American Journal of Case Reports

e-ISSN 1941-5923
© Am J Case Rep, 2026; 27: e952855
DOI: 10.12659/AJCR.952855

Received: 2026.01.19
Accepted: 2026.06.03
Available online: 2026.07.10
Published: 2026.XX.XX

# Simultaneous Acute Multiple Ischemic Strokes of the Left Cerebral Hemisphere and Signs of Acute Anterior STEMI: 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

ABCDEF Xiaotong Wei
ACDG Zhonghua Liu

The First School of Clinical Medicine, Guangdong Medical University, Zhanjiang, Guangdong, PR China



**Corresponding Author:** Zhonghua Liu, No. 2 Sunwen East Road, Shiqi District, Zhongshan City, Guangdong Province, China, Phone: 0086-13923308898, e-mail: zhonghua_reha@163.com

**Financial support:** None declared
**Conflict of interest:** None declared

**Patient:** Male, 62-year-old
**Final Diagnosis:** Acute multiple infarcts in the left cerebrum • acute myocardial infarction • hemiplegia of the right limb • limited ability in activities of daily living • limited social participation ability • sudden cardiac arrest
**Symptoms:** Right-sided limb weakness • slurred speech
**Clinical Procedure:** —
**Specialty:** Cardiology • Neurology • Rehabilitation

**Objective:** Rare coexistence of disease or pathology

**Background:** Acute cerebral infarction and acute myocardial infarction are both common life-threatening conditions with high incidence rates. The simultaneous occurrence of these conditions is extremely rare. Most documented cases are limited to case reports, and no specific treatment guidelines have been established. Treatment strategies considerably vary among individuals; the prognosis is often poor, particularly in older patients. Studies of cardio-cerebral infarction are important to reduce mortality and disability.

**Case Report:** A 62-year-old man was admitted to the hospital with right-sided limb weakness and inability to speak for more than 4 hours, without chest pain. Electrocardiographic findings were consistent with acute anterior ST-segment elevation myocardial infarction. Computed tomography angiography revealed acute occlusion of the left internal carotid artery, and magnetic resonance imaging demonstrated multiple infarctions in the left cerebral hemisphere. The patient had a National Institutes of Health Stroke Scale score of 13 and underwent emergency thrombectomy of the left internal carotid artery. He recovered well after the procedure, with only mild hemiplegia, and regained independence in activities of daily living. However, during the stable recovery phase, he suddenly experienced cardiac and respiratory arrest, resulting in death.

**Conclusions:** Although cardio-cerebral infarction is rare, it is associated with a very high mortality rate. Survivors often have poor outcomes, underscoring the need for greater clinical awareness. This case highlights the importance of early coronary artery evaluation even in patients with asymptomatic myocardial infarction, given that cardio-cerebral infarction can increase cardiogenic shock risk. The rehabilitation phase should also receive close attention, with appropriate monitoring to detect potentially fatal complications.

**Keywords:** Cardiology • Case Reports • Cerebral Infarction • Myocardial Infarction • Stroke • Thrombectomy
**Full-text PDF:** https://www.amjcaserep.com/abstract/index/idArt/952855





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

## Introduction

Cardio-cerebral infarction (CCI) was first described in 2010 as the simultaneous occurrence of acute ischemic stroke and acute myocardial infarction. It is classified as synchronous (both conditions occur simultaneously) or metachronous (1 condition precedes the other). CCI is relatively rare in clinical practice, with most published evidence limited to case reports; no specific treatment guidelines have been established [1]. Although acute cerebral infarction and acute myocardial infarction are both common diseases with high incidence and mortality rates, the reported incidence of CCI is extremely low (approximately 0.009%) [2,3]. Coexistence of these conditions may reflect an underlying pathophysiological association, rather than a coincidental simultaneous occurrence. Studies of CCI diagnosis, treatment, and prognosis, along with analyses of reported cases, may help establish evidence-based diagnostic and treatment strategies for this condition [4].

## Case Report

A 62-year-old man was admitted to the emergency department with right-sided weakness and aphasia lasting for more than 4 hours. He had a history of lacunar infarction without residual deficits and a 30-year smoking history. He denied a history of hypertension, diabetes mellitus, or previous surgery.

On admission, his blood pressure was 145/80 mm Hg. He was conscious but presented with mixed aphasia. Muscle strength was grade 1/5 in the right upper and lower limbs and grade 4+/5 in the left upper and lower limbs. Bilateral Babinski signs were present. The National Institutes of Health Stroke Scale score [5] was 13, and the pre-stroke modified Rankin Scale score was 0.

Emergency laboratory testing revealed a troponin T level of 299 ng/L (normal, < 50 ng/L) and an N-terminal pro-B-type natriuretic peptide (NT-proBNP) level of 153 pg/mL (normal, < 125 pg/mL). Electrocardiography demonstrated sinus rhythm with findings suggestive of acute anterior ST-segment elevation myocardial infarction (STEMI), as well as evidence of prior inferior and right ventricular myocardial infarction (**Figure 1**). Although limited by poor image quality due to pulmonary interference, bedside echocardiography demonstrated mild mitral and tricuspid regurgitation, incoordinate left ventricular wall motion, preserved systolic function, impaired diastolic function, and otherwise normal cardiac structure. Takotsubo

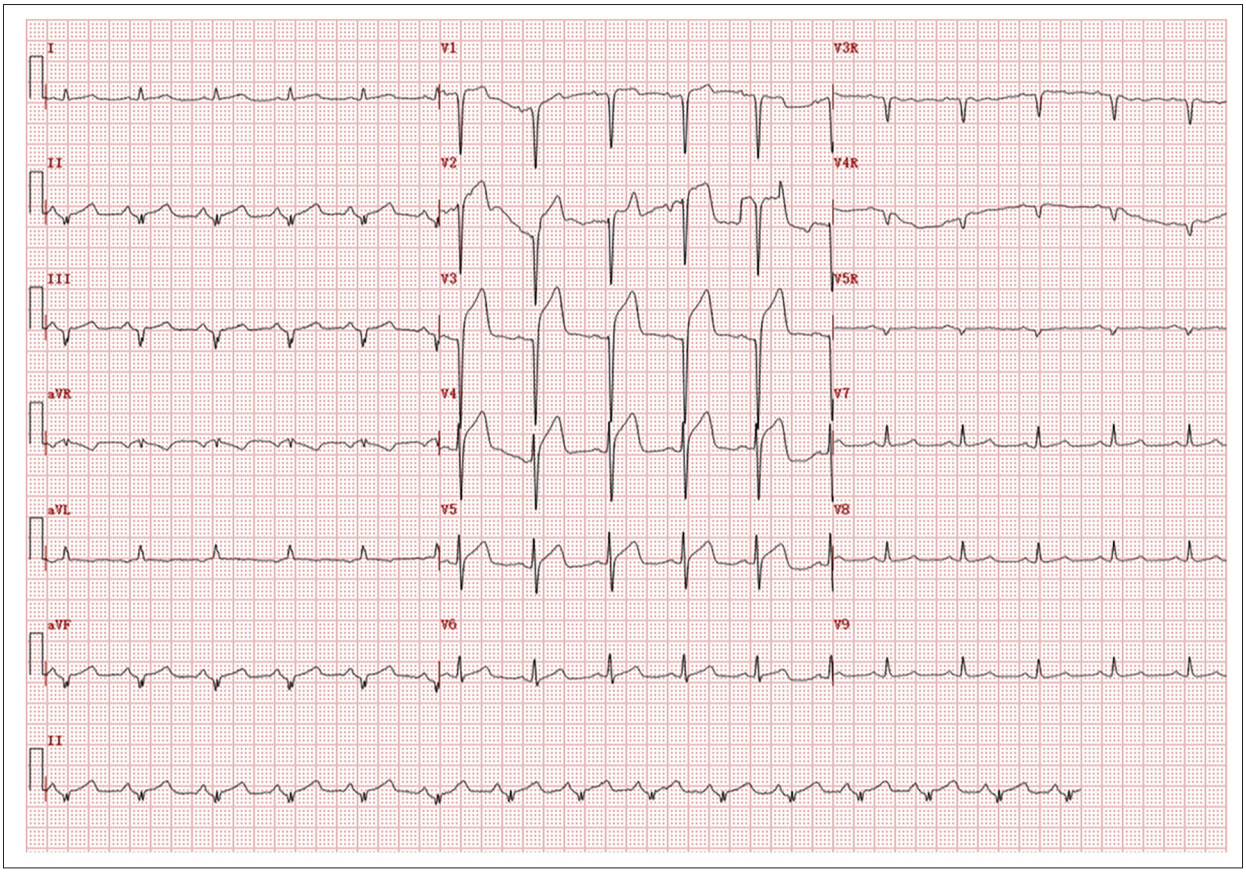


**Figure 1.** Electrocardiogram demonstrating ST-segment elevation in leads V1-V4.





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

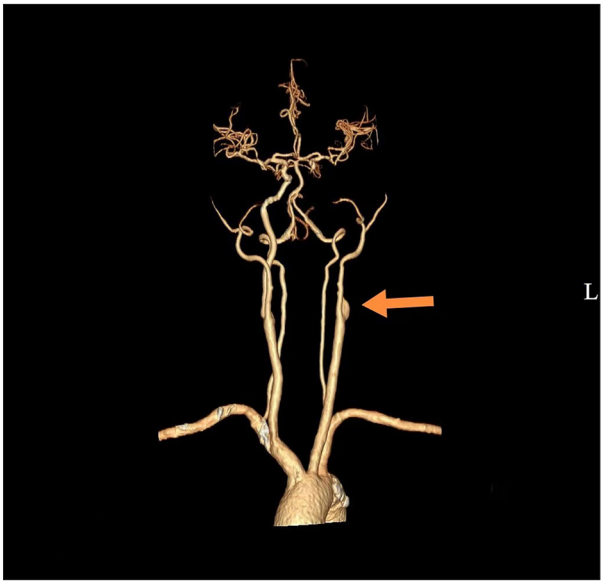


**Figure 2.** Cerebral computed tomography angiography demonstrating patent bilateral common carotid, internal carotid, external carotid, vertebral, middle cerebral, anterior cerebral, and posterior cerebral arteries, as well as the basilar artery and its branches. The arterial walls are irregular with calcified plaques, and the vessel lumens display multifocal narrowing. Occlusion of the left internal carotid artery and approximately 70%-80% stenosis at the origin of the left vertebral artery are present.

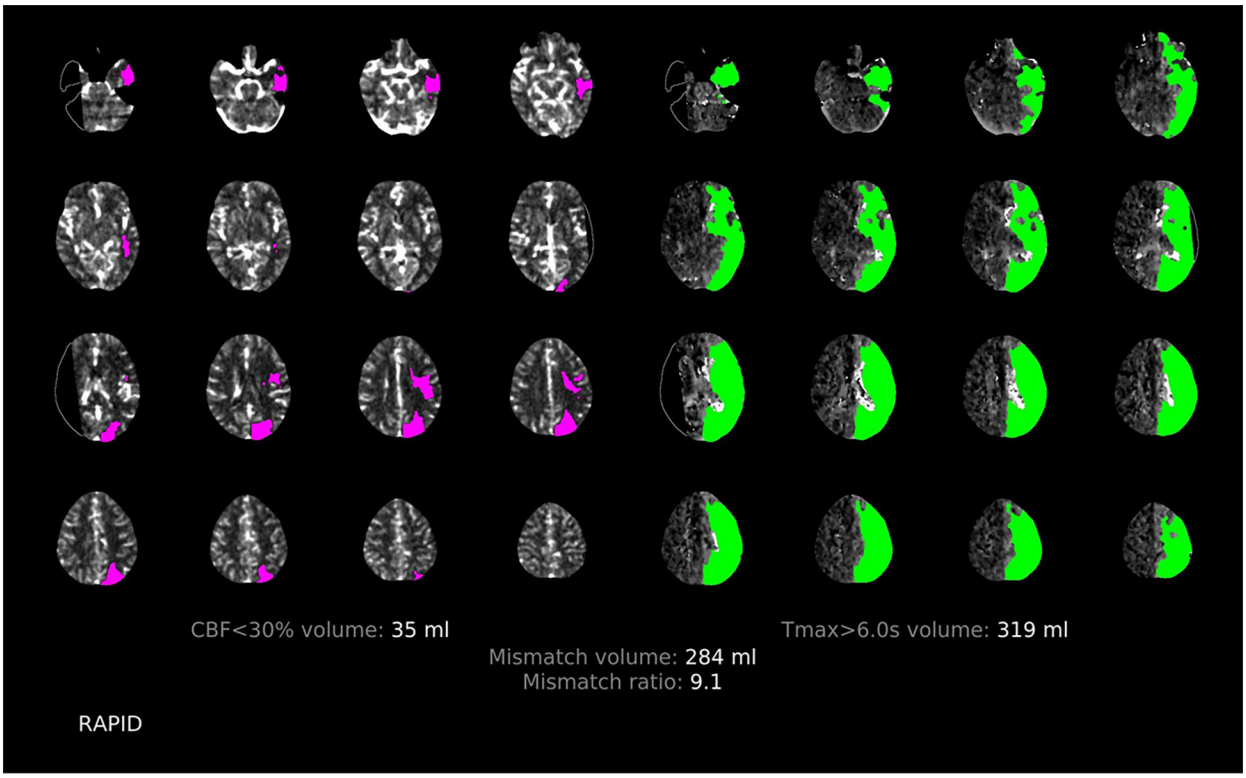


**Figure 3.** Computed tomography perfusion imaging demonstrating extensive regions of prolonged mean transit time and time to maximum (Tmax) in the left cerebral hemisphere. Corresponding regions showed decreased cerebral blood volume (CBV) and cerebral blood flow (CBF). The volume of CBF < 30% was approximately 35 mL, volume of Tmax > 6 seconds was approximately 319 mL, mismatch volume was approximately 284 mL, and mismatch ratio was 9.1. The hypoperfusion intensity ratio (Tmax > 10 seconds/Tmax > 6 seconds) was 0.6, and the CBV index (relative CBV within the Tmax > 6 seconds region) was 0.8.







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

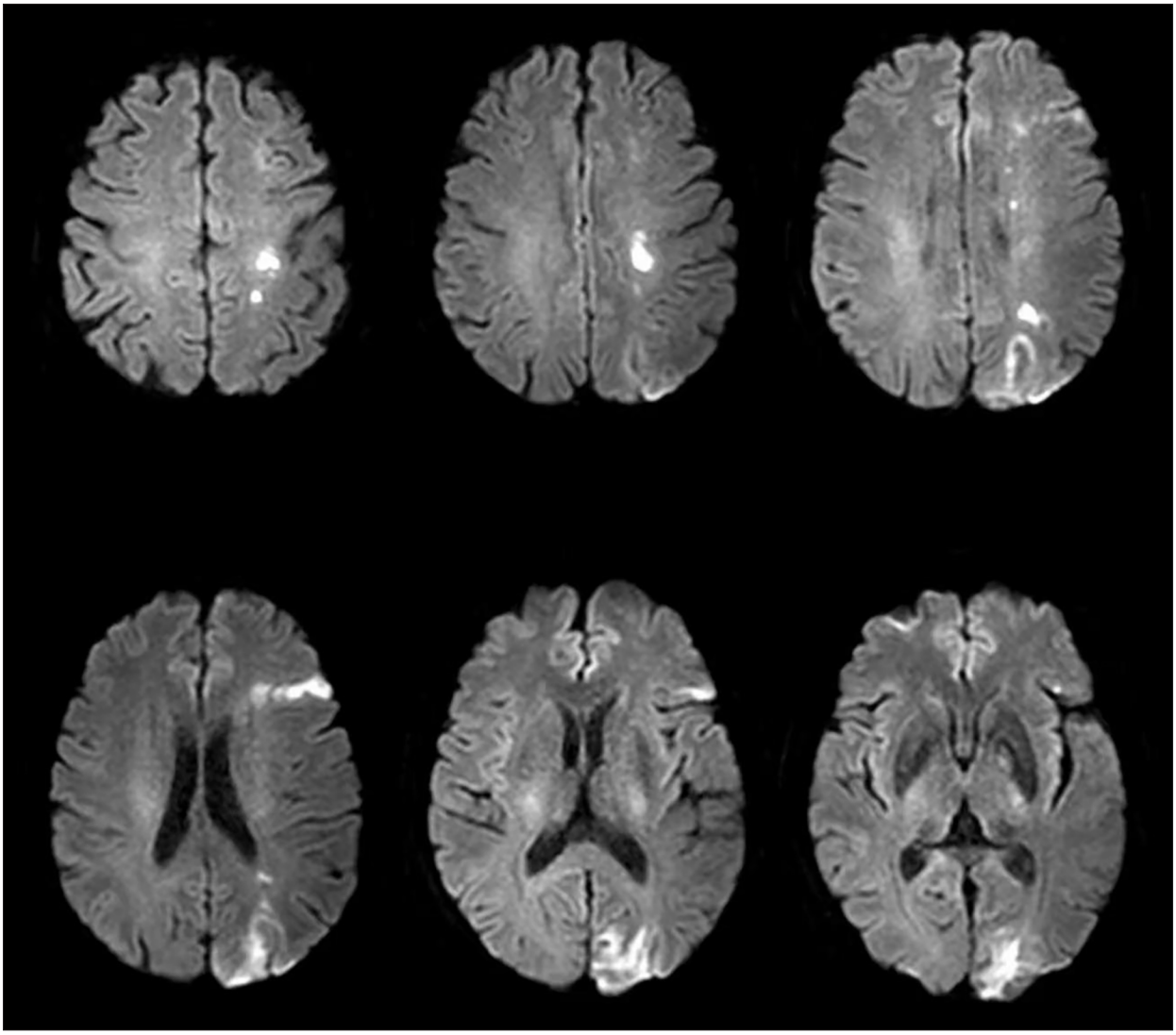

**Figure 4.** Cranial magnetic resonance imaging demonstrating cerebral atrophy, cerebral white matter hyperintensities (Grade I, modified Fazekas scale), and multiple acute cerebral infarctions involving the left frontal lobe, occipital lobe, and corona radiata.

cardiomyopathy was considered in the differential diagnosis but was deemed unlikely because characteristic echocardiographic features were absent. Brain computed tomography (CT) excluded intracranial hemorrhage, whereas chest CT showed mild inflammatory changes in the right upper lobe. Screening results for metabolic disorders, autoimmune disease, thrombophilia, and malignancy were unremarkable.

Cranial CT angiography demonstrated occlusion of the left internal carotid artery, severe stenosis at the origin of the left vertebral artery, and calcified plaques throughout the arterial system (**Figure 2**); CT perfusion revealed extensive hypoperfusion in the left cerebral hemisphere (**Figure 3**) [3]. After multidisciplinary evaluation, the patient underwent emergency cerebral angiography and mechanical thrombectomy of the left internal carotid artery without stent implantation to promptly restore cerebral blood flow and prevent further brain injury [2,6].

Neurological symptoms improved after the procedure. Continuous electrocardiographic monitoring revealed occasional premature ventricular contractions and non-sustained ventricular tachycardia that spontaneously resolved. Postoperatively, tirofiban was administered as a continuous intravenous infusion for 24 hours for antithrombotic therapy, followed by bridging to dual antiplatelet therapy [7,8]. Secondary prevention for coronary heart disease was initiated, including antiplatelet therapy, lipid-lowering therapy, and blood pressure and heart rate control [9]. Additional supportive measures included acid suppression, gastric mucosal protection, promotion of collateral circulation, fluid replacement, and hydration; blood pressure





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

was maintained within the target range of 100 to 130 mm Hg. Coronary angiography was planned when the intracranial condition had stabilized. Follow-up examinations demonstrated a decline in myocardial enzyme levels, and the patient's vital signs remained stable. Given the simultaneous occurrence of acute ischemic stroke and myocardial infarction, as well as the uncertain etiology, additional investigations were performed for metabolic syndrome, autoantibody profiles, thrombophilia, and malignancy—all yielded normal results.

On postoperative day 1, the patient developed a low-grade fever and hypoxemia. Chest CT demonstrated progression of pulmonary inflammation, hypostatic pneumonia, and small bilateral pleural effusions. Piperacillin-tazobactam therapy was initiated. On postoperative day 3, inflammatory markers were elevated (interleukin-6, 64.1 pg/mL; procalcitonin, 0.13 ng/mL; troponin T, 3593 ng/L; NT-proBNP, 1852 pg/mL); pulmonary infection complicated by cardiac insufficiency was suspected. Diuretics and high-flow oxygen therapy were administered. Pathogen test findings were negative.

On postoperative day 9, brain magnetic resonance imaging (**Figure 4**) demonstrated multiple acute infarctions in the left frontal lobe, occipital lobe, and corona radiata, despite substantial clinical improvement.

Two weeks after surgery, the patient was transferred to the rehabilitation department. He was conscious, with nonfluent speech and partially appropriate responses. Right hemiparesis persisted, although he was able to ambulate independently. Brunnstrom stages were IV-IV-V. Antiplatelet and lipid-lowering therapies were continued along with rehabilitation training. Blood pressure ranged from 97 to 120/67 to 73 mm Hg and heart rate was 75 to 90 beats/min; oxygen saturation remained above 96%.

At 11: 00 PM on postoperative day 16, the patient became unresponsive during sleep, without a carotid pulse or spontaneous respiration. Cardiac arrest was diagnosed. Return of spontaneous circulation was temporarily achieved after cardiopulmonary resuscitation, endotracheal intubation, intravenous epinephrine administration, and dopamine support. Cardiac monitoring showed a heart rate of 121 beats/min and blood pressure of 96/43 mm Hg. However, cardiac arrest recurred 30 minutes later. Despite 35 minutes of resuscitation, spontaneous circulation was not restored. The patient was pronounced dead after confirmation of absent pulse, dilated pupils, and asystole on electrocardiography. During the resuscitation period, only continuous cardiac monitoring was performed, and no electrocardiographic recordings were retained. Thus, the occurrence of ventricular tachycardia or ventricular fibrillation could not be confirmed.

## Discussion

CCI is a rare and complex condition with multifactorial pathophysiological mechanisms. Atherosclerotic plaque instability and rupture are considered the most common underlying causes, leading to simultaneous thrombosis in the coronary and cerebral arteries. Additionally, cardiogenic embolism—particularly secondary to ventricular thrombus formation or arrhythmias—may contribute to cerebral ischemia after myocardial infarction [10]. Hemodynamic instability, such as severe hypotension, can further reduce cerebral perfusion and precipitate stroke. Less common mechanisms include aortic dissection and paradoxical embolism [4]. Overall, these mechanisms often coexist, reflecting the systemic nature of CCI.

In the present case, simultaneous CCI was most likely attributable to systemic atherosclerotic disease with multivessel involvement. The patient had a long history of smoking, a major risk factor for atherosclerosis [11]. Vascular CT angiography demonstrated occlusion of the left internal carotid artery and severe stenosis at the origin of the left vertebral artery. Calcified plaques were observed throughout the arterial system, with varying degrees of luminal stenosis, supporting the presence of large-artery atherosclerotic disease in the cerebral circulation. In parallel, electrocardiographic findings were consistent with acute anterior myocardial infarction, suggesting concomitant coronary involvement within the same underlying pathological process. Takotsubo cardiomyopathy was considered in the differential diagnosis but was deemed unlikely because characteristic echocardiographic features were absent.

Taken together, these findings suggest a systemic atherosclerotic process involving both the cerebral and coronary vascular beds, in which plaque instability and local thrombosis may have contributed to simultaneous ischemic events. Although no direct evidence of an embolic source was identified, large-artery atherosclerosis remains the most plausible unifying mechanism.

Cardiogenic embolism was considered less likely because no intracardiac thrombus was identified on echocardiography; evaluations for thrombophilia and malignancy showed negative findings. However, occult paroxysmal atrial fibrillation could not be completely excluded. Additionally, postoperative ventricular arrhythmias were more likely secondary to myocardial ischemia, whereas the distribution of cerebral infarctions did not suggest a neurogenic mechanism mediated through the brain-heart axis.

Overall, the present findings are most consistent with a systemic atherosclerosis-related mechanism underlying CCI, with potential contributions from multiple interacting factors.







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

## Conclusions

In patients with simultaneous CCI, concomitant coronary artery disease may represent a major risk factor for subsequent cardiovascular events, and delayed coronary evaluation can result in missed opportunities for intervention [12]. In this case, monitoring during the rehabilitation period may have been insufficient—the absence of continuous electrocardiographic monitoring or nighttime cardiac surveillance might have delayed the detection of clinically significant arrhythmias. These limitations reflect competing treatment priorities when cardiocerebral emergencies coexist, along with the complexity of the condition and the challenges of providing intensive monitoring [13]. This case highlights the need for more proactive coronary evaluation and enhanced cardiovascular and cerebrovascular risk monitoring in patients with simultaneous CCI.



### Institution Where Work Was Done

Zhongshan People's Hospital, Zhongshan, Guangdong, PR China.

### Patient Consent

Given the deceased status of the patient, this report was approved by the Medical Ethics Committee of Zhongshan People's Hospital.

### Declaration of Figures' Authenticity

All figures submitted have been created by the authors who confirm that the images are original with no duplication and have not been previously published in whole or in part.

## References:

1. Akinseye OA, Shahreyar M, Heckle MR, Khouzam RN. Simultaneous acute cardio-cerebral infarction: Is there a consensus for management? Ann Transl Med. 2018;6(1):7
2. Yeo LLL, Andersson T, Yee KW, et al. Synchronous cardiocerebral infarction in the era of endovascular therapy: Which to treat first? J Thromb Thrombolysis. 2017;44(1):104-11
3. de Castillo LLC, Diestro JDB, Tuazon CAM, et al. Cardiocerebral infarction: A single institutional series. J Stroke Cerebrovasc Dis. 2021;30(7):105831
4. Omar HR, Fathy A, Rashad R, Helal E. Concomitant acute right ventricular infarction and ischemic cerebrovascular stroke; Possible explanations. Int Arch Med. 2010;3(1):25
5. You S, Wang Y, Wang X, et al. Twenty-four-hour post-thrombolysis NIHSS score as the strongest prognostic predictor after acute ischemic stroke: ENCHANTED study. J Am Heart Assoc. 2024;13(18):e036109
6. Powers WJ, Derdeyn CP, Biller J, et al. 2015 American Heart Association/American Stroke Association focused update of the 2013 guidelines for the early management of patients with acute ischemic stroke regarding endovascular treatment. Stroke. 2015;46(10):3020-35
7. Tang L, Tang X, Yang Q. The application of tirofiban in the endovascular treatment of acute ischemic stroke: A meta-analysis. Cerebrovasc Dis. 2021;50(2):121-31
8. Dannenberg L, Wolff G, Naguib D, et al. Safety and efficacy of tirofiban in STEMI-patients. Int J Cardiol. 2019;274:35-39
9. Lewis SJ. Prevention and treatment of atherosclerosis: A practitioner's guide for 2008. Am J Med. 2009;122(1):S38-S50
10. Gao W, Yu L, She J, et al. Cardio-cerebral infarction: A narrative review of pathophysiology, treatment challenges, and prognostic implications. Front Cardiovasc Med. 2025;12:1507665
11. Ji X, Leng XY, Dong Y, et al. Modifiable risk factors for carotid atherosclerosis: A meta-analysis and systematic review. Ann Transl Med. 2019;7(22):632
12. Gunnoo T, Hasan N, Khan MS, et al. Quantifying the risk of heart disease following acute ischaemic stroke: A meta-analysis of over 50 000 participants. BMJ Open. 2016;6(1):e009535
13. Ibekwe E, Kamdar HA, Strohm T. Cardio-cerebral infarction in left MCA strokes: A case series and literature review. Neurol Sci. 2021;43(4):2413-22





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