Cause of ST-segment elevation on electrocardiogram

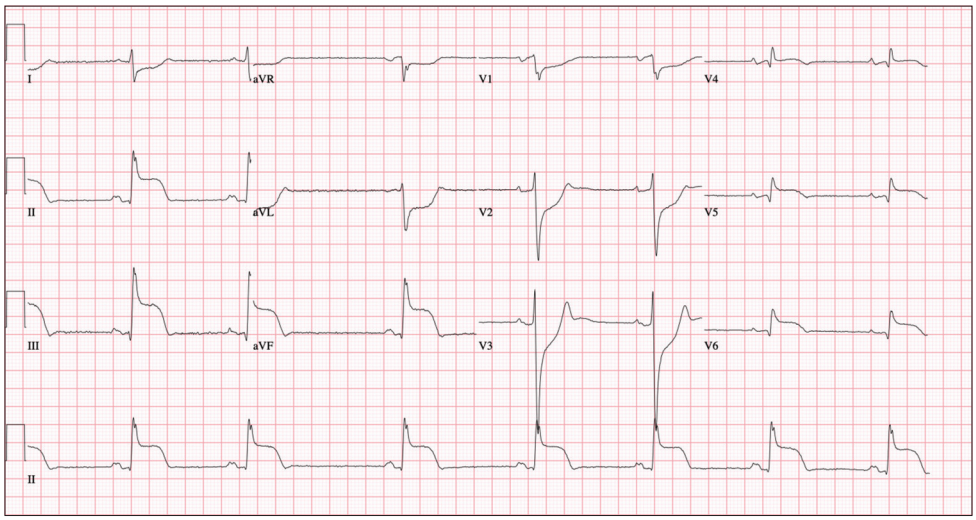

Figure 1: Case 1: 12-lead ECG on admission. ECG = electrocardiogram.

CASE 1

Clinical presentation

An 80-year-old man was referred to the emergency department with vertiginous giddiness. He had no chest pain, shortness of breath or palpitations. The patient had a past medical history of hypertension, chronic smoking of 30 pack-years and previous subdural haemorrhage status post surgical drainage 15 years ago. Computed tomography of the brain did not show any intracranial haemorrhage. Troponin levels were elevated at 613 ng/L (normal range 0–17.4 ng/L). What does the electrocardiogram (ECG) in Figure 1 show?

ECG interpretation

The ECG shows sinus bradycardia. It also demonstrates ST elevations over the inferior leads II, III and aVF with reciprocal ST depressions over the lateral leads I and aVL. There are also ST depressions over the precordial leads, V1–V3, with upright T waves, especially noted in V3. The ECG is suggestive of inferoposterior ST-segment elevation myocardial infarction (STEMI).

Clinical course

The patient underwent an emergency coronary angiogram, which showed diffuse coronary vasospasm (CVS) involving all three major coronary arteries. Intracoronary glyceryl trinitrate was given with resolution of the CVS. The patient was subsequently discharged after observation in the coronary care unit and started on long-acting calcium channel blockers with long-acting nitrates. He was counselled for smoking cessation. He remained well clinically with no complains during outpatient cardiology clinic review.

CASE 2

Clinical presentation

A 58-year-old man presented with an asystolic out of hospital cardiac arrest with return of spontaneous circulation after a downtime of 15 min. He had a past medical history of hypertension, hyperlipidaemia, chronic smoking of 15 pack-years and microvascular disease with a normal coronary angiogram 1 year before presentation. He was newly started on bisoprolol for stable angina just 2 weeks before presentation. Troponin I was elevated at 363 ng/L (normal range 0–39 ng/L). The ECG on arrival showed normal sinus rhythm. During the intensive care unit stay, he developed cardiac arrest [Figure 2]. Post-resuscitation, there were ECG changes as shown in Figure 3. What do the rhythm strip in Figure 2 and ECG in Figure 3 show?

ECG interpretation

In Figure 2, the cardiac rhythm strip demonstrates development of ventricular tachycardia from normal sinus rhythm in leads II and V5. In Figure 3, the ECG shows normal sinus rhythm with ST elevations over leads II, III and aVF with reciprocal ST depressions over lead I and aVL. This was consistent with inferior STEMI.

Clinical course

He underwent emergency coronary angiogram in view of the clinical and ECG findings. The coronary angiogram

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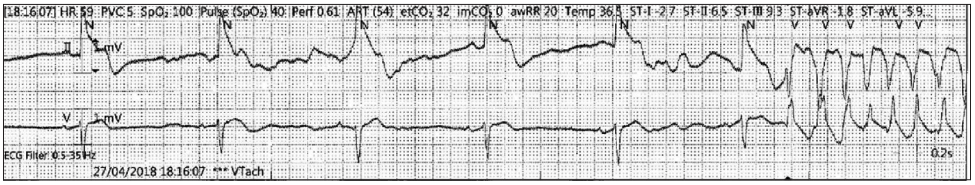

Figure 2: Cardiac rhythm strip during the intensive care unit stay.

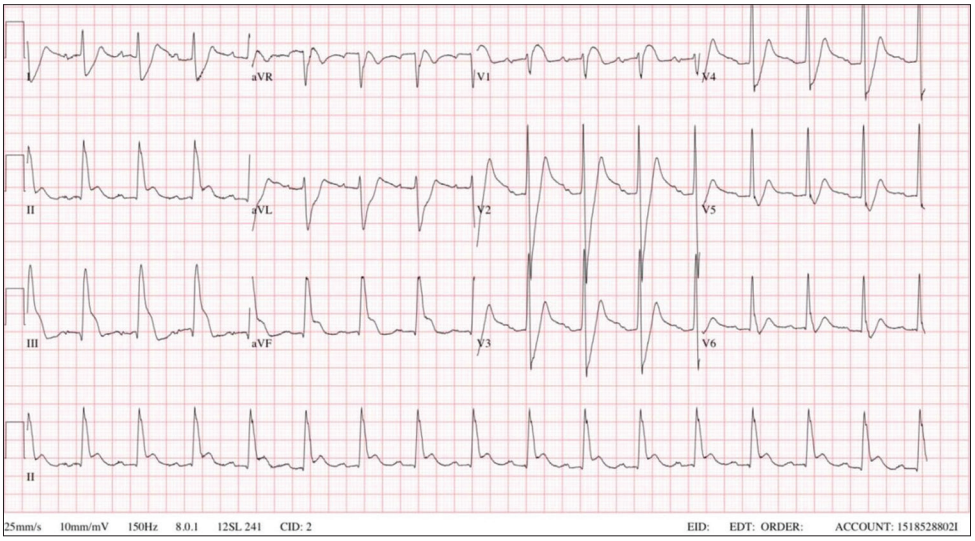

Figure 3: ECG post-resuscitation in the intensive care unit. ECG = electrocardiogram.

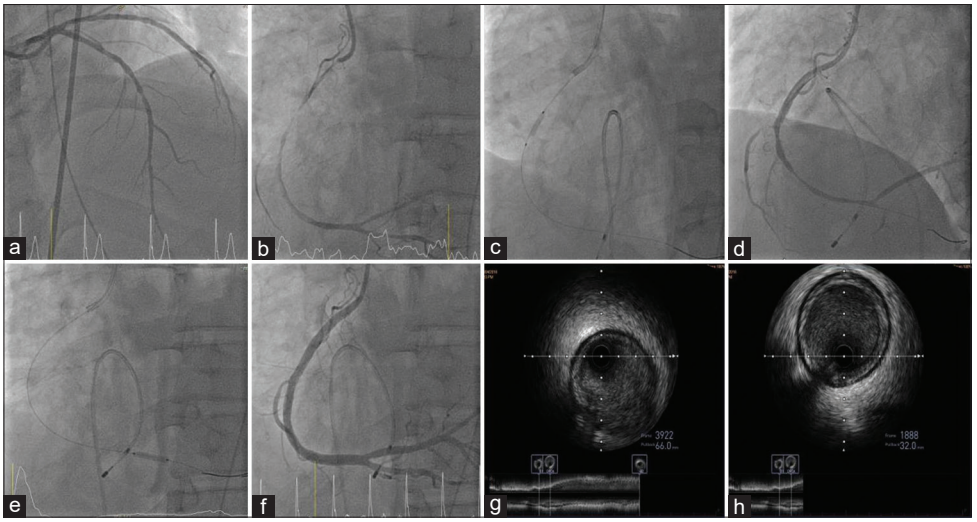

Figure 4: Coronary angiogram images. (a) Left anterior descending artery and left circumflex artery (AP cranial). (b) Right coronary artery (LAO). (c) Balloon angioplasty of the proximal right coronary artery lesion (LAO). (d) Right coronary artery post-angioplasty with new stenoses appearing in the distal right coronary artery and right posterior descending artery (LAO cranial). (e) Balloon angioplasty of the distal right coronary artery lesion (LAO). (f) Right coronary artery post-angioplasty (LAO) (given intracoronary glyceryl trinitrate 200 mcg before the final shot). (g) Intravascular ultrasound of the proximal right coronary artery. (h) Intravascular ultrasound of the distal right coronary artery. AP = anteroposterior view, LAO = left anterio-oblique view.

demonstrated new diffuse non-obstructive lesions with a 'string of beads' appearance in the left anterior descending artery [Figure 4a], minor irregularities in the left circumflex artery and a critical ostial to proximal right coronary artery stenosis [Figure 4b]. Balloon angioplasty was performed to the proximal right coronary artery stenosis [Figure 4c]. During balloon angioplasty, the patient developed complete heart block with junctional rhythm of 30 beats per minute, and a temporary transvenous pacing wire was placed in the right ventricle. The patient suffered pulseless electrical activity arrest shortly after, which was followed by a ventricular tachycardia cardiac arrest requiring management with advanced cardiac life support. After he was stabilised with resuscitation, he continued with balloon angioplasty, which led to resolution of proximal

Table 1: Reported electrographic differences between coronary vasospasm and acute myocardial infarction from coronary thrombosis

Entity	Coronary vasospasm	Acute myocardial infarction
ST elevation	- Negative u waves may be present, typically in the same lead as ST elevation - ST elevations resolve with symptom resolution	- More likely to have inversion of the terminal portion of T waves - Prolonged ST elevation
In general	- q waves are rarely seen and transient - More likely to have transient intraventricular conduction disorders - Alternans of elevated ST-segment and negative T wave deepness	- Presence of q waves that persist beyond day 2 of the event - May present with permanent conduction disorder such as left bundle branch block

stenosis and no underlying stenosis or dissection, but new critical stenoses were seen in the distal right coronary artery and posterior descending artery [Figure 4d]. Intracoronary glyceryl trinitrate 200 mcg was administered with improvement of the distal lesions. Further balloon angioplasty was performed and it resolved the stenoses [Figure 4e, f]. Intravascular ultrasound evaluation of the right coronary artery showed no significant plaque burden or disruption [Figure 4g, h]. Upon transfer to the coronary care unit, the patient went into polymorphic ventricular tachycardia/ventricular fibrillation storm and required multiple defibrillations. The impression was that of significant recurrent CVS of the right coronary artery causing cardiac arrest and ventricular fibrillation storm. Further management thereafter included intravenous glyceryl trinitrate with eventual conversion to oral isosorbide mononitrate, a long-acting nitrate. The patient's bisoprolol was also stopped, and he was started on the calcium channel blocker, diltiazem. He also received an implantable cardioverter defibrillator (ICD) as secondary prevention for ventricular tachycardia and ventricular fibrillation before discharge. The patient was advised on smoking cessation.

DISCUSSION

CVS is a non-structural cause of cardiac arrest, with coronary arteries' vasoconstriction, eventual vessel occlusion and subsequent sudden cardiac death. CVS continues to be a diagnostic challenge as its presentation is variable and subtle.[1] CVS patients have a wide range of ECG manifestations. They may present with ST elevations or depressions with prominent T waves. The ST elevations often correlate with the culprit artery of vasospasm and are accompanied by ST depressions in the reciprocal leads. There is variable predisposition to spasm in different parts of coronary circulation in CVS, with a higher probability of CVS (spontaneous and catheter induced) in the right coronary artery, compared to the left circumflex artery as seen in our second case.[2] In some cases, there may be negative U waves observed at the beginning or near the end phase of the CVS, which were not observed in our two cases.[3] It is often difficult to differentiate between CVS and coronary thrombosis with ECG. For CVS, the ST elevations are present only during the presence of symptoms, which typically happens from midnight to early morning, peaking at 5 am, compared to a case of acute coronary thrombosis with no direct association with circadian rhythm.[4] In most cases of CVS, there may be q waves observed in the ECG, but it is often transient and does not translate to permanent myocardial necrosis as ischaemia is often short lasting. In 20% of the patients, there have been reports of alternans of elevated ST-segment and negative deep T wave.[5]

Furthermore, patients with CVS may present with transient intraventricular conduction disorders such as bifascicular block, left anterior or posterior hemiblock and right bundle branch block, although this is rare.[6] CVS is also associated with various arrhythmias such as sinus bradycardia, sinus arrest with or without junctional escape beats, ventricular tachycardia, ventricular fibrillation and asystole.[3] Right coronary artery vasospasms often cause bradyarryhthmias as in Case 1, while left anterior descending artery vasospasms are prone to tachyarrhythmias.[3,7] Diffuse multivessel vasospasm may potentially lead to either of them. A summary of electrographic differences between CVS and acute myocardial infarction from coronary thrombosis is presented in Table 1.[3-6,8]

In contrast to stable angina, CVS is usually not inducible by exercise and occurs during rest. It is often transient, lasting a few seconds and is unpredictable. Ambulatory ECG can be considered for workup to detect attacks; however, the attacks may not appear during periods of monitoring. Hence, an implantable loop recorder with continuous ECG monitoring may serve as a better option. Definitive diagnosis of this entity thus requires invasive provocative testing with ergonovine or acetylcholine. Despite the proven overall safety of using ergonovine or acetylcholine, serious complications such as ventricular fibrillation, myocardial infarction or death may occur, although rarely.[3] As such, these tests are not commonly or routinely performed, perhaps with the exception of Japan. In the recent guidelines, the Japanese Circulation Society Joint Working Group had stated that CVS can even be diagnosed without coronary angiography if it fulfils certain criteria and characteristics.[9] Patients with chest pain, ST elevation on ECG and normal coronary arteries are usually considered to be affected by CVS if the clinical scenario is consistent with the characteristics of spasm, namely, the presence of risk factors, circadian pattern (night–morning), female and occurring at a younger age.[9]

Precipitating factors of CVS include physical and/or mental stress, magnesium deficiency, hyperventilation, alcohol consumption and notably administration of pharmaceuticals like beta-blockers.[3] There are case reports that suggest the

potential association between beta-blockers and the increased frequency as well as duration of CVS.[10-12] The main significant risk factors identified for CVS are smoking, age and elevated high-sensitivity C-reactive protein.[3] The common risk factor in both cases is chronic smoking, with the addition of beta-blockers initiation as precipitant in Case 2, given the chronology of events.

Advice to avoid precipitating factors is key. Calcium channels blockers and long-acting nitrates have also been proven effective in preventing an attack.[3] Evidence for using ICD in the management of CVS is unclear, and further research is needed. Although medical therapy is imperative for patients with CVS and lethal arrhythmia, ICD therapy should be considered as secondary prevention in high-risk groups such as patients with CVS and lethal ventricular arrhythmias above medical treatment.[9,13,14]

In conclusion, this paper discussed two cases of CVS with ECG manifestations, which demonstrated that CVS is an uncommon but potential mimicker of coronary thrombosis in recurrent cardiac arrest. With the identification of key risk factors and clinical features, clinicians will have heightened sense of suspicion to make an earlier diagnosis of this elusive condition.

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Conflicts of interest

Poh KK and Loh JPY are members of the SMJ Editorial Board, and were thus not involved in the peer review and publication decisions of this article.

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SMC CATEGORY 3B CME PROGRAMME

Online Quiz: https://www.sma.org.sg/cme-programme

Question	True	False
1. The following features on 12-lead electrocardiogram (ECG) may be suggestive of coronary vasospasm:		
(a) Presence of negative u waves in the same lead as the ST elevation		
(b) Persistent ST elevation beyond 24 h		
(c) Prominent T waves		
(d) Transient intraventricular conduction disorders		
2. The following are the precipitating factors for coronary vasospasm:		
(a) Usage of calcium channel blocker		
(b) Magnesium deficiency		
(c) Physical/mental stress		
(d) Usage of betablocker		
3. The following clinical features point toward coronary vasospasm:		
(a) Circadian pattern (night-morning) of symptoms		
(b) Female patients		
(c) Younger patients		
(d) Attacks are suppressed by beta-blockers		
4. Regarding investigations for coronary vasospasm:		
(a) Ambulatory ECG can be considered for workup to detect attacks		
(b) An implantable loop recorder with continuous ECG monitoring can be offered in selected patients		
(c) Provocative testing with intracoronary administration of ergonovine or acetylcholine can be done		
(d) One set of 12-lead ECG is adequate to confirm the diagnosis		
5. The following are the possible pharmacological treatments for coronary vasospasm:		
(a) Calcium channel blocker		
(b) Diuretics		
(c) Long-acting nitrates		
(d) Fenofibrate		