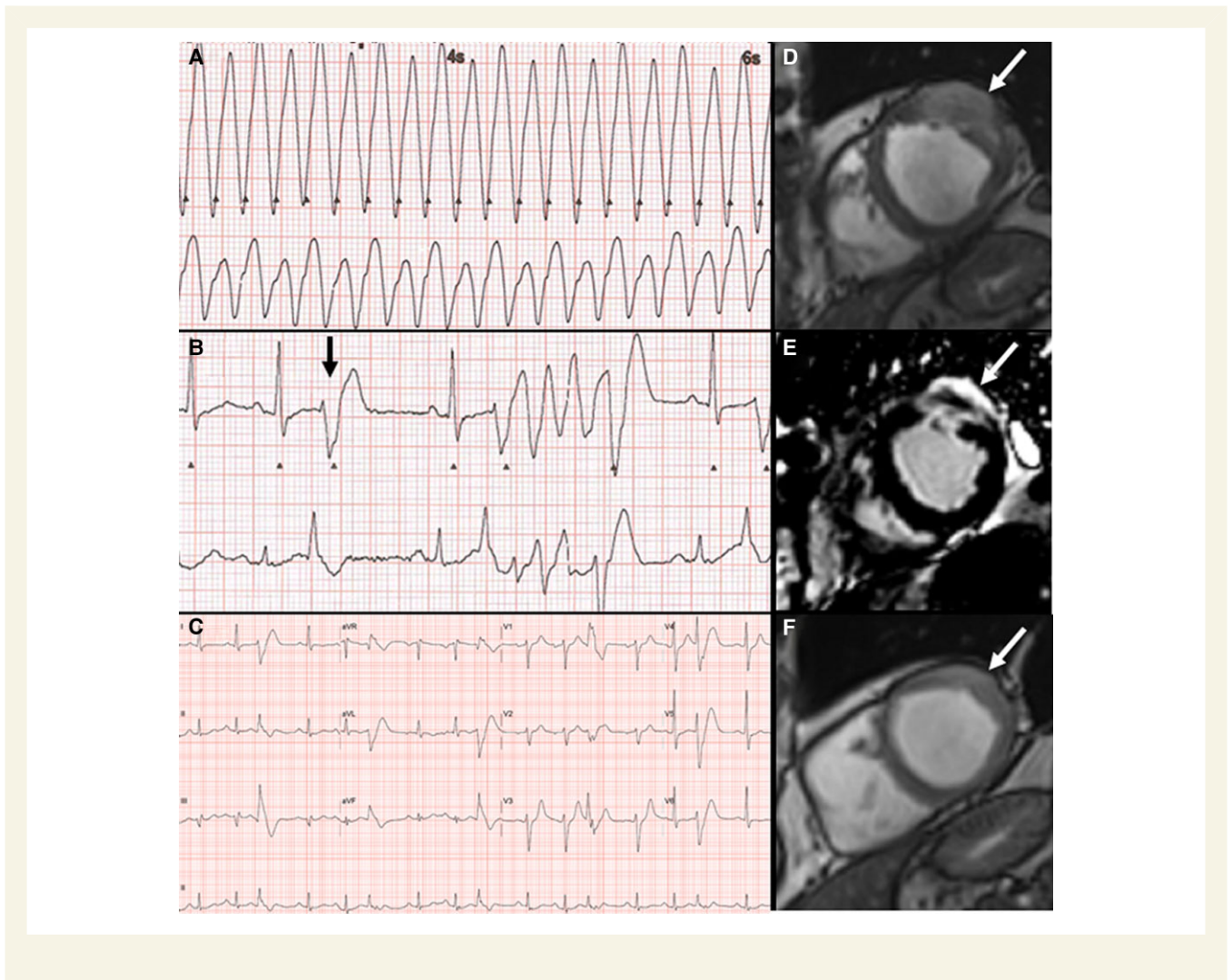


Out-of-hospital cardiac arrest secondary to intramyocardial metastasis

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Emergency services were contacted for an out-of-hospital cardiac arrest of a 65-year-old man with no previous cardiovascular history. Furthermore, there was no family history of sudden cardiac death, internal cardiac defibrillator (ICD), or pacemaker. In addition, no pro-arrhythmic drugs were taken by the patient. The patient was recently diagnosed with a carcinoma of the lung with bone metastases, for which treatment with pembrolizumab and analgic radiotherapy was initiated.

The first rhythm documented was a monomorphic ventricular tachycardia (VT) (*Panel A*). After direct current conversion, repetitive short runs of polymorphic VT were detected, always triggered by an identical premature ventricular complex (PVC) falling at the end of the T-wave (*Panel B*, arrow). Electrocardiogram demonstrated PVC originating from the basal anterior wall and a normal QTc (*Panel C*).

In the pre-hospital setting, the patient was intubated and mechanically ventilated. He was haemodynamically stable without the need for intravenous inotropes. The intravenous amiodarone started during the cardiopulmonary resuscitation was continued. The patient's blood work showed no electrolyte disturbances and a normal first lactate. The lactate reached its maximum of 3.5 mmol/L (normal: 0.5–2.0 mmol/L) after ~6 h.

Transthoracic echocardiography revealed a preserved left ventricular ejection fraction (LVEF = 50%) without significant valvular disease or pericardial effusion. Coronary angiography showed no significant coronary stenosis (see [Supplementary material online](#)). The presence of a pulmonary embolism was excluded by computed tomography angiography.

Cardiac magnetic resonance (CMR) imaging a few days after admission revealed a normal LVEF. A myocardial wall thickening in

the mid- and apicolateral region (2.5 × 4.5 cm) with irregular non-ischaemic type early and late gadolinium enhancement, compatible with intramyocardial metastasis was found (*Panels D and E*, arrows). Given the recent medical history, clinical presentation, PVC origin, and metastasis location, a diagnosis of short-coupled PVCs from the metastatic region inducing polymorphic VT was made. Likely, one of these runs organized into a sustained monomorphic VT. Unfortunately, no 12-lead ECG of this monomorphic VT was available.

During hospitalization, no VTs occurred after the initiation of amiodarone and a beta-blocker. The treatment with pembrolizumab was continued. In accordance with the current guidelines, no ICD was implanted in secondary prevention due to the poor oncological prognosis. Furthermore, in consultation with the patient, a do not attempt resuscitation decision was made. A CMR after 6 months showed regression of the cardiac metastasis (*Panel F*, arrow).

Supplementary material

[Supplementary material](#) is available at *European Heart Journal – Quality of Care and Clinical Outcomes* online.

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