A 76-year-old man with ventricular tachycardia

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ECG Education articles are inspired by, but not based on, real cases to illustrate the importance of knowledge about ECGs in relation to clinical situations in general practice. Management is not discussed in detail.

James is 76 years old and exercises regularly. He has noted some irregularity and rapidity of his pulse rate directly after exercise on two occasions and felt weak and unwell at that time. He had no chest pain. You arrange a 24-hour Holter monitor and an ECG. Both show the same rhythm (see Figure of ECG result below).

Q1. What does this patient's ECG show?
The ECG shows a short run of monomorphic, nonsustained ventricular tachycardia (VT).

Q2. What is ventricular tachycardia (VT)?
VT is three or more heart beats with a rate over 100 beats per minute. The QRS is widened over 120 msec. The origin of the VT will be below the level of the bundle of His and will have a cycle length of 140 to 250 beats per minute typically, with a regular rhythm and a constant QRS morphology. This will be different from the usual sinus rhythm QRS morphology, will have a different axis, and may have atrioventricular dissociation (which is diagnostic). VT may be monomorphic (each QRS complex is identical and arising from a single focus) or polymorphic (the QRS complexes are irregular and arise from multiple foci). Monomorphic VT is dangerous as it may degenerate into ventricular fibrillation. Polymorphic VT is also dangerous as this is indicative of active myocardial ischaemia or torsades de pointes, which may also degenerate into ventricular fibrillation. If the VT lasts over 30 seconds it is called 'sustained VT' and if it is under 30 seconds, it is called 'nonsustained VT'.

Q3. What is 'torsades de pointes'?
Torsades de pointes is a polymorphic VT (rate typically from 200 to 250 beats per minute) with a prolonged QT interval (typically over 600 msec), which may be congenital or acquired. The amplitude of the QRS complex changes in a sinusoidal fashion, twisting around the isoelectric line, so there is variation from beat to beat – this is similar to a ballet movement from which the abnormality derives its name. It is usually associated with QT prolongation (normal sinus rhythm with a QTc of >500 msec). Torsades de pointes is usually self-limiting but may transform into ventricular fibrillation.

Q4. What is a re-entrant VT?
Re-entrant (or monomorphic) VT is a paroxysmal, regular VT in which the electrical impulse follows an abnormal electrical pathway that loops back into itself. This stable circuit usually involves abnormal scarring of the ventricular myocardium or a ventricular specialised conduction system (usually seen in myocardial ischaemia or cardiomyopathy). The VT is often also triggered by hypoxia or electrolyte disturbances. Re-entrant VTs require a unilateral block and a slowed conduction to develop.

Q5. What may trigger VTs?
Common triggers of VTs are myocardial ischaemia and inflammation, cardiomyopathy, genetic syndromes (e.g. long QT syndrome, Brugada syndrome), drug use (cocaine, amphetamines),

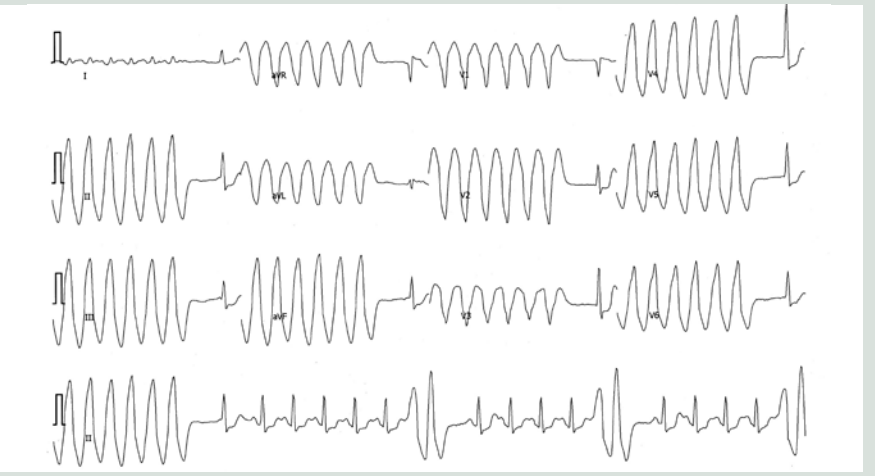

Figure. An ECG showing nonsustained ventricular tachycardia.

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medications that prolong the QT interval, hypokalaemia and hyperkalaemia, hypomagnesaemia, hypoxia (sleep apnoea), congenital heart disease (e.g. tetralogy of Fallot and right ventricular dysplasia), digitalis toxicity, sympathomimetic medications, infiltrative diseases of the myocardium, systemic lupus erythematosus and rheumatoid arthritis. VTs may also be idiopathic and in the absence of cardiac disease. It should be noted that any arrhythmia occurring during exercise should be taken seriously and fully investigated. Exercise is a known trigger of VT.

Q6. Why is a VT so dangerous?

The rapidity of the heart rate and the abnormal ejection volumes cause less filling pressure in the ventricles and thus a reduced cardiac output. This frequently results in hypotension, cerebral and myocardial ischaemia and the risk of consequent ventricular fibrillation. It follows that VT is more dangerous in people with underlying cardiac conditions, such as ischaemia, fibrosis, cardiomyopathy and structural cardiac abnormalities.

Q7. What are the differential diagnoses of VT?

Idioventicular rhythms are much slower than VTs (usually 60 to 100 beats per minute) and as a result are usually better tolerated haemodynamically. They occur in the setting of ischaemic or structural cardiac disease and are quite often seen after successful defibrillation during cardiac resuscitation. If the rate is accelerated they may be confused with VT.

Supraventricular tachycardia with rate-related bundle branch block (such as an atrioventricular re-entrant tachycardia, an atrioventricular nodal re-entrant tachycardia with aberrant conduction or a focal atrial tachycardia, flutter or atrial fibrillation) mimics VT because of the rapidity of heart rate and the broadening of the QRS complex secondary to the atrioventricular block. Calcium channel blockers are avoided in cases of wide complex tachycardias of uncertain diagnosis as they are negative ionotropes and may precipitate sudden death in patients with VT with supraventricular tachycardia and bundle branch block.

Traces from dual chamber pacemakers tracking a fast sinus tachycardia or other atrial arrhythmia will cause a wide complex QRS mimicking VT, as may those from inappropriate rate-responsive pacemakers.

Q8. What medications are used to treat acute VT?

The medications used to treat acute VT depend on whether there is normal left ventricular function and whether the patient has major contraindications to beta blockers. Medications are used in patients who are haemodynamically stable with VT (i.e. not requiring immediate defibrillation) and to prevent recurrent VT after defibrillation. Medications include (in the absence of contraindications) intravenous or oral beta blockers such as sotalol, metoprolol, intravenous or oral amiodarone and intravenous lignocaine. Sotalol and amiodarone are avoided in patients with torsades de pointes as they may further prolong the QT interval.

Q9. What are the indications for use of medications to prevent VT?

The original studies concluding that medications were a safe alternative to implantable catheter defibrillators to prevent VT have been shown to be untrue. An implantable catheter defibrillator is first line management as the only proven therapy to save lives. The only exception to these statements are the truly curable idiopathic VTs or bundle branch re-entrant VT, which should be ablated. Medications (beta blockers, sotalol, amiodarone and rarely flecainide) may also be used in conjunction with implantable cardioverter defibrillators to reduce the number of defibrillations occurring. Amiodarone may reduce events but does not reduce the risk of death. Ablation may be considered if VT is refractory or highly recurrent and is not adequately controlled by medications (and coronary revascularisation, if appropriate).

Q10. What are the indications for an implantable cardioverter defibrillator to prevent sudden death in individuals at risk?

The indications are: New York Heart Association functional class II or III; a left ventricular ejection fraction of 30 to 40% or less; left ventricular dysfunction for at least 40 days following a myocardial infarction; receiving chronic optimal medical therapy; life expectancy is at least a year with reasonable function; or recurrent Torsades de pointes.

Q11. What are the indications for endocardial cardiac ablation?

Endocardial catheter ablation is used in idiopathic monomorphic VT (by definition, there is no structural abnormality causing the arrhythmia). It also has a use in patients with cardiomyopathy to reduce arrhythmias and in those with implantable cardioverter defibrillators to reduce the number of defibrillations occurring.

Outcome

James' cardiovascular risk factors were assessed and he underwent cardiac echocardiography and CT coronary angiography. His heart was structurally sound with no clinically significant atheroma. Two options were discussed: electrophysiological studies with a view to catheter ablation versus a trial of a beta blocker or verapamil (this would be the only setting that verapamil can be used as the right ventricular outflow tract VTs are described as 'verapamil sensitive VTs'). James proceeded to electrophysiological studies (which offers an 80% chance of permanent cure from a single procedure, but not without a low risk of complications). He was ultimately diagnosed with idiopathic monomorphic VT related to exercise and successfully underwent endocardial catheter ablation. **CT**

Key points

- **Ventricular tachycardia (VT) is three or more heart beats with a rate over 100 beats per minute. The QRS is widened over 120 msec and there is atrioventricular dissociation.**
- **Any arrhythmia occurring during exercise should be taken seriously and fully investigated as exercise is a known trigger of VT.**
- **An implantable catheter defibrillator is usually first-line management for VT.**
- **Medications (beta blockers, sotalol, amiodarone and rarely flecainide) may be used in conjunction with implantable cardioverter defibrillators to reduce the number of defibrillations occurring.**
- **Ablation is considered if VT is refractory or highly recurrent and not adequately controlled by medications.**