EP CASE REPORT

Dynamic J-point elevation associated with bigeminy in a patient with early repolarization phenotype

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Early repolarization syndrome (ERS) is the combination of J-point elevation and spontaneous VF.[1] We present a case demonstrating association between the J-point amplitude and arrhythmia.

Case

A 54-year-old Caucasian gentleman underwent treatment for stable angina in our institution. There were no symptoms suggestive of arrhythmia. The electrocardiogram (ECG) revealed inferolateral J-point elevation (present in Leads I, II, III, aVF, V5–V6, with greatest amplitude 0.2 mV

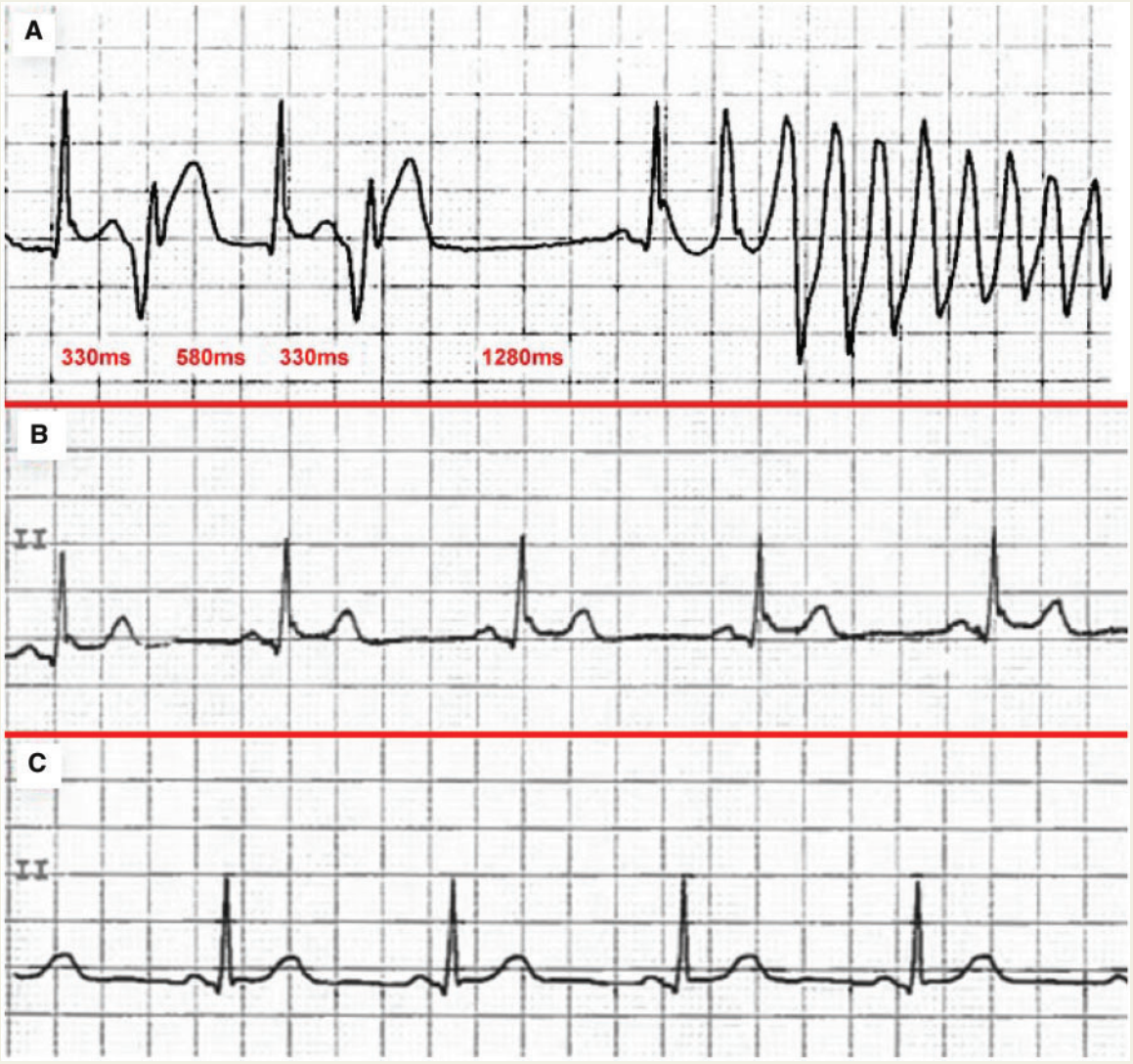

Figure 1 (*A*) Surface ECG Lead II during dysrhythmia. R–R coupling intervals shown. J-point accentuation following short-long R–R intervals, then subsequent polymorphic VT. (*B*) Resting ECG in clinic before treatment. (*C*) ECG in clinic after initiation of Quinidine.

in Lead II), notching of the S-wave and horizontal ST segments, a high-risk ER pattern.[2] The corrected QT interval (Bazett's, QTcB) was 433 ms. Coronary angiography revealed an aberrant circumflex artery arising from the right aortic sinus before coursing retroaortically. Minor disease within the left anterior descending artery (LAD) and right coronary artery was observed and a discrete stenosis in the mid-circumflex artery was stented, apparently without complication. Medication included dual antiplatelet agents and Bisoprolol 2.5 mg once daily.

Twenty-eight days after this intervention, he represented following an out-of-hospital ventricular fibrillation (VF) cardiac arrest. This was treated promptly with defibrillation, with good recovery. Elevation in J-point amplitude was observed during therapeutic cooling in the Intensive Therapy Unit (ITU). Serial biomarkers did not suggest an acute ischaemic event, and repeated coronary angiography confirmed stent patency. Magnetic resonance imaging demonstrated a normal size left ventricle with mild systolic dysfunction. Mid-wall late gadolinium enhancement was noted in the lateral basal-mid left ventricle suggestive of infarction. Our patient was discharged following implanted cardiac defibrillator (ICD) placement. He declined genetic testing.

A further two weeks later, he presented in VF electrical storm with five discrete VF episodes with appropriate ICD discharges. Rhythm monitoring during an episode is shown in *Figure 1A*. Note ventricular bigeminy resulting in short–long R–R coupling, and greatest J-point amplitude immediately preceding VF. From the data obtained, it was not possible to localize the ectopic focus.

Beta-blockers and amiodarone did not completely control the dysrhythmia, but the I_{to}-blocking agent Quinidine (200 mg four times daily) resulted in near-normalization of the electrocardiogram (*Figure 1B* and *C*). This has been well tolerated with no further arrhythmia.

Discussion

Early repolarization syndrome is incompletely understood, but experimental models have shown that transmural gradients in the transient outward potassium current (I_{to}) can lead to phase 2 re-entry, J-point elevation on the surface ECG and arrhythmia.[3] The reduction in dysrhythmia with the I_{to} blocker Quinidine supports this mechanism. The J-point amplitude can be increased by bradycardia, parasympathetic activity, hypothermia, and hypercalcaemia. The diagnosis is usually made in patients with cardiac arrest, ER pattern, and an otherwise normal heart. Our patient does not satisfy this definition, although the polymorphic dysrhythmia favours ERS rather than scar as an underlying mechanism. We hypothesize that the combination of a subclinical infarction with ventricular bigeminy (causing beat-to-beat R–R variation) provided the substrate required for re-entry in a patient with a pathological ER pattern.

Conflict of interest: none declared.

References

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