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CASE REPORT
Coronary heart disease

ST-segment elevation myocardial infarction with normal coronary arteries secondary to anterior communicating cerebral artery aneurysmal rupture: a case report

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Background Myocardial infarction (MI) with non-obstructive coronary arteries presenting with ST-segment elevation can be challenging. Understanding the cardiac and non-cardiac causes aid in identifying the underlying diagnosis and deciding on the management. Neurological insult resulting in a mismatch of oxygen supply or demand to cardiomyocytes can lead to type 2 MI. Acute brain injury, such as intracranial haemorrhage, can induce cardiac dysfunction secondary to brain–heart interaction via hypothalamic–pituitary–adrenal axis and catecholamine surge.

Case summary A 50-year-old Caucasian male who vaped cannabis presented with epileptic seizures. A Glasgow coma scale of 7/15 necessitated urgent intubation. Electrocardiogram showed ST-segment elevation in inferior leads. Computed tomography of the head suggested intracerebral haemorrhage. He was stabilized in the intensive care unit (ICU). Subsequent imaging confirmed anterior communicating cerebral artery aneurysm and haematoma. Echocardiogram showed severe left ventricular dysfunction and hypokinesia in the left circumflex (LCx) territory. After step down from ICU, cardiac magnetic resonance imaging revealed transmural MI and myocardial oedema at LCx territory. Coronary angiogram was normal. Patient was treated with Levetiracetam and heart failure regimen. A cardiac defibrillator was implanted for secondary prevention and he was scheduled for elective neurosurgical intervention. A follow-up outpatient echocardiogram was normal.

Discussion Myocardial infarction with non-obstructive coronary arteries is uncommon. Though the majority is due to either plaque disruption or myocarditis, non-cardiac causes, such as acute neurological insults and substance use, should be considered. Scrutinizing the clinical presentation and using a meticulous approach with appropriate investigations are required to reach the correct diagnosis and appropriate management.

Keywords Myocardial infarct with non-obstructive coronary artery • Intracranial haemorrhage • Brain • heart interaction • Case report

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Learning points

- Myocardial infarction with non-obstructive coronary arteries can present as ST-segment elevation myocardial infarction where the investigation of underlying cause is paramount for appropriate management.
- When clinical presentation is atypical, non-cardiac causes such as acute neurological insult or substance use should be sought before considering emergent invasive coronary intervention.

Introduction

The majority of ST-elevation myocardial infarction (STEMI) is type 1 myocardial infarction (MI) with angiographic evidence of coronary thrombus or plaque disruption. However, STEMI can occur without obstructive coronary artery disease and is classified as MI with non-obstructive coronary artery disease (MINOCA).[1,2] Up to 6–8% of MI present as MINOCA.[1] Extracardiac causes resulting in a profound mismatch in oxygen supply and demand to the myocardium can lead to type 2 MI, which is a subgroup of MINOCA.[3] Stimulation of the hypothalamus following acute central nervous system insult (ACNSI) can also induce electrocardiographic changes, arrhythmias, and MI. The hypothesis explaining MINOCA in ACNSI is a catecholamine surge following brain–heart interaction causing increased myocardial oxygen demand and coronary vasospasm instigating myocardial insult.[4,5] We report a patient who presented with status epilepticus secondary to cerebral artery aneurysmal (CAA) rupture, where the electrocardiogram (ECG) demonstrated an acute inferior STEMI. MINOCA was confirmed by a normal coronary angiogram (CA) and evidence of MI on cardiac magnetic resonance imaging (CMRI).

Case presentation

A 50-year-old previously healthy Caucasian male who was a long-term cannabis vape user with a strong family history of CAA was brought to the emergency department with status epilepticus. Initial blood tests showed metabolic acidosis with a pH of 7.27, bicarbonate of 20 mEq/L, and troponin's of 1886 ng/L. All other blood tests were normal. Electrocardiogram showed an inferior STEMI (*Figure 1*). As the Glasgow coma scale (GCS) was 7 out of 15, the patient was emergently intubated and ventilated. Urgent non-contrast computed tomography (CT) of head suggested haemorrhage in the left lateral ventricle. Based on neurosurgical opinion, the patient was admitted to intensive care unit (ICU) and was initiated on 4 hourly nimodipine 60 mg. Invasive CA was deferred since the risk of further bleeding outweighed the benefits of revascularization at that time. On the first day of admission, the patient developed episodes of ventricular tachycardia (VT) with haemodynamic compromise, which necessitated synchronized DC cardioversion and IV amiodarone 300 mg over 30 min and subsequently 900 mg over 24 h. Transthoracic echocardiogram revealed left ventricular (LV) systolic dysfunction with an ejection fraction (EF) of 30% (Supplementary material online, *Videos S1–S3*). On

Timeline

Initial presentation to emergency department with status epilepticus	Electrocardiogram demonstrated inferior ST-segment elevation myocardial infarction (MI) and non-contrast computed tomography (CT) head suggested intracerebral haemorrhage.
(March 2020—Day 0)	Patient was intubated and transferred to intensive care unit (ICU).
ICU admission for stabilization and mechanical ventilation. (Day 0)	Anti-epileptics initiated.
Developed multiple episodes of ventricular tachycardia (Day 0–1)	Underwent CT head with contrast which suggested left anterior communicating artery (ACA) aneurysm and haemorrhage.
	Neurosurgical and neuroradiology multi-disciplinary team suggested initial stabilization.
	Synchronized direct current cardioversion and initiation of amiodarone.
	Two-dimensional echocardiogram revealed severe left ventricular systolic dysfunction (Day 1).
ICU stay complicated with aspiration pneumonia and acute kidney injury	Initiated on intravenous antibiotics and continuous veno-venous haemofiltration.
(Day 2–25)	He underwent magnetic resonance imaging brain with angiography which suggested left ACA aneurysm measuring 4.5 mm with haemorrhage (Day 6).
	Subsequently underwent CT angiogram brain following neurosurgical advice (Day 20).
Stepped down to coronary care unit.	He underwent a coronary angiogram which showed no significant obstructive coronary artery disease (Day 27).
Complete neurological recovery with no deficit.	Cardiac magnetic resonance imaging showed left ventricular ejection fraction (EF) of 48% with regional wall motion abnormalities and findings suggestive of MI at the left circumflex artery territory (Day 30).
Improvement of acute kidney injury and resolution of infection	He was initiated of heart failure treatment and underwent insertion of implantable cardiac defibrillator for secondary prevention.
(Day 26–35)	

Continued

Continued

Transferred to a tertiary neurosurgical unit for further evaluation and intervention	He was reviewed by the neurosurgical team and the multi-disciplinary team's (MDT's) decision was for neurosurgical intervention.
(Day 36)	
Inpatient neurosurgical evaluation for intervention was performed in neurosurgical unit (Day 37)	Repeated CT brain with angiography elicited additional aneurysms. Surgical option was offered to patient after MDT discussion. However, patient preferred staged procedure.
Outpatient (OP) course:	Currently, he is waiting for surgery.
Patient reviewed again by neurosurgery as OP	Normal findings with normal EF.
(3 months following initial presentation)	He is asymptomatic from a cardiac perspective.
Patient reviewed in cardiology OP with follow-up echo requested (8 months following initial presentation)	

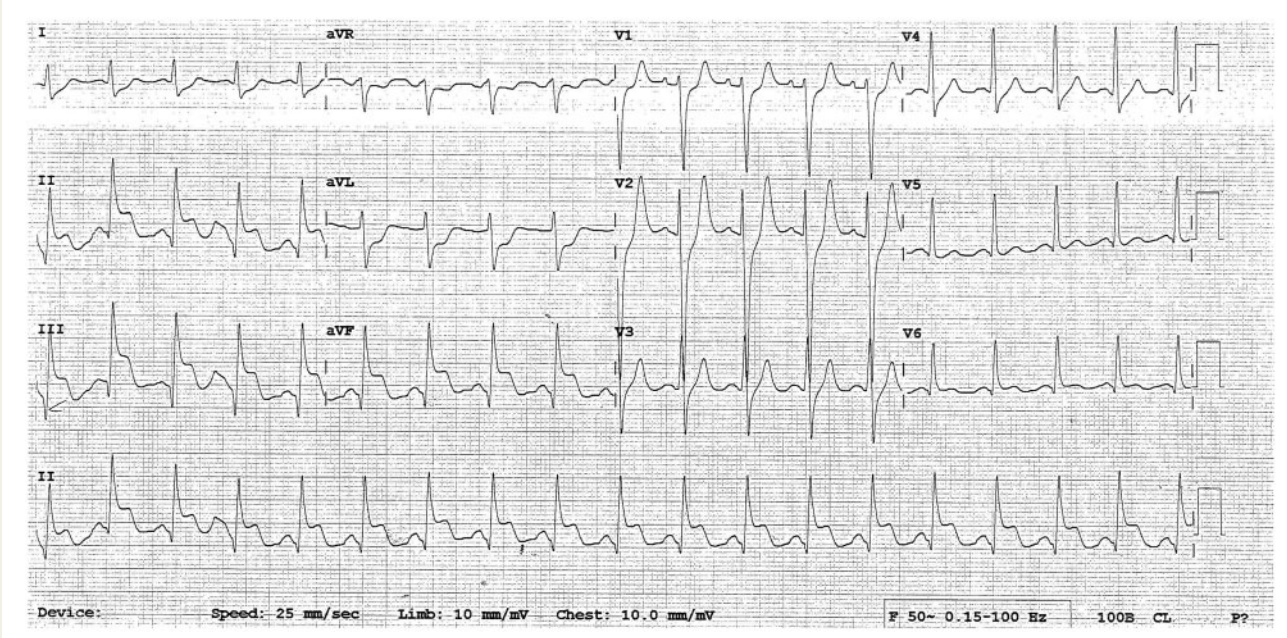

Figure 1 Preliminary 12-lead electrocardiogram on arrival to emergency department demonstrates ST-segment elevation in inferior territory represented noticeable in leads II, III, aVF with reciprocal ST depression in aVL.

Day 2, he developed aspiration pneumonia and acute kidney injury (AKI) requiring 10 days of intravenous meropenem and continuous veno-venous haemofiltration. Subsequently, he underwent a contrast CT head, which revealed an area of high attenuation suggestive of a haemorrhage adjacent to the left anterior communicating artery (ACA) with a 4 mm aneurysm (*Figure 2*). Thereafter, a magnetic resonance imaging brain with angiography suggested a 4.5-mm aneurysmal dilatation, at the origin of left A2 segment of ACA with a small intra-parenchymal subacute haemorrhage in the medial part of the left frontal lobe with surrounding gliosis (*Figure 3*). He was treated with IV Levetiracetam 500 mg BD and IV Phenytoin 100 mg TDS for seizures in the ICU (later converted to oral) and following renal stabilization, he was initiated and gradually up-titrated on Ramipril 5 mg o.d. (later changed to Entresto 97/103 mg b.i.d.), Bisoprolol 10 mg o.d., and Eplerenone 25 mg o.d. for heart failure.

On Day 20 of admission, patient recovered from AKI and CT angiogram of brain was performed which confirmed an aneurysm at the A2 segment of the left anterior communicating artery with an established haematoma (Supplementary material online, *File S1*). The patient made a complete neurological recovery with no residual deficit and hence detailed history was taken from patient retrospectively which revealed he had vaped Cannabis 1 hour before his initial presentation. The multidisciplinary team involving neurosurgeons and

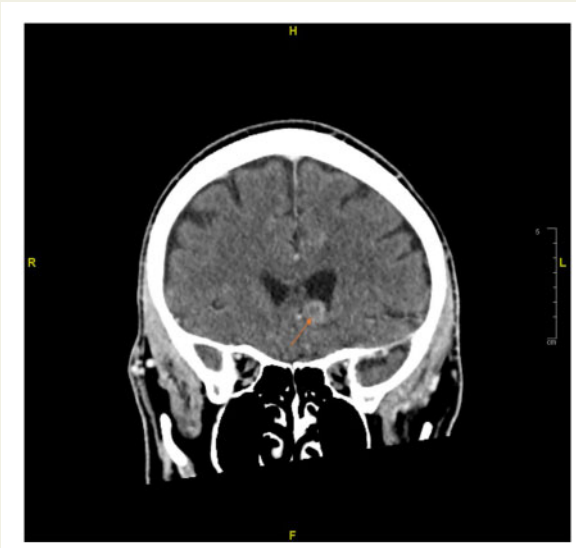

Figure 2 Coronal view of contrast computed tomography head shows an area of high attenuation medial to the left frontal lobe (indicated by red arrow) the appearances are atypical of a haemorrhage, and this may represent a mass or a bleed into a mass. There is no post-enhancement in the mentioned areas of interest. There is a probable small 4-mm aneurysm at the junction of the anterior communicating artery on the left.

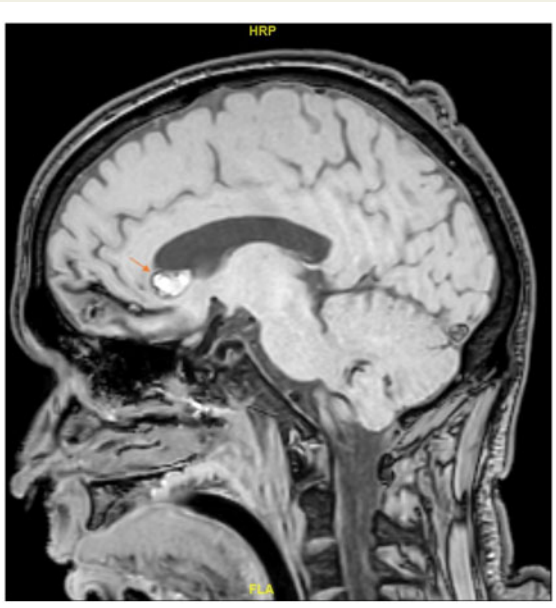

Figure 3 Magnetic resonance imaging head—T1 flair sagittal view shows there is a small intra-parenchyma subacute haemorrhage (red arrow) in the medial part of the left frontal lobe adjacent to the inferior margin of anterior horn of the left lateral ventricle with a haemorrhage measuring 17 mm.

neuroradiologists agreed that the final neurological diagnosis was a rupture of a CAA at A2 segment of left ACA causing haemorrhage, but recommended to complete cardiac investigations and stabilization before neurosurgical intervention. Hence, on Day 26, as he remained stable and independent of haemofiltration, he was transferred to the coronary care unit for further evaluation. Elective CA was performed, which demonstrated non-obstructive coronary arteries (Supplementary material online, *Videos S4–S6*). Subsequently, CMRI showed normal LV size with moderately impaired systolic function (EF 38%) with akinesia of mid-distal inferolateral and the whole inferior wall. On the late gadolinium enhancement images (Supplementary material online, *Videos S7–S9*), there was patchy enhancement with transmural involvement at focal areas of the mid and distal inferior wall (*Figure 4*) and myocardial oedema was seen at these areas on short-tau inversion recovery (STIR) images consistent with MI at left circumflex artery territory (Supplementary material online, *Files S2 and S3*). His subsequent ECG's showed established infarction in inferior territory (*Figure 5*). Also, he underwent an implantable cardiac defibrillator (ICD) implantation for secondary prevention. He was transferred afterwards to a tertiary neurosurgical centre for further evaluation and intervention, where he underwent further neuroimaging which suggested existence of additional aneurysms for which neurosurgery was offered to the patient. However, following the discussion of risks vs. benefits, the patient preferred elective procedure as outpatient. On cardiology follow-up 8 months later, the repeat transthoracic 2D echocardiogram showed normalization of left ventricular ejection fraction (Supplementary material online, *Video S10*). He remains asymptomatic from a cardiac perspective.

Discussion

Acentral nervous system insult, such as subarachnoid haemorrhagic (SAH), acute ischaemic stroke, mass lesions with oedema, and seizures, can cause ECG abnormalities including ST-segment changes.[4,5] Ruptured CAA can cause seizures because of SAH, intracerebral haematoma, vasospasm causing infarction, and worsening hydrocephalus.[6] Arrhythmias may also be appreciated following ACNSI and include life-threatening rhythms, such as ventricular fibrillation, VT, and AV blocks. Consensus by authors of similar case reports of CAA rupture and haemorrhage complicated with simultaneous acute coronary event favours initial cerebral aneurysmal coiling before endovascular cardiac intervention if clinically stable and feasible.[7,8] Additionally, a cohort analysis by Ahmadian *et al.*,[9] questioned the helpfulness of coronary angiography in this populace given the reversibility and catecholamine mediation of cardiac insult. The postulated theories supporting the relationships between brain damage and heart dysfunction include catecholamine mediation via epinephrine and norepinephrine and also the direct effect of sympathetic and parasympathetic nervous system. Resultant hypertension, elevated oxygen demand and vasospasm mediate the cardiac insult leading to type 2 MI and MINOCA. The ECG changes represent underlying myocardial tissue injury appreciable with subendocardial damage characterized by myocytolysis, myofibrillar degeneration, and fuchsinophilic degeneration with elevated cardiac enzymes and echocardiographic abnormalities keeping in with an acute MI.[5]

Cannabis use is known to cause cardiovascular events including acute MI, cardiomyopathy, fatal arrhythmias, and even sudden cardiac deaths.[10,11] Our patient used cannabis 1 h before the onset of his

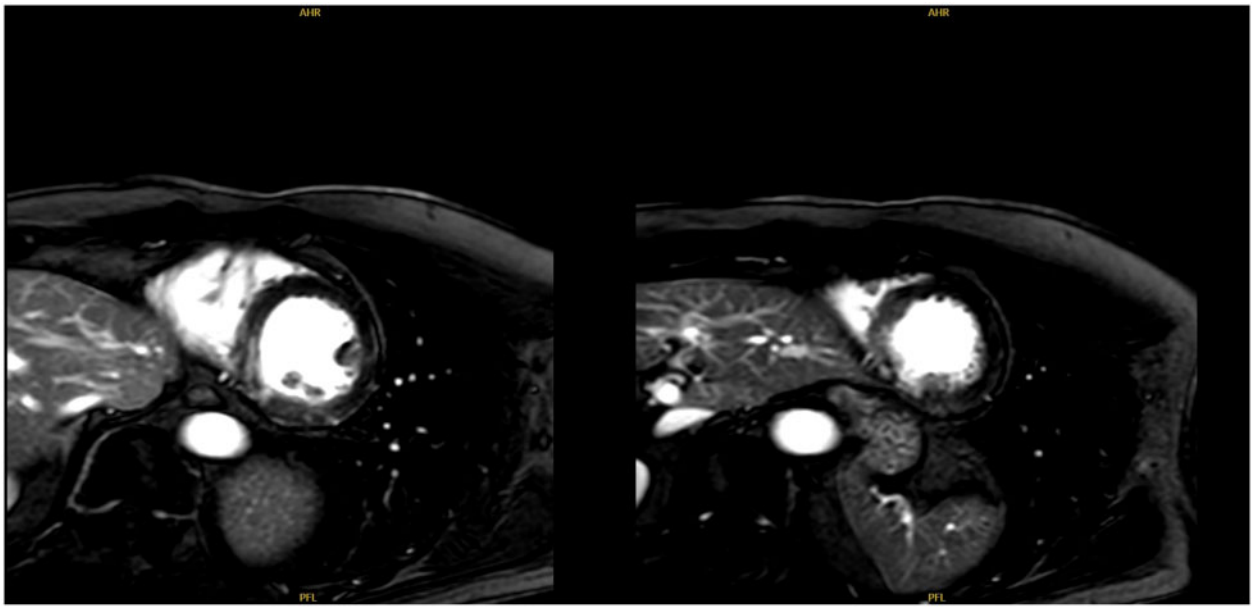

Figure 4 Cardiac magnetic resonance imaging in the short-axis view sequences demonstrate near patchy late gadolinium enhancement with focal areas of transmural involvement at the left circumflex artery territory consistent with myocardial infarction.

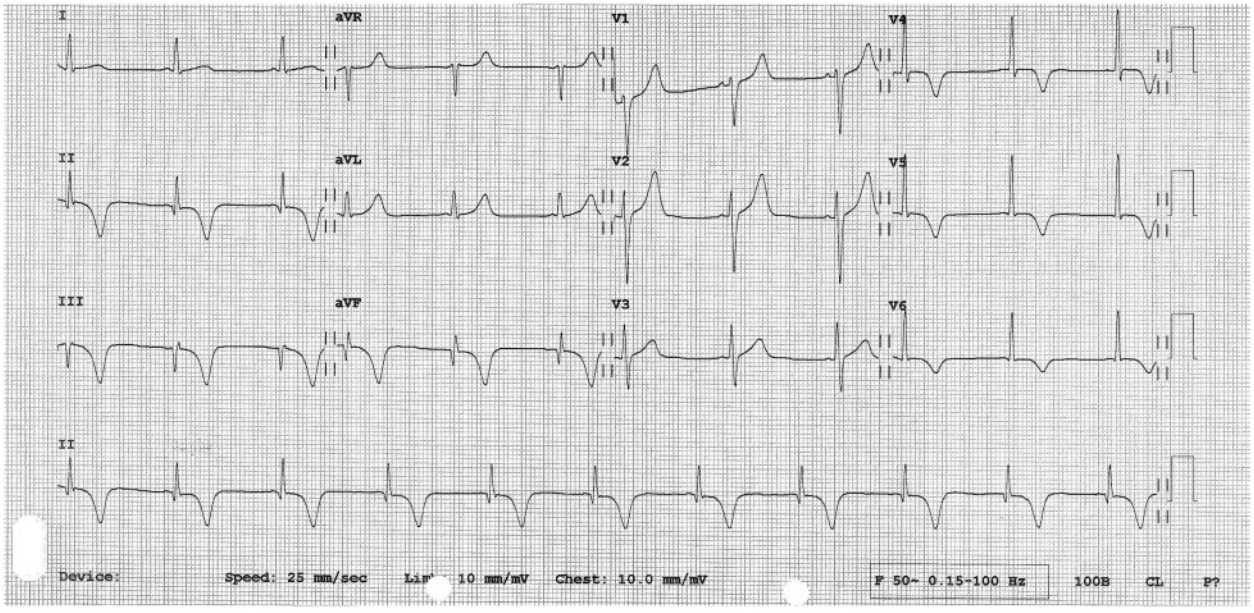

Figure 5 Subsequent 12-lead electrocardiogram demonstrates ST changes keeping with established infarction with Q waves in leads III, aVF, and deep T-wave inversion in leads II, III, aVF, and V4–V6.

presentation, and studies have shown the first hour following cannabis use confers a 5-time risk for acute MI.[12] An alternate consideration is whether the coronary event was induced by cannabinoids, which was complicated by non-perfusing VT or VF leading to seizures. Acute MI following cannabinoid use can be in the backdrop of established coronary artery disease or occurs in de-novo due to increased factor VII levels, or carboxyhaemoglobin levels with subsequent hypoxia and coronary artery spasm.[11–13] Cannabinoids,

however, are suggested to be protective against new-onset seizures and even considered in drug-resistant epilepsy[14–16] which makes it an unlikely aetiology in our patient.

Our patient had an acute cerebrovascular accident with simultaneous occurrence of an inferior STEMI confirmed by ECG, raised troponins, and CMRI findings. In the context of a normal CA, the unifying diagnosis was MINOCA due to brain–heart interaction brought about by the ACNSI due to CAA rupture and haemorrhage. His initial cardiovascular instability and AKI prompted a staged neurosurgical intervention given his neurological stability and recovery. This case highlighted the interplay between neurological injury and cardiac dysfunction via hypothalamic–pituitary–adrenal axis and catecholamine surge.

Lead author biography

Tin Sanda Lwin was graduated in Myanmar. She worked as a cardiology registrar in Singapore after she completed her MRCP. She worked as a registrar in cardiology at Kettering General Hospital before entering the national cardiology trainee program. Currently, she is working as a cardiology specialty trainee at the Castle Hill Hospital.

Supplementary material

Supplementary material is available at *European Heart Journal—Case Reports* online.

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Slide sets: A fully edited slide set detailing this case and suitable for local presentation is available online as Supplementary data.

Consent: The authors confirm that written consent for submission and publication of this case report including images and associated text has been obtained from the patient in line with COPE guidance.

Conflict of interest: None declared.

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