Wide-complex tachycardia

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Cardiac arrhythmia is a common cause for patient presentation in the emergency department. If the arrhythmia is a wide-complex tachycardia, it needs rapid decision-making and treatment in order to avoid further deterioration.

Introduction

Two ECG features define wide-complex tachycardia: a QRS complex >120 ms and a heart rate of >100 beats per minute [1].

Patients with wide-complex tachycardia can present at the emergency department (ED) haemodynamically stable or unstable. ECG algorithms, as well as knowledge about pre-existing cardiac diseases, can help to identify ventricular tachycardia. Symptoms often are not reliable and helpful for differentiation of the origin of the wide-complex tachycardia.

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The following examples illustrate the approach in patients presenting with wide-complex tachycardia in the ED.

Two patients presenting with wide-complex tachycardia in the ED

Patient 1: a 26-year-old man with palpitations for 3 hours. Vital signs: blood pressure 120/70 mm Hg, oxygen saturation (SpO$_2$) 98%, Glasgow Coma Scale (GCS) score 15. His ECG is shown in figure 1.

Patient 2: a 48-year-old man with chest pain for
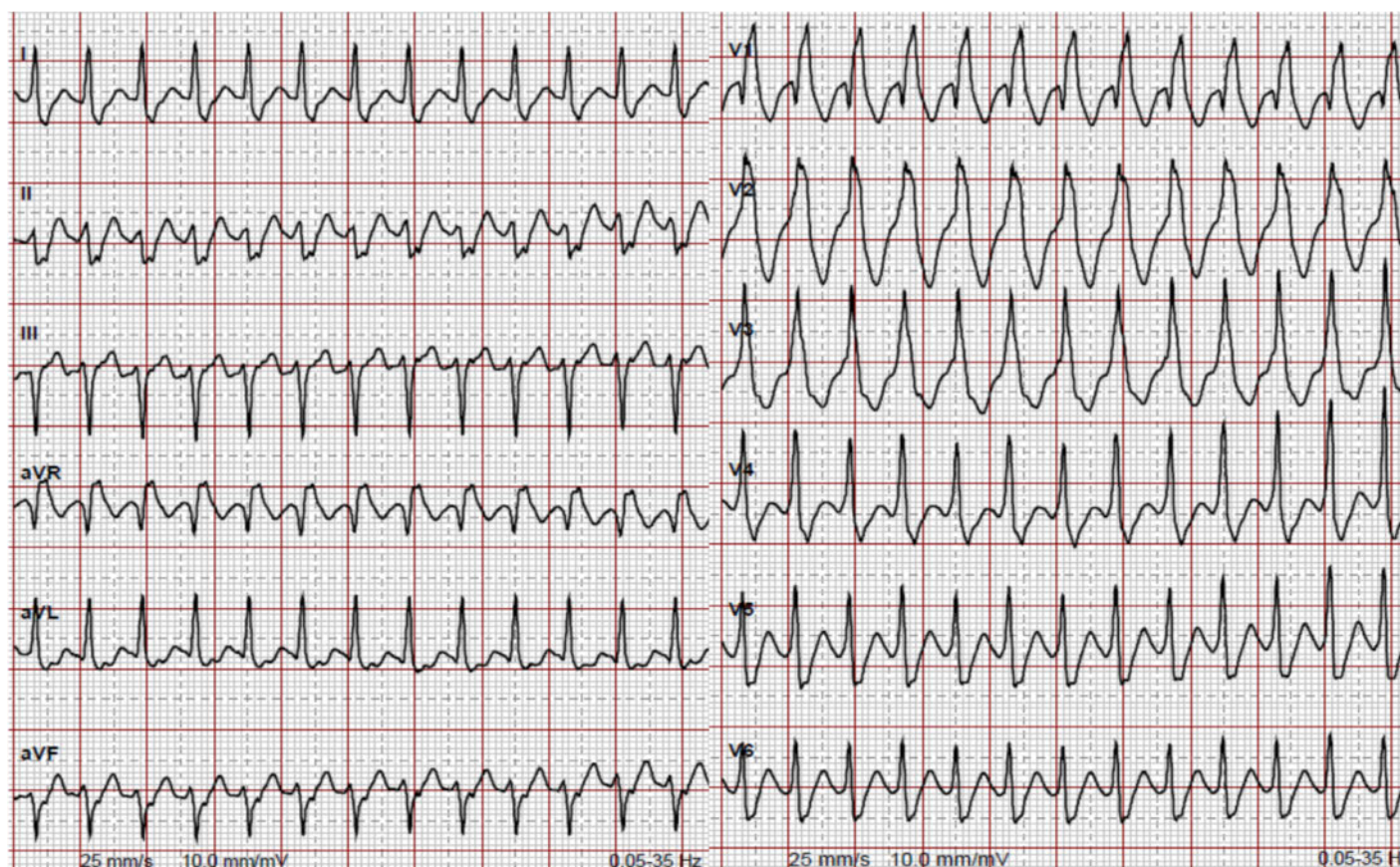
Figure 1: 12-lead ECG from patient 1 at emergency department admission with left ventricular fascicular tachycardia.

Figure 2: 12-lead ECG from patient 2 at emergency department admission with ventricular tachycardia.

40 minutes. Vital signs: blood pressure 90/40 mm Hg, SpO$_2$ 94%, GCS score 12. His ECG is shown in figure 2.

The first questions are: Is the patient having a ventricular tachycardia or not? Is the wide-complex tachycardia a ventricular tachycardia?

The Brugada criteria [2] and the Vereckei algorithm [3] are the most commonly used diagnostic standards for ventricular tachycardia. Both algorithms are a four-step decision procedure in which four criteria for ventricular tachycardia are considered (fig. 3). These algorithms allow correct identification of ventricular tachycardia in about 90% of cases and are not always reliable. However, correct analysis of the ECG takes time if ventricular tachycardia is not obvious after the first or second step.

Regardless of the correct identification of ventricular tachycardia, from the emergency point of view, the question of whom you treat first depends on the patient's stability. Patient 2 was unstable on arrival at the ED. He was immediately transferred to the shock room where the advanced cardiac life support (ACLS) algo-

rithm for stable/unstable tachycardia was applied (fig. 4, adapted from [4]). As the patient had hypotension, altered mental state and ischaemic chest discomfort, he immediately underwent synchronised cardioversion, in accordance with the algorithm. Most important is to record a 12-lead ECG after successful cardioversion, which in our case showed an anterior ST-segment elevation myocardial infarction (fig. 5A). The patient was directly transferred to the heart catheterisation laboratory, where an in-stent thrombosis of the left anterior descending artery was found and treated with percutaneous transluminal coronary angioplasty and stenting. He remained stable after this intervention (fig. 5B).

The vital signs of patient 1 remained stable during his stay in the ED. Detailed analysis of the 12-lead ECG (fig. 1) identified left ventricular fascicular ventricular tachycardia with right bundle branch block and left axis deviation. Again on the basis of the ACLS algorithm for stable/unstable tachycardia, the patient received antiarrhythmic treatment because of his
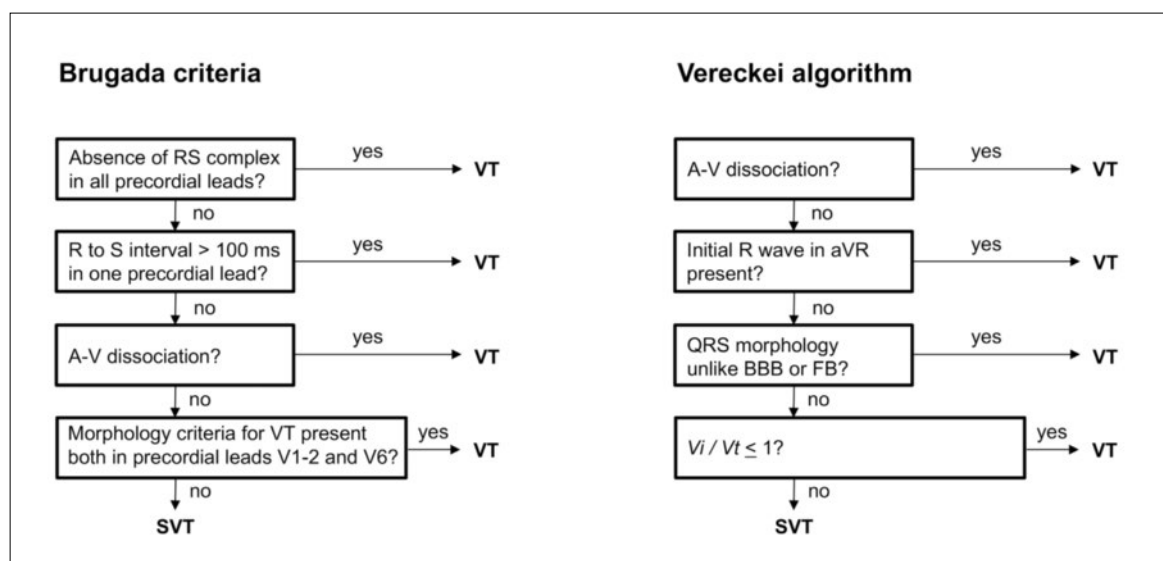
Figure 3: Brugada criteria and Vereckei algorithm to identify ventricular tachycardia (adapted from [2, 3]). A-V = atrio-ventricular; BBB = bundle branch block; FB = fascicular block; VT = ventricular tachycardia.
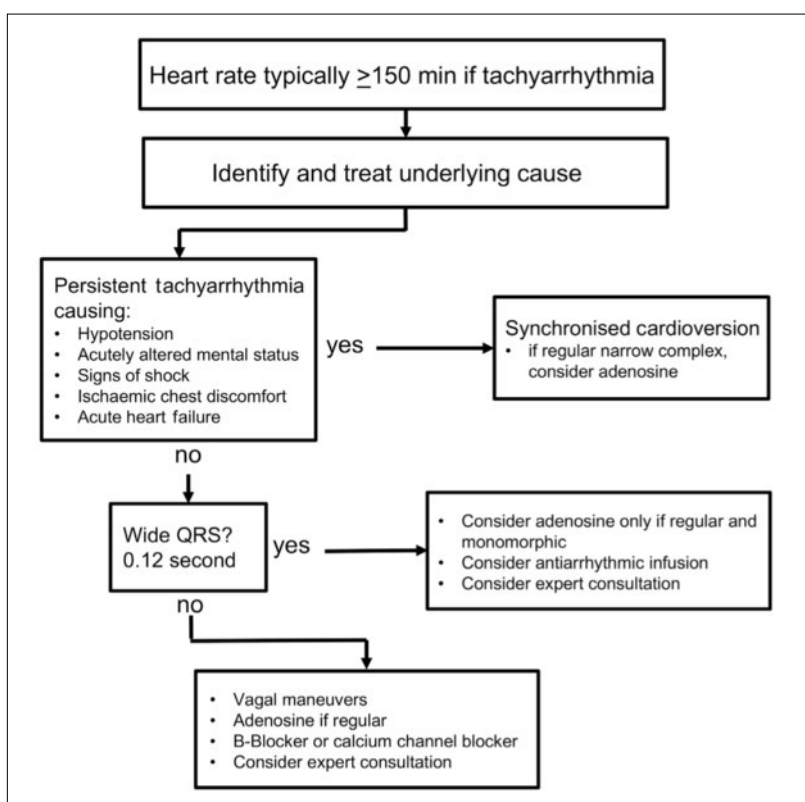
Figure 4: ACLS algorithm for stable/unstable tachycardia: tachycardia with a pulse algorithm. Adapted from https://www.acls.net/images/algo-tachycardia.pdf. © for the original version: 2016 ACLS Training Center, https://www.acls.net. The original document is current with respect to 2015 American Heart Association Guidelines for CPR and ECC. These guidelines are current until they are replaced on October 2020. If you are reading this page after October 2020, please contact ACLS Training Center at support@acls.net for an updated document.

haemodynamic stability. After intravenous administration of 5 mg verapamil, ventricular tachycardia converted to sinus rhythm (fig. 6).

If these patients had presented with pulseless ventricular tachycardia, immediate cardiopulmonary resuscitation would have had to be started according to common ACLS or European Resuscitation Council (ERC) guidelines [5]. As ventricular tachycardia, and ventricular fibrillation as consequence of ventricular tachycardia deterioration, are shockable rhythms, early defibrillation must be attempted.

Wide-complex tachycardia: general considerations and differential diagnosis

A wide-complex tachycardia can arise from a ventricular or a supraventricular origin with a regular or irreg-

Table 1: Wide-complex tachycardia.

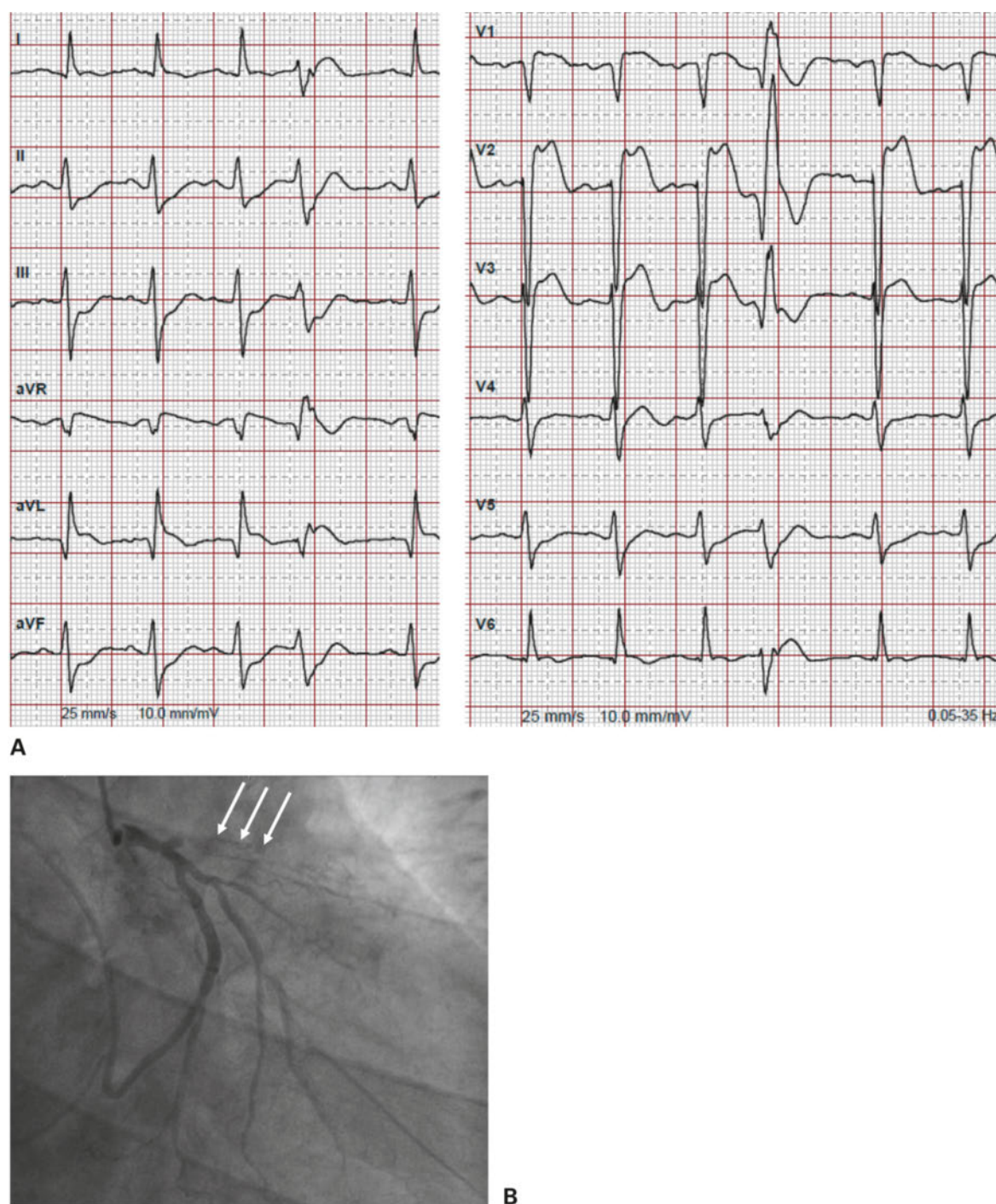| Regular QRS complex |
|---|
| Monomorphic ventricular tachycardia |
| Origin from LV / LVOT / RV / RVOT |
| Supraventricular tachycardia |
| With aberrant conduction in bundle branch block |
| With aberrant conduction in Wolff-Parkinson-White syndrome |
| **Irregular QRS complex** |
| Polymorphic VT, torsades des pointes |
| Atrial fibrillation with bundle branch block |

LV = left ventricle; LVOT = left ventricular outflow tract; RV = right ventricle; RVOT = right ventricular outflow trac; VT = ventricular tachycardia

Figure 5: A. 12-lead ECG from patient 2 after cardioversion showing anterior ST-segment elevation myocardial infarction. B. Coronary angiography showing in-stent thrombosis of the left anterior descending artery (white arrows).

ular QRS complex (table 1). Brugada criteria and the Vereckei algorithm or the simplified avR algorithm [3] are helpful in correctly identifying ventricular tachycardia. Nonetheless, 10% of cases remain misdiagnosed.

Besides ventricular tachycardia, the differential diagnosis of wide-complex tachycardia includes supraventricular tachycardia with aberrant conduction due to bundle branch block or Wolff-Parkinson-White syndrome if the QRS complex is regular. If the QRS complex is irregular, polymorphic ventricular tachycardia such as torsades des pointes Tachycardia or atrial fibrillation with bundle branch block must be considered.
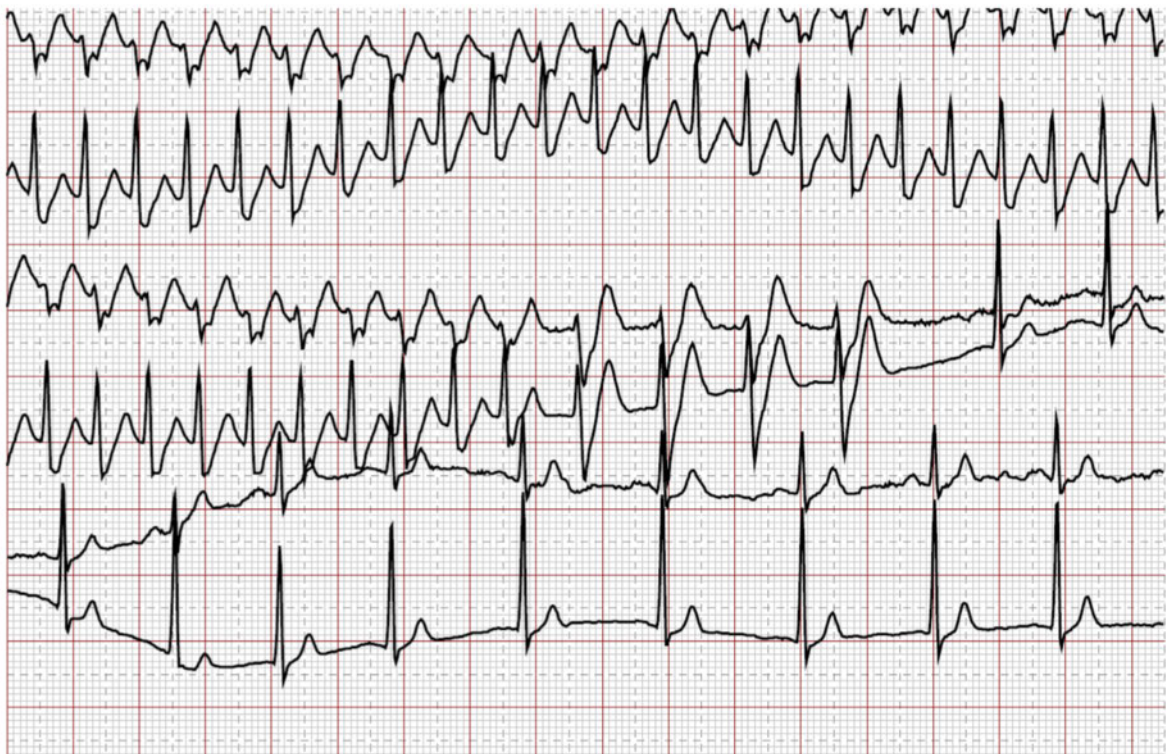
Figure 6: 12-lead ECG from patient 1 during intravenous administration of verapamil showing conversion of ventricular tachycardia into sinus rhythm.

As ventricular tachycardia is still the most common cause for sudden cardiac death rapid identification and immediate treatment are the corner stones of patient survival.

Clinical symptoms

Symptoms during wide-complex tachycardia basically depend on the ventricular rate, left ventricular func-tion and the existence or absence of an atrioventricular synchronicity and include palpitations, (near) syncope, chest pain and dyspnoea and pre-shock.

Underlying diseases

The patient's medical history can be helpful as wide-complex tachycardia can result from different underlying cardiac disorders (table 2). Most commonly, wide-complex tachycardia or ventricular tachycardia originates from coronary artery disease. The presence of cardiomyopathies with or without left ventricular dysfunction are often already known. In addition, a family history of sudden cardiac death is important in patients with cardiomyopathies and channelopathies, as these inherited cardiac disorders can unfortunately present with a fatal ventricular tachycardia as first manifestation. In cases with a structurally normal heart, long-term prognosis is usually better.

Morphology of the wide-complex tachycardia

The morphology of the tachycardia also give hints concerning the origin. If a wide-complex tachycardia is monomorphic, its origin can be ventricular tachycardia in a structurally abnormal heart, most com-

Table 2: Wide-complex tachycardia: most common underlying cardiac disorders.

Coronary artery disease
Acute coronary syndrome / early after myocardial infarction / scar
Left ventricular dysfunction
With or without heart failure
Cardiomyopathies
Hypertrophic cardiomyopathy / dilated cardiomyopathy
Arrhythmogenic right ventricular cardiomyopathy
Channelopathies
Long QT syndrome / short QT syndrome / Brugada syndrome
Catecholaminergic polymorphic ventricular tachycardia
Congenital heart disease
Valvular heart disease
Inflammatory and rheumatic heart disease
Structural normal heart
Toxic / metabolic

monly scar re-entry in coronary artery disease or cardiomyopathies such as hypertrophic or dilated cardiomyopathy, or arrhythmogenic right ventricular cardiomyopathy (table 2). In polymorphic wide-complex tachycardia, the QT interval has to be analysed firstly. If it is normal (*cave*: the Bazzet formula is not valid for correcting the QT interval during tachycardia), ischaemia or electrolyte imbalance should be considered. Torsade des pointes tachycardia results from QT interval prolongation and originates from inherited long QT syndrome, drugs, intoxication or electrolyte imbalance, to name the most common causes.

Ventricular or supraventricular tachycardia?

Regular wide-complex tachycardia can be either ventricular tachycardia or supraventricular tachycardia. Ventricular tachycardia originates from the left ventricle, the left ventricular outflow tract, the right ventricle or the right ventricular outflow tract. In contrast, wide-complex supraventricular tachycardia arises from aberrant conduction in bundle branch block or, rarely, in an accessory bundle (Wolff-Parkinson-White syndrome).

Clinical symptoms are not reliable in the differentiation between ventricular tachycardia and supraventricular tachycardia. The patient's age might be helpful: the older the patient, the more probable is ventricular tachycardia. Haemodynamics as shown in the two examples above are not reliable markers. In contrast, the patient's cardiovascular history might be helpful: for example, if an ischaemic or structural heart disease, previous myocardial infarction, congestive heart failure, a family history of sudden cardiac death is known, ventricular tachycardia is more probable than supraventricular tachycardia. In supraventricular tachycardia with aberration, previous ECGs often show a bundle branch block pattern with morphology identical to that of a wide-complex tachycardia, or evidence of Wolff-Parkinson-White syndrome (short PQ interval, delta-wave), and the patient's history often includes recurrent paroxysmal tachycardia with sudden onset and termination with vagal manoeuvres or adenosine.

Therefore, to clearly distinguish ventricular tachycardia from supraventricular tachycardia, the above named criteria and algorithms always should be used at the onset.

Management of wide-complex tachycardia

Tachycardia with a pulse

The first steps are maintenance of the patient's airway with assisted breathing if necessary, cardiac monitoring to identify the heart rhythm, monitoring of blood pressure and oximetry, and establishing intravenous access [4, 5]. Whenever possible, a 12-lead ECG should be recorded, and reversible causes, such as electrolyte imbalance, should be identified and treated.

In tachycardia causing hypotension, acutely altered mental status, signs of shock, ischaemic chest discomfort or acute heart failure, immediate synchronised cardioversion is attempted. Sedation is required. Adenosine can be considered if a narrow QRS complex is evident.

If wide-complex tachycardia is irregular, possibilities include polymorphic ventricular tachycardia such as torsade des pointes, where immediate administration of magnesium 2 g intravenously over 10 minutes is needed, while stand-by for defibrillation is provided. In atrial fibrillation with bundle branch block causing irregular wide-complex tachycardia, the same treatment as for small complex tachycardia is recommended [5].

For a stable and regular wide-complex tachycardia, if ventricular, amiodarone 2 × 150 mg intravenously over 20–60 minutes is a safe treatment of choice. In previously known left ventricular fascicular ventricular tachycardia, verapamil and a beta-blocker are first-line options. If previously confirmed supraventricular tachycardia with bundle branch block is present, adenosine can be given as for treatment of regular small complex tachycardia [4, 5]. In implantable cardioverter-defibrillator carriers with slow ventricular tachycardia, overstimulation with the device should be attempted.

Tachycardia without pulse

In tachycardia without pulse, advanced cardiac life support is mandatory [5]: start cardiopulmonary resuscitation (CPR), beginning with chest compressions and with a compression : ventilation ratio of 30:2. When the defibrillator arrives, continue chest compressions while applying the defibrillation electrodes. For shockable rhythms, apply defibrillator shock and continue CPR according to guidelines.

Once a wide-complex tachycardia converts into sinus rhythm, a 12-lead ECG helps to identify the underlying cause. According to common guidelines, further diagnostic work-up in patients presenting with sustained ventricular tachycardia should be performed in the

heart catheter laboratory, in the chest pain unit or intensive care unit [6].

Conclusion

In the emergency setting, a wide-complex tachycardia always should be considered as ventricular tachycardia unlike proven otherwise, as treatment has to be initiated immediately to avoid degeneration into ventricular fibrillation.

The Brugada criteria and Vereckei algorithm are helpful for correct ECG analysis and rapid treatment of stable and unstable ventricular tachycardia is based on the common ERC and ACLS algorithms.

Disclosure statement

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