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ORIGINAL ARTICLE

Cardio-cerebral infarction in left MCA strokes: a case series and literature review

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Abstract
The objective of this manuscript is to describe the challenges of Cardio-Cerebral Infarction (CCI) treatment and to highlight the variable approaches in management. CCI is a rare clinical presentation of simultaneous acute ischemic stroke (AIS) and acute myocardial infarction (AMI) and poses a therapeutic challenge for practitioners. Each disease requires timely intervention to prevent irreversible damage; however, optimal management remains unclear. We describe three cases of CCI. All three patients presented with symptomatic left MCA (M1) occlusion, with ST elevation myocardial infarction (STEMI) and left ventricular apical thrombus. Fibrinolysis and mechanical thrombectomy (MT) were discussed in all cases, but only one patient received alteplase (0.9 mg/kg) and none underwent MT. Percutaneous intervention (PCI) was done in only one case. The two patients that did not receive thrombolysis were treated with modified therapeutic heparin (no bolus), and all received antiplatelet therapy. Ultimately, all three patients passed away. CCI poses a clinical challenge for physicians including (1) optimal strategies to enable swift mechanical reperfusion to both the brain and myocardium; (2) difference in dosage of thrombolytics for AIS versus AMI; (3) risk of symptomatic intracerebral hemorrhage following administration of anticoagulation and/or antiplatelet therapy; and (4) caution with use of thrombolytics in the setting of acute STEMI due to the risk of myocardial rupture. In the absence of high quality evidence and clinical guidelines, treatment of CCI is highly individualized.

Keywords Acute ischemic stroke · Cardio-cerebral infarct · Cerebral ischemia · Myocardial infarct · Alteplase

Introduction

Acute ischemic stroke (AIS) and acute myocardial ischemia (AMI) are the leading causes of morbidity and mortality globally [1]. Optimal management prioritizes revascularization via time-sensitive fibrinolysis, percutaneous intervention (PCI), mechanical thrombectomy (MT), and antiplatelet or anticoagulation therapy [2]. Simultaneous presentation of AIS and AMI within 48 h, termed Cardio-Cerebral Infarction (CCI), is quite rare and may be associated with high morbidity and mortality [3]. In patients with CCI, optimal administration of fibrinolytics (dose and agent), timing of intervention (PCI versus MT), treatment with anticoagulation and antiplatelet therapies, and rates of symptomatic ICH are unclear [4, 5]. Thus, optimal management is controversial, with added challenges in the event of a left ventricular (LV) thrombus.

Our objective is to review the epidemiology, pathophysiology, and management of CCI, along with the many management challenges.

Methods

This is a case series presenting observational data collected from three patients at The Ohio State University, Wexner Medical Center with acute presentation of CCI within a 1-month period in the year 2020. Due to the COVID-19 pandemic, verbal consent was obtained via telephone.

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Furthermore, a literature review of CCI was performed in PubMed and EMBASE. No time limitation filter was applied. The following MeSH terms were used as follows: "Cardio cerebral infarct," "stroke and MI," "stroke and myocardial infarction," "STEMI and stroke," "NSTEMI and stroke." Articles included, contained a full description of the case, outlining the presentation and management steps, and with evidence of both cerebral and cardiac infarction within 48 h.

Case summary

We describe three cases of CCI in a 43-year-old male, a 72-year-old male, and an 80-year-old female (Table 1). All three patients presented with symptomatic proximal left MCA (M1) occlusion (Fig. 1), ST elevation myocardial infarction (STEMI) (Fig. 2), and left ventricular apical thrombus. Examinations revealed full left MCA syndrome with aphasia and R sided weakness (NIHSS 17–25). Interventional therapies such as alteplase and mechanical thrombectomy (MT) were discussed in all cases, but alteplase was utilized in only one. In one case, left heart catheterization was done without intervention/stenting. Two patients received modified therapeutic heparin (no bolus) for LV thrombus; all patients received antiplatelet therapy with aspirin and/or clopidogrel. Ultimately, all three patients expired.

Cases

Case presentation 1

A 43-year-old right handed male with past medical history of gun-shot wound, and nephrolithiasis presented to our medical center with symptoms of right hemiplegia and global aphasia (NIHSS 25) with unknown last known well (LKW). Vitals on admission were stable. Labs revealed Troponin of 49 ng/ml, with ST elevation in leads II, III, aVF, V2-6 (Fig. 2). Left heart coronary (LHC) angiography revealed 60% stenosis with associated non-atherosclerotic

Table 1 Summary and comparison of patient presentation, management, and outcomes

	Patient 1	Patient 2	Patient 3
Age	43	80	72
Gender	Male	Female	Male
Past medical history	Nephrolithiasis, gun shot wound	COPD, tobacco use	Pancreatitis
CTH findings	Acute left MCA infarction	Acute Left MCA Infarction	Acute left MCA infarction
CTA brain findings	Left M1 thrombus	Left M1 thrombus	Left M1 thrombus
CT Perfusion findings	> CBF < 30% volume: 55 cc > Tmax > 6.0 s volume: 96 cc > Mismatch volume: 41 cc > Mismatch ratio: 1.7	> CBF < 30% volume: 28 cc > Tmax > 6.0 s volume: 36 cc > Mismatch volume: 8 cc > Mismatch ratio: 1.3	> CBF < 30% volume: 12 cc > Tmax > 6.0 s volume: 48 cc > Mismatch volume: 36 cc > Mismatch ratio: 4
NIHSS on presentation	25	23	17
EKG findings	Anterior inferior STEMI in leads II, III, aVF, V2-6	Anterior STEMI in leads V3-V5	Anteroseptal STEMI
Troponin level	49 ng/ml	1.1 ng/ml	6.5 ng/ml
Echo and LHC findings	Echo: EF 30% and LV thrombus LHC: distal LAD non-artherosclerotic thrombus	Echo: EF 29% and LV thrombus LHC: N/A	Echo: EF 30% and LV thrombus LHC: N/A
Intervention			
Medical	> No intravenous alteplase > Received non-bolus neuro-scale heparin > Started long-term aspirin > Started long-term statin	> Received intravenous alteplase > No heparin > Started long-term aspirin > Started long-term statin	> No intravenous alteplase > Received non-bolus neuro scale heparin drip > Started long-term aspirin + clopidogrel > Started long-term statin
Surgical	> PCI without intervention/stenting > No mechanical thrombectomy	> No PCI > No mechanical thrombectomy	> No PCI > No mechanical thrombectomy
Outcome	Transitioned to comfort care died on hospital day 7	Transitioned to comfort care and discharged to hospice. Died 3 days after discharge	Transitioned to comfort care Died on hospital day 7

CTH computed tomography of head, *CTA* brain computed tomography angiogram of brain, *EKG* electrocardiogram, *Echo* echocardiogram, *LHC* left heart catheterization, *LAD* left anterior descending, *LV* left ventricle, *PCI* percutaneous intervention

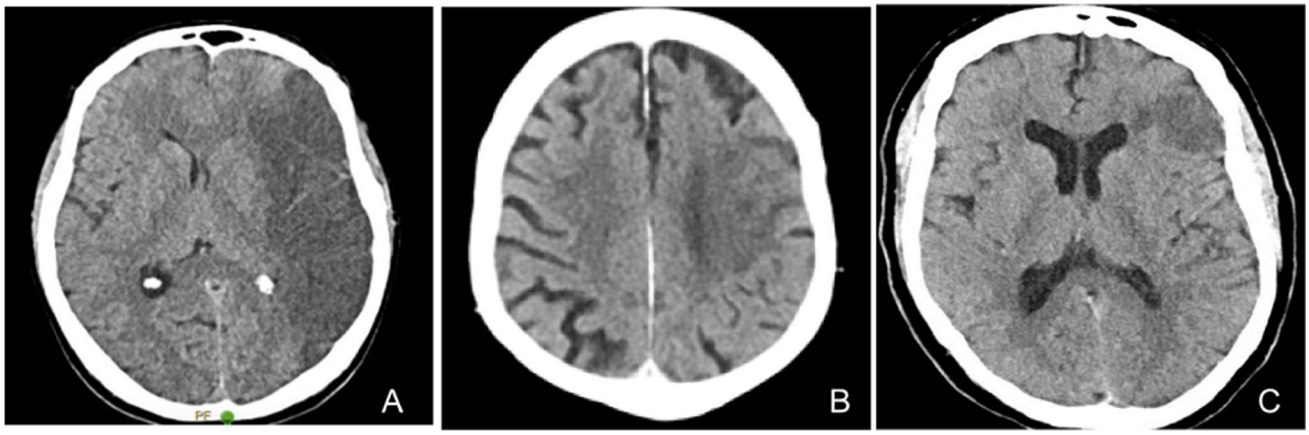

Fig. 1 CT Head imaging on admission showing evolving acute left MCA infarction in patient 1 (**A**), patient 2 (**B**), and patient 3 (**C**)

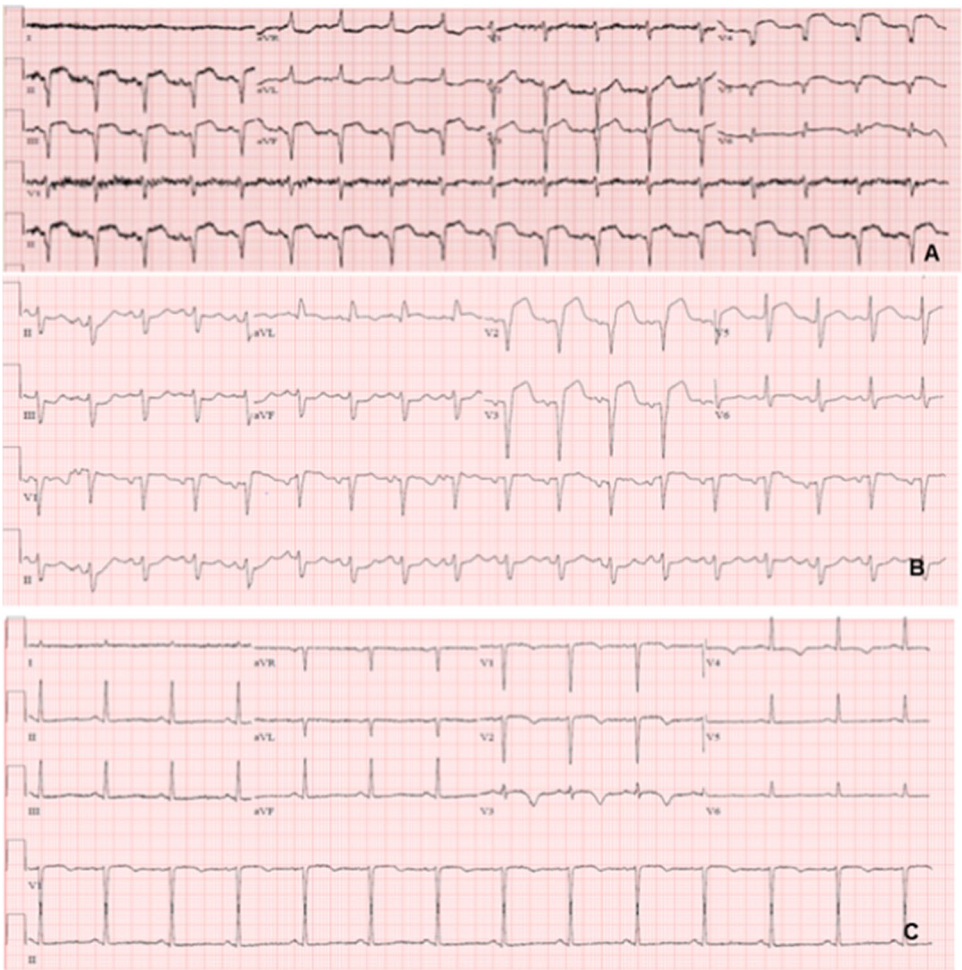

Fig. 2 EKG showing anterior-inferior STEMI in patient 1 (**A**), anterior STEMI in patient 2 (**B**), and anterior STEMI in patient 3 (**C**)

thrombus in the distal left anterior descending (LAD) artery. Echocardiogram revealed left ventricle (LV) apical thrombus with ejection fraction (EF) of 30%. CT cerebral angiogram (CTA) showed a long segment intraluminal thrombus within the left M1 segment with distal M1 occlusion. CT perfusion showed core of 55 cc with a mismatch volume and ratio of 41 cc and 1.7 respectively.

Given unknown LKW, with unavailability of MRI FLAIR-diffusion mismatch protocol at our institution, acute management ischemic stroke did not include alteplase, nor

MT. Due to STEMI on presentation, he was loaded with aspirin and Ticagrelor, with only aspirin continued after, and taken for emergent percutaneous coronary angioplasty. PCI was aborted due to need of bolus heparin prior to catheterization, and dual anti-platelet therapy "DAPT" post- catheterization, which would put him at increased risk of hemorrhagic conversion. Subsequent medical management was optimized for acute ischemia with goal directed medical therapy (GDMT) including aspirin. Immediate anticoagulation was not started for LV thrombus given risk of hemorrhagic conversion of large acute ischemic stroke. On day 5 of admission, "neuro scale" heparin drip (aPTT goal 52–75, no bolus) was started for LV thrombus, without transitioning to "cardiac scale" (aPTT goal of 72–95). His hospital course was further complicated by wide complex tachycardia secondary to known LAD occlusion, which was medically managed with lidocaine and amiodarone. He was ultimately transitioned to comfort care and passed away on day 7 of admission.

Case presentation 2

An 80-year-old right handed female with past medical history of COPD, and tobacco use presented initially to an outside hospital with right hemiplegia and global aphasia (NIHSS 23) with LKW $\leq$ 4.5 h. She was given alteplase at 3.2 mg bolus with infusion at 28.7 mg/h, and transferred to OSU for further evaluation. On arrival, CTA revealed left M1 occlusion and CT Perfusion revealed core of 28 cc, mismatch volume and ratio of 8 cc and 1.3 respectively. She was deemed to not be a MT candidate given her low mismatch ratio. EKG revealed an acute STEMI with mild elevation of Troponin to 1.1 ng/ml and ST elevation in leads V3-5 (Fig. 2). Echocardiography showed an EF of 29% and apical LV thrombus.

Stroke management included intravenous (IV) alteplase (0.9 mg/kg), aspirin at 24-h post alteplase, statin, blood pressure control, and preventing edema by induced hypernatremia (140–145 mmol/L) using 0.9% normal saline. Due to down-trending troponin, lack of new EKG changes, and the need for DAPT increasing the risk of hemorrhagic conversion, she was not offered PCI, nor started on a heparin drip. Her AMI was managed with GDMT. Given her comorbidities, age, and wishes, she was transitioned to comfort care and discharged to hospice with NIHSS of 25. She passed away at day 3 of discharge.

Case presentation 3

A 72-year-old right handed male with past medical history of pancreatitis presented with right hemiplegia and facial droop (NIHSS 27) with LKW > 4.5 h. Vitals were pertinent for tachycardia to 110 on admission. Labs revealed Troponin of 6.5 ng/ml. His EKG showed an acute STEMI in the anteroseptal region (Fig. 2). CTA showed a Left M1 occlusion. CT perfusion showed a core of 12 cc with mismatch volume and ratio of 36 cc and 4.0 respectively. Echocardiogram revealed an EF 30% and LV thrombus. As with the cases above, etiology of acute cerebral and myocardial ischemia was presumed secondary to his LV thrombus.

His AIS was managed medically with dual anti-platelet with aspirin/ clopidogrel, and statin. He did not receive alteplase due to being outside the window, nor did he get mechanical thrombectomy due to inability to perform the procedure per neurosugery. His AMI was managed medically, with non-bolus "neuro scale" heparin drip and antiplatelet and statin. His hospital course was complicated by ventricular tachycardia. He continued to have significant neurological disability with significant dysarthria and dysphagia requiring PEG tube. Family elected to transition him to comfort care and he passed away on hospital day 7.

Discussion

Epidemiology

The incidence and prevalence of simultaneous AIS and AMI are unknown due to rarity of presentation. The reported incidences in literature are from observational studies and case reports. Rokey et al. found 58% prevalence of CAD in patients presenting with AIS/ TIA [6]. Chin et al. reported the incidence of CCI as 12.7% in geriatric patients who were screened for AMI within 72 h of admission for acute stroke [7]. Findings from the Global Registry of Acute Coronary Event (GRACE) trial reported an incidence of in-hospital stroke as 0.9% in a cohort of patients presenting with acute coronary syndrome, with a higher incidence in STEMI compared to non-STEMI [8]. The most recent data in 2017 from Yeo et al. showed that 6% of patients with acute stroke had ST segment elevation [9, 10]. Notably, these reported incidences are mostly for non-synchronous presentation, rather than simultaneous presentation of AIS and AMI.

Pathophysiology and pathogenesis

There are four major pathophysiologic mechanisms of CCI including (1) cardioembolic, (2) neurogenic stunned myocardium, (3) hypotensive, and (4) dissection.

Cardioembolism to the cerebral and myocardial arteries can lead to AIS and AMI and is typically secondary to either LV thrombus formation associated with heart failure, atrial fibrillation, or paradoxical embolization via patent foramen ovale in patients with RV thrombus or deep vein thrombosis [9, 11–13].

Ischemic infarcts, especially ones involving the insular cortex, are associated with higher risk of neurogenic stunned myocardium and arrhythmias which can mimic ST-elevation myocardial ischemia [14, 15]. This myocardial stunning is a result of dysfunction of CNS structures that control autonomic nervous system regulation of myocardium leading to sympathetic dominance. At the molecular level, there is reduced duration of delayed rectifier K^+ channels and alteration in L-type Ca^{++} channels. Alterations of K^+ channels lead to shortened diastolic interval (DI), reduced action-potential duration (APD), and insufficient myocardial refractory period prior to generation of new stimulus. Concurrently, abnormal sympathetic nervous system (SNS) stimulation of L-type Ca^{++} channels triggers frequent afterdepolarization. Ultimately, this affects myocardium rhythm and integrity, predisposing to wall motion abnormalities and formation of cardiac thrombus [15, 16].

Another reported mechanism of CCI is hypotension, which occurs as a sequela of AMI causing dysregulation of cerebral blood flow leading to watershed infarcts [3, 9]. Although rare, extension of an ascending aortic dissection to the coronary ostia and a subsequent extension to the carotid, vertebral, or basilar arteries may also explain the simultaneous occurrence of a cerebral and a coronary infarction [17].

Management

Management of AIS prioritizes thrombolytic administration and revascularization with MT. Alteplase is administered if ischemic stroke is within 4.5 h of LKW (Table 2), and MT is considered if there is a large vessel occlusion within 24 h. Although less commonly used in the USA, tenectaplase, which is more fibrin-specific and has a longer mechanism of action, may be considered well [18].

Management of AMI is similar, prioritizing timely restoration of blood flow to coronary vessels using PCI and/or fibrinolytic therapy [22, 23]. PCI is the preferred method of revascularization when it can be performed within 120 min, and in patients presenting with AMI within 12 h. Fibrinolysis via tenecteplase, alteplase, or streptokinase is indicated for symptom onset within 12 h followed by transfer for PCI, or if PCI cannot be completed within 120 min (Table 2) [19, 20]. Peri-procedural heparin and dual antiplatelet therapy are recommended following PCI.

Management of CCI is individualized (Table 3). Among the twenty-five cases reviewed within the literature review performed, 7 (28%) utilized MT, 15 (60%) underwent PCI, 9 (36%) received fibrinolysis (alteplase), and 12 (48%) had concurrent cardiac thrombus. 4 (16%) received mono antiplatelet (MAP) only, 3 (12%) DAP only, 4 (16%) MAP plus AC, 3 (12%) DAP plus AC, 10 (40%) heparin therapy, and 8 (32%) AC other than, and/ or with heparin. Outcomes were reported in 16 (64%), with 4 (16%) and 6 (24%) having MRS ≤ 3 at 1 and 3 months respectively. Approximately 20% expired during hospitalization. This highlights the inconsistency in practice and outcomes, thus the need for clear guidelines.

Stroke and LV thrombus

The management of AIS in the setting of LV thrombus is centered on the decision of anticoagulation timing, since much of the cardiac literature recommends prompt treatment of thrombus. Management of LV thrombus includes anticoagulation therapy for 3–6 months with serial echocardiogram to monitor resolution of thrombus. Anticoagulation can be discontinued after 6 months if there is resolution of LV thrombus, low risk of thrombus re-formation, and no significant wall motion abnormality. Timing on initiation of AC in setting of CCI has no clear guidelines with variations as noted in Table 3.

Controversies

Dilemmas associated with management of CCI include the following:

Table 2 Dose of fibrinolytic therapy for acute ischemic stroke and acute myocardial ischemia [19–21]

	Acute ischemic stroke	Acute myocardial ischemia
Alteplase	- 0.9 mg/kg - 10% given as initial bolus over 1 min - 90% as a continuous infusion over 60 min	-15 mg Bolus - then 0.75 mg/kg (maximum 50 mg) over 30 min - then 0.5 mg/kg (maximum 35 mg) over the next 60 min
Tenecteplase	- 0.25 – 0.4 mg/ kg - consider 0.25 mg/kg for LVO - consider 0.4 mg/kg for minor, non- LVO	-Single bolus over 5 to 10 s based upon body weight: - < 60 kg: 30 mg - 60 to 69 kg: 35 mg - 70 to 79 kg: 40 mg - 80 to 89 kg: 45 mg - ≥ 90 kg: 50 mg
Reteplase	- Not indicated	- 10 units over 2 min then repeat 10 unit bolus at 30 min

LVO large vessel occlusion

Table 3 Approach to cardio- cerebral infarct and outcomes from worldwide reported cases [9, 24–39]

Case	Sex/age	CTH/CTA findings	NIHSS	Fibrinolysis	PCI	MT	AC/AP	Cardiac thrombus	Outcome
Eskandarani 2021 [26]	62 years old/M	LCCA occlusion	22	None reported	None reported	None reported	ASA loading dose Clopidogrel loading dose Continued with DAP	None reported	Death
Eskandarani 2021 [26]	50 years old/M	BL cerebral ischemia	21	None reported	None reported	None reported	MAP AC (unknown type and dosage)	LV thrombus	None reported
Iqbal 2021 [30]	65 years old/M	LMCA ischemia	None reported	IV alteplase at 0.9 mg/kg (discontinued after bolus due to bleeding)	None reported	None reported	DAP LMWH	LV thrombus	None reported
Ibekwe 2021	43 years old/M	LM1 occlusion	25	None reported	None reported	None reported	ASA 325 mg load Ticagrelor load MAP after Heparin infusion on day 5	LV thrombus	Death
Ibekwe 2021	80 years old/F	LM1 occlusion	23	IV alteplase at 039 mg/kg	None reported	None reported	ASA	LV thrombus	Death
Ibekwe 2021	72 years old/M	LM1 occlusion	27	None reported	None reported	None reported	DAP Heparin infusion	LV thrombus	Death
Abe 2019	73 years old/F		21	IV alteplase at 0.6 mg/kg	Yes, no stenting	Yes	Rivaroxaban on day 5	None reported	MRS 2 at 3 month
Katsuki 2019 [37]	72 years old/M	Lt Cerebellar ischemia	29	None reported	Yes, without stenting	None reported	Heparin infusion	LAA thrombus	None reported
Sakuta 2019 [36]	55 years old/F	LMCA occlusion	23	None reported	Yes, without stenting	Yes	Heparin infusion	None reported	MRS 3 at 3 month
Wan Asyraf 2019 [39]	33 years old/M	LMCA occlusion	11	IV Tenecteplase	None reported	None reported	Heparin infusion DAP	None reported	MRS 1 at 1 month
Plata 2018 [27]	46 years old/M	RM2 occlusion	15	IA alteplase at 5 mg Bolus	Yes with Drug Eluting Stent	Yes	MAP Warfarin	LV thrombus	MRS 0 at 1 month
Yeo 2017 [9]	45 years old/M	RICA occlusion	None reported	None reported	Yes with Drug Eluting Stent	Yes	Heparin bridge to Warfarin	LV thrombus	MRS 3 at 3 month
Yeo 2017 [9]	53 years old/M	LMCA occlusion	None reported	None reported	Yes with Bare-Metal Stent	Yes	ASA 81 mg	None reported	MRS 4 at 3 month
Yeo 2017 [9]	71 years old/F	RPCA occlusion	None reported	None reported	Yes	Yes	None reported	LV thrombus	None reported

Table 3 (continued)

Case	Sex/age	CTH/CTA findings	NIHSS	Fibrinolysis	PCI	MT	AC/AP	Cardiac thrombus	Outcome
Yeo 2017 [9]	Middle age/M	LMCA occlusion	None reported	IV alteplase at 0.9 mg/kg	None reported	None reported	None reported	None reported	MRS 1 at 3 month
Kijpaisalratana 2017 [25]	65 years old/M	RM1 occlusion	12	IV alteplase at 0.9 mg/kg	Yes with DES	None reported	DAP with ASA/ Clopidogrel	None reported	None reported
Hosoya 2017 [28]	50 years old/M	Multifocal Ischemia	None reported	None reported	Yes, no stenting	None reported	ASA	None reported	None reported
Tokuda 2016 [35]	87 years old/F	RMCA and ACA occlusion	19	None reported	Yes	Yes	Rivaroxaban	None reported	MRS 3 at 3 month
Maciel 2015 [24]	44 years old/M	RMCA ischemia	11	IV alteplase at 0.9 mg/kg	None reported	None reported	None reported	None reported	MRS 2 at 3 months
Wee 2015 [29]	49 years old/M	RPCA ischemia	None reported	None reported	Yes, no stenting	None reported	Clopidogrel 300 mg load Heparin infusion at 48 h (apt goal 60) Warfarin on Day 3	LV thrombus	MRS 2 at 1 month
Gonzalez-Pacheco 2014 [33]	66 years old/F	Rt FrontoParietal Ischemia	16	IV alteplase at 0.9 mg/kg	Yes with DES	None reported	ASA 81 mg at 48 h DAP with clopidogrel / ASA on day 4	LV thrombus	None reported
Hashimoto 2014 [34]	84 years old/M	Multifocal ischemia	1	None reported	Yes, without stenting	None reported	Heparin infusion Warfarin	None reported	MRS 0
Kim 2013 [32]	58 years old/M	LMCA ischemia	None reported	None reported	Yes, with DES	None reported	Clopidogrel 600 mg load	None reported	None r eported
Klecynski 2012 [31]	62 years old/M	BL Frontal and Lt Parietal ischemia	None reported	None reported	Yes, no stenting	None reported	Clopidogrel 600 mg load ASA 300 mg load Heparin bolus and infusion Warfarin	LAA thrombus	None reported
Omar 2010 [3]	48 years old/M	BL occipital and cerebellar ischemia	25	None reported	None reported	None reported	None reported	None reported	Death

AC anti-coagulation, *AP* anti-platelet, ASA aspirin, *CTH* computed tomography of head, *CTA* computed tomography angiogram, *DAP* dual anti-platelet, *DES* drug eluting stent, *IA* intra-arterial, *ICH* intra cerebral hemorrhage, *IV* intra-venous, *LAA* left atrial appendage, *LMWH* low molecular weight heparin, *LV* left ventricle, *NIHSS* National Institute of Health Stroke Scale, *MAP* mono anti-platelet, *MRS* Modified Rankin Score, *MT* mechanical thrombectomy, *PCI* per-cutaneous intervention, *PEA* pulseless electrical activity

1. The different dosages and duration of IV alteplase for management of AIS versus AMI (Table 2).
2. The use of PCI with stenting requires peri-procedural heparin infusion, and prolonged use of dual-anti platelet therapy. This has been associated with increased risk of hemorrhagic conversion in AIS, but evidence is limited regarding long-term clinical outcomes [40].
3. Caution with use of thrombolytics in the setting of acute STEMI due to the risk of myocardial rupture [41].

Risk of hemorrhagic transformation

Currently, data on risk factors associated with hemorrhagic transformation in the setting of CCI is lacking. A small retrospective study from Schmidbauer et al. did not show increased incidence of ICH in AIS patients treated with PCI compared to those not getting PCI [42]. Several trials, such as Troponin Elevation in Acute Ischemic Stroke (TRELAS), which evaluated the diagnostic yield of troponin levels in AIS, did not report rates of symptomatic ICH. We await the results of ongoing trial Prediction of Acute Coronary Syndrome in Acute Ischemic Stroke (PRAISE), which is assessing the diagnostic yield of biomarkers, clinical scores, signs and symptoms, EKG, and echocardiography findings to identify patients in need of PCI. We hope that rates of hemorrhagic transformation are reported.

Given the lack of high quality data, clinicians should individualize management—prioritizing fibrinolytic therapy and mechanical revascularization—with full assessment of bleeding risk, and other comorbidities.

Treatment strategies

Regarding CCI necessitating acute intervention, we propose combined treatment of myocardial and cerebral vascular territories with administration of alteplase (0.9 mg/kg) followed by PCI with stenting if indicated [9]. Concomitant need for mechanical cerebral thrombectomy can then be assessed using cerebral angiogram. Another approach could be combining mechanical thrombectomy with PCI in select cases, with close monitoring of neurological status to detect symptomatic hemorrhagic conversion.

Limitations

This case series only reports observational findings and is not structured to evaluate for associations between different management options and clinical outcome (i.e., symptomatic ICH, morbidity/mortality). Furthermore, since all of the patients were transitioned to comfort care within a week of admission, it is impossible to predict overall outcomes. Lastly, the search strategy and inclusion criteria for the literature review focused on the current definition of CCI necessitating AMI and AIS within 48 h of each other, leading to exclusion of many studies.

Conclusion

Appropriate management of CCI poses a great challenge for practitioners. Clinicians must be mindful of the following: (1) treatment strategies to enable swift mechanical reperfusion to both the brain and myocardium; (2) risk of cardiac tamponade and myocardial rupture with intravenous thrombolytic therapy; (3) difference in dosage of thrombolytics for AIS versus AMI; and (4) risk of symptomatic intracerebral hemorrhage following administration of anticoagulation and/or antiplatelet therapy. There is an urgent need for future studies to guide optimal management.

Author contribution Dr. Hera Kamdar and Dr. Tamara Strohm were involved in manuscript review and editing. Dr. Elo Ibekwe is the main author of the manuscript.

Declarations

Ethics approval No ethics approval was necessary for this manuscript.

Informed consent Informed consent was obtained from health-care power of attorney over telephone.

Conflict of interest The authors declare no competing interests.

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