**CASE REPORT**

**Diagnosis of acute myocardial infarct with ventricular paced rhythm**

Jonathan Knott
Department of Emergency Medicine, Royal Melbourne Hospital, Parkville, Victoria, Australia

**Abstract**

Management of acute myocardial ischaemia is dependent upon interpretation of the 12-lead electrocardiogram. The presence of ventricular pacing and acute myocardial infarction makes electrocardiogram interpretation difficult. This may impact upon patient management if treating staff are unaware of the expected electrocardiogram morphology or do not have a rapidly available means to make the diagnosis. This case highlights the difficulty with diagnosis of acute myocardial infarction in the presence of ventricular paced rhythm and demonstrates the electrocardiogram changes that occur with myocardial infarction.

**Key words:** acute myocardial infarction, electrocardiogram, pacemaker, ventricular paced rhythm.

**Introduction**

Acute myocardial infarction (AMI) is associated with high mortality and morbidity. Outcomes are improved with early treatment using beta-blockers\(^1\) aspirin\(^2\) and relief of vascular occlusion with thrombolytic drugs\(^2,3\) or coronary angioplasty.\(^4\) However, coronary reperfusion therapies are only of proven benefit in patients with a clear clinical picture of AMI and specific changes on the 12-lead ECG.\(^2\) Patients with a permanent pacemaker pose a particular diagnostic dilemma due to their ventricular paced rhythm (VPR). The morphology of the VPR restricts utilization of the ECG criteria for thrombolysis to treat AMI. Lack of access to potentially beneficial treatment modalities may adversely affect outcomes in the increasing patient population with a permanent cardiac pacemaker.

**Case study**

A 58-year-old man self-presented to the emergency department complaining of chest pain. He was grey and sweaty and was moved rapidly to the resuscitation area. His pain had begun 45 min prior to presentation. It was on the left side of his chest, heavy and radiated to his bottom teeth which were aching. He was nauseated and short of breath. Initial examination revealed him to be sweaty and anxious, with a respiratory rate of 16 breaths per minute, blood pressure of 135/75 mmHg and a regular pulse of 70 b.p.m. There were two heart sounds with no murmurs and a clear chest without evidence of cardiac failure.

His initial ECG (Fig. 1) showed a VPR with visible pacing spikes. There was a monophasic R wave in leads I and aVL, deep Q waves across leads V1 to V6.
Diagnosis of AMI with VPR

101

and discordant ST segments in all leads except V2 which showed marked ST depression.

He was treated with 300 mg of aspirin and two sublingual glyceryl trinitrite (GTN) tablets. This therapy had minimal effect so he was given 2.5 mg of intravenous morphine and a GTN infusion was commenced. As a result, his pain score decreased from 8/10 to 6/10.

Although AMI was the most likely diagnosis, thrombolysis was initially withheld due to the possibility of alternative diagnoses including aortic dissection. Fortunately, he spontaneously reverted to his native rhythm allowing a repeat ECG to be obtained (Fig. 2). This showed an infero-lateral AMI with reciprocal ST depression in aVL and V2.

He had no contra-indications for thrombolysis and was treated with reteplase (Recombinant Plasminogen Activator marketed as Rapilysin®) (Roche Products, NSW, Australia) approximately 30 min after arrival. He became mildly hypotensive and responded to a rapid infusion of 1000 mL of normal saline. His ST segment elevation appeared to decrease on the cardiac monitor but, prior to obtaining a repeat ECG, he returned to a VPR. He became pain free 35 min after the initial bolus of reteplase, which was 110 min after the onset of his symptoms.

Results of blood tests taken on arrival were available 90 min after presentation: normal full blood examination, urea and electrolytes, creatinine kinase (CK) 140 U/L, CK-MB 28 U/L and troponin-I 5.3 ng/mL. His CK peaked at 2078 U/L 12 h after the onset of pain, whilst his troponin-I was > 500 ng/mL.

The following morning he felt well, had been pain free overnight and his ECG demonstrated evolving Q waves.

A coronary angiogram 36 h after presentation revealed a tightly narrowed right coronary artery that was stented. He had good left ventricular function.

He was discharged from hospital 6 days after presentation.

Figure 1. 12-lead electrocardiogram showing a ventricular paced rhythm 45 min after onset of crushing central chest pain.
Discussion

Several large studies have shown an improvement in outcomes following AMI with the use of thrombolytic drugs in specific patient populations. The earlier the drugs are given, the greater the improvement.\textsuperscript{2,3,5} To be eligible for thrombolysis, patients must have a ‘good’ clinical picture for myocardial ischaemia and an ECG with a new left bundle branch block (LBBB) pattern or specific ECG changes. These changes consist of 1 mm ST elevation in two contiguous inferior leads or 2 mm ST elevation in two contiguous antero-septal leads.

As permanent cardiac pacemakers are usually inserted into the right ventricle, the spread of electrical conduction occurs from right to left, simulating a LBBB. This pattern is expected to show discordant ST segments, i.e. the ST changes are in the opposite direction to the QRS complex and the complex should be longer than 0.12 s. A monophasic R wave is present in leads I and aVL and a dominant S wave occurs in lead III, aVF and across the ventricular leads. Sgarbossa \textit{et al.} compared 17 patients from the GUSTO trial (0.04\% of total) with a VPR to matched controls and identified criteria ‘useful’ for identifying an AMI.\textsuperscript{6} These were: discordant ST elevation $\geq 5$ mm (sensitivity 53\%, specificity 88\%, $P = 0.025$), concordant ST elevation $\geq 1$ mm (sensitivity 18\%, specificity 94\%, $P$-value not significant) and ST depression $\geq 1$ mm in leads V1, V2 or V3 (sensitivity 29\%, specificity 82\%, $P$-value not significant). The low sensitivity for identifying an AMI has important clinical implications when rapid diagnosis is required for the most effective therapy. In Fig. 1, discordant ST elevation $> 5$ mm can be seen in leads III and aVF, whilst ST depression is observed in V2. These correspond with the ST changes in Fig. 2, Figure 1 also has ST elevation in II, V5 and V6 that does not meet Sgarbossa’s criteria of $\geq 5$ mm; however, also corresponds to changes in Fig. 2.

![Figure 2. 12-lead electrocardiogram 10 min after arrival. Non-paced rhythm with evidence of acute, infero-lateral, myocardial infarction.](image-url)
If access to acute coronary angioplasty is available then a definitive diagnosis can be made and treatment implemented early. For patients with a clinical picture suggesting AMI but a VPR this might be regarded as the optimal approach. There has been one observational study of patient outcomes with an AMI and VPR. This showed that acute coronary angioplasty lowered mortality at long-term follow-up but that this benefit was not conferred by thrombolytic therapy. Unfortunately, access to acute angioplasty is restricted to a few centres, especially after hours.

If a pacemaker technician is available then modification of the pacemaker output should allow the underlying ST morphology to be analysed. This is also limited by the general lack of rapid availability of this intervention when required. In addition, there may be haemodynamic deterioration when adjusting the pacemaker output that also limits this approach.

Finally, as the pacemaker is responding to an underlying bradycardia, titration of a drug that increases cardiac rate, such as atropine, may result in a dominant native rhythm that allows ST segment analysis. Although this approach has been suggested, there is no evidence to support this intervention and the risk of a relative tachycardia during an AMI must be weighed against the benefit of early therapy (or the risk of inappropriate therapy).

The population with a permanent cardiac pacemaker is growing and overlaps considerably with that of patients with ischaemic heart disease. The presence of a VPR may lead to a delay in definitive treatment of an AMI. Conversely, an awareness of this problem may decrease the delay to vessel patency and improve individual outcomes. Management of a patient with a VPR and a suspected AMI must be tailored to account for available interventions, especially outside normal office hours.

References