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Inferior ST-Elevation Myocardial Infarction With Complete Heart Block Precipitating Posterior Circulation Ischaemia in Haemodynamic Cardio-Cerebral Infarction: A Case Report and Focused Literature Review

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Abstract

Cardio-cerebral infarction (CCI) is a rare clinical condition in which acute myocardial infarction and acute ischaemic stroke occur simultaneously. Management is challenging because both conditions require urgent treatment, and the most appropriate treatment sequence is not always clear. We present a case of a 76-year-old man who was admitted with severe neurological deficits and an inferior ST-elevation myocardial infarction complicated by complete heart block and profound bradycardia. Initial CT angiography suggested bilateral internal carotid artery occlusion. However, these findings were considered more likely to reflect delayed contrast flow due to reduced cardiac output rather than true arterial occlusion. Following multidisciplinary discussion, intravenous tenecteplase was administered at the stroke dose. Repeat brain imaging demonstrated an evolving right occipital infarction, although additional thalamic involvement was suspected because the severity of his impaired consciousness was not fully explained by the imaging findings. Despite treatment, the patient's condition remained poor, and he died on the fifth day of admission. This case suggests that cerebral ischaemia in CCI may sometimes result from reduced cardiac output rather than an independent cerebral arterial occlusion. Recognising this possibility is important because restoring cardiac output through appropriate cardiac interventions may be essential to improving cerebral perfusion. The case highlights the need to consider the underlying mechanism of CCI when deciding treatment priorities.

Categories: Neurology, Cardiology, Emergency Medicine
Keywords: acute ischaemic stroke (ais), cardio-cerebral infarction, cerebral hypoperfusion, complete heart block (chb), pseudo-occlusion, st-elevation myocardial infarction (stemi), tenecteplase (tnk)

Introduction

Cardio-cerebral infarction (CCI) describes the occurrence of acute myocardial infarction (AMI) and acute ischaemic stroke (AIS) in the same patient within a narrow time window. Omar et al. discussed this association in 2010, although concurrent myocardial and cerebral infarctions had been described previously [1]. Contemporary registry data give the condition firmer epidemiological footing than the frequently quoted figure of 0.009% suggests. In a linkage of the Singapore national stroke and myocardial infarction registries comprising 120,531 patients, synchronous (same-day) CCI accounted for 0.5% of cases - approximately one in 200 presentations of AMI or AIS - with a further 0.8% classified as metachronous [2]. Reported prevalence elsewhere ranges from 0.009% to 0.9% depending on case definition and ascertainment method [3,4,5].

The prognostic significance is substantial and consistent. In the Singapore cohort, one-year all-cause mortality was 56.3% for synchronous CCI, compared with 29.9% for AMI alone and 15.4% for AIS alone; after adjustment for baseline characteristics and treatment, synchronous CCI carried approximately twice the 30-day mortality risk of AMI alone and nearly three times that of AIS alone [2]. Patients with synchronous CCI were also markedly less likely to receive percutaneous coronary intervention (PCI), dual antiplatelet therapy or secondary prevention medication than patients with AMI alone, highlighting the challenges of managing competing time-critical conditions [2].

The management difficulty is well described. Both conditions have narrow therapeutic windows, and treatment directed at one may delay or contraindicate treatment of the other [6,7]. A frequently cited American Heart Association/American Stroke Association recommendation - first articulated in the 2016 scientific statement on alteplase inclusion and exclusion criteria and carried into the 2019 guideline update - states that for patients with concurrent AIS and AMI, it is reasonable to give intravenous alteplase at the dose appropriate for cerebral ischaemia, followed by coronary angioplasty and stenting if indicated [8,9]. This is a consensus recommendation supported by expert opinion rather than trial data, and neither the

European Society of Cardiology nor NICE offers a dedicated pathway.

An important consideration is that cerebral ischaemia in CCI may involve different, potentially overlapping mechanisms. While thromboembolic occlusion is one possibility, reduced cardiac output secondary to myocardial infarction and associated bradyarrhythmia may also contribute to cerebral hypoperfusion. Distinguishing these mechanisms can be challenging, particularly in the absence of confirmatory haemodynamic measurements and vascular imaging. In patients with significant cardiac dysfunction, assessment and optimisation of cardiac output may therefore be important alongside time-critical reperfusion strategies.

We report a case of synchronous CCI in which inferior ST-elevation myocardial infarction (STEMI), complicated by complete heart block and profound bradycardia, was associated with posterior circulation ischaemia. We explore cerebral hypoperfusion as a plausible contributing mechanism, while acknowledging that a thromboembolic mechanism cannot be excluded. We use the case to discuss treatment sequencing in CCI, describe two diagnostic pitfalls encountered during the admission, and critically examine the decisions we made - including those we would reconsider. The report is prepared in accordance with the CARE guidelines [10].

Case Presentation

Patient information

A 76-year-old Caucasian man was brought to the emergency department by ambulance after being found collapsed and unresponsive at home by his wife at approximately 10:00 AM. His wife reported that he had been neurologically well approximately five minutes before being found collapsed. He arrived at the emergency department shortly before 12:00 PM. He regained consciousness in the presence of paramedics but remained vacant and non-verbal. A pre-hospital alert was issued simultaneously for an acute STEMI and stroke.

His past medical history comprised hypertension and a previous myocardial infarction treated with coronary stenting. Details of the previous coronary intervention, including the date and number of stents, were unavailable. Regular medications were felodipine, tamsulosin and rosuvastatin. He was not taking an antiplatelet agent or anticoagulant at presentation. He was a lifelong smoker and drank alcohol regularly. His baseline functional status was good, and he lived independently with a pre-stroke modified Rankin Scale score of 0.

Clinical findings

Observations on arrival are summarised in Table *1*.

Parameter	Value	Comment
Heart rate	47 beats/min (36 beats/min during complete heart block on pre-hospital ECG)	Marked bradycardia
Blood pressure	140/68 mmHg	Systolic pressure maintained
Respiratory rate	18 breaths/min	
Oxygen saturation	98% on air	
Temperature	36.3 °C	
Capillary blood glucose	9.1 mmol/L	Hypoglycaemia excluded as a cause of reduced GCS
Peripheral pulses	Weak radial pulses bilaterally	Clinical evidence of low cardiac output despite recorded cuff pressure

TABLE 1: Observations on arrival

Neurological examination revealed a GCS of 9 (E4V1M4), left conjugate gaze deviation, dense right-sided weakness, and absent verbal output, precluding formal assessment of comprehension. Pupils were equal and reactive to light, and the NIHSS score was 25.

Diagnostic assessment

Electrocardiography

The pre-hospital and admission electrocardiograms (Figures *1*-*2*) demonstrated inferior STEMI with ST elevations of 3 mm in lead II, 2 mm in leads III and aVF, with intermittent complete atrioventricular block and a ventricular escape rate of approximately 36 beats/min. Pathological Q waves were also noted in leads III and aVF, although the precise onset of myocardial infarction remained uncertain. The pattern was consistent with acute right coronary artery territory infarction involving the atrioventricular nodal branch. Atropine was administered up to the maximum recommended dose, with only a transient response.

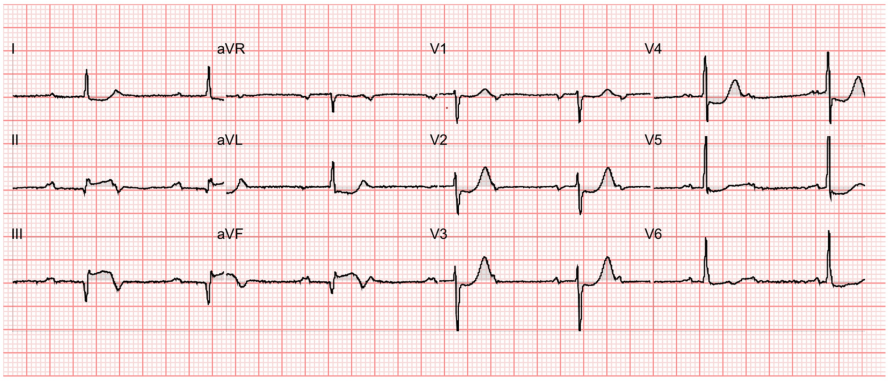


FIGURE 1: Pre-admission ECG recorded by paramedics

ECG recorded by paramedics showed ST elevation in ECG leads II, III and aVF.

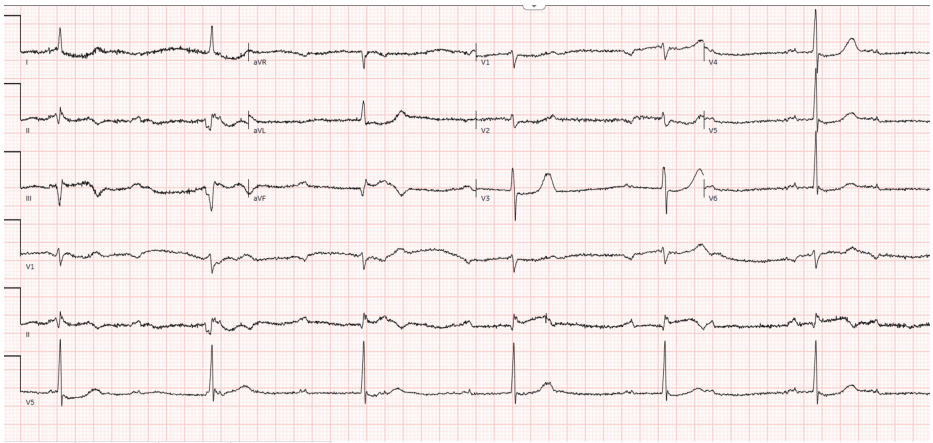


FIGURE 2: ECG on admission

This is the first ECG done in A&E, showing ST elevation in leads II, III and aVF with significant bradycardia. Intermittent episodes of complete atrioventricular block were noted on the cardiac monitor and documented by the cardiologist and stroke physician.

Laboratory investigations

Test	Value	Normal range
Sodium	136 mmol/L	133-146 mmol/L
Potassium	3.8 mmol/L	3.5-5.3 mmol/L
Urea	8.7 mmol/L	2.5–7.8 mmol/L
Creatinine	76 µmol/L	64-111 µmol/L
eGFR	84 mL/min/1.73m^2	>59 mL/min/1.73m^2
Haemoglobin	144 g/L	130-180 g/L
White blood cell count	11.00 * 10^9/L	4.0-11.0 × 10^9/L
Platelet count	304 * 10^9/L	150-450 × 10^9/L
CRP	120 mg/L	0.0-5 mg/L
Prothrombin time	13.1 seconds	9.2-13.5 seconds
Activated partial thromboplastin time	23.5 seconds	25-35 seconds
INR	1.1	0.8 to 1.2
0-hour troponin	8141 ng/L	0-4.9 ng/L
3-hour troponin	N/A	0-63.9 ng/L
Haemoglobin A1c	43 mmol/mol	20-41 mmol/mol
NT-proBNP	1790 ng/L	<300 ng/L: heart failure unlikely; 300 to <1,800 ng/L: consider echocardiography if clinically suspected; ≥1,800 ng/L: heart failure very likely (Black Country Pathology Services)*
Fasting total cholesterol	5.1 mmol/L	< 5.0 mmol/L
Fasting LDL	3.7 mmol/L	< 3.0 mmol/L
Fasting triglycerides	1.2 mmol/L	<1.7 mmol/L
Magnesium	1.03 mmol/L	0.8-1.5 mmol/L

TABLE 2: Laboratory tests

Laboratory tests with their values and normal ranges.

CRP: C-reactive protein; eGFR: estimated glomerular filtration rate; INR: international normalised ratio; LDL: low-density lipoprotein; NT-proBNP: N-terminal pro-B-type natriuretic peptide

*These are interpretive thresholds for acute heart failure rather than a single reference interval.

Echocardiography

Bedside transthoracic echocardiography, which was done after thrombolysis, demonstrated preserved left ventricular systolic function, impaired right ventricular systolic function, moderate mitral regurgitation, with regional wall motion abnormality in the basal to mid inferior wall and basal inferoseptal atrial wall. No intracardiac thrombus was seen.

Neuroimaging

Non-contrast CT of the brain on arrival (Figure *3*) showed no acute intracranial haemorrhage and no established infarct. CT angiography (Figure *4*) demonstrated severe bilateral internal carotid artery flow limitation with the appearance of occlusion. Given the profound bradycardia, weak peripheral pulses, and clinical evidence of low cardiac output, this was interpreted as contrast-transit pseudo-occlusion arising

from inadequate arterial opacification rather than true bilateral thrombotic occlusion. Repeat non-contrast CT on Day 3 (Figure 5) demonstrated evolving right occipital infarction. Magnetic resonance imaging with diffusion-weighted sequences was requested to characterise the full extent of infarction, and specifically to confirm or exclude thalamic involvement. The patient did not become well enough to be transferred to the scanner and died before it could be performed.



FIGURE 3: CT on admission

This is the CT slide on A&E presentation showing no acute intracranial abnormality.



FIGURE 4: CT angiogram slide with no carotid visualisation

CT angiogram slide showing poor opacification of B/L internal carotid arteries (marked in red circles are the areas where we normally see carotid arteries).

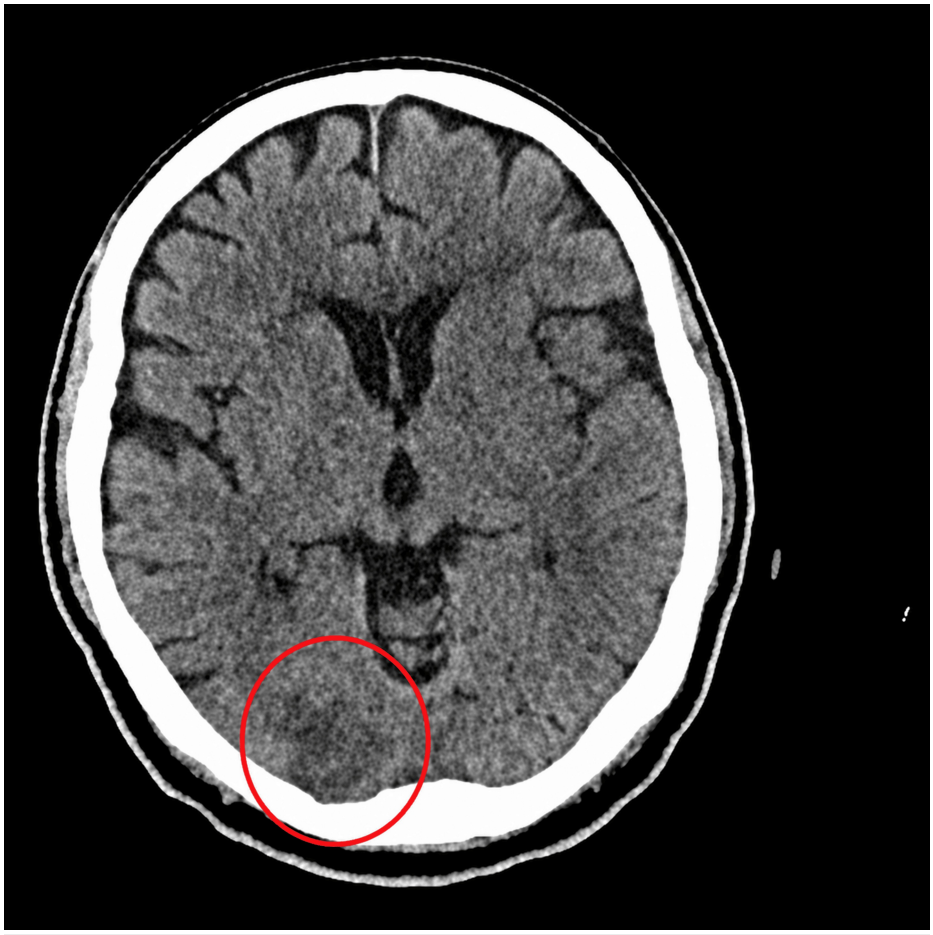


FIGURE 5: CT brain on Day 3

Repeat CT brain on Day 3 showing evolving right occipital infarction (marked in a red circle).

Therapeutic intervention and multidisciplinary decision-making

An immediate joint discussion took place between the stroke, cardiology, and emergency medicine teams. The competing considerations included a severe neurological deficit (NIHSS 25) with imaging suggestive of bilateral anterior circulation compromise; a concurrent inferior STEMI requiring immediate reperfusion; intermittent complete heart block with profound bradycardia; uncertainty regarding the chronology of events, as it was not possible to establish whether the cardiac or cerebral event occurred first; and the recognition that antiplatelet loading for PCI, once administered, would make subsequent intravenous thrombolysis substantially more hazardous, whereas the reverse sequence remained an option.

The cardiology team's initial preference was for primary PCI. The stroke team's position was that the neurological deficit was severe, haemorrhage had been excluded, and the patient was within the thrombolysis window. The considerations recorded in favour of a thrombolysis-first strategy were the severity of the neurological presentation, the maintained systolic blood pressure, the implications of antiplatelet loading for subsequent thrombolysis, and the potential benefit of thrombolysis in both vascular territories. As his approximate body weight was 90 kg, he received intravenous tenecteplase 22.5 mg (0.25 mg/kg) with a door-to-needle time of 1 hr 14 min.

The patient was admitted under cardiology for consideration of coronary angiography, PCI and pacing. Single antiplatelet therapy was commenced following thrombolysis on specialist advice after 24 hours.

Despite documented intermittent complete heart block, he was subsequently assessed as unsuitable for temporary or permanent pacing based on his overall clinical condition and anticipated poor outcome by the cardiology team on Day 2, without any trial of isoprenaline or transcutaneous pacing. Coronary angiogram could not be performed in view of the patient's deteriorating clinical status.

Follow-up and outcome

The patient was transferred to the stroke unit for continued supportive management. His neurological status

did not improve, and he deteriorated progressively. Following discussion with his family and the treating teams, care was redirected towards comfort-focused management. He died on the fifth day of admission.

The recorded cause of death was ischaemic stroke as the immediate cause, with inferior wall myocardial infarction listed as the underlying cause. No post-mortem examination was performed.

Discussion

Literature review

Search Strategy

We searched PubMed/MEDLINE and Embase from database inception to 15/09/2026 using the terms "cardio-cerebral infarction", "simultaneous myocardial infarction and stroke", "concurrent myocardial infarction stroke", and "synchronous cardio-cerebral infarction", combined with terms for management ("thrombolysis", "percutaneous coronary intervention", "thrombectomy"). English-language case reports, case series, cohort studies, systematic reviews, and guidelines were eligible. Reference lists of included articles were hand searched.

Definitions And Classification

Terminology in this field remains inconsistent, which limits the comparability of published series. Two classification schemes are in common use. The first distinguishes synchronous CCI, in which AMI and AIS occur simultaneously or within a short interval, from metachronous CCI, in which one event follows the other over a longer period [1,2,6]. Definitions of the interval vary: the largest registry study defined synchronous CCI as both events within 24 hours and metachronous CCI as both events within one week, the latter threshold chosen because European Stroke Organisation guidance treats STEMI within the preceding week as relevant to thrombolysis eligibility [2]. The second scheme, attributed to Habib, divides the syndrome into type I (concurrent), type II (AIS following recent AMI within three months) and type III (AMI following recent AIS within three months). The present case is synchronous, Habib type I.

Reported Outcomes And Management Patterns

Key studies are summarised in Table *3*. Two consistent findings emerge. First, mortality is high and exceeds that of either condition alone even after adjustment. Second, patients with CCI receive lower rates of PCI and guideline-directed secondary prevention compared with patients with isolated AMI or AIS [2]. Whether this reflects appropriate individualised management in a high-risk population or potentially avoidable treatment delays cannot be determined from registry data.

Individual case reports have also highlighted the risks associated with sequential reperfusion strategies, including fatal cardiac rupture following thrombolysis after PCI [11]. They further illustrate the variability in clinical presentation and management of simultaneous cardio-cerebral infarction. Yadav et al. described concurrent myocardial infarction and ischaemic stroke, highlighting the challenges of managing two time-sensitive vascular emergencies [12]. Obaid et al. reported a patient with anterior STEMI and left middle cerebral artery infarction who received intravenous alteplase at the cerebral dose, followed by delayed percutaneous coronary intervention [13]. Similarly, de la Torre et al. reported successful treatment of simultaneous STEMI and right middle cerebral artery occlusion using intravenous tenecteplase followed by mechanical thrombectomy [14]. These reports demonstrate the variation in reperfusion strategies and reinforce the importance of individualising treatment according to the patient's neurological and cardiovascular status.

Study (year)	Design and size	Findings relevant to management
Omar et al. (2010) [1]	Case report	Introduced the term cardiocerebral infarction; proposed intravenous thrombolysis as a means of addressing both territories, acknowledging the absence of supporting trial data.
Yeo et al. (2017) [3]	Single-centre series, 555 AIS patients	CCI identified in 0.9%; framed the sequencing question ("which to treat first?") in the endovascular era.
Akinseye et al. (2018) [7]	Narrative review	Concluded that no consensus exists; noted that thrombolysis is the only single therapy approved for both conditions.
de Castillo et al. (2021) [4]	Institutional series, 1,683 AIS and 1,983 AMI patients	Synchronous CCI prevalence 0.25%; all-cause mortality 45%.
Ng et al. (2022) [5]	Meta-analysis	Pooled analysis of reported cases; confirmed heterogeneity of management and high case fatality.
Bao et al. (2022) [11]	Two cases with review	One patient sustained fatal cardiac rupture after alteplase given following PCI; illustrates the hazard of sequential reperfusion therapies.
Eskandarani et al. (2021) [15]	Case series during COVID-19	Reported clustering of CCI during a period of increased large-vessel arterial thrombosis, supporting a systemic prothrombotic mechanism in some cases.
Ho et al. (2024) [2]	Linked national registries, 120,531 patients	Synchronous CCI in 0.5% (1 in 200); 1-year mortality 56.3% versus 29.9% (AMI alone) and 15.4% (AIS alone); adjusted 30-day mortality HR 2.41 versus AMI alone and 2.90 versus AIS alone; PCI and secondary prevention markedly underused in CCI.
Gao et al. (2025) [6]	Narrative review	Synthesised pathophysiology and treatment challenges; reiterated the absence of any guideline or expert consensus specific to CCI.

TABLE 3: Selected published evidence on cardio-cerebral infarction.

A possible haemodynamic mechanism in cardio-cerebral infarction

Several mechanisms have been proposed for CCI, summarised in Table *4*. These include shared atherosclerotic disease, cardioembolism, aortic thromboembolism and haemodynamic compromise secondary to cardiac dysfunction. Although these mechanisms are presented separately, they may overlap in individual patients. In particular, significant cardiac dysfunction may contribute to cerebral hypoperfusion even when an underlying embolic or atherothrombotic process is present.

Mechanism	Description	Implication for treatment sequencing
Shared atherosclerotic disease	Simultaneous plaque rupture in coronary and cerebral circulations in a patient with diffuse atherosclerosis.	Two independent occlusions; both require reperfusion. Thrombolysis-first then PCI is rational.
Cardioembolism	Intracardiac thrombus (atrial fibrillation, left ventricular dysfunction or aneurysm) embolising to coronary and cerebral vessels.	Cerebral large vessel occlusion likely; thrombectomy should be actively considered alongside coronary reperfusion.
Aortic arch thromboembolism	Fragmentation of proximal aortic thrombus with embolisation into multiple arterial territories.	As above; consider aortic imaging and exclude dissection before thrombolysis.
Haemodynamic failure	Cardiogenic shock, severe arrhythmia or high-grade conduction block producing global cerebral hypoperfusion and border-zone or perforator-territory infarction.	No cerebral thrombus to lyse. Restoration of cardiac output (pacing, inotropy, coronary reperfusion) is the cerebral reperfusion therapy. Thrombolysis contributes bleeding risk without a clear cerebral target.

TABLE 4: Proposed mechanisms of cardio-cerebral infarction and their treatment implications.

The findings in this case are consistent with a haemodynamic mechanism, which we propose as a plausible hypothesis. Acute right coronary artery occlusion produced inferior myocardial infarction with ischaemia of the atrioventricular nodal artery, resulting in intermittent complete heart block with a ventricular rate of approximately 36 beats/min. The clinical correlate was weak peripheral pulses and marked bradycardia. Global cerebral perfusion pressure fell as a consequence, and the pattern of subsequent infarction - posterior circulation infarction, becoming radiologically apparent only at Day 3 - is consistent with hypoperfusion in a watershed or perforator distribution rather than with acute large vessel occlusion, which typically declares itself earlier on CT.

The alternative explanation, that simultaneous coronary and cerebral thromboembolic events occurred as part of a systemic thrombotic process, cannot be excluded and is acknowledged. Two observations argue against it. Bedside echocardiography demonstrated preserved left ventricular systolic function without an identified intracardiac thrombus, and the patient was not known to be in atrial fibrillation, removing the two most common cardioembolic substrates. In addition, a simultaneous embolic shower producing both bilateral internal carotid occlusion and posterior circulation infarction would be an extraordinary event, whereas transient global hypoperfusion accounts for the entire picture parsimoniously.

Clinico-radiological correlation: impaired consciousness disproportionate to the demonstrated occipital infarction

A notable feature of this case was the severity of neurological impairment relative to the infarction demonstrated on subsequent CT imaging. The patient presented with a GCS of 9, absent verbal output, left gaze deviation, and dense right-sided weakness, while repeat CT on Day 3 demonstrated a right occipital infarction

An isolated unilateral occipital infarction would not ordinarily explain this degree of impaired consciousness or the full neurological presentation. Additional ischaemic involvement of structures responsible for arousal, including the thalami or brainstem, was therefore considered.

The bilateral paramedian thalami and rostral midbrain receive their arterial supply from branches of the posterior cerebral circulation. In some individuals, the artery of Percheron provides a common arterial supply to both paramedian thalami. Infarction involving this territory may present with impaired consciousness, ocular motor abnormalities and memory disturbance [16,17].

In this patient, possible thalamic or midbrain involvement could have contributed to the impaired consciousness. However, this remained a clinical hypothesis. Alternative explanations, including additional hemispheric ischaemia or systemic factors contributing to neurological deterioration, also remained possible.

MRI with diffusion-weighted sequences was requested to characterise the full extent of cerebral infarction but could not be performed because of the patient's clinical condition. Consequently, the proposed thalamic involvement was not radiologically confirmed.

The recorded left gaze deviation and right-sided weakness also raise the possibility of additional left hemispheric involvement, which was not demonstrated on the available CT imaging. These findings reinforce the limitations of anatomical localisation based solely on the available investigations.

Possible contrast-transit pseudo-occlusion on CT angiography

CT angiography demonstrated poor opacification of both internal carotid arteries, raising concern regarding bilateral carotid occlusion. In the context of marked bradycardia and weak peripheral pulses, delayed contrast transit secondary to impaired cardiac output was considered as a possible explanation.

Reduced cardiac output and altered contrast transit may affect arterial opacification during CT angiography, potentially creating the appearance of vascular occlusion. However, inadequate opacification may also arise from technical factors, and the appearance alone cannot reliably distinguish pseudo-occlusion from true arterial obstruction.

In this case, repeat vascular imaging was not performed, and true bilateral carotid occlusion could not be definitively excluded. The CTA findings therefore remain an important source of diagnostic uncertainty rather than confirmation of a haemodynamic mechanism.

The clinical implication is that apparent vascular occlusion should be interpreted in conjunction with the patient's haemodynamic condition, contrast acquisition and neurological examination. Where uncertainty persists, delayed or repeat vascular imaging may be valuable if clinically feasible. Importantly, consideration of contrast-transit artefact should not delay appropriate assessment of a potentially treatable large vessel occlusion.

Treatment sequencing and haemodynamic considerations

The simultaneous presence of severe neurological impairment, inferior STEMI and intermittent complete heart block created competing management priorities. Intravenous thrombolysis was selected following multidisciplinary discussion and assessment of the available clinical and imaging findings.

Existing AHA/ASA guidance considers intravenous alteplase at the cerebral dose, followed by coronary angioplasty and stenting when indicated, a reasonable approach in selected patients with concurrent acute ischaemic stroke and myocardial infarction [8,9]. However, this guidance is based largely on expert opinion, and its application depends on the individual patient's eligibility for treatment and clinical circumstances.

In the present case, tenecteplase was administered at the acute ischaemic stroke dose of 0.25 mg/kg. The decision was made in the setting of severe neurological impairment and concurrent myocardial infarction, with CT excluding intracranial haemorrhage. CT perfusion imaging was unavailable at the treating hospital.

An important consideration is that the haemodynamic mechanism, if present, may require treatment directed at restoring cardiac output in addition to conventional reperfusion strategies. However, the available evidence does not establish whether a different treatment sequence would have improved neurological or cardiac outcomes in this patient.

Haemodynamic assessment

Marked bradycardia and intermittent complete heart block may adversely affect cardiac output despite a maintained systolic blood pressure. Cardiac output depends on both heart rate and stroke volume, and blood pressure alone is an imperfect surrogate for systemic or cerebral perfusion.

In this case, weak peripheral pulses and marked bradycardia raised concern regarding haemodynamic compromise. However, quantitative cardiac output measurements and serial objective assessments of systemic perfusion were unavailable.

These findings highlight the importance of considering haemodynamic assessment alongside neurological examination and vascular imaging in patients presenting with concurrent cardiac and cerebral emergencies.

Tenecteplase and coronary reperfusion

Tenecteplase is increasingly used in acute ischaemic stroke at a dose of 0.25 mg/kg, to a maximum of 25 mg, supported by randomised clinical trial evidence [18,19]. In contrast, established fibrinolytic regimens for STEMI use higher weight-adjusted doses, up to a maximum of 50 mg.

Consequently, administration of tenecteplase at the cerebral dose should not be assumed to provide reliable coronary reperfusion. Although thrombolysis may have activity in both vascular territories, the extent of coronary reperfusion cannot be established without further assessment.

In the present case, coronary angiography and PCI were considered but could not be performed because of the patient's deteriorating clinical condition. The coronary anatomy and degree of reperfusion therefore remained unknown.

This case illustrates the difficulty of selecting a reperfusion strategy when both cerebral and coronary vascular emergencies are present, particularly when the underlying mechanism of neurological impairment remains uncertain.

The antithrombotic sequence

Antithrombotic management is particularly challenging in CCI because the requirements of acute ischaemic stroke and acute coronary syndrome may conflict.

Following intravenous thrombolysis, antiplatelet therapy is generally deferred for 24 hours because of concern regarding intracranial haemorrhage. In contrast, acute coronary syndrome management frequently requires early antiplatelet and anticoagulant treatment, particularly when coronary intervention is performed.

In this case, antiplatelet therapy was commenced after 24 hours on specialist advice. Therapeutic-dose enoxaparin was not administered because of concern regarding intracranial bleeding risk following thrombolysis.

The competing risks of cerebral haemorrhage and ongoing coronary ischaemia underline the importance of individualised multidisciplinary decisions regarding antithrombotic treatment.

Consideration of cardiac pacing

Complete atrioventricular block complicating inferior STEMI may occur because of ischaemia involving the atrioventricular nodal circulation. In some patients, conduction abnormalities improve following coronary reperfusion, while temporary pacing may be required when clinically significant bradycardia persists.

In the present case, atropine was administered up to the maximum recommended dose, producing only a transient response. Temporary cardiac pacing was considered by the cardiology team but was not undertaken because the patient was considered clinically too unwell to tolerate the procedure.

Given the possibility of impaired cerebral perfusion secondary to marked bradycardia, restoration of cardiac output might theoretically have improved cerebral haemodynamics. However, this effect was not demonstrated, and it cannot be concluded that temporary pacing would have reversed the neurological impairment or altered the outcome.

The case highlights the importance of assessing cardiac rhythm, circulatory status and the feasibility of supportive interventions alongside cerebral and coronary reperfusion strategies.

Pacing as cerebral therapy

The most consequential decision in this admission may have been the decision against pacing. Complete heart block complicating inferior STEMI is frequently transient, arising from atrioventricular nodal ischaemia and vagal effects, and often resolves with coronary reperfusion; temporary pacing is a bridging measure in this setting rather than a commitment to permanent device therapy. If the cerebral injury was driven by low output secondary to bradycardia, then temporary pacing was not merely cardiac supportive care - it was the cerebral reperfusion intervention. The reasoning recorded for withholding it, namely overall clinical condition and anticipated poor outcome, is legitimate and may well have been correct; but it risks circularity, in that a poor neurological prognosis was used to justify withholding the intervention most likely to improve cerebral perfusion, whose failure then confirmed the prognosis.

We raise this not to second-guess a difficult bedside judgement but because the sequencing logic deserves scrutiny. Where the cerebral deficit is plausibly perfusion-dependent, a time-limited trial of pacing with reassessment of neurological status at restored cardiac output is a defensible strategy and offers a diagnostic as well as a therapeutic yield: improvement would confirm the haemodynamic mechanism, while absence of improvement would clarify prognosis on firmer ground.

A proposed mechanistic approach to sequencing in cardio-cerebral infarction

We propose that the first question in suspected CCI should be mechanistic rather than procedural. The following framework is offered as a pragmatic aid to structured discussion, not as a validated pathway.

Step 1

Exclude intracranial haemorrhage and aortic dissection. Non-contrast CT of the brain and, where clinically suspected, aortic imaging precede any reperfusion decision.

Step 2

Characterise the haemodynamic state using stroke volume surrogates rather than blood pressure alone: heart rate and rhythm, pulse pressure, peripheral perfusion, lactate, urine output and bedside echocardiographic assessment of left and right ventricular function.

Step 3

Interrogate the cerebral vasculature with the haemodynamic state in mind. Treat bilateral or symmetrical apparent occlusions in a low-output patient as pseudo-occlusion until proven otherwise and confirm with delayed or repeat acquisition once output is restored.

Step 4

Classify the probable mechanism. Where an intracardiac thrombus, atrial fibrillation, or a focal large vessel occlusion is identified, treat as embolic CCI. Where high-grade conduction block, bradyarrhythmia or cardiogenic shock coexists with diffuse, watershed or perforator-territory ischaemia and no demonstrable cerebral occlusion, treat as haemodynamic CCI.

Step 5

In embolic CCI, follow the existing consensus: intravenous thrombolysis at the cerebral dose, with mechanical thrombectomy for large vessel occlusion and coronary angiography with PCI thereafter, sequenced by the relative acuity of the two territories and by institutional capability.

Step 6

In haemodynamic CCI, prioritise restoration of cardiac output - temporary pacing for high-grade block, correction of arrhythmia, inotropic or mechanical circulatory support as indicated, and coronary reperfusion - and reassess the neurological deficit once output is restored before committing to systemic thrombolysis.

Step 7

Document the mechanistic reasoning, not only the decision. In a condition where the evidence base is composed of individual cases, a recorded rationale is what makes a case contributory rather than anecdotal.

The dominant theme across the published literature remains the value of a rapidly convened heart-brain team spanning emergency medicine, cardiology, stroke medicine, neuroradiology and critical care [6,7]. Registry data showing systematic undertreatment of CCI patients suggest that the purpose of such a team is not only to choose between competing therapies but to guard against the outcome in which, faced with two urgent indications, neither is adequately addressed [2].

Limitations

The principal limitation of this case is the absence of direct confirmation of the proposed haemodynamic mechanism. Cardiac output and cerebral perfusion were not quantitatively measured, and repeat vascular imaging was not performed. Therefore, neither cerebral hypoperfusion nor contrast-transit pseudo-occlusion could be established definitively.

The suspected thalamic or brainstem involvement was not confirmed. Diffusion-weighted MRI was requested but could not be performed because of clinical instability. Consequently, the full extent and distribution of cerebral ischaemia remain uncertain.

Although the patient was reportedly neurologically well shortly before being found collapsed, the precise chronology of the cardiac and cerebral events could not be established. The reported prehospital times were approximate, limiting precise reconstruction of the onset-to-treatment interval. CT perfusion imaging was unavailable.

Coronary anatomy was not defined because angiography could not be performed. Attribution of the conduction block to right coronary artery involvement therefore rests on the electrocardiographic findings rather than angiographic confirmation. The status of the previously implanted coronary stent also remained unknown.

Finally, the absence of follow-up cardiac and vascular investigations limits assessment of treatment response. Alternative explanations, including concurrent thromboembolic events, cannot be excluded, and the effect of alternative management strategies on clinical outcome remains uncertain.

Learning points

Cardio-cerebral infarction may involve thromboembolic, atherothrombotic and haemodynamic mechanisms, which can coexist. Identifying the probable mechanism is important when considering treatment priorities.

A maintained systolic blood pressure does not necessarily establish adequate cardiac output or cerebral perfusion, particularly in patients with marked bradyarrhythmia or high-grade atrioventricular block.

Poor arterial opacification on CT angiography in a patient with suspected haemodynamic compromise should prompt consideration of contrast-transit artefact, while ensuring that true vascular occlusion is not overlooked.

Neurological impairment disproportionate to the infarction demonstrated on CT should prompt consideration of additional cerebral involvement, including thalamic or brainstem ischaemia, where clinically appropriate.

Stroke-dose tenecteplase should not be assumed to provide reliable coronary reperfusion. Coronary assessment and reperfusion strategies must be considered separately according to the patient's clinical condition.

Temporary cardiac pacing and other measures to support cardiac output may be important considerations in

selected patients with haemodynamically significant bradyarrhythmia, although their effect on neurological outcomes in CCI remains uncertain.

Management of simultaneous cardiac and cerebral infarction requires individualised multidisciplinary decision-making, balancing time-critical reperfusion strategies, haemodynamic support and haemorrhagic risk.

Conclusions

Cardio-cerebral infarction is an uncommon condition associated with high mortality and limited evidence to guide treatment sequencing. This case describes simultaneous inferior STEMI complicated by complete heart block, marked bradycardia, and posterior circulation infarction. The possibility that impaired cardiac output contributed to cerebral hypoperfusion represents an important consideration, although the underlying mechanism could not be definitively established. The severity of impaired consciousness also raised suspicion of additional thalamic or brainstem involvement, which could not be confirmed by MRI.

This case highlights the importance of considering haemodynamic compromise alongside thromboembolic mechanisms when evaluating synchronous cardio-cerebral infarction. Assessment and optimisation of cardiac output should be considered alongside time-critical cerebral and coronary reperfusion strategies, with management individualised through multidisciplinary decision-making. Further observational evidence and detailed case reporting may help clarify the mechanisms and optimal treatment approaches in this challenging clinical condition.

Additional Information

Author Contributions

All authors have reviewed the final version to be published and agreed to be accountable for all aspects of the work.

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Disclosures

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