CASE REPORT

A 54-year-old woman with premature ventricular complexes and a rapidly changing ECG

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SUMMARY

Premature ventricular complexes (PVCs) are common findings in asymptomatic participants during ECG recordings. While most PVCs are treated conservatively, some patients may develop adverse cardiac events needing further investigations and definitive ablation by radiofrequency. Spectral strain ultrasonography has demonstrated ventricular dysfunction as a sequel to high burden PVCs, but the exact underlying pathophysiology is still uncertain. Cohort studies have associated PVCs with increased risks for overall cardiovascular morbidity and mortality, and yet consensus is still lacking for threshold in treating PVCs. Our case is that of a 54-year-old woman with an unusual bradycardiac presentation of PVCs and an ECG that changed rapidly within minutes.

BACKGROUND

Premature ventricular complexes (PVCs) is a common diagnosis among the general population and often presents as an incidental finding in up to 10.7% of individuals.¹ There are many aetiologies underlying PVCs and some of them may precede more ominous diagnoses including ventricular fibrillation (VF) and death. In a broad sense, PVCs refer to all depolarisation signals outside the sinoatrial node that leads to activation of the ventricular myocardium. It has been shown that frequent PVCs are associated with left ventricular dysfunction as measured by speckled strain echocardiography.² Also, catheter ablation studies of PVCs consistently improved left^{2–4} and right² ventricular functions. Thus said, current views are still dissident regarding increased risks of mortality or morbidity due to PVCs. Lin *et al*⁵ reviewed 24 h Holter tracings of 3351 individuals with apparently normal heart and concluded that multiform PVCs were associated with a significant higher HR for mortality, hospitalisation, transient ischaemic attacks and new-onset heart failure and new-onset atrial fibrillation in the following 10 years. On the contrary, study by Qureshi *et al*⁶ in an 18-year follow-up of 7504 patients with arrhythmias from standard 12-lead ECGs showed increased mortality and morbidity with premature atrial complexes and not PVCs. One meta-analysis study suggested that the poor prognosis of PVCs maybe confounded by occult structural heart diseases in those so-called 'normal' patients which are not divulged by advanced cardiac tests.⁷ Here, we present a patient with unusual presentation of PVCs with multiple forms that change within 10 min as revealed by sequential ECG. We also discuss the various treatments of PVCs and the main differential diagnoses.

CASE PRESENTATION

The patient is a 54-year-old woman who presented to our family medicine clinic for regular follow-up of her heartburn symptoms. She has type 2 diabetes controlled by diet, and comorbidities of hypertension and obstructive sleep apnoea syndrome. She admitted recent stressors due to family conflicts. She smokes 5–10 cigarettes a day and consumes up to 50 units of alcohol a week. She also smokes marijuana up to 1 g occasionally. A stress test was attempted but results were inconclusive due to poor exercise capacity. Cardiology referrals had been made but patient defaulted on both occasions. On routine examination, patient developed mild chest tightness and dizziness with a radial pulse of 45/min bilaterally. She appeared pale with no diaphoresis or sweating. She was breathing spontaneously with no respiratory distress or cyanosis. Her sitting blood pressure was 140/87 mm Hg, respiratory rate of 18/min with oxygen saturation at 98% on room air.

INVESTIGATIONS

A 12-lead ECG was performed immediately when the patient was in maximum discomfort ([figure 1](#)). There were PVCs coupled with nodal complexes in bigeminy of duplet and triplet, with no obvious ST-segment changes denoting cardiac ischaemia.

A second ECG was performed 5 min later, when the symptoms started to relieve ([figure 2](#)). Here, PVCs were still in bigeminy with only one duplet seen.

A third ECG was performed when the patient said her symptoms had completely subsided ([figure 3](#)). Note the persistence of PVC in constant bigeminy with nodal QRS complexes.

Owing to patient's reporting of chest tightness, blood was also sent off for serum troponin-I level.

DIFFERENTIAL DIAGNOSIS

Differential diagnosis include premature atrial complexes (PACs), third-degree AV heart block, Wolf-Parkinson-White syndrome (WPW) and Torsade de Pointes (TdP).

Premature atrial complexes arise from the atria and hence, these are invariably accompanied by a P-wave due to atrial depolarisation. Also the extra complex follows the physiological conduction pathway and hence, carries a normal upward T-wave, which contrast the PVCs which are characterised by inverted T-waves. Third-degree AV (complete) heart block leads to classic bradycardia with QRS complexes completely decoupled with the P-waves. These complexes arise from an accessory

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Figure 1 First 12-lead ECG revealing an irregular sinus rhythm with frequent premature ventricular complex bigeminy occurring in duplets and triplets.

pacemaker which is outside the sinoatrial node. The interval between P-waves is constant, so is the interval between R-waves of the QRS complexes. In our patient's tracing, a P-QRS complex with constant P-R interval is present and coupled with each PVC.

WPW syndrome is a common pre-excitation syndrome with an aberrant conduction pathway (Bundle of Kent) between the atria and ventricles, predisposing to a re-entrant type of supra-ventricular tachycardia. Owing to their sinoatrial origin, the depolarisation complexes of WPW are characterised by the presence of P-waves with a slurred upstroke and short P-R interval. In our patient, the PVC has neither P-wave nor slurred upstroke.

TdP describes a polymorphic tachycardia which often precedes VF. The QRS complexes arise from the ventricles and are, therefore, wide with no recognisable P-waves; as the TdP complexes typically twist around the isoelectric axis, hence the name. The PVCs described in our case have a constant isoelectric axis and are coupled with narrow nodal QRS complexes.

TREATMENT

As the patient became asymptomatic while being assessed and troponin level was negative for myocardial damage, patient was discharged home with a referral to repeat 48 h Holter monitor and an urgent appointment with the arrhythmia clinic to consider radiofrequency ablation (RFA) therapy. In asymptomatic patients with frequent PVCs but preserved left ventricular (LV)

function, the clinician can reassure the patient and continue to follow-up with them should they become symptomatic.⁸ Treating PVCs is important because they have been associated with LV enlargement and cardiomyopathy.⁹ The treatment of PVCs typically includes one of two modalities: RFA to remove the focus of arrhythmogenic electrical current or using antiarrhythmic drugs. Patients are generally considered candidates for catheter ablation or a pharmacotherapy if >10% of their total heart beats over 24 h are PVCs and they have LV dysfunction.⁸ Both treatment modalities aim to eliminate PVCs and improve LV systolic function.¹⁰ Current consensus favours RFA over pharmacotherapy, in particular for patients with PVCs from the right ventricular outflow tract.^{10 11} RFA is also reserved for drug-intolerant or refractory patients who are heavily symptomatic.¹² RFA has been shown to halt the progression of LV dilation, which in turn reduces the risk of cardiovascular events.¹³ Studies also showed restoration of LV function after ablation therapy for PVCs.^{4 14 15} For patients not suitable for ablation, PVCs can also be effectively treated pharmacologically using medications like β -blockers or calcium channel blockers.⁷ The standard follow-up for patients who had either ablation or start of pharmacotherapy is a repeat Holter monitor and echocardiography within 3 months.⁷

DISCUSSION

PVCs occur in 1.2–10.7%^{1 16 17} of the general population and are often asymptomatic findings in 12-lead ECG and Holter

Figure 2 Second 12-lead ECG showing continued premature ventricular complexes in bigeminy with one duplet in 1 min tracing.

Figure 3 Third 12-lead ECG showing continued premature ventricular complexes in constant bigeminy.

tracings. It is an age-dependent condition with prevalence increasing drastically in people older than 75 years old.⁸ Studies have shown that PVCs originate preferentially from the right ventricular outflow tract rather than the left, with the approximate ratio of 2:1.^{18–19} While episodic occurrences generally lead a benign course, persistent PVCs that occurs >20% of times will lead to compromise of ventricular outflow function² and higher risks of cardiovascular events and overall mortality.⁵ One possible mechanism involves PVC-induced cardiomyopathy, which has been well documented clinically when patients had their cardiomyopathy successfully reversed after ablation of PVCs.^{14–15} The exact underlying pathophysiology is still unknown, with cellular mechanisms involving membrane cyclic adenosine monophosphate (cAMP)¹⁵ and the voltage-dependent calcium channel Cav_{1.2} receptors being implicated.²⁰

Owing to their ectopic non-sinoatrial origin, PVCs are prone to subsequent re-entrant currents that can predispose more sinister wide QRS complexes with prolonged QT intervals, leading to ventricular tachycardia, TdP or even VF. Ephrem *et al*²¹ showed that multiform PVCs confirmed by Holter monitoring are associated with fourfold increase in adverse cardiac events. A recent meta-analysis by Ataklte *et al*¹ also confirmed a 2.64 times higher risk for sudden cardiac death associated with presence of PVCs.

Learning points

- ▶ Premature ventricular complexes (PVCs) are not uncommon among the general population and can be asymptomatic.
- ▶ Though most PVCs are managed conservatively, clinicians should be cognizant of their potential sequelae and be prepared to further investigate.
- ▶ PVCs may mimic other ventricular arrhythmias and should be differentiated accordingly.
- ▶ PVCs may lead to ventricular dysfunction and reduced cardiac output, and are implicated as a cause of cardiomyopathy.
- ▶ Most PVCs arise from the right ventricular outflow tract; radiofrequency ablation is the treatment of choice and offers better prognosis than pharmacotherapy.

Our patient, described earlier, was unusual in that she presented with a very slow palpable pulse with minimal symptoms in contrast to most PVCs which often lead to tachyarrhythmias. Moreover, in her resting asymptomatic state, her PVCs were tightly coupled with physiological nodal QRS complexes in a 1:1 bigeminy manner. Yet, only her PVCs were leading to effective ventricular contractions which are barely sufficient to sustain a functional cardiac output. Hence, there was an urgency for cardiology referral and definitive ablation by radiofrequency.

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Competing interests None.

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