

An unusual case of pulseless electrical activity arrest associated with Prinzmetal's angina

Niranjan J Gaikwad, Martin McNamara, Ravindra Batra, Gregory Aroney and Rohan Jayasinghe

Cardiac arrest due to ventricular tachyarrhythmia in the setting of Prinzmetal's angina (PA) has been well reported.¹⁻³ Increased QT dispersion has been proposed as a marker of increased risk of ventricular arrhythmias in this setting.^{4,5} However, to our knowledge, pulseless electrical activity (PEA) arrests in patients with PA have not been reported.

Clinical record

A 61-year-old woman presented to hospital with an infection in the first web-space of the left hand after minor penetrating trauma. The abscess was drained under a general anaesthetic, and intravenous ticarcillin/clavulanate treatment was commenced. She had a history of osteoporosis, depression, hyperlipidaemia and Raynaud's phenomenon, and was an active smoker. She also had PA, diagnosed less than 12 months before the current admission after a positive stress test showed transient ST elevation in inferior leads and reciprocal ST depressions in

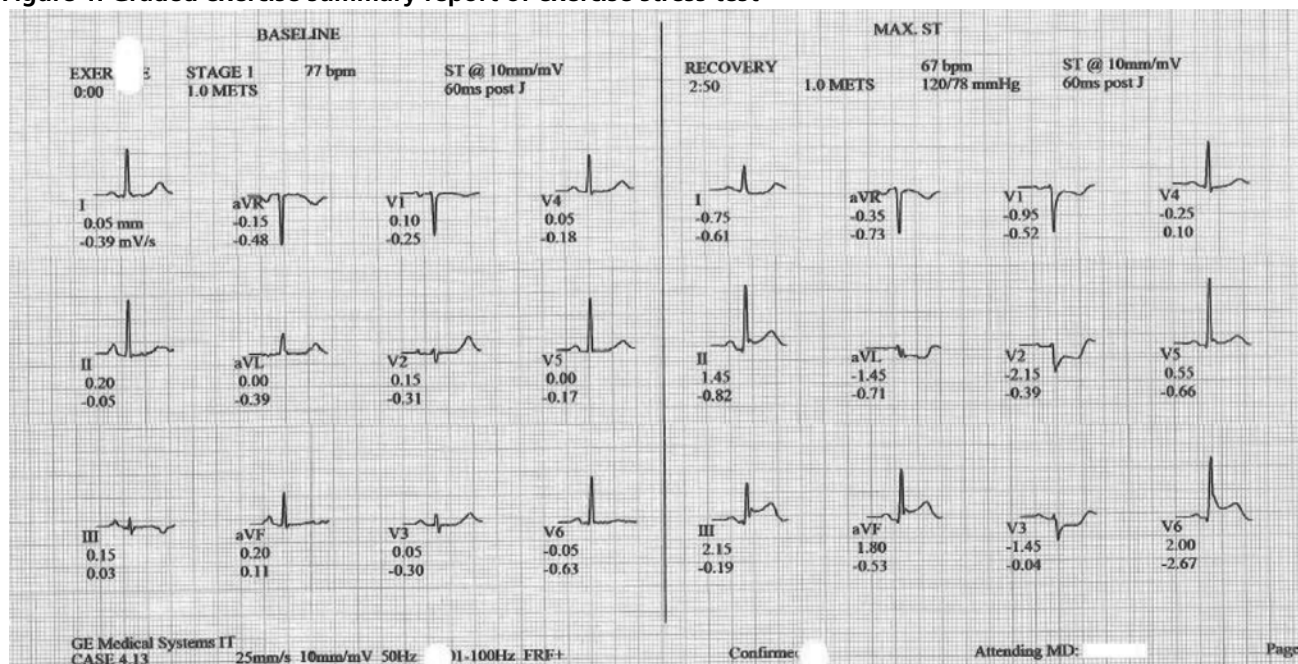
ABSTRACT

- Cardiac arrest due to ventricular tachyarrhythmia in the setting of Prinzmetal's angina (PA) has been well reported in the literature. However, to our knowledge, there have been no reports of a pulseless electrical activity (PEA) arrest in patients with PA.
- We report a case of PEA arrest in a patient with PA after surgical drainage of an abscess in the first web-space of the left hand.
- We propose that the Bezold–Jarisch reflex may be the underlying mechanism responsible for this arrest. The case also highlights the danger of ceasing treatment with nitrates and calcium channel blockers in patients with PA.

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anterior chest leads. An angiogram done at the same time was normal. (Figure 1).

Figure 1. Graded exercise summary report of exercise stress test*



* During this test, the patient developed chest pain with ST segment elevation in inferolateral leads (II, III, aVF and V6), with reciprocal changes in reciprocal leads (V1, V2, V3 and aVL). These changes resolved during recovery after administration of sublingual nitrate. This is considered to be diagnostic of Prinzmetal's angina.

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The patient's medications were isosorbide mononitrate 60 mg once daily, amlodipine 10 mg once daily, aspirin 100 mg once daily, metoprolol 12.5 mg twice daily, atorvastatin 80 mg once daily, sodium valproate 200 mg once daily, venlafaxine 300 mg once daily and omeprazole 20 mg once daily. Significantly, the patient had missed her isosorbide mononitrate and amlodipine tablets during her 3-day surgical admission. On the fourth day in hospital, she had a sudden deterioration with agitation, hypotension and hypoxia on the ward, leading to a medical emergency team call. She progressed to a PEA arrest within minutes of the arrival of the cardiac arrest team. She received cardiopulmonary resuscitation for less than 1 minute, with return of circulation before any administration of adrenaline/atropine. After the arrest she was agitated, hypotensive and hypoxic (Glasgow Coma Score 10; blood pressure 90/60 mmHg; SaO₂ 86% after receiving 15 L oxygen via a non-rebreather mask). The patient was intubated and ventilated for agitation and hypoxia. A 12-lead electrocardiogram (ECG) recorded during the arrest showed bradycardia with 1 mm ST elevation in inferior leads and reciprocal ST depression in anterior chest leads (Figure 2). A repeat ECG after return of spontaneous circulation showed T wave inversion in inferior leads, with no ST segment depression or elevation. Blood biochemical measurements were pH 7.07, PO₂ 98 mmHg, PCO₂ 43 mmHg, HCO₃ 12 mEq/L, base deficit 17.4, SaO₂ 94% and lactate 9.6 mmol/L.

A chest x-ray ruled out spontaneous pneumothorax, but did show some pulmonary congestion consistent with left ventricular failure. There was no evidence of aspiration at the time of intubation. The patient was normothermic and had a blood glucose level of 15.8 mmol/L.

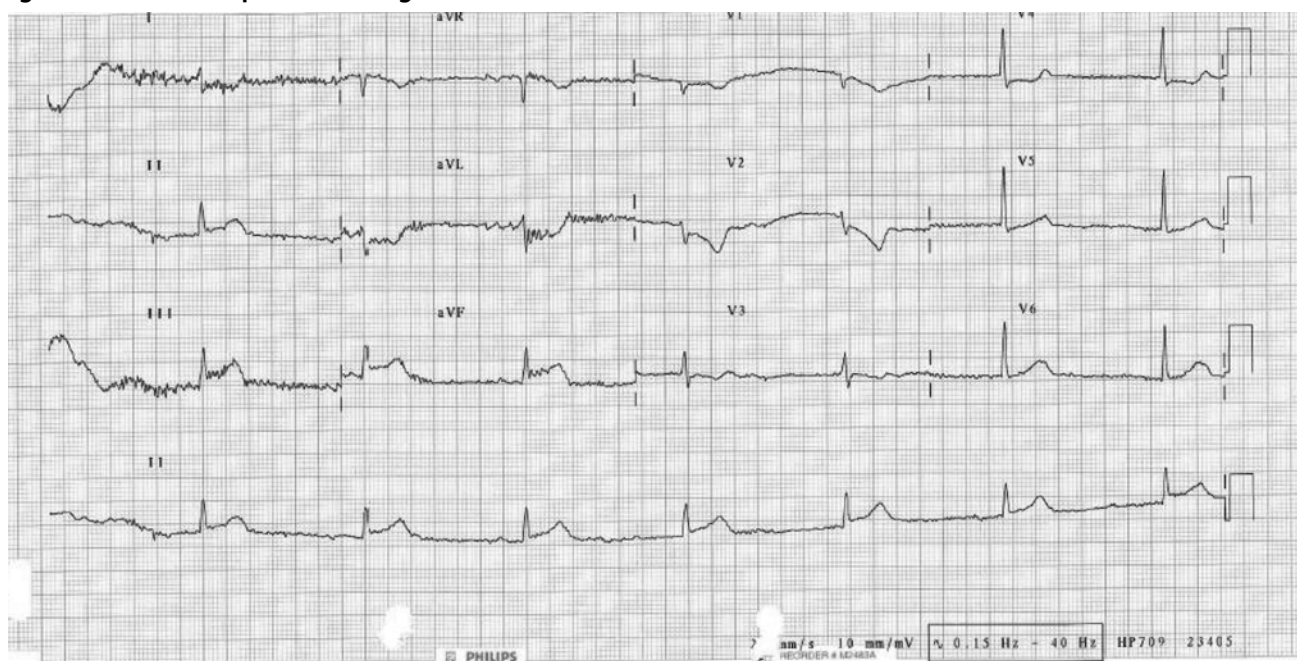
A bedside transthoracic ECG revealed globally reduced left ventricular function. There were no regional wall motion abnormalities. Right ventricular systolic pressure was not elevated and the right ventricle was not dilated. There was no pericardial effusion.

A computed tomography pulmonary angiogram was negative for pulmonary embolus. A subsequent coronary angiogram showed diffuse spasm in the circumflex artery which was promptly relieved with glyceryl trinitrate. (Figure 3, Figure 4). The rest of the coronary vessels were normal. The patient was extubated the same morning and transferred to the ward. She had an uneventful recovery in hospital and was discharged.

Discussion

In their first case reports of patients with variant angina, Prinzmetal and colleagues described patients having anginal symptoms at rest.⁶ The syndrome was defined by absence of symptoms with exercise and emotion. However, PA has since been reported in the setting of exercise.^{7,8} The diagnosis of PA in this case was made on the basis of

Figure 2. ECG of the patient during PEA arrest*



ECG = electrocardiogram. PEA = pulseless electrical activity. * The ECG shows ST elevation in inferior leads (II, III and aVF), with reciprocal changes in anterior leads (V1, V2 and V3). These changes resolved within 5 minutes on a subsequent ECG.

Figure 3. Post-arrest angiogram showing diffuse spasm in the circumflex artery

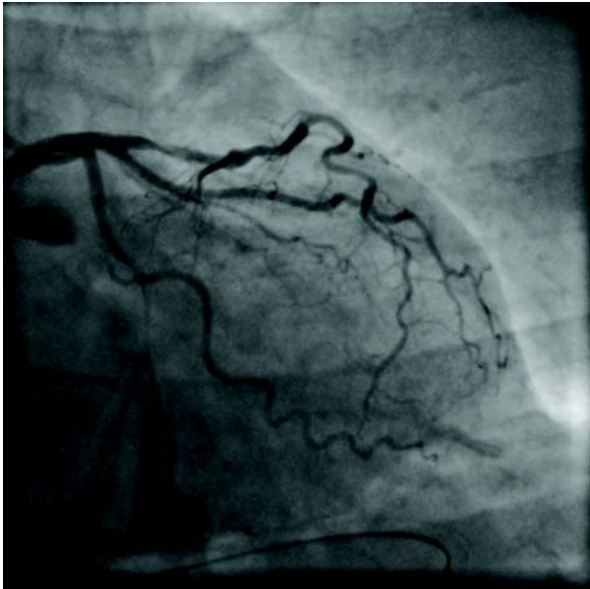
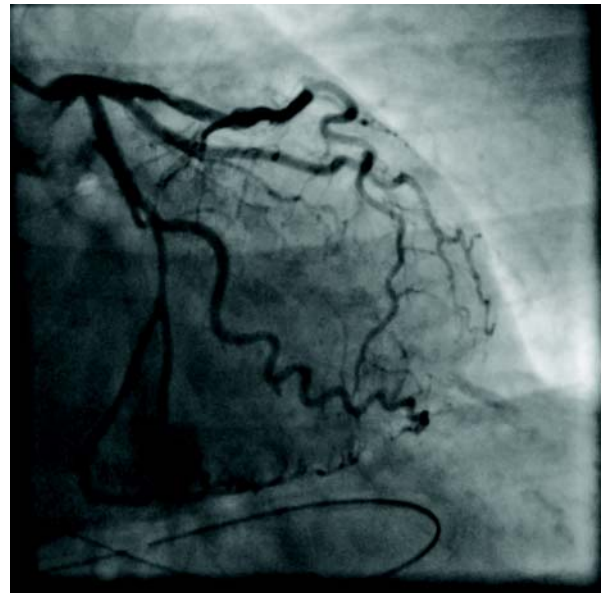


Figure 4. Image taken during angiogram after intracoronary GTN administration*



GTN = glyceryl trinitrate. * The image shows normalisation of blood flow in the circumflex artery.

transient reversible ST elevations during a stress test followed by a normal coronary angiogram.

Our patient's normal angiogram is also of note. PA is known to occur both in the presence of normal coronary arteries and also in the presence of plaques. The significance of a normal coronary vasculature in PA with regard to mortality risk is debatable. Bory et al,⁹ in a study of 277 patients with confirmed PA, concluded that normal or near-normal coronary arteries confer a low risk of mortality and myocardial infarction. Chevalier et al,¹⁰ reporting on a series of seven patients, also concluded that patients with PA had a favourable outcome in the absence of significant coronary disease. In contrast, MacAlpin's study of 81 patients demonstrated a 150% increase in risk of a serious arrhythmia in the absence of a high-grade stenosis.¹¹ In a study by Yasue et al, 50% of deaths were in people with normal angiograms.¹²

Our patient was a current smoker, which is a risk factor for PA. Other disorders that have been found to be associated with the condition include Raynaud's phenomenon (seen in our patient), migraine, and cocaine and marijuana misuse.

Calcium channel blockers and nitrates are the mainstay of treatment for PA. Their role in preventing vasospasm is well established. However, two small studies of the role of calcium channel blockers in PA^{10,13} revealed contradictory findings with regard to prevention of ventricular tachyarrhythmias. Significantly, there is some evidence that withdrawal of calcium channel blockers can lead to rebound vasospasm, causing increased symptoms.¹⁴ Another point of

interest is that our patient had been prescribed small doses of metoprolol for persistent symptomatic tachycardia. However, she was not taking them, as she was intolerant to them. Though we did not come across any data on the effect of highly selective β -blockers in PA, there is some evidence that non-selective β -blockers may precipitate coronary vasospasm by allowing unabated alpha stimulation with catecholamines.¹⁵ Aspirin in high doses is also best avoided.¹⁶ Our patient was discharged on the following cardiac medications: amlodipine 10 mg once daily, aspirin 100 mg once daily, isosorbide mononitrate 60 mg once daily and atorvastatin 80 mg once daily.

We hypothesise activation of the Bezold–Jarisch reflex as a possible mechanism for the PEA arrest. The Bezold–Jarisch reflex is a cardioinhibitory reflex characterised by the triad of bradycardia, hypotension and peripheral vasodilatation.¹⁷ Interestingly, it has been reported in the setting of acute inferior ST elevation myocardial infarction during primary angioplasty.¹⁸ Certainly, in the literature, severe hypotension has been well reported both in the setting of right ventricular myocardial infarction¹⁹ and right coronary artery angioplasty after right coronary artery occlusion.²⁰

Conclusion

Prinzmetal's angina can present as sinus bradycardia proceeding to pulseless electrical activity cardiac arrest. Physi-

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cians should be mindful of the other ways in which PA can cause death, such as myocardial infarction, spontaneous coronary artery dissection, ventricular tachyarrhythmia, atrioventricular nodal blocks, sinus bradycardia and sinus arrest.

This case also highlights the danger of ceasing treatment with nitrates and calcium channel blockers in patients with PA. Hence, the importance of compliance should be emphasised with patients and medical practitioners.

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