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Case Report

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Cardio-cerebral infarction following syncope

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ABSTRACT

Cardio-cerebral infarction (CCI) initially introduced by Omar et al. in 2010, pertains to the concurrent manifestation of acute ischemic stroke (AIS) and acute myocardial infarction (AMI). In this study, we presented a 50-year-old man who arrived at the emergency department with syncope 6 hours prior. Neurological examination revealed left nasolabial fold flattening and left hemiparesis. Computed tomography brain scan showed no intracranial hemorrhage, while diffusion brain magnetic resonance imaging displayed restricted diffusion of the right centrum ovale in the periventricular space. Electrocardiogram indicated subacute ST-segment elevation in V2-V4, which resulted in the diagnosis of CCI. Due to ST-elevated myocardial infarction, he received aspirin, ticagrelor, and heparin before undergoing emergent coronary angiography. After multidisciplinary discussion, the patient was planned for coronary artery bypass graft surgery. His AIS was medically managed with antiplatelet and anticoagulant therapy. CCI is a rare and high-mortality disease arising from the simultaneous occurrence of AIS and AMI. Due to its rarity, there's no consensus on its treatment. The treatment process for AIS is limited to the patient's suitability for thrombolytic and thrombectomy therapy. We advise using a hybrid angiography laboratory for AIS patients.

Keywords: Cardio-cerebral infarction, acute myocardial infarction, acute ischaemic stroke, syncope

INTRODUCTION

Cardio-cerebral infarction (CCI), first defined in 2010 by Omar et al., refers to the simultaneous occurrence of acute ischemic stroke (AIS) and acute myocardial infarction (AMI).[1] Its incidence rate has been documented at 0.009%.[2] CCI can result from aortic dissection, hypotension, AMI, atrial fibrillation, and embolus originating from prosthetic valves.[3] Due to the limited number of cases, there is no consensus on the treatment management of CCI patients. The objective of this case presentation is to illustrate a patient with CCI who presented to the emergency department (ED) following a syncopal episode, with a detailed outline of our treatment approach, following the acquisition of informed consent.

CASE

A 50-year-old man was admitted to the ED with syncope 6 hours before arrival. His medical history was significant for type 2 diabetes mellitus (DM) and chronic tobacco use. He was on metformin 500 mg twice a day (2×1) orally and was not taking any antiplatelet or anticoagulant therapy. He reported mild diaphoresis. His vital signs were normal. Glucose level was 125 mg/dl. Glasgow Coma Scale score was 15. Neurologic examination revealed flattening of the left nasolabial fold and left hemiparesis. His National Institutes of Health Stroke Scale (NIHSS) score was 2. Magnetic resonance imaging (MRI) showed acute cytotoxic edema compatible with restricted diffusion of the right centrum ovale in periventricular space (Figure 1). Computed tomography (CT) angiography of the aorta didn't show aortic dissection (Figure 2). Electrocardiogram (ECG) showed subacute ST-segment elevation in V2-V4 (Figure 3). hs-TroponinT was 754 ng/L. Echocardiography showed a left ventricular ejection fraction (LVEF) of 40% associated with hypokinetic apex. He was loaded with 300 mg aspirin, 180 mg ticagrelor and 5000 IU heparin, then taken for emergent coronary angiography (CAG). CAG revealed a filling defect in the left coronary artery but distal flow was adequate. Because he was hemodynamically stable and distal Thrombolysis in Myocardial Infarction (TIMI) grade flow was 3 (complete perfusion), percutaneous coronary intervention (PCI) was not performed. The patient planned for coronary artery bypass graft (CABG) surgery. His AIS was managed medically with antiplatelet and anticoagulant therapy. The patient was discharged on the 7th postoperative day with a modified Rankin score of 1, and was prescribed aspirin 100 mg once daily and enoxaparin 40 mg subcutaneously once daily for ten days. At the one-week follow-up, the patient was started on dual antiplatelet therapy with lifelong aspirin 100 mg daily and clopidogrel 75 mg daily.

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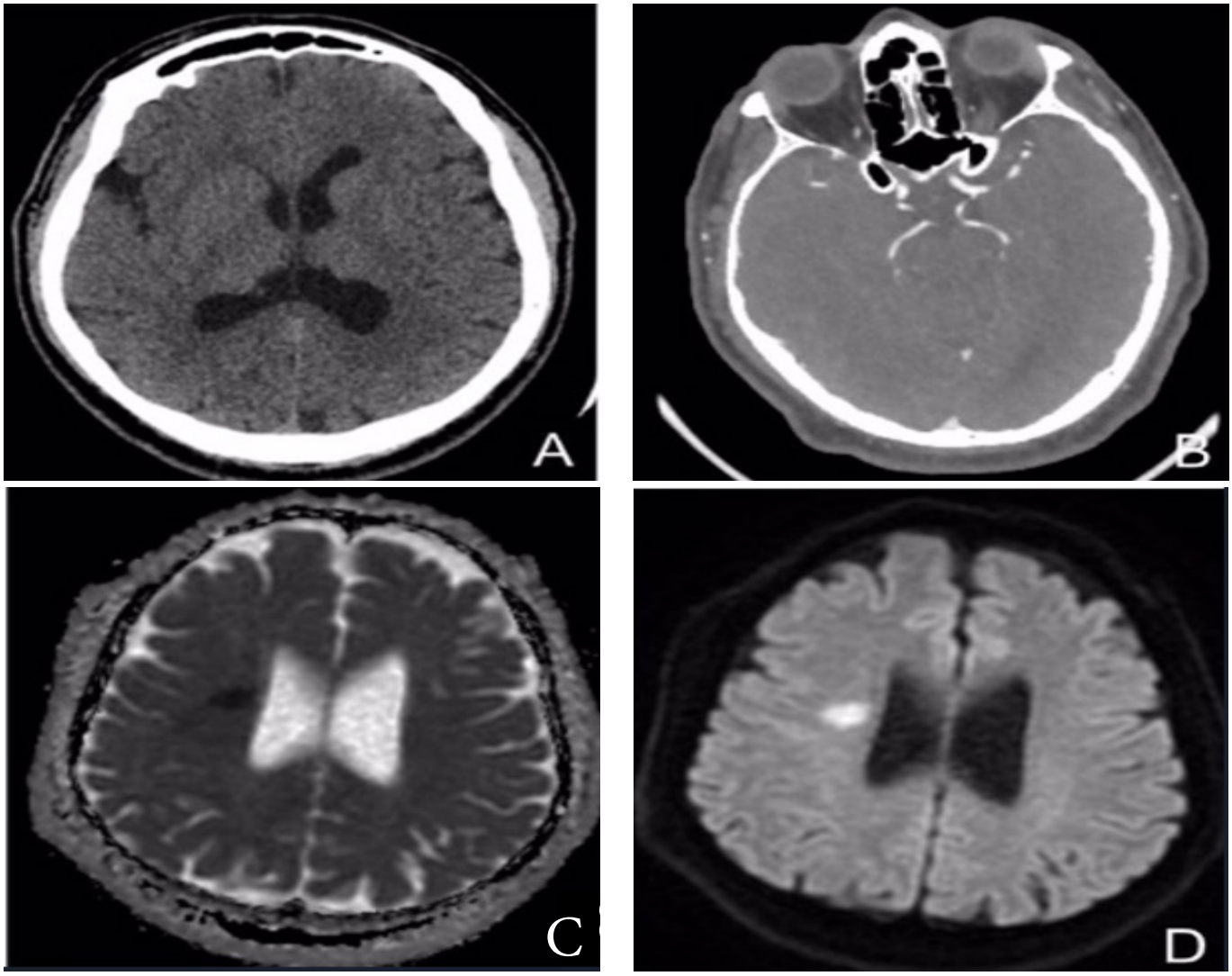

Figure 1. **A**) The brain computed tomography scan did not reveal any signs of intracranial bleeding. **B**) There was no major vessel occlusion in the cerebral angiography. **C**) Acute diffusion restriction detected in the centrum ovale on apparent diffusion coefficient (ADC) magnetic resonance imaging sequence. **D**) The hyperintensity observed in the diffusion-weighted imaging (DWI) scan is consistent with acute ischemic stroke

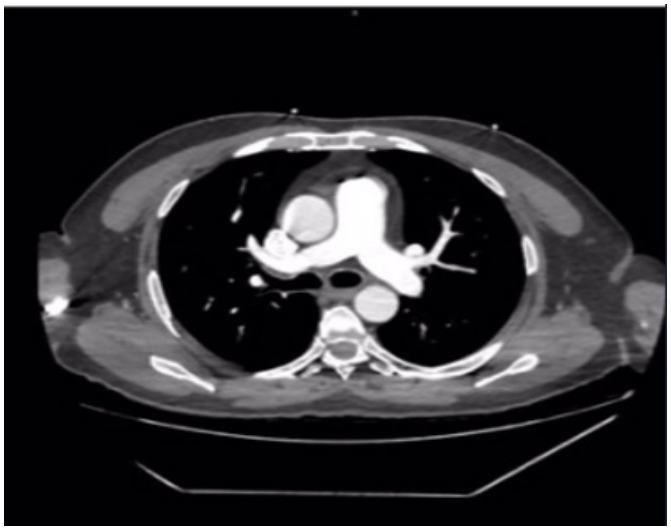

Figure 2. Aortic dissection is not present in the computed tomography angiography

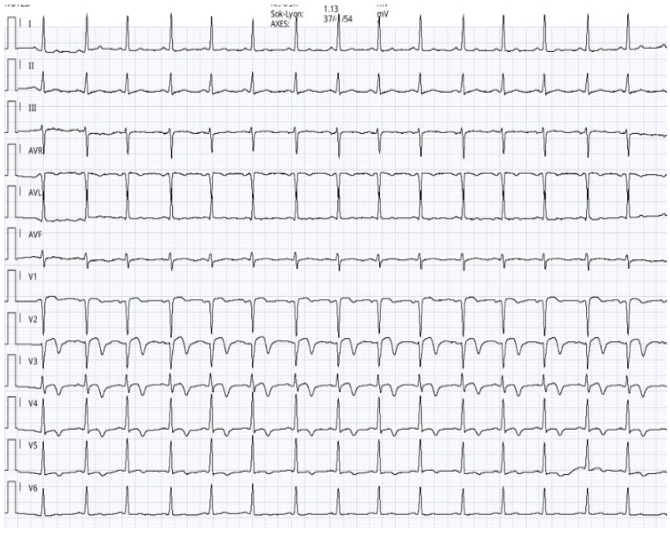

Figure 3. The electrocardiogram reveals signs consistent with anterior myocardial infarction

DISCUSSION

CCI is a condition characterized by the simultaneous occurrence of AIS and AMI, resulting in high mortality rates.[4] CCI can be categorized into two groups based on the timing of component occurrences. Synchronized CCI refers to the simultaneous infarction of coronary and cerebral vessels, while metachronous CCI involves sequential infarctions of coronary and cerebral vessels.[2,4] It has been reported that 66% of cases are synchronized, and 33% are metachronous. Men are found to be more susceptible than women, and the average age is commonly in the fifth decade of life. The most prevalent comorbidities are smoking, DM, and hyperlipidemia.[1] In our presented case, we observed a metachronous CCI in a 50-year-old male with a history of smoking and DM. Thus, our case is consistent with the demographic characteristics reported in the literature.

Left ventricular dysfunction arising from AMI increases the risk of left ventricular thrombus formation and embolism.[5,6] In the presented case, echocardiography revealed an LVEF of 40% and apical hypokinesis. Therefore, we believe that CCI may stem from central hypoperfusion due to reduced cardiac output or cardiac microthrombi. A similar case in the literature describes a 71-year-old patient who presented to the ED with speech impairment following syncope. Diffusion MRI revealed acute diffusion restriction in the left parietal cortex. CAG was performed on the patient with a De Winter pattern on the ECG. Subsequently PCI was performed on the circumflex artery (CX). In this CCI case, the development of cerebral infarction was attributed to hypoperfusion. The patient was discharged seven days later with antiplatelet and anticoagulant therapy without any neurological impairment.[7]

Due to the rarity of CCI, there is no consensus on its treatment. Personalized treatment modalities include PCI and cerebral thrombectomy, only 0.9 mg/kg (cerebral dose) intravenous (IV) thrombolytic, 0.9 mg/kg IV thrombolytic and PCI, dual antiplatelet therapy and anticoagulant treatment were applied according to the patient.[3] In this case, the patient exceeded the therapeutic window and had a low NIHSS score, so thrombectomy and IV thrombolytic therapy were not administered. For AMI, the patient underwent CAG revealing an 80% stenosis before the diagonal branch of the left anterior descending artery (LAD), an 80% stenosis at the ostium of first diagonal artery (D1), and a 98% stenosis after D1, leading to CABG surgery.

CONCLUSION

The medical history of patients arriving at the ED with acute neurological symptoms should be taken comprehensively, followed by a performed ECG. In the treatment process, if simultaneous intervention is required for stroke and AMI, intervention can be performed in the hybrid angio laboratory. If thrombolysis and thrombectomy are not suitable for stroke, AMI treatment should be done first.

ETHICAL DECLARATIONS

Informed Consent

The patient signed and free and informed consent form.

Referee Evaluation Process

Externally peer-reviewed.

Conflict of Interest Statement

The authors have no conflicts of interest to declare.

Financial Disclosure

The authors declared that this study has received no financial support.

Author Contributions

All of the authors declare that they have all participated in the design, execution, and analysis of the paper, and that they have approved the final version.

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