Wolff–Parkinson–White Syndrome and myocardial infarction in ventricular fibrillation arrest: a case of two one-eyed tigers

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Case

A 42-year-old man was carrying a set of goalposts at his son’s football match when he slumped against a fence and collapsed. Two off-duty nurses commenced cardiopulmonary resuscitation (CPR) and called an ambulance. When the crew arrived, ventricular fibrillation (VF) was identified and the patient was defibrillated (Figure 1). After a short period of asystole, he reverted into sinus rhythm. He was admitted to the hospital for further assessment.

The patient made a rapid neurological recovery and was conscious on arrival. He had felt unwell and experienced palpitation only for a few seconds prior to collapse, but had no chest pain or other cardiovascular or neurological symptoms. He described intermittent and self-resolving palpitation when he was in his teens, which was not investigated. He had previously undergone treatment for Helicobacter pylori and gastroesophageal reflux disease, and had had three minor head injuries in the previous month. He took no medications, and had no significant family history. He was an ex-smoker, having stopped 8 years before. He normally drank ~16 U of alcohol per week, although on the evening prior to admission he had consumed six cans of lager (~16 U).

Examination was unremarkable. Troponin I taken 12 h after cardiac arrest was slightly elevated at 0.21 ng/ml (reference threshold <0.05 ng/ml). Chest X-ray was normal. A 12-lead electrocardiogram (ECG) (Figure 2) showed sinus rhythm with a short P-R interval and delta waves suggestive of Wolff–Parkinson–White Syndrome (WPW). A computed tomography head scan revealed no abnormality. An echocardiogram showed normal left ventricular function, except for reduced wall motion in the apical septal segment, extending to the left ventricular apex.

He was referred for an electrophysiology study (EPS) with a working diagnosis of pre-excited atrial fibrillation (AF) precipitating VF. Left atrial pacing, from a catheter placed in the coronary sinus, revealed a pre-excitation pattern suggestive of a left anterolateral accessory pathway. The pathway had a relatively long effective refractory period of 300 ms, and pre-excitation was intermittent. After intravenous isoprenaline the refractory period shortened to 260 ms. During the study, atrial fibrillation occurred as a result of catheter manipulation, and the ventricular response was not very rapid, at ~130 beats/min. This, along with the findings of a relatively long accessory pathway refractory period suggestive of a left anterolateral accessory pathway, cast some doubt over the diagnosis of pre-excited atrial fibrillation as a precipitant for the patient’s cardiac arrest.

The accessory pathway was successfully ablated. Although the delta wave was eliminated in the inferior leads, the ECG after ablation remained abnormal with anterior Q-waves and a slurred downstroke of the QRS complex in leads V1 and V2 (Figure 3). Further electrophysiological testing eliminated the possibility of a second accessory pathway, and a diagnosis of prior myocardial infarction was considered. Programmed ventricular stimulation, using the Wellens protocol,
did not produce sustained ventricular tachycardia (VT), but non-sustained VT of 10–15 s was produced using two extra stimuli. With these findings, it was felt that an accessory pathway may have been a bystander finding and that myocardial infarction may have caused the patient’s cardiac arrest.

Cardiac magnetic resonance imaging (MRI) demonstrated thinned akinetic mid-ventricular anterior wall, septum and apical segments with extensive delayed enhancement. A mural thrombus was also identified. These findings were consistent with an established myocardial infarction. Coronary angiography demonstrated single vessel disease with an occluded left anterior descending coronary artery, and the vessel was successfully treated with percutaneous coronary intervention utilizing a drug-eluting stent.

The timing of the myocardial infarction was not easy to determine as the patient had no symptoms prior to his cardiac arrest. In the absence of sequential ECG changes, and with a relatively low peak troponin I of 0.21 ng/ml in a patient with transmural infarction, it seemed likely that the myocardial infarction may have occurred several days before the cardiac arrest. A final diagnosis was made of late ventricular fibrillation following myocardial infarction, and bystander WPW syndrome. A single chamber implantable cardiac defibrillator was implanted for secondary prevention.

Figure 1. Electrocardiogram showing ventricular fibrillation, defibrillation and asystole.

Figure 2. A 12-lead electrocardiogram suggestive of Wolff-Parkinson-White syndrome.
Discussion

A total of 56–66% of out-of-hospital deaths have a cardiac aetiology, and out-of-hospital cardiac arrest has been reported to have an incidence between 36 and 128/100 000 per year.\(^1\) These numbers are likely an underestimate however, since not all cases are seen by emergency medical services. VF is the first recorded rhythm in 75–80% of patients presenting with sudden cardiovascular collapse.\(^2\) Causes include cardiomyopathies, valvular and congenital heart diseases and primary electrophysiological disorders. Electrophysiological disorders included WPW syndrome, where ventricular pre-excitation (i.e. the earlier activation of the ventricles via an accessory pathway than would be expected through the atroventricular node and bundle of His) may deteriorate to VF. This may be the case as with AF and a rapidly conducting accessory pathway. Patients may be treated with catheter ablation. However, coronary artery disease remains the most common cause of fatal arrhythmia and sudden cardiac death, accounting for 80% of cases.\(^1,3,4\)

In this case of cardiac arrest, it is interesting to note two potential aetiologies for the presentation were identified: an accessory pathway and myocardial infarction. Professor Calbert I Phillips once wrote on logic in medicine, ‘If you see two eyes staring at you out of the jungle at night it may not be a man-eating tiger, it may be two one-eyed man-eating tigers.’\(^5\) A thorough and systematic assessment of a patient is indispensible. Keeping an open mind to differential diagnoses is vital, because the initial ‘obvious’ diagnosis is not always the correct one. Moreover, as doctors we are taught that common is common. Almost ubiquitous in medicine are concepts and maxims such as Occam’s razor, and ‘When you hear hoof beats think horses not zebras.’ Ultimately, the underlying pathology responsible for our patient’s presentation was the most common cause for cardiac arrest: obstructive coronary artery disease.

Conflict of interest: None declared.

References