



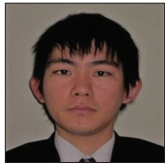
Case Report

A case of cardiac tamponade during the treatment of simultaneous cardio-cerebral infarction associated with atrial fibrillation – Case report

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Received : 04 October 19

Accepted : 23 November 19

Published : 06 December 19

DOI

10.25259/SNI_504_2019

Quick Response Code:



ABSTRACT

Background: Simultaneous cerebral and myocardial infarction is called cardio-cerebral infarction (CCI). It is a rare condition, and its management strategy has yet to be determined. We report a case of cardiac tamponade during the treatment of CCI associated with atrial fibrillation.

Case Description: A 72-year-old man presented with loss of consciousness after chest discomfort. He had taken rivaroxaban for paroxysmal atrial fibrillation. Twelve-lead electrocardiography showed ST elevation at II, III, and aVF. His National Institutes of Health Stroke Scale was 29. We diagnosed him with synchronous cardioembolic stroke and acute myocardial infarction due to atrial fibrillation. The coronary angiography revealed distal occlusion in the posterior descending branch of the right coronary artery, and overall myocardial perfusion seemed sufficient. The diffusion-weighted image showed hyperintense lesions at the cerebellum, and magnetic resonance angiography did not reveal the flow of the basilar artery. The patient's NIH score improved immediately, so we did not perform intravenous tissue plasminogen activator (IV-tPA) administration nor endovascular treatment. Heparin administration was started. After 38 h from the onset, he suffered from hydrocephalus, and cerebral ventricular drainage was performed. Subsequently, circulatory dynamics worsened, and he was diagnosed with cardiac tamponade. Emergency pericardiectomy was performed, and he has been taking intensive care.

Conclusion: Some cases with CCI treated with IV-tPA and endovascular intervention were reported, but the treatment strategy should be still discussed multidisciplinary. Especially, the administration of antithrombotic drugs for CCI should be carefully performed because fatal hemorrhage such as cardiac tamponade can occur.

Keywords: Acute ischemic stroke, Acute myocardial infarction, Atrial fibrillation, Cardiac tamponade, Cardio-cerebral stroke

INTRODUCTION

Simultaneous cerebral and acute myocardial infarction (AMI) is called cardio-cerebral infarction (CCI)^[12] and is as rarely encountered as 0.009% of ischemic stroke patients.^[15] Because of the narrow time window for treatment and the complex pathophysiology, CCI is challenging to diagnose and treat immediately. However, its management strategy has yet to be determined due to its rarity. We described a case of CCI treated with conservative therapy, which led to cardiac tamponade treated by pericardiectomy.

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CASE PRESENTATION

A 72-year-old man presented with sudden onset of loss of consciousness after chest discomfort and numbness in his left

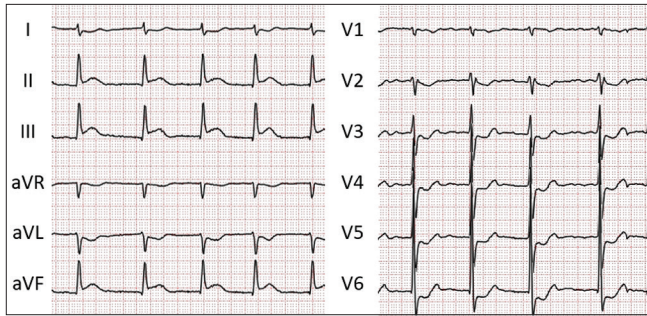


Figure 1: Twelve-lead electrocardiography on admission showed atrial fibrillation with a heart rate of 78 beats/min and ST elevation at II, III, and aVF and ST depression at V2–V6.

upper limb. His medical history included hypertension and paroxysmal atrial fibrillation, and he had taken rivaroxaban. His blood pressure was 150/101 mmHg without any significant difference between right and left limbs. The Japan coma scale was III-100, and the Glasgow Coma Scale was 8 (E1V2M5) on the admission. His National Institutes of Health Stroke Scale score was 29. Twelve-lead electrocardiography showed atrial fibrillation with a heart rate of 78 beats/min and ST elevation at II, III, and aVF and ST depression at V2–V6 [Figure 1]. The head computed tomography (CT) image showed no intracranial hemorrhages [Figure 2a], but the hyperdense arterial sign was observed at the basilar artery [Figure 2b], which suggested the basilar artery occlusion. The contrast-enhanced chest CT did not indicate aortic dissection nor cardiac tamponade [Figure 2c]. The platelet count was $13.2 \times 10^4/\mu\text{L}$, and D-dimer was 7.5 mg/dL, and the other laboratory data were almost within normal limits.

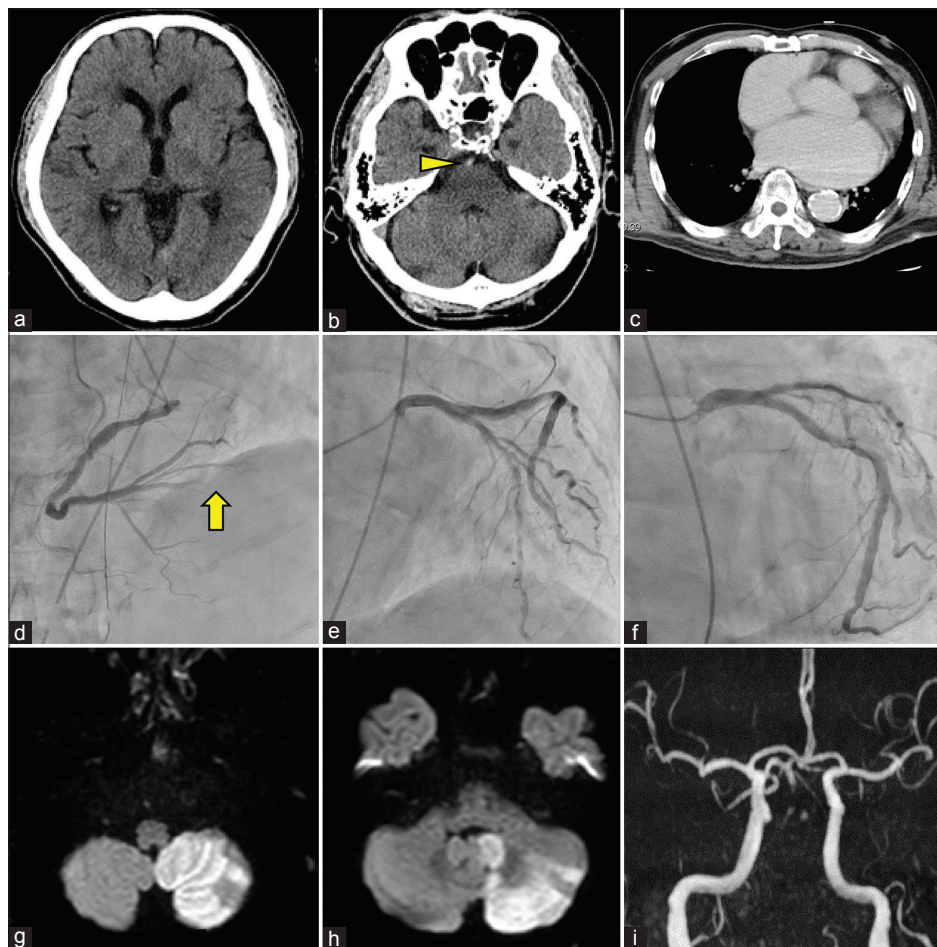


Figure 2: The head computed tomography (CT) image showed no intracranial hemorrhages (a), but the hyper-dense arterial sign was observed at the basilar artery (b, arrowhead), which suggested the basilar artery occlusion. The contrast-enhanced chest CT did not indicate aortic dissection nor cardiac tamponade (c). The coronary angiography revealed distal occlusion in the posterior descending branch of the right coronary artery (d, arrow). Mild stenotic lesions suggestive of atherosclerotic pathology were observed in some parts (e and f). The diffusion-weighted image showed hyper-intense lesions in the region of the posterior inferior cerebellar artery (g and h). Magnetic resonance angiography did not reveal the flow of the basilar artery, and severe stenotic lesions of the major arteries were scarcely observed (i).

The patient was diagnosed with CCI, and a multidisciplinary discussion was done. Due to the current use of rivaroxaban and high NIH score, he was not considered as a candidate for intravenous tissue plasminogen activator (IV-tPA) treatment. We immediately performed coronary angiography for salvage of the myocardial ischemic area, and it revealed distal occlusion in the posterior descending branch of the right coronary artery [Figure 2d]. Mild stenotic lesions suggestive of atherosclerotic pathology were observed in some parts [Figure 2e and f]. We considered that overall myocardial perfusion was sufficient and that immediate further investigation on stroke was needed, so percutaneous coronary intervention was not performed. Magnetic resonance imaging was subsequently performed, and the diffusion-weighted image showed hyperintense lesions in the region of the posterior inferior cerebellar artery [Figure 2g and h]. Magnetic resonance angiography did not reveal the flow of the basilar artery, and severe stenotic lesions of the major arteries were scarcely observed [Figure 2i]. Therefore, we diagnosed him with cardioembolic stroke. We considered percutaneous endovascular recanalization of the basilar artery, but the fatal hemorrhagic risk of the ischemic cerebellum was deeply concerned. At that time, the patient's Japan coma scale was improved as I-1 and his NIH score was four. Due to the rapid improvement of stroke symptoms,

we started medical treatment with 10,000 unfractionated heparin administration per day. He started rehabilitation, and his blood pressure was around 140/90 mmHg without any vasopressors.

After 38 h from the onset, his consciousness level was depressed as Japan coma scale I-3. The head CT showed hydrocephalus [Figure 3a] due to cerebellar edema and hemorrhagic infarction of the ischemic cerebellum, which compressed the aqueduct [Figure 3b]. We stopped the heparin administration and performed cerebral ventricular drainage under local anesthesia to control hydrocephalus. External decompression for cerebellar edema and hemorrhagic infarction was subsequently planned, but he suddenly presented shock after intubation by an anesthesiologist. After stabilizing circulatory dynamics with adrenaline and noradrenaline, the head CT showed improvement in hydrocephalus [Figure 3c]. The chest CT showed cardiac tamponade [Figure 3d], considered as the cause of shock. Emergency pericardiotomy was immediately performed to allow the pericardial fluid to drain [Figure 3e]. The pericardial fluid was bloody and over 500 mL. A left mural and atrial appendage thrombi were observed. There were no apparent bleeding sources, aortic dissections, nor myocardial ruptures intraoperatively, suggesting that his cardiac tamponade seemed idiopathic or to be resulted from oozing type cardiac

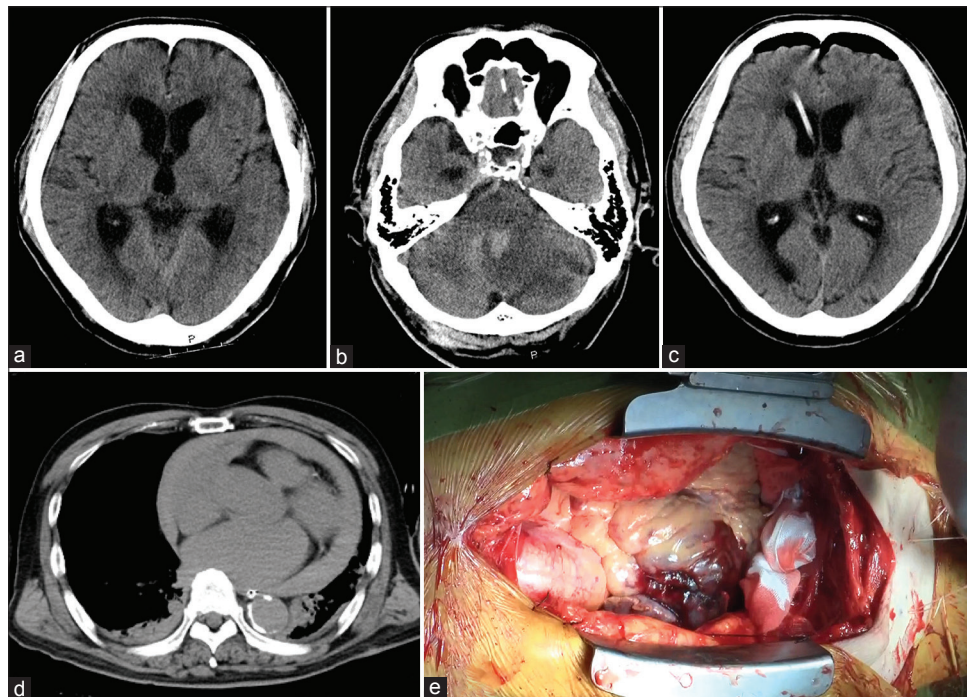


Figure 3: After 38 h from the onset, his consciousness level was depressed as Japan coma scale I-3. The head computed tomography (CT) 38 h after the onset showed hydrocephalus (a) due to cerebellar edema and hemorrhagic infarction of the ischemic cerebellum, which compressed the aqueduct (b). The head CT after cerebral ventricular drainage showed improvement in hydrocephalus (c). The chest CT showed cardiac tamponade (d). Emergency pericardiotomy was immediately performed to allow the fluid to drain. A left mural and atrial appendage thrombi were observed. There were no apparent bleeding sources, aortic dissections, nor myocardial ruptures intraoperatively (e).

Table 1: Previous reports on cardio-cerebral embolization.^[1,6-9,11,12,15]

Author/year	Age/Sex	Chief complaint	Premorbid antithrombotic drugs	NIHSS	Stroke territory	MI territory	Treatment	Outcome
Omar/2010	48/M	Chest pain, vomiting	None	40	Basilar artery	Inferoposterior wall	Antiplatelet, anticoagulant	Poor
González/2014	66/W	Left hemiparesis, chest pain	None	16	Right MCA	Inferior wall	IV-tPA, aspirin 48 h after the onset, PCI (DES) on day 4, DAPT	-
Maciel/2015	44/M	Left hemiparesis, dysarthria, left hemineglect	None	11	Right MCA	Inferior wall	IV-tPA	Good
Kijpaisalratana /2017	65/M	Left hemiparesis, dysarthria, left hemineglect	None	12	Right MCA	Inferior wall	IV-tPA, PCI (DES)	Poor
Yeo/2017	67/M	Left hemiparesis, chest pain	None	13	Possible right MCA	Anterior and inferior wall	PCI (ballooning)	Poor
	45/M	Chest pain, left hemiplegia, visual neglect	Heparin, warfarin	16	Right ICA	Possible LAD territory	PCI (stenting), cerebral thrombectomy	Poor
	53/M	Right hemiplegia, aphasia	None	23	Left MCA	LAD territory	PCI (stenting), cerebral thrombectomy	Poor
	71/W	Left hemiplegia, vomiting	None	27	PCA	LAD territory	Cerebral embolectomy, PCI (stenting)	Poor
	55/M	Right hemiplegia, aphasia	None	18	Left MCA	Lateral wall	IV-tPA	Good
	57/M	Right hemiplegia, aphasia	None	16	Left MCA	Lateral wall	IV-tPA	Good
Abe/2019	73/W	Right hemiplegia, aphasia	None	21	Left MCA	Posterior wall	IV-tPA, cerebral thrombectomy	Good
Gungoren/2018	69/M	Chest discomfort, right hemiplegia, aphasia	None	13	Possible right MCA	Anterior wall	IV-tPA, PCI (DES), DAPT	Good
Obaid/2018	41/W	Syncope, facial droop	DAPT	6	Left MCA	Anterior wall	IV-tPA, PCI (DES) 48 h after the onset, DAPT	Poor
Our case/2019	72/M	Chest discomfort, coma	Rivaroxaban	29	Basilar artery	Possible inferior wall	Heparin	Under treatment

DAPT: Dual antiplatelet therapy, DES: Drug-eluting stent, Good: Modified Rankin Scale 0-2, ICA: Internal carotid artery, IV-tPA: Intravenous tissue plasminogen activator, LAD: Left anterior descending artery, MCA: Middle cerebral artery, MI: Myocardial infarction, NIHSS: National Institutes of Health Stroke Scale, PCA: Posterior cerebral artery, PCI: Percutaneous coronary intervention, Poor: Modified Rankin Scale 3-6

rupture due to AMI. The blood pressure gradually increased, and the patient no longer needed any vasopressors. Seven days after pericardiectomy, his Japan coma scale was III-100. He has been undertaking rehabilitation.

DISCUSSION

Table 1 shows previously reported 13 patients with CCI as far as we know.^[1,6-9,12,15] Among the 13 reported patients, the 11 patients did not receive oral anticoagulation before the event, and the ones^[11] had both heparin and warfarin, or dual antiplatelet therapy each. Our patient had rivaroxaban but suffered from CCI. Despite adequate rivaroxaban treatment, stroke or systemic embolism can occur as 2.1% per year.^[13] Besides, 6% of patients with acute stroke had ST-segment elevation,^[15] and 12.7% of geriatric patients had CCI who were screened for AMI within 72 h of admission for acute stroke.^[3] In the case of cardioembolic stroke, it is important to pay attention to the possibility of simultaneous AMI. Our patients first complained of chest discomfort, so we could relatively easily detect AMI. However, most patients with embolic stroke cannot complain of typical symptoms of AMI due to aphasia, impaired consciousness, and depressed level of consciousness, so electrocardiogram monitoring and repeated 12-lead electrocardiogram are considered for early detection of CCI.

There are several mechanisms to explain CCI occurrence. The anterior and apical wall infarction associated with a decrease of the left ventricular systolic function induces the formation of the left ventricular mural thrombus,^[5] and this thrombus might cause CCI. A case of embolization to the coronary and cerebral arteries with atrial fibrillation has been also reported.^[4] Adrenergic surge associated with stroke may result in catecholamine-induced myocardial stunning associated with takotsubo syndrome that may mimic ST-elevation AMI. Takotsubo syndrome also forms thrombus^[2] which may cause CCI. The left mural and atrial appendage thrombi were observed intraoperatively, but our case did not show anterior and apical wall infarction nor decrease of the left ventricular systolic function, so the thrombus due to atrial fibrillation seemed to cause CCI.

The American Heart Association and American Stroke Association in 2018 recommended that in the setting of hyper-acute simultaneous CCI, treatment with IV-tPA at the dose used for cerebral ischemia followed by PCI is reasonable (Class IIa; level of evidence C).^[14] The previously reported three patients treated with IV-tPA alone^[9,15] and one of the four patients treated with IV-tPA and PCI^[6-8,11] had good outcomes (modified Rankin Scale 0-2). In our case, IV-tPA and endovascular intervention for cerebral and coronary arteries were not necessarily required considering the risk of bleeding and rapid improvement of the stroke symptoms. A patient with CCI is rare, but the high risk of mortality of

AMI and stroke, hemorrhagic conversion, and their narrow therapeutic time window are challenges for stroke neurologists and cardiologists. Treatment of CCI should be individualized with multidisciplinary communication and management because there are no clinical trials or a consensus.

Cases of cardiac tamponade during the treatment of acute cerebral infarction were reported, and the causes were aortic dissections and carcinomatous pericarditis.^[10] Our patient presented cardiac tamponade and underwent pericardiectomy. His cardiac tamponade seemed idiopathic or to be resulted from oozing type cardiac rupture due to AMI, according to the intraoperative findings. Cardiac rupture often occurs at the anterior wall of the left ventricle, but his myocardial ischemic area was not there. Therefore, his cardiac tamponade subsequently to AMI was considered atypical, so there may be an association between ischemic stroke and cardiac tamponade. Furthermore, the administration of anticoagulant or antiplatelet for CCI should be carefully performed to patients with CCI in preparation for hemostasis with thoracotomy and pericardiectomy for cardiac tamponade. Administration of oral antithrombotic drugs for CCI should be cautiously performed, and antagonists against heparin, warfarin, and dabigatran or prothrombin complex concentrate should be prepared to be usable in case of emergent bleeding.

CONCLUSION

CCI is an infrequent and devastating clinical condition. Some cases with CCI treated with IV-tPA and endovascular intervention were reported, but the treatment strategy should be still discussed multidisciplinary. Especially, the administration of anticoagulant or antiplatelet for CCI should be carefully performed because fatal hemorrhage such as cardiac tamponade can occur.

Acknowledgments

MK wrote this manuscript. SK approved the final manuscript. We are grateful to the patients and their families who willingly provided their medical data.

Declaration of patient consent

The authors certify that they have obtained all appropriate patient consent forms.

Financial support and sponsorship

Nil.

Conflicts of interest

There are no conflicts of interest.

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How to cite this article: Katsuki M, Katsuki S. A case of cardiac tamponade during the treatment of simultaneous cardio-cerebral infarction associated with atrial fibrillation – Case report. *Surg Neurol Int* 2019;10:241.