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Concurrent Management of Acute Ischemic Stroke and Myocardial Infarction: A Case Report

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ABSTRACT

Background: Acute myocardial infarction (AMI) and acute ischemic stroke (AIS) are two leading causes of morbidity and mortality. The simultaneous occurrence of these two events is rare and its management is challenging.

Case presentation: We report the case of a 59-year-old postmenopausal woman with a history of heavy smoking who presented with sudden-onset right hemiplegia. She was diagnosed with an acute ischemic stroke. Although she did not experience chest pain, the initial electrocardiogram revealed an acute simultaneous ST-elevation myocardial infarction (STEMI). Cerebral computed tomography angiography ruled out aortic dissection and cerebral hemorrhage. The patient was treated invasively for the STEMI by a left anterior descending angioplasty and anticoagulants were stopped immediately after angioplasty while the patient continued to receive dual antiplatelet therapy. The immediate evolution was favorable. The patients did not have heart failure or rhythm complications and the modified Rankin scale score at discharge was 2.

Conclusion: Cardio-cerebral infarction Syndrome is a rare but life-threatening condition that requires individualized management. Further studies are needed to develop standardized guidelines for the optimal treatment.

Keywords: Myocardial infarction, stroke, revascularization

INTRODUCTION

Acute myocardial infarction (AMI) and acute ischemic stroke (AIS) are two leading causes of mortality(1) Concomitant occurrence of these two events, known as Cardio-Cerebral Infarction Syndrome (CCIS), is rare. A reported incidence ranged between 0.009% to 0.29% (2,3), ST-elevation myocardial infarction (STEMI) was more often at the forefront of the clinical presentation(4).

Prompt action is essential in this situation which involves many therapeutic issues (5) and is not supported by clear guidelines.

This report aimed to elucidate the main difficulty points and elements to consider for a balanced risk assessment and adapted management through an illustrative clinical case.

CASE PRESENTATION

A 59-year-old menopausal, heavy smoker female with no other known cardiovascular risk factors nor further addictions and no hormone replacement therapy, presented to the emergencies of the National Institute of Neurology for a sudden onset of right hemiplegia with a time from last known well to presentation of 4 hours. There were no witnesses at the time of the index event. Immediate physical examination rated the National Institutes of Health Stroke Scale (NIHSS) score at 8. The patient had not been reporting any chest pain or

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dyspnea. She was conscious, oriented, and sweaty, without respiratory distress. The body temperature was 37°C. The neurological assessment showed slight hemiparesis, normal cranial nerve function, motor and sensory grades, and deep tendon reflexes. Her Glasgow Coma Scale score was 15/15. The Cardiovascular examination revealed a symmetric blood pressure of 120/60 mmHg in both arms and a regular heart rate of 80 beats per minute. There were no murmurs on the heart and along the main arterial axes (carotid, subclavian, renal, and femoral). The initial electrocardiogram (ECG) made three minutes after the presentation (Figure 1), showed a sinus rhythm and a convex, three millimeters ST-segment elevation in leads anterior and inferior leads (V_2 to V_6 DII, DIII, and aVF) and Q waves in the anteroseptal leads V_1 to V_4. QRS width was 85 milliseconds and corrected QT was 445 milliseconds. Aorta and cerebral vasculature were investigated by computed tomography thoracic and cerebral angiography; aortic dissection was invalidated and there was no thrombus in the cerebral arteries and no carotid stenosis. The patient's blood glucose level was 4.26 g/L without ketosis.

High-sensitivity cardiac Troponin I (hs-cTnI was 1.123 ng/ml (normal < 0.01 ng/ml)

The complete blood count was within normal range, with a white blood cell count of 9,280/mm3, hemoglobin of 12.6g/dl, and a platelet count of 260,000/mm3. The patient's blood type was O-positive, and the prothrombin time was 85%. The creatinine level was 0.9 mg/dL, and the C-reactive protein was 8 mg/L. The transthoracic (Figure 2) and transesophageal echocardiograms revealed a symmetrical left ventricular hypertrophy with anterior and septal akinesis, ejection fraction was estimated at 36%, and global longitudinal strain was -12%. Additionally, it showed a moderate rheumatic mitral valve stenosis with a surface of 1,9 cm^2 and a mean gradient of 6 mmHg, a dilated left atrium 43 ml/m^2body surface with moderate contrast but no thrombus. The left atrial reservoir strain was reduced at 21%, suggesting a possible atrial cardiomyopathy. There was no evidence of a patent foramen ovale. There were grade 3 atheromatous descending aorta plaques without ulceration or calcification. She did not receive alteplase since the therapeutic window time for thrombolysis of acute ischemic stroke was over, and the Magnetic resonance imaging fluid-attenuated inversion recovery (MRI FLAIR)-diffusion mismatch protocol was unavailable. Nor did she undergo mechanical thrombectomy due to the absence of intraluminal thrombus in the cerebral angiogram. Following the neurologist's expert advice, she received 200 mg aspirin, 300 mg clopidogrel, 20 mg Rosuvastatin, and a 70 IU/kg IV unfractionated heparin with close monitoring of secondary brain insults of systemic origin (SBISOs). Additionally, her blood glucose was corrected to 1.5 g/L through intravenous insulin therapy. After the neurological stabilization, a primary percutaneous coronary angiography (Figure 3) was performed at 2 hours 15 minutes from initial presentation through the right radial artery. It revealed a total occlusion of the proximal left anterior descending artery and diffuse disease in the right coronary artery with a maximum stenosis of 80% in the mid-part. Thrombo-aspiration was deemed non-feasible by the operator. It was determined that stent insertion was required. The coronary wire passed freely in the LAD, and a 3,00 x 18 mm drug-eluting stent was deployed successfully in the proximal LAD. The final angiogram confirmed thrombolysis in myocardial infarction risk score III flow, indicating successful restoration of blood flow. The patient was transferred to the intensive care unit for close monitoring. The patient's neurological status remained stable.

The ST-segment elevation regressed upon leaving the catheterization laboratory. Anticoagulation was stopped due to the cerebral bleeding risk. Bisoprolol, Aldactone and ramipril were introduced after 24h. The patient was then monitored for 5 days in the hospital, then she was discharged on dual antiplatelet therapy with concomitant gastric protection. The patient's modified Rankin Scale (mRS) score was 2, and she was scheduled for regular follow-up visits at the outpatient clinic for 3 months.

DISCUSSION

This case highlighted the complexities involved in the management of cardio-cerebral infarction syndrome as far as risk assessment, etiology investigation, and therapeutic management are concerned

In this case, stroke was the first clinical presentation, and STEMI diagnosis was based on ECG and cardiac biomarkers.

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Epidemiology

The epidemiological data on simultaneous cardio-cerebral infarction is quite limited. The reported incidence has been estimated to range from 0.009% to 0.29% (2,3,6) .

The most common type of myocardial infarction associated with this condition was anterior ST-elevation myocardial infarction (38,3%) (4) which was the case in our clinical presentation.

Simultaneous cardio-cerebral infarction reveals critical considerations. The narrow therapeutic time window presents challenges. The incidence and sequence of the two conditions, as well as the associated morbidity when they occur concurrently, warrant further investigation (2,7).

Pathogenesis

The mechanism contributing to CCIS remains debated and may be multifactorial.

CCIS can be classified into three types (7) type I with concurrent cardio-cerebral infarction syndrome which was observed in our patient. Type II involved Acute ischemic stroke (<4.5 hours) after a recent myocardial infarction. Type III with an Acute myocardial infarction (< 12 hours) after a recent ischemic stroke. The occurrence of simultaneous or nearly simultaneous cardio-cerebral infarction involves three main mechanisms, First, cardiovascular causes including atrial fibrillation, acute aortic dissection with extension to the coronary and carotid arteries, and arterial vasospasm due to electrical shock. Furthermore, a pre-existing left ventricular thrombus secondary to impaired left ventricular function or thrombosis of a prosthetic valve (i.e. from suboptimal anticoagulation) can lead to this condition. Thrombus formation in the right ventricle during acute right ventricular infarction, combined with a patent foramen ovale, can result in embolization to both cerebral and coronary territories. Severe hypotension or cardiogenic shock following acute myocardial infarction can also contribute to concurrent stroke and myocardial infarction. We discussed in the case of our patient a cardiac cause like a left atrium thrombus due to mitral moderate stenosis and/or a paroxysmal atrial fibrillation, however, we did not obtain any echocardiographic or electrocardiographic sign of this hypothesis. Brain causes, consist of autonomic dysfunction, indeed, the insular cortex plays a critical role in central autonomic system regulation with the right-side stimulation of the insular cortex resulting in a predominant sympathetic activation. This cause remained a plausible hypothesis in our patient, AIS was in the front frame of clinical presentation. Finally, non-cardiac and non-brain causes were observed for example in coronavirus disease 2019 (COVID-19) infection with an increased risk of both AIS and AMI.

Diagnostic and Management

Given the scarcity and intricacy of concurrent cardio-cerebral infarction, there are limited guidelines for its management, treating one condition will necessarily delay managing the other, the reason why handling this situation should be personalized. While intravenous alteplase may be an option, alteplase is used at a higher dose in acute coronary syndrome, which could lead to intracranial bleeding. However, if the patient is within the time window for thrombolysis, the alternative would be Stroke-dose alteplase thrombolysis typically 0.9 mg/kg avoiding the cardiac dose which can reach up to 1.0 mg/kg as a bolus (with a maximum of 100 mg), to reduce the risk of intracranial hemorrhage, this would then be followed by percutaneous coronary angioplasty.(8). Our patient had surpassed the time window for thrombolysis after she had undergone brain computed tomography angiography. Additionally, there was no large vessel occlusion on cerebral imaging, making mechanical thrombectomy futile, otherwise, we could opt for thrombolysis before the coronary angiography, which might have improved the neurological recovery. Furthermore, the inability to determine the exact timing of the ST-segment elevation and the presence of akinetic wall segments complicated the management.

The primary dilemma lies in determining which organ's condition should be prioritized for treatment. Performing percutaneous coronary intervention may delay the management of AIS, potentially missing the therapeutic window for thrombolysis. Conversely, undergoing mechanical thrombectomy to address cerebral

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infarction could delay coronary revascularization, leading to further cardiac damage and increasing the risk of arrhythmias and cardiogenic shock.

Postprocedural management following CCIS presents several clinical challenges. AMI typically requires dual antiplatelet therapy (DAPT), with aspirin combined with either ticagrelor or prasugrel, as these agents have demonstrated superiority over clopidogrel in improving outcomes(9,10). For stroke patients, the standard of care generally involves single antiplatelet therapy, DAPT (usually aspirin and clopidogrel) is indicated only for specific presentations such as transient ischemic attacks or severe intracranial stenosis (11), where the benefits must be carefully weighed against the risk of bleeding.

Although anticoagulation is a standard treatment for AMI, it is typically avoided in acute ischemic stroke (AIS) due to the risk of hemorrhagic transformation, unless there is a compelling indication, such as atrial fibrillation or the presence of a mechanical heart valve.

Concerning adjuvant medical treatment, beta-blockers, and angiotensin-converting enzyme inhibitors are beneficial and standard treatments in AMI management to reduce morbidity and sudden death. Still, blood pressure management following a stroke should be more precautious and progressive, it is recommended not to reduce blood pressure in the first hours unless it is above 220 mmHg (or 180 mmHg when thrombolysis is indicated). Our patient had left ventricular dysfunction indicating the need for Renin–Angiotensin–Aldosterone System inhibitors and beta-blockers, which can lead to hypotension and hypoperfusion, potentially compromising collateral cerebral circulation and worsening cerebral infarct size. We delayed their use by 24 hours after the index event.

CONCLUSION

CCIS is a rare phenomenon that remains poorly studied. Prompt revascularization is crucial and Treatment should be personalized. The prognosis is often unfavorable. Patient management must be personalized based on, the underlying mechanism, and the timing of presentation. A multidisciplinary approach involving radiologists and cardiologists is essential. Further studies are needed to provide better guidance for the optimal management of CCIS.

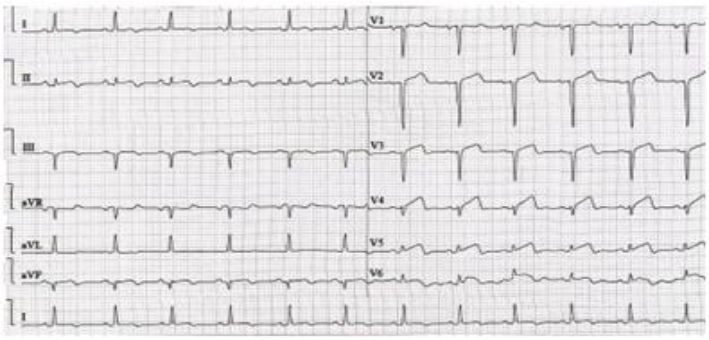

Figure 1: Twelve-lead electrocardiography (ECG); ST-segment elevation in anterior and inferior leads (V1 to V5 and in leads DII, DIII, and aVF). With a Q wave anterior leads V1 to V4.

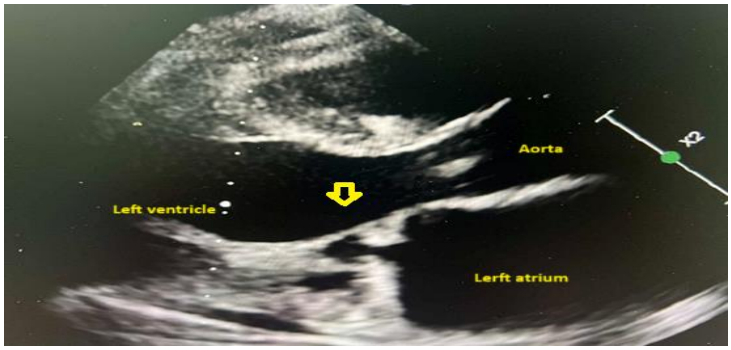

Figure 2: Transthoracic echocardiography; rheumatic mitral valve stenosis and left atrial dilation. The yellow arrow shows a thickened mitral apparatus and mitral stenosis

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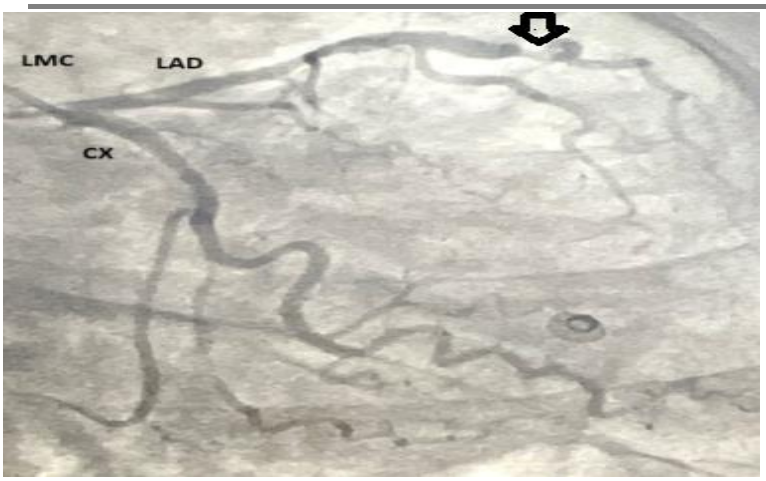

LMC: Left main coronary artery, LAD left anterior descending artery, CX: circumflex artery

Figure 3: Coronary angiogram. Right anterior oblique caudal incidence showing a recent total occlusion of the mid-left anterior descending artery (arrow).

Abbreviations

AMI: acute myocardial infarction
AIS: acute ischemic stroke
CCIS: cardiac cerebral infarction syndrome
CX: circumflex artery
DAPT: dual aggregation antiplatelet therapy
LAD: left main descending artery
LMC: Left main coronary artery
FLAIR: Fluid-attenuated inversion recovery
MRI: Magnetic resonance imaging
SBISOs: secondary brain insults of systemic origin
STEMI: ST-elevation myocardial infarction

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The authors declare that they do not have a competing/conflict of interest in this paper.

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Data interpretation:

The patient's data were anonymized.

Patient's consent:

The patient gave her informed consent for this publication. When fully conscious and after getting clear and well understood explanations

Ethical considerations:

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Authors contributions:

Abdelmoula Yecine: patient's clinical care and investigation, main writer of the paper

Mghaieth Zghal Fathia: echocardiography scanning and interpretation, main senior writer of the paper

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Saied Zakaria: neurological care and writing of the neurological care chapter of the paper

Ben Halima Manel: bibliography and revision of the paper

Jebberi Zeynab: patient's care, ECG interpretation, iconography choice and bibliography

Daly Foued: reanimation of the patient, writing of reanimation aspects and revision of the paper

Ouali Sana: english correction and overall valuable remarks about the structure of the article and its plan

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Ben Sassi Samia: neurology imaging interpretation and many valuale advices about how guidelines guided neurological treatment and its writing in the paper

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