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Simultaneous cardiocerebral embolization in patients with atrial fibrillation

Abbreviated title: Simultaneous cardiocerebral embolization

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Summary

Atrial fibrillation is a common cause of ischaemic stroke, but is also the most common cause of coronary artery embolism. Many cases of coronary and cerebral embolism coexistence have been reported in the literature. The objective of this article is to provide an update on the clinical signs, biological data and reperfusion strategy for simultaneous cardiocerebral embolization.

Résumé

La fibrillation atriale est une cause fréquente d'accident vasculaire cérébral ischémique mais aussi la cause la plus fréquente d'embolie coronaire. De nombreux cas précédents de coexistence d'embolie coronaire et cérébrale ont été rapportés dans la littérature. L'objectif de cet article est de faire le point sur les signes cliniques, les données biologiques et la stratégie de reperfusion concernant les embolies coronaires et cérébrales concomitantes sur fibrillation atriale de novo.

KEYWORDS

Coronary embolism;

Simultaneous;

Myocardial infarction;

Atrial fibrillation;

Ischaemic stroke

MOTS CLÉS

Embole coronaire ;

Concomitant ;

Infarctus du myocarde ;

Fibrillation atriale ;

Accident vasculaire cérébral ischémique

Abbreviations: AF, atrial fibrillation; CE, coronary artery embolism; CI, confidence interval; MRI, magnetic resonance imaging; NSTEMI, non-ST-segment elevation myocardial infarction; NT-proBNP,

N-terminal fragment of prohormone brain-type natriuretic peptide; OR, odds ratio; STEMI, ST-segment elevation myocardial infarction.

Background

Acute myocardial infarction and ischaemic stroke are the leading causes of morbimortality worldwide. Prompt diagnosis and treatment are needed in both diseases to limit cell necrosis immediately after thrombotic arterial occlusion. The reperfusion strategies in acute myocardial infarction and ischaemic stroke are pharmacological and/or percutaneous interventions. The strategies for ST-segment elevation myocardial infarction (STEMI) have been used for decades, and the first guidelines for acute ischaemic stroke were published in 2003 [1]. In addition, atrial fibrillation (AF) is one of the most frequent causes of ischaemic stroke. In contrast, myocardial infarction related to a coronary artery embolism (CE) is rare; only 2.9% of all myocardial infarctions are caused by CE, but AF is the most common cause of CE (73.0%) [2]. The objective of this article is to provide an update on current knowledge regarding simultaneous cardiocerebral embolization in patients with AF.

Method

We searched the PubMed database up to February 2020 with the following key words: “coronary embolism”; “simultaneous”; “myocardial infarction”; “atrial fibrillation”; and “ischaemic stroke”.

Coexisting acute myocardial infarction and ischaemic stroke with coexisting clinical signs

We found 10 published clinical cases that reported coexisting acute myocardial infarction and CE causing ischaemic stroke in patients with AF. In these cases, there were clinical signs of stroke, but also indisputable clinical and/or electrocardiographic signs of acute cardiac syndrome. These clinical cases are summarized in [Table 1](#). It should be noted that there was only one case of non-STEMI (NSTEMI), whereas the majority of cases were STEMI [3-11]. Moreover, CE was confirmed in only seven cases.

Stroke is a serious complication after acute myocardial infarction. In a recent study of 8485 patients admitted for myocardial infarction, the incidence of in-hospital stroke was 1.4% (123/8485) [12]. The leading subtypes of in-hospital ischaemic stroke, according to the TOAST classification, were cardioembolic stroke ($n = 64$, 60.0%) and stroke of undetermined pathogenesis ($n = 38$, 36.0%). In this study, all ischaemic strokes were recorded after admission; 65 occurred on the first day following admission (52.8%), and 108 within the first 5 days (87.0%). This series did not include any

cases of concomitant stroke/MI. As shown in [Table 1](#), neurological signs were always the initial reason for presenting at the emergency room.

In the acute phase of myocardial infarction, several complications have been found to be associated with an excess risk of ischaemic stroke, such as AF, hypotension, intracardiac thrombus or cardiogenic shock. The incidence of AF may be underestimated given that recent studies have demonstrated that, in the acute phase, silent AF is 3 times more frequent than symptomatic AF [13].

Recent studies in patients with AF treated with oral anticoagulants found that the ratio of non-cerebral ischaemic systemic embolism/ischaemic stroke was 0.08%. So far, no data are available on the simultaneous occurrence of these events [14-16].

Acute ischaemic stroke with coexisting biological signs of myocardial infarction

Atrial dysfunction or cardiomyopathy, defined by the presence of specific serum biomarkers and electrocardiographic or echocardiographic findings, has been implicated in the mechanisms of ischaemic stroke. Among the serum biomarkers is cardiac troponin T, a marker of ischaemic heart disease that is used to diagnose acute myocardial infarction. Several studies have shown that 10–20% of patients with acute ischaemic stroke have troponin T-positive status. In a recent paper by Yaghi et al., out of a cohort of 1129 patients with ischaemic stroke, 113 (10.0%) had positive troponin. Interestingly, there was an association between troponin positivity and embolic stroke of unknown source (odds ratio [OR] 4.46, 95% confidence interval [CI] 1.03–7.97; $P = 0.003$) and the cardioembolic stroke subtype (OR 5.00, 95% CI 1.83–13.63; $P = 0.002$) [17]. A positive troponin test is highly specific to a cardioembolic source, but intermediate concentrations or a negative troponin test does not rule out a cardioembolic source. There are several potential reasons for early troponin elevation in cardioembolic stroke: (1) a mechanical injury to the myocardium in the course of clot migration from its cardiac origin to the brain; (2) a myocardial stress-induced injury mediated by sympathetic stimulation (which has been described in ischaemic insular stroke) [4]; (3) a reflection of myocardial fibrosis in patients without known cardiac disease; and (4) myocardial infarction related to CE.

Simultaneous cardiocerebral infarction can be diagnosed following simultaneous acute onset of a focal neurological deficit, indicating acute stroke, and chest pains or typical electrocardiogram signs.

However, two very different situations are possible, complicating the analysis of electrocardiogram changes and the elevation of cardiac enzymes. The first situation is STEMI, as in the cases presented in [Table 1](#), and the second potential situation groups together all the other conditions that can influence troponin concentrations or electrocardiogram features. In all 10 cases of simultaneous cardiocerebral embolization presented here, the ischaemic strokes occurred in large vessel territories and were, for the most part, associated with STEMI. For instance, in a case described by Kim et al., the patient presented a 2 mm ST-segment depression with a blood pressure drop to 71/53 mmHg 4 hours after admission, prompting a coronary angiography that identified CE [7].

Besides STEMI or cardiogenic shock, electrocardiographic analysis of the ST segment is very complex in patients with ischaemic stroke. In a small study including 244 consecutive patients with acute ischaemic stroke and without ischaemic heart disease, only 3% of stroke patients had troponin positivity, whereas 24% of cases had ST-T electrocardiogram changes; it was concluded that the observed electrocardiogram changes were not caused by myocardial infarction [18].

A prospective observational study demonstrated that 14% of patients with ischaemic stroke had elevated concentrations of cardiac troponin [19]. When compared with age- and sex-matched patients presenting with NSTEMI without ischaemic stroke, coronary culprit lesions were significantly less frequent among patients with acute ischaemic stroke. Moreover, half of all cases of ischaemic stroke had no angiographic evidence of coronary artery disease.

No case series has investigated the presence of CE in patients with cardioembolic stroke. Several additional factors must be taken into account regarding this specific issue: (1) the time between coronary angiography and stroke, as the anticoagulants and/or thrombolytics used for ischaemic stroke can contribute to the dissolution of the thrombus; and (2) CE of small volume, which would not lead to STEMI, but which could leave sequelae of necrosis on magnetic resonance imaging (MRI).

The case presented by Hashimoto et al. is notable because the author highlights multiple CEs, but also diffuse myocardial abnormalities on MRI [5]. In addition, the MRI on day 18 showed delayed gadolinium enhancement, revealing the presence of subendocardial myocardial infarction of the apical to mid-anterior and mid-lateral walls. T2-weighted imaging showed slight myocardial oedema in the infarcted segments, suggesting acute infarction.

Cardiac MRI can highlight ischaemic myocardial sequelae in cases where there is a suspicion of CE and, in particular, in cases with no obstructive coronary artery disease. In addition, MRI can highlight small sequelae that would not have caused STEMI, or older embolisms that would not have had clinical consequences.

AF is often associated with underlying heart failure, valvular heart disease, ischaemic heart disease and other structural heart diseases, but it can also occur as an independent entity, which is known as pure AF or lone AF.

The N-terminal fragment of the prohormone brain-type natriuretic peptide (NT-proBNP) is a powerful predictor of adverse outcomes in patients with coronary artery disease and/or heart failure, whatever the aetiology. In addition, a recent meta-analysis including 10,498 patients demonstrated that, in patients with ischaemic stroke, elevated plasma NT-proBNP concentrations were associated with an increased risk of all-cause mortality (OR 2.43, 95% CI 1.62–3.64; $P < 0.001$) and cardiovascular mortality (OR 2.01, 95% CI 1.55–2.61; $P < 0.001$) [20].

Cases in the literature include patients with signs of STEMI, but data on the kinetics of biomarkers (troponin and NT-proBNP) emphasize that the number of simultaneous cardiocerebral embolizations is probably underestimated. Systematic screening on brain imaging for acute embolization in cases of CE – and vice versa – should be considered, even with only minor neurological or cardiac signs.

Acute myocardial infarction with CE and non-acute stroke

CE is recognized as an important non-atherosclerotic cause of STEMI. The prevalence of CE as mechanism of STEMI ranges from 4% to 13% according to angiographic and autopsy studies [21, 22]. Moreover, CE is associated with clinical conditions, such as aortic and mitral prosthetic heart valves, AF, dilated cardiomyopathy, neoplasia, infective endocarditis, atrial septal defect and intracardiac tumours. In many cases, an underlying hypercoagulable state was found to be a potential predisposing prothrombogenic factor. However, most previous reports describing the prevalence and clinical characteristics of CE were based on small series of case reports. The larger cohort published by Popovic et al. included 1232 consecutive patients who presented de novo STEMI. The case definition of CE used in this study consisted of major and minor criteria according to Shibata et al. [2]. The three major criteria were: (1) angiographic evidence of coronary artery thrombosis without

atherosclerotic components; (2) concomitant multisite CE; and (3) concomitant systemic embolization excluding left ventricular thrombus attributable to STEMI. The three minor criteria were: (1) coronary angiography showing < 25% luminal stenosis, except for the culprit lesion; (2) evidence of an embolic source detected by any imaging modality; and (3) coexistence of a potential for thromboembolic disease such as intracardiac tumour, infective endocarditis, prosthetic valve implantation, AF, any condition or haemostasis disorder favouring hypercoagulable state, patent foramen ovale and atrial septal defect. The diagnosis of definite coronary embolus was based on the presence of at least two major criteria, one major criterion plus at least two minor criteria or three minor criteria. Only 53 patients with STEMI (4.3%) with CE were identified; 12 of these patients (22.6%) had multisite CE and nine patients (17.0%) had another extracoronary embolic localization. AF was the most frequent cardiac cause of CE (41.5%), whereas 15 patients had paroxysmal or permanent AF (28.3%) and seven patients had new-onset AF (13.2%). The results reported by Popovic et al. are in agreement with the results published by Shibata et al. in 2015; in their cohort of 52 patients, the authors demonstrated that the most frequent cause of CE was AF (38 patients, 73.0%), and that 13 patients had paroxysmal AF, 25 patients had chronic AF and 25 patients had non-valvular AF [2]. The authors found no significant difference in CE location (i.e. left anterior descending, left circumflex or right coronary artery), but CE was observed at multiple sites in eight patients (15.0%). In addition, 12 patients (23.0%) presented with concomitant systemic embolization: eight patients with stroke (67.0%); three patients with acute limb ischaemia (25.0%); and three with renal infarction (25.0%). In an aging population, the reduced rate of rheumatic valvular disease and an increase in the prevalence of AF could explain why the most frequent cause of CE was AF. The rate of AF was lower in the cohort analysed by Popovic et al. than in that analysed by Shibata et al., but this discrepancy may be the result of unrecognized AF in the acute phase. It should be noted that, in the majority of series, approximately one quarter of patients had no identified cause of CE.

Reperfusion therapy in cases of coexisting acute myocardial infarction and ischaemic stroke

Because of the rarity of simultaneous cardiocerebral infarction, recommendations for optimal reperfusion strategies are still lacking for this scenario [6, 23-27]. The initial treatment of ischaemic stroke will delay subsequent events (i.e. treatment for myocardial infarction). Intravenous thrombolytic

therapy with recombinant tissue plasminogen activator is currently recommended within 4.5 hours of onset. On the other hand, primary percutaneous coronary intervention is the first-line therapy for acute cardiac syndromes such as STEMI or selected NSTEMI. Fibrinolytic therapy can be used for both acute stroke and STEMI, but with different dosages and timing. Moreover, the higher doses and longer infusion duration for the treatment of STEMI may increase the haemorrhagic transformation of stroke. With the advent of endovascular therapy for ischaemic stroke in the large vessels, in combination with thrombolysis, a combined endovascular approach with the use of percutaneous coronary intervention/thrombectomy for myocardial infarction and thrombectomy devices has been suggested [6]. The American Heart Association/American Stroke Association have recommended treatment with intravenous tissue plasminogen activator at stroke dose followed by percutaneous coronary intervention if indicated.

Long-term follow-up after CE

After 5 years of follow-up, Shibata et al. found a recurrence of thromboembolism after a CE in five patients (5/52, 10.4%), including two new cases of CE (4.2%) and three cases of stroke (6.3 %), but no case of embolism in the other organs [2].

So far, no prospective study has systematically searched for myocardial infarction by CE in patients with embolic stroke associated with de novo AF. In all published cases, chest pain or electrographic signs began after the stroke was treated with thrombolysis or thrombectomy.

Conclusions

Except in rare clinical situations, we do not commonly find multiple embolic locations in patients with AF, but simultaneous cardiocerebral embolizations are probably underestimated. Their management is complex, with a temporal issue of whether to rescue the brain or the heart first; therefore we propose an algorithm for patient management based on haemodynamic stability and the presence or absence of an ST-segment elevation on the electrocardiogram (Fig. 1). In addition, within the first days after an ischaemic stroke linked to AF, cardiac MRI screening for acute infarction or previous myocardial infarction may help to identify acute cardiocerebral infarction (Fig. 2). Finally, randomized clinical trials will be difficult to carry out. However, prospective studies are ongoing to provide long-term data on pharmacological and non-pharmacological management and prognosis.

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Disclosure of interest

G. L. Consultant or speaker for the companies **Abbott, Biotronik, Boston, Medtronic** and **Microport**.

L. F. Consultant or speaker for the companies **Bayer, BMS/Pfizer, Boehringer Ingelheim, Novartis, Sanofi** and **Servier**.

Y. C. Consultant or speaker for the companies **Bayer, BMS/Pfizer, Boehringer Ingelheim, Novartis, Sanofi** and **Servier**.

The other authors declare that they have no conflicts of interest concerning this article.

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Figure legends

Figure 1. Management algorithm for simultaneous cardiocerebral infarction at the acute phase. CI: contraindication; CT: computed tomography; CTA: computed tomography angiography; ECG: electrocardiogram; IV: intravenous; MRI, magnetic resonance imaging; rtPA: recombinant tissue plasminogen activator; STEMI: ST-segment elevation myocardial infarction; tPA: tissue plasminogen activator.

Figure 2. Cardiac imaging management after the acute phase in case of suspected or confirmed simultaneous cardiocerebral infarction. CMRI, cardiac magnetic resonance imaging; CT: computed tomography; EDV: end-diastolic volume; ESV: end-systolic volume; LGE: late gadolinium enhancement; LVEF: left ventricular ejection fraction; MI, myocardial infarction.

Table 1 Clinical cases reporting coexisting acute myocardial infarction and cerebral embolism.

	MI type; ECG location and clinical status	MI location	IS location	MI treatment strategy			IS treatment strategy	Time to cardiac signs (from admission)
				CE	Culprit lesion	Other coronary arteries		
Chlapoutakis et al. ^a (2008) [4]	STEMI; DII-DIII-aVf	N/A	N/A	N/A	N/A	N/A	None	8 hours
Omar et al. (2010) [10]	STEMI; DII-DIII-aVf	RCA	VBA	N/A	N/A	N/A	None	Concomitant
Kleczyński et al. (2012) [8]	STEMI; V2-V4	LAD	Frontal	Yes	Thrombectomy without stenting	Normal	None	1-day delay
Kim et al. (2013) [7]	NSTEMI	LAD	Left MCA	Yes	Thrombectomy + stenting (DES)	Normal	None	4 hours
Hashimoto et al. (2014) [5]	STEMI V1-V6	LAD and Cx	Frontal	Yes	Thrombectomy without stenting	Normal	None	Concomitant
Maciel et al. (2015) [9]	STEMI; DII-DIII-aVf	No coronary angiography	Right MCA	N/A	N/A	N/A	IV tPA	10 hours
Tokuda et al. (2016) [11]	STEMI; DII-DIII-aVf	RCA	Left MCA + ACA	Yes	Thrombectomy without stenting	Normal	Thrombectomy	6 hours after admission, during

Kijpaisalratana et al. (2017) [6]	STEMI; DII-DIII-aVf	Cx	Right MCA	Yes	Stenting (DES)	Normal	IV tPA and thrombectomy	thrombectomy
Kijpaisalratana et al. (2017) [6]	STEMI; DII-DIII-aVf + RIVA then cardiac arrest	LAD occlusion and 99% RCA stenosis	Right MCA	Yes	Balloon on RCA	LAD occlusion	None	Minutes after CT scan
Abe et al. (2019) [3]	STEMI; DII-DIII-aVf	RCA	Right MCA proximal	Yes	Nothing	CE in distal Cx and diagonal branch	IV tPA and thrombectomy	Minutes after cerebral thrombectomy

ACA: anterior cerebral artery; CE: coronary artery embolism; CT: computed tomography; Cx: circumflex; DES: drug-eluting stent; ECG: electrocardiogram; IS: ischaemic stroke; LAD: left anterior descending; MCA: middle cerebral artery; MI: myocardial infarction; N/A: not available; NSTEMI: non-ST-segment elevation myocardial infarction; RCA: right coronary artery; RIVA: rythme idioventriculaire accéléré (accelerated idioventricular rhythm); STEMI : ST-segment elevation myocardial infarction; tPA: tissue plasminogen activator; VBA: vertebrobasilar artery.

^a Patient had transient ischaemic attack, coronary angiography at 9 days, without coronary embolism.

Acute cardio-cerebral infarction
Ischemic stroke onset < 4-5 hours and myocardial infarction < 12 hours

Brain MRI / CT / CTA / CT perfusion and 17-lead ECG

STEMI and / or cardiogenic shock ?

Yes

No

Simultaneously treat
brain and heart

Treat brain occlusion
first

According to facilities and CI*

Intravenous rtPA at brain
standard dose (0.9 mg/Kg)

IV tPA for
heart and brain

Thrombectomy ±
angioplasty for heart
Thrombectomy for brain

Combined approach
IV-tPA for brain
Thrombectomy ±
angioplasty for heart

No CI*

CI*

Endovascular treatment

If stable, cardiac evaluation at
distance from the stroke :
angiography and MRI

Acute cardio-cerebral infarction
Cardiac imaging management after acute phase

Coronary embolism at angiography

Yes

CMRI during hospitalization :
- LGE, T2 weight sequence
(prior and acute MI)
- LVEF, ESV, EDV to prognosis

Cardiac CT scan at 1 month
to evaluate
coronary therapy success

No

CMRI during hospitalization :
- LGE, T2 weight sequence
(prior and acute MI)
- LVEF, ESV, EDV to prognosis