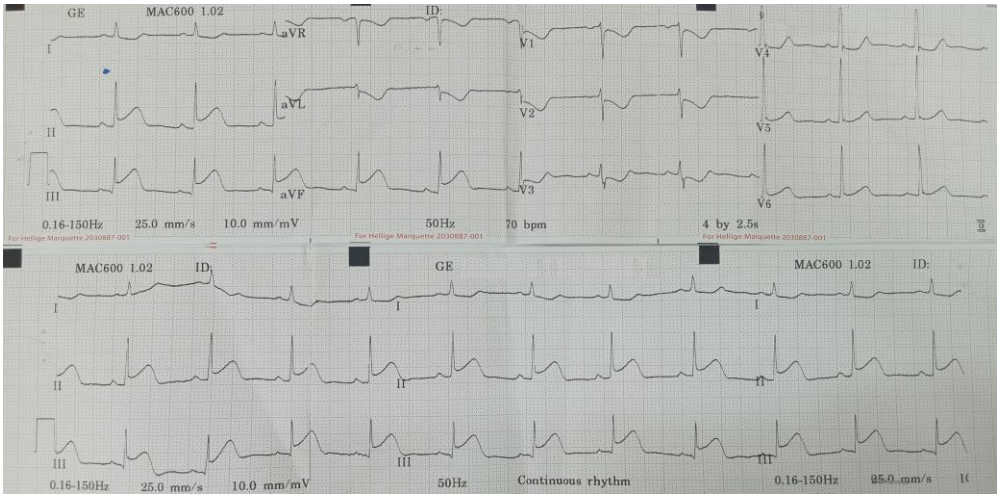

Fig. 1. ECG showed ST elevation in II, III and aVF with reciprocal ST depression in V1–V3

Troponins were elevated (540 ng/mL). Coronary angiogram revealed total occlusion of the dominant right coronary artery with thrombus, while the left coronary system was normal. A JR4 6F guiding catheter and a Fielder FC 0.014-inch wire were used to access the right coronary artery, followed by balloon angioplasty using a 2.5×15 mm coronary balloon inflated to 14 atm for 5 seconds. Two sirolimus-eluting stents (DES) (3.5×27 mm and 2.75×37mm) were implanted, followed by post-dilation with an NC Accuforce 3.5×15 mm balloon at 18 atm. A final angiogram confirmed successful PCI to RCA-PDA with two DES, resulting in complete distal flow (Fig. 2A, 2B).

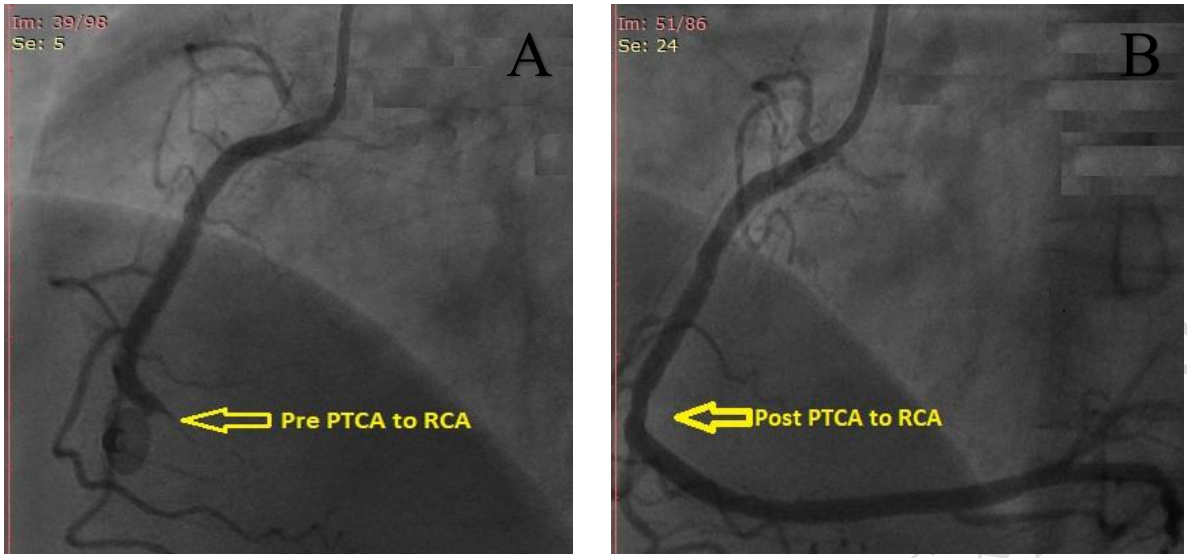

Fig. 2. A: Total occlusion of the dominant right coronary artery with thrombus, 2B: PCI to RCA-PDA with 2 DES resulting in TIMI 3 flow distally

Six hours post- PCI, the patient suddenly developed neurological symptoms, including memory impairment, reduced consciousness, disorientation, right-sided hemiparesis, right upper motor neuron facial palsy, and a right extensor plantar response, with a National Institutes of Health Stroke Scale (NIHSS) score of 15. A magnetic resonance imaging (MRI) revealed acute ischemic lesions in the left globus pallidus and multiple small acute lacunar infarcts in the left thalamus region and lenticulostriate branches of the left middle cerebral artery (MCA) territory (Fig. 3A–D).

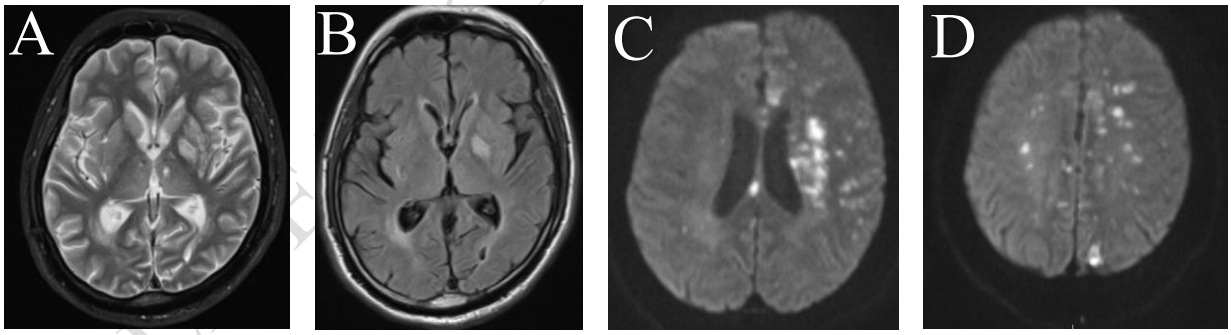

Fig. 3. A and B: Acute lacunar infarct in left globus pallidus, C and D: Small acute lacunar infarcts in left thalamus

Given the recent AMI and infarct location, a conservative approach was taken for AIS. The patient was discharged on the seventh post-procedure day, showing gradual improvement in memory and orientation. Continuous ECG monitoring during hospitalization showed no atrial fibrillation or arrhythmias. At three- and six- month follow-ups, the patient remained well, with no recurrence of chest pain or neurological symptoms. The modified Rankin Scale (mRS) at six months was 1, indicating slight disability but overall

favorable functional recovery. Serial ECGs demonstrated normal sinus rhythm without new ischemic changes.

Discussion

The term "concurrent cardio-cerebral incident (CCI)" was first introduced by Omar et al. in 2010 to describe the co-occurrence of AMI and AIS.[5] CCI can manifest as either synchronous, where both events occur simultaneously, or metachronous, where one precedes the other.[6] Type 1 CCI refers to simultaneous or within 12 hours of each other, with subcategories: type IA (cardiac factors responsible), type IB (brain-related factors), and type IC (neither cardiac nor brain-related factors). Type 2 involves AIS within 4.5 hours of recent AMI, but more than three hours after onset. Type 3 CCI involves AMI within 12 hours of recent AIS, but more than 4.5 hours after AIS.[7] Our case fits into Type 1 CCI, with symptoms of AIS six hours after MI onset.

The prevalence of CCIs varies from 0.009% to 0.29% with AIS following AMI occurring in 0.7% to 2.2% of hospitalized patients with peak occurrence in the early days post-AMI, but remains elevated for up to 12 weeks post-MI.[1,2,7,8] A single-center study by Olivier Hachet found that most stroke and TIAs occur within the first five days post-AMI, with 52.8% occurring on the first day and 87% within five days.[9] In our case, the development of neurological symptoms within six hours post-PCI highlights this early period and emphasizes the need for vigilance in the immediate post-revascularization phase. In addition, studies by Chin et al. and Yeo et al. have demonstrated that 6-12.7% of patients with acute stroke have a history of recent AMI.[6,10] A retrospective study revealed that among over 11 million patients hospitalized for AMI between 2000 and 2017, 1.6% developed AIS within 24 hours.[3] CCIs are associated with high mortality; meta-analyses report in-hospital mortality of 33.3% and a three-month mortality of 49.2%.[11]

The overlap of risk factors such as hypertension, hypercholesterolemia, smoking, diabetes, and advanced age contributes to both conditions via inflammation and atherosclerosis.[12] Our patient's hypertension, diabetes, and CKD likely contributed both coronary and cerebral artery disease. The pathophysiology of CCI involves mechanisms like intra-cardiac thrombogenesis due to coronary vasospasm, ventricular dysfunction, atrial fibrillation, and type A aortic dissection, as well as thrombotic viral infections, like COVID-19.[1,4,13] Cardiomyopathies, such as left ventricular non-compaction, predispose to intra-cardiac thrombus and systemic embolization.[14] Furthermore, right ventricular infarction or extensive MI complicated by cardiogenic shock can rapidly compromise hemodynamics, leading to watershed brain infarction (hemodynamic stroke) in patients with a prolonged history of hypertension.[5]

Management of CCIs requires a multidisciplinary approach. Prompt brain imaging is essential to determine stroke type; MRI is often necessary when CT is inconclusive, as seen in our case.[15] In metachronous CCIs, treatment focuses on the initial event. Both conditions carry high mortality and narrow therapeutic windows, so delays can worsen outcomes. Furthermore, thrombolytic therapy for AIS may increase the risk

of cardiac rupture post-MI, although two large studies (SMART and SITS-MOST) show no significant difference.[16] The use of anticoagulants and antiplatelet agents during PCI may also increase the risk of thrombolysis-associated hemorrhagic conversion in AIS. Therefore, individualized treatment plans are vital.

The American Heart Association/American Stroke Association (AHA/ASA) recommends that stable patients with hyperacute CCI without contraindications receive IV alteplase followed by PCI (Class IIa: level of evidence C).[17] However, the 2019 European Stroke Organization (ESO) guidelines advise against IV alteplase within 4.5 hours in patients with recent MI; instead, mechanical thrombectomy and PCI are recommended.[18] Our conservative approach was guided by infarct location, size, and bleeding risk, with mechanical thrombectomy contraindicated due to the infarct's deep location, and thrombolysis avoided because of recent MI. Instead, we prioritized dual antiplatelet therapy and supportive care, leading to neurological improvement.

In CCIs, dual antiplatelet therapy (DAPT) should continue for 12 months, then transition to single antiplatelet therapy (SAPT) for life. When cardioembolic stroke accompanies CCI, especially with atrial fibrillation, triple therapy (aspirin, clopidogrel, and a non-vitamin K oral anticoagulant, NOAC or warfarin) is recommended for one week, then dual therapy (SAPT and NOAC) for 6–12 months, depending on bleeding risk. Long-term, single-agent therapy is advised. The MIRACL study and GRACE registry have shown that early high-intensity statins reduce recurrent non-fatal ischemic stroke in AMI, by lowering low-density lipoprotein cholesterol (LDL-C) by over 50%, aiming for levels below 1.4 mmol/L (55 mg/dL).[7]

Key clinical messages

- CCI needs timely recognition and a team-based approach due to the challenges posed by simultaneous AMI and AIS.
- Immediate brain imaging, especially MRI, is crucial for diagnosing AIS after AMI, guiding treatment decisions and outcomes.
- Treatment should be personalized based on the timing and type of infarcts, while carefully weighing thrombolysis risks.
- Ongoing monitoring and follow-up are important as patients may gradually improve in neurological function after CCI despite initial severity.
- Increasing clinician awareness of CCI can enhance early detection and improve management, reducing mortality and morbidity rates.

Conclusion

Concurrent CCI is a rare and complex condition requiring a multidisciplinary approach. Prompt brain imaging is critical for accurate diagnosis. In this case, conservative stroke management was chosen due to

cerebral infarct location, which contraindicated mechanical thrombectomy. The patient was maintained on DAPT for 12 months, then transitioned to SAPT. Early recognition and management are vital to reduce high mortality and morbidity. Despite progress, gaps remain in evidence-based guidelines for managing CCI, particularly concerning the timing and choice of thrombolytic and revascularization strategies. Further research should focus on establishing standard guidelines through large-scale prospective studies for better patient outcomes.

Declarations

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Author contributions

Conceptualization, M.T.K.; Validation, M.T.K.; Formal analysis, M.T.K.; Investigation, M.T.K.; Resources, M.T.K.; Data curation, M.T.K.; Writing – Original Draft Preparation, M.T.K.; Writing – Review & Editing, M.T.K.

Conflicts of interest

There are no conflicts of interest.

Data availability

No new data generated.

Ethics approval

This case report does not require ethical approval because it is not considered human subject research.

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