

# Image in cardiology

## An interesting cause of syncope

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Coronary artery spasm or Prinzmetal variant angina is seldom considered as a cause of symptomatic bradyarrhythmias in a patient not presenting with chest pain. The images shown here were recorded in a 48-year-old lady referred to cardiology with a history of repeated episodes of syncope over the preceding week. On initial evaluation the syncope was not related to postural change or exercise. Both episodes occurred in the early morning while sitting in bed. No associated angina or dyspnoea was noted. There was no history of palpitations, dyspnoea, Raynaud's phenomenon or migraine. She had no previous medical history of note. She had a 30 pack year smoking history. She was not taking any chronic medication. General examination was normal with no features of postural hypotension. Cardiac and systemic examination was normal. Her referral ECG was normal, but a bradycardia of 20 beats per minute was documented during a witnessed event at the referral hospital. Electrolytes and thyroid function were normal and serial cardiac markers negative. The diagnosis of symptomatic sinus node dysfunction was made and a VVI pacemaker was inserted with a backup rate of 40 beats per minute.

After VVI insertion she had another witnessed event. The patient developed chest discomfort with nausea and autonomic symptoms. Serial ECG recordings revealed transient sinus bradycardia progressing to 3rd degree atrioventricular (AV) block (VVI activated) (Figure 1, ECG 1) and return to sinus rhythm with clear inferior

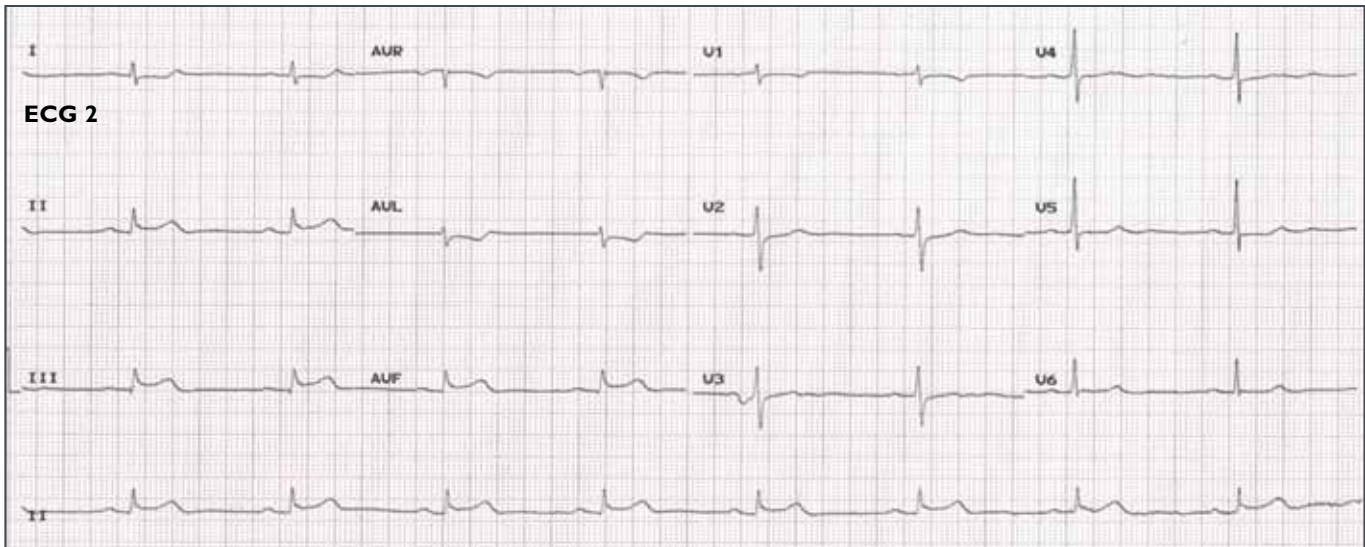
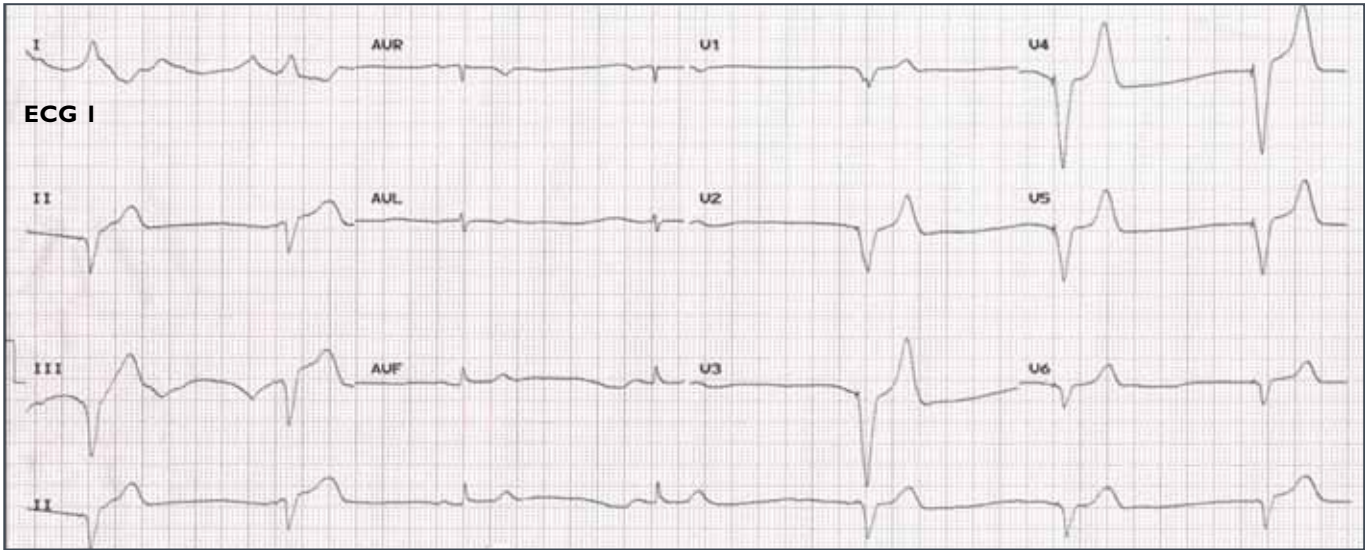
ST elevation (Figure 1, ECG 2). This was followed by spontaneous resolution of symptoms and complete ST resolution.

An angiogram was performed, with a similar unprovoked event occurring during the procedure. Angiography at this time revealed complete right coronary artery occlusion at mid-vessel level (Figure 2.1 and 2.2) with TIMI 3 flow after intra-coronary administration of 200ug of nitrates (Figure 2.3 and 2.4). Mild irregularity of the right coronary artery was noted. The diagnosis of syncope secondary to transient sinus bradycardia/3rd AV block caused by vasospasm of the right coronary artery, i.e. Prinzmetal variant angina was made. We concluded that the initial history did not reveal angina as the patient had syncope with these episodes and thus could not give a clear account of preceding events. The patient was treated with a Ca<sup>++</sup> channel blocker and has not suffered any further events.

Prinzmetal variant angina was first described by Myron Prinzmetal in 1959 with the features of chest pain at rest with transient ST segment elevation due to coronary spasm.<sup>(1)</sup> Numerous arrhythmias have been described with these events.<sup>(2)</sup> A few case reports of right coronary artery spasm with 3rd degree AV block have been described.<sup>(3)</sup> Younger females with a paucity of cardiovascular risk factors (except for smoking) are the typical patient group. The exact pathogenesis is not well understood, but endothelial dysfunction and the autonomic nervous system causing alpha adrenergic activation is thought to be the mechanism of this entity. Multiple triggers have been identified including cigarette smoking, hyperventilation, alcohol, cocaine and acetylcholine.<sup>(2)</sup> Prinzmetal variant angina has a circadian rhythm to it and usually occurs during the early morning hours. Vasodilators are the mainstay of treatment which includes nitrates and Ca<sup>++</sup> channel blockers. Beta receptor blockers and high doses of aspirin should be avoided. The prognosis of Prinzmetal variant angina is usually good except if there is significant coronary artery stenosis or if arrhythmias develop secondary to coronary artery spasm.<sup>(2)</sup>

### REFERENCES

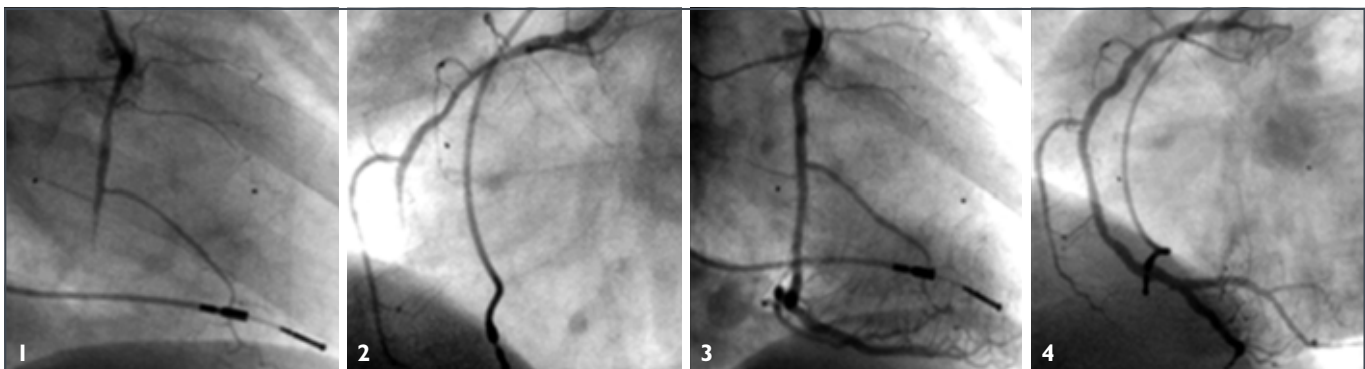
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**FIGURE 1:** Serial ECGs were recorded during a witnessed pre-syncope event associated with chest discomfort and autonomic symptoms occurring days after implantation of a VVI pacemaker. The ECGs revealed transient sinus bradycardia progressing to 3rd atrioventricular (AV) block with activation of the pacemaker.

ECG 1: The initial ECG recording revealed a paced rhythm alternating with marked sinus bradycardia.

ECG 2: Spontaneous return to sinus rhythm with inferior ST elevation.



**FIGURE 2:** Following the event described in Figure 1 coronary angiography was performed. Another unprovoked pre-syncope event associated with chest pain occurred during the procedure. Shown here are four images from the angiogram of the right coronary artery taken at the time. Coronary flow in the right coronary artery was interrupted at mid-vessel level – right anterior oblique (RAO) view (1) and left anterior oblique (LAO) view (2). Following the intra-coronary administration of nitrates (200ug) TIMI 3 flow was restored – RAO view (3) and LAO view (4).