Evolving J-wave electrocardiographic pattern precipitating polymorphic ventricular tachycardia in a patient with probable silent coronary artery spasm

We present a case of probable silent coronary artery spasm (CAS) presenting as an out-of-hospital cardiac arrest with electrocardiographic dynamic J-point elevation precipitating malignant ventricular arrhythmia.

Case History:

A 46-year-old male was admitted following out-of-hospital cardiac arrest. On initial assessment, the patient was in polymorphic ventricular tachycardia (VT), which degenerated into ventricular fibrillation (VF) and required multiple shocks to restore sinus rhythm. Past medical history included significant smoking & alcohol excess, and childhood asthma. There was no family history of sudden cardiac death. The admission electrocardiogram (ECG) showed sinus rhythm with infero-lateral ST-segment depression (figure 1). Emergent coronary angiography showed minor atheroma in the proximal right coronary artery but otherwise unobstructed epicardial coronary arteries. Cardiac MRI showed mild LV impairment with global hypokinesia with absent scar or other focal abnormality. Pending further investigation, a provisional diagnosis of idiopathic VF was made and a subcutaneous implantable cardiac defibrillator (ICD) was implanted and the patient discharged on bisoprolol and amiodarone with advice on smoking cessation and cutting down alcohol. After 3 weeks he was readmitted following 2 shocks from his ICD for VT/VF. Whilst under continuous cardiac monitoring, he developed further episodes of polymorphic VT degenerating to VF. Dynamic electrocardiographic changes in the J-point was seen, with extreme J-point elevation in the infero-lateral leads immediately prior to VF (figure 2). The patient had no chest pain during these episodes. Electrophysiology study with VT stimulation protocol was negative. The working differential diagnosis was early repolarization syndrome or coronary artery spasm. Quinidine failed to suppress further VT/VF episodes but he had no episodes after commencing verapamil. He was scheduled for a repeat coronary angiogram with vasospasm provocation testing. However, he developed recurrent ventricular tachycardia culminating in two further ICD shocks on withdrawal of verapamil prior to the test. At the time of intracoronary injection of contrast into the right coronary artery, there was electrocardiographic loss of S wave, QRS prolongation, and J-point elevation (figure 3). He also developed some discomfort and anxiety which led to him refusing to proceeding with the provocation component. Subsequent ajmaline testing was negative. Collectively these findings confirmed our potential diagnosis of silent CAS and verapamil was restarted. No further ventricular arrhythmias occurred and the patient was discharged. The patient has had no further arrhythmic events since.

Although we were unable to definitively confirm the diagnosis of CAS using intracoronary provocation testing, the investigative results which excluded other causes, the electrocardiographic changes seen during right intracoronary contrast injection, and the marked response to calcium channel blockade, make the diagnosis of silent CAS causing ventricular arrhythmia probable. This case serves as a reminder to include coronary artery spasm as a differential diagnosis in cases of suspected ERS even in the absence of typical anginal chest pain.
Figures and legends

Figure 1. Admission electrocardiogram showing sinus rhythm and inferolateral ST-segment depression.

Figure 2. Dynamic J-point elevation prior to ventricular tachycardia and ICD discharge. Electrocardiograms captures on continuous cardiac monitoring whilst inpatient. Corresponding times on bottom of each trace.

Figure 3. Electrograms at baseline and during right coronary artery contrast injection. Notice loss of S wave most noticeable in lead III, QRS broadening, and J-point elevation.