Hyperacute Cardio-cerebral Infarction: a Therapeutic Challenge

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Accepted: 21 April 2021 / Published online: 3 May 2021
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Abstract
A 72-year-old woman was admitted to the Intensive Care Unit (ICU) with right-sided hemiparesis and mixed aphasia; an axial brain computed tomography (CT) with no acute injuries and the angiotomography showed a 50% stenosis of the intracranial left internal carotid. Presented a National institute of Health Stroke Scale (NIHSS) score of 23, whereby she received fibrinolytic treatment with endovenous tissue plasminogen activator (rTPA) 170 min after the symptoms started. An electrocardiogram (ECG) and echocardiogram revealed an acute myocardial infarction (MI) with a ST-segment elevation at the inferior face. After the fibrinolytic treatment, the patient presented criterion for myocardial reperfusion. She evolved favourably and was discharged from the ICU after 48 h with a NIHSS score of 9. The aim of the present case report is, therefore, to suggest a clinical approach to help physicians in the decision-making process for treatment in patients with concurrent hyperacute cardio-cerebral infarction (CCI).

Keywords Cardiocerebral infarction · Acute ischemic stroke · Myocardial infarction

Introduction
Both acute ischemic stroke (AIS) and myocardial infarction (MI) are medical emergencies that require early diagnosis and treatment. The linkage between cerebrovascular disease and the MI and other cardiac pathologies is part of a phenomenon known as brain-heart interaction: the AIS increases the risk of an MI, and vice versa. However, simultaneous cardio-cerebral infarction (CCI) is a rare condition, first used in 2010 [1], to describe a concomitant AIS and MI, which could be further divided into synchronous (thrombosis of two vessels at the same time) or metachronous (thrombosis of one vessel precedes the other), making it a therapeutic challenge. It is defined as the simultaneous presence of an acute focal neurological deficit (AIS indicative) and MI evidence as precordial pain or modifications in the ECG (mainly ST-segment elevation) and cardiac enzymes increase within 4.5 h after the neurological symptoms appeared [2]. Actually, there are no clear recommendations for ideal management because of the rarity of this scenario.

Case Presentation
A 72-year-old woman, with a history of dyslipidemia, smoking, and arterial hypertension was admitted to the ICU with severe right hemiplegia and mixed aphasia with a NIHSS score of 23 after 3.30 h of symptoms onset. A CT showed no severe injuries (Fig. 1a and b), and the angiotomography showed a 50% stenosis of the intracranial left Internal Carotid, which led to performing rTPA treatment. The admission ECG showed a ST-segment elevation at the inferior face (Fig. 2a and b), with high levels of cardiac enzymes, and an echocardiogram which discarded an ascending aorta dissection and patent foramen ovale (PFO), with a 55% ejection fraction of the left ventricle, inferobasal, inferomedial, and inferolateral basal akinesis.

An ECG was performed after the fibrinolytic treatment that showed criterion for reperfusion. A percutaneous coronary intervention (PCI) was considered, although it was decided to continue medical treatment for hemodynamic stability (Killip-Kimbal I), plus the risk of central nervous system bleeding due to the NIHSS (taking into account the possibility of double antiaggregation after PCI). She evolved favourably and was discharged from the ICU after 48 h with a NIHSS...
score of 9 with no deterioration of the cardiac function or progress of the akinesis in the control echocardiogram.

Discussion and Conclusions

The simultaneous CCI is a highly unlikely frame, but it carries a high morbidity and mortality; there is not a specific therapeutic reperfusion strategy for both organs, presenting the treating physician challenges and dilemmas, having to prioritize the most affected organ in a narrow time window. In addition, treating one of the organs will indubitably delay the other, with possible consequences.

Several mechanisms operate in the pathogenesis of CCI and can be summarized into three groups [3]. The first group can trigger a simultaneous CCI, the second group is cardiac originated cerebral infarctions, and the third group consists of secondary cardiac infarctions to ischemic strokes (Frame 1)

Within the ethyologies with vascular components that can cause a simultaneous CCI, the auricular fibrillation stands out, also being exceptional causes the cerebral and coronary embolisms. The type 1 aortic dissection is another CCI cause. An infrequent cause is the coronary and cerebral vasospasm posterior to an electric shock.

Within the cardiac causes, there are anterior and apical wall MI associated with a decrease of the left ventricular systolic function, myocardial stunning associated (Takotsubo syndrome also forms thrombus that mimic ST MI elevation), and ventricular thrombus associated with PFO. Likewise, arterial hypotension after a MI can trigger a simultaneous CCI. In the case of cardioembolic stroke, it is important to pay attention to the possibility of simultaneous MI.

Regarding CNS causes, right insular strokes play a key role in the regulation of the autonomous nervous system, triggering cardiac arrhythmias, QT interval prolongation interval, and several cardiac injuries. In addition, aphasia and reduced pain perception might lead to delay in the diagnosis of MI in
some patients. The American Heart Association recommends cardiac monitoring for at least 24 h after AIS [4]. Moreover, some ECG changes are commonly seen in patients with AIS, making ECG interpretation challenging.

The ideal management of simultaneous CCI is a treatment strategy that benefits both vascular territories. The fibrinolytic treatment in AIS doses, followed by PCI, is sensible [4]. Even if the use of rtPA can be applied to strokes and MI with a ST segment elevation, the dosage is different and the time periods differ, which limits the use of this drug as a definitive treatment for both pathologies. In addition, the use of antithrombotics that are inherently part of a PCI for MI may increase the risk for hemorrhagic transformation associated with intravenous thrombolysis [5] and the use of thrombolytic

Fig. 2 a ECG on admission shows elevation of the ST segment at DII, DIII, and aVF. b ECG 2 h after the beginning of the fibrinolytics in which there is evidence of a decrease in the J point of more than 70% with respect to admission as a criterion of reperfusion

Frame 1 Simultaneous cardio-cerebral infarction (CCI) causes. Type 1 aortic dissection compromises the arch’s ascending aorta and extends to the descending aorta (according to Bakey and Standford’s aortic dissection classification). LV left ventricle, EF ejection fraction, RV right ventricle, PFO patent foramen ovale, MI acute ischemic stroke

Simultaneous cardio-cerebral infarction (CCI) causes:

Group 1: vascular causes.

a. Auricular fibrillation
b. Type 1 aortic dissection
c. Secondary cardio-cerebral vasospasm due to electrical injury

Group 2: cardiac causes.

a. LV thrombus with a low EF
b. RV thrombus with a low EF plus PFO
c. MI with Killip and Kimbal III and IV

Group 3: Cerebral Causes

a. Insular ischemia
b. Parietal ischemia
Stroke onset < 4.5 hours with myocardial infarction < 12 hours

CT brain/CTA/CT perfusion + Electrocardiogram

Hemodynamic unstable or STEMI

Yes

Emergency PCI

No

Intravenous rtPA (0.9mg/kg)

Large vessel occlusion

Yes

Endovascular treatment for stroke

No

Medical management

MCA infarction

Yes

Close monitoring for arrhythmia

No

PCI*/ Medical management

*In case of no initial PCI
in AIS increases the risk of cardiac wall rupture in the setting of MI.

The AHA/ASA recommendation does not propose a specific treatment regarding the categories and severity of the MI [4]. Additionally, even if the coronary reperfusion therapeutic window is longer compared to the AIS, urgent treatment is still necessary.

It can be highlighted that, mainly, the evaluation of the hemodynamic state will influence the decision of which organ to treat first.

Kijpaisalratana et al. [3] proposed a management algorithm (Fig. 3). They recommend, to evaluate if there is hemodynamic instability, ST-segment elevation, and evaluate the NIHSS. In the case that cardiocirculatory instability or ST-segment elevation was found, it performs an urgency PCI, otherwise pharmacological and/or mechanical reperfusion of the stroke accordingly.

Another issue is that the administration of anticoagulant or antiplatelet for CCI should be carefully performed because although rare, delayed fatal haemorrhage such as cardiac tamponade (idiopathic or resulted from oozing type cardiac rupture due to MI) can occur, even in cases without rTPA [6].

The patient in our case was hemodynamically stable (Killip and Kimbal I) and neurological compromised (NIHSS 23). We decided to prioritize AIS treatment with intravenous rtPA.

In conclusion, although uncommon, hyperacute simultaneous CCI is among one of the most challenging medical emergency conditions, with a narrow therapeutic time-window if not addressed promptly. The bibliography is scarce, and given the current knowledge limitations, the approach to management should be individualized as outlined above based on the stroke severity, type of MI, and hemodynamic stability, with close cardiac monitoring in order to facilitate the decision to rescue the brain or the heart first.

**Author Contribution** Dr Matías Casanova was in charge of literature review and wrote up the paper, Dr Rubén Mesa was in charge of literature review, and Dr Emiliano Comú supervised the case write up.

**Availability of Data and Material** Figure 3 was allowed for publication by Frontiers in Neurology Editorial Office. All Frontiers articles are open-access and distributed under the terms of the Creative Commons Attribution License (CC BY)—which means you are free to use them, as well as images and any other content, provided the original author(s) are credited and the original Frontiers publication is cited. For online use, we also ask that you link to the original research article.

**Declarations**

**Ethics Approval** All procedures performed in studies involving human participants were in accordance with the ethical standards of the institutional and/or national research committee and with the 1964 Helsinki declaration and its later amendments or comparable ethical standards.

**Consent to Participate** Not applicable.

**Consent for Publication** Written consent was not obtained from the patient.

**Conflict of Interest** The authors declare no competing interests.

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