A memorable experience
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The electrocardiographic phenomenon of cardiac memory results from aberrant ventricular activation and causes transient ECG features that can mimic ischaemia. We present the case of a patient with ischaemic cardiomyopathy who exhibited evidence of T-wave memory in the setting of multiple episodes of non-sustained ventricular tachycardia that were triggered by an insect bite.

Keywords Ventricular tachycardia; Implantable cardioverter-defibrillator

A 60-year-old man with ischaemic heart disease and a history of cardiac arrest presented following ICD discharge.
On the afternoon prior to admission, he was stung by a wasp, but had no immediate symptoms except for pain at the site of the sting. Early the following morning, he received an ICD shock without prodrome. Interrogation of the device revealed no use of pacing for bradycardia and no detected episodes in the previous 9 months, but showed 88 episodes of non-sustained ventricular tachycardia (NSVT) at rates of 140–150 bpm, starting within minutes of the reported time of the insect sting. Electrograms showed the VT to be monomorphic with similar morphology for all episodes. Antitachycardia pacing terminated seven events, 80 terminated spontaneously, and a shock was required for the last episode, which occurred ~12 h after the initial occurrence of NSVT. There were no further episodes following ICD discharge.

The admission ECG, obtained ~10 h after the ICD shock, showed sinus rhythm with deep symmetric anterolateral T-wave inversions, which were new since previous tracings (Figure 1A). There had been no recent changes to the patient’s medical regimen, which included ACE inhibitor, beta-blocker, and mexiletine. Serial cardiac enzymes were normal. Adenosine myocardial perfusion testing showed severely depressed LVEF (20%), and no reversible perfusion or

Figure 1  (A) The patient’s ECG on presentation. Device interrogation showed nearly 100 episodes of non-sustained ventricular tachycardia in the preceding 12 hours, culminating in ICD discharge. The marked T-wave inversions are thought to represent the phenomenon of cardiac memory, as serial cardiac enzymes and myocardial perfusion imaging did not show evidence of ischaemia. (B) Follow-up ECG 8 weeks after presentation. The T-wave changes present on admission have resolved. (C) ECG during VT. The predominantly negative QRS complexes in the lateral leads during VT correspond to the repolarization abnormalities observed in panel (A).

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wall motion abnormalities. The patient felt well throughout his hospital course and had no further tachyarrhythmias. An ECG 10 days later was unchanged, whereas 8 weeks after presentation, the T-wave changes had reverted to the previous baseline (Figure 1B). A subsequent ECG showed a wide-complex tachycardia with evidence of a prior inferior infarction, right bundle branch block, right superior axis, and a late-reversal R-wave pattern suggestive of VT with an inferobasal LV origin (Figure 1C). At electrophysiology study, the VT mapped to this region, but was unable to be ablated.

The striking T-wave changes seen on the presenting ECG are not explained by a major ischaemic event, and represent the transient sequelae of aberrant ventricular activation, the phenomenon of cardiac memory. The predominantly negative QRS complexes in the lateral leads during VT are consistent with the repolarization abnormalities seen in the presenting ECG (Figure 1A). Well-recognized causes of cardiac memory include intermittent left bundle branch block, pre-excitation, or tachyarrhythmias. Although usually reported after prolonged periods of arrhythmia, cardiac memory has been observed following even shorter periods of abnormal ventricular activation than were present in our patient. In this case, an insect bite triggered the frequent NSVT that created this ‘memorable’ ECG.

Conflict of interest: none declared.