



## Editorial

## Incessant ventricular tachyarrhythmia in the emergency room

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Ventricular tachyarrhythmia  
Hyperkalemia  
Digitalis toxicity

Incessant ventricular tachyarrhythmia (including ventricular fibrillation, polymorphic and monomorphic ventricular tachycardia), which can be a cause of sudden cardiac death, occurs in patients with various cardiac and non-cardiac diseases. Such an arrhythmia may also happen in healthy subjects and is called “idiopathic ventricular tachyarrhythmia”. To rescue patients from these life-threatening ventricular tachyarrhythmias, initial management is essential. However, appropriate therapy is not identical in each case (or background) of ventricular tachyarrhythmia. Of course, for termination of ventricular fibrillation or hemodynamically unstable ventricular tachycardia, defibrillation (or cardioversion) shock is the only reliable therapy to restore sinus rhythm. However, the same ventricular tachyarrhythmia may recur within a short period repetitively unless the underlying arrhythmogenic causes are successfully treated.

For the suppression of incessant episodes of ventricular tachyarrhythmia in patients with impaired cardiac function due to structural heart diseases, treatment combining beta-blockers, deep sedation, and/or intravenous administration of amiodarone (or nifekalant) would be chosen [1]. Twelve-lead electrocardiogram (ECG), echocardiogram, chest X-ray, and/or serological test including the measurement of brain natriuretic peptide, etc., are all useful in estimating the magnitude of the cardiac dysfunction and to identify the underlying heart disease(s). Repetitive form of ventricular tachyarrhythmias can also develop during acute myocardial ischemia. In such situations, coronary revascularization may be the primary therapy in suppressing the ventricular tachyarrhythmias.

Therapeutic approaches for ventricular tachyarrhythmias in patients without structural heart diseases are different from those in patients with structural heart disease. For example, intravenous isoproterenol infusion would be the first treatment for repetitive arrhythmic events in patients with Brugada syndrome or in patients with J-wave associated idiopathic ventricular fibrillation [2]. Twelve-lead ECG is an important clue (coved-type ST-segment elevation in V1–V3 leads for Brugada syndrome and slurred or notched J wave in inferior and/or lateral leads in J-wave syndrome) to reach a correct diagnosis of these syndromes. Similarly,

abnormal QT interval prolongation on 12-lead ECG is important to reach a diagnosis of congenital (or acquired) long QT syndrome (LQTS). Magnesium infusion, faster-pacing and/or supplemental treatment with potassium would be chosen in suppressing recurrent episodes of polymorphic ventricular tachycardia in LQTS patients [2].

As a case presented in this journal [3], drug toxicity and/or electrolyte disturbance is another important cause of repetitive attacks of ventricular tachyarrhythmia. However, in the emergency room, reaching a diagnosis of these conditions may not always be easy, especially in patients first arriving by ambulance.

**Arrhythmias induced by digitalis toxicity or hyperkalemia**

In the article by Mori et al. [3], the patient showed repetitive episodes of polymorphic ventricular tachycardia and accelerated idioventricular rhythm. Basic beats, which were recorded between the intervals of the repetitive ventricular arrhythmias, showed a widening QRS complex and normal QT interval suggesting slowed ventricular conduction and non-prolonged ventricular repolarization, respectively.

It is well known that many arrhythmias (including paroxysmal atrial tachycardia with block, various degrees of atrio-ventricular block, premature ventricular tachycardia, accelerated idioventricular rhythm, etc.) can be induced by digitalis toxicity. Although characteristically tall T wave is considered an initial sign of hyperkalemia, PR interval prolongation, flattening of P wave, widening of QRS complex, and/or development of life-threatening ventricular tachyarrhythmia would emerge on the ECG as the stage of hyperkalemia progressed. Therefore, from only the ECGs in the patient reported by Mori et al., it might be difficult to obtain an immediate diagnosis of digitalis toxicity combined with hyperkalemia as a cause of the repetitive episodes of ventricular tachyarrhythmias.

**Therapeutic approach for ventricular tachyarrhythmias associated with digitalis toxicity and hyperkalemia**

Although the authors used lidocaine as the first treatment, intravenous administration of lidocaine is not indicated for the treatment of ventricular arrhythmias associated with digitalis toxicity [4]. However, at emergency admission, therapeutic intervention for malignant ventricular arrhythmias may need to be started before obtaining complete laboratory results. As an appropriate treatment for digitalis toxicity, the American Heart Association/American College of Cardiology guideline (2006)

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recommends the following [4]. Among these options, an antidigitalis antibody is unavailable in Japan.

#### Class I

An antidigitalis antibody is recommended for patients who present with sustained ventricular arrhythmias, advanced atrio-ventricular block, and/or asystole that are considered due to digitalis toxicity. (Level of Evidence: A)

#### Class IIa

1. Patients taking digitalis who present with mild cardiac toxicity (e.g. isolated ectopic beats only) can be managed effectively with recognition, continuous monitoring of cardiac rhythm, withdrawal of digitalis, restoration of normal electrolyte levels (including serum potassium greater than 4 mM/L), and oxygenation. (Level of Evidence: C)
2. Magnesium or pacing is reasonable for patients who take digitalis and present with severe toxicity (sustained ventricular arrhythmias, advanced atrio-ventricular block, and/or asystole). (Level of Evidence: C)

#### Class IIb

Dialysis for the management of hyperkalemia may be considered for patients who take digitalis and present with severe toxicity (sustained ventricular arrhythmias; advanced atrio-ventricular block, and/or asystole). (Level of Evidence: C)

#### Class III

Management by lidocaine or phenytoin is not recommended for patients taking digitalis and who present with severe toxicity (sustained ventricular arrhythmias, advanced atrio-ventricular block, and/or asystole). (Level of Evidence: C)

As a treatment for hyperkalemia, emergent interventions are usually recommended either when any findings indicating hyperkalemia are recorded on ECG or serum potassium level is elevated to more than 6.0 mEq/L. Calcium gluconate hydrate, glucose fluid plus insulin, sodium bicarbonate, calcium polystyrene sulfate, and/or furosemide are considered to be effective in lowering the serum level of potassium, but hemodialysis may be required in serious cases (including patients with renal insufficiency). The patient reported by Mori et al. showed life-threatening ventricular tachyarrhythmias, thus she required emergent treatment for hyperkalemia (8.3 mEq/L). Indeed, the authors successfully treated the hyperkalemia by use of the combined treatment of glucose fluid plus insulin, sodium bicarbonate, and calcium polystyrene sulfate. Following the treatment of the hyperkalemia and digitalis toxicity, the incessant form of ventricular tachyarrhythmias spontaneously disappeared.

In the patient reported by Mori et al. [3], dehydration, which was caused by vomiting and anorexia 2 days before the admission, was the most likely trigger of the digitalis toxicity and hyperkalemia. Although laboratory tests before the arrhythmic event did not indicate severe renal dysfunction (serum creatinine was 0.76 mg/dL and estimated glomerular filtration rate was 56.1 mL/min/1.73 m<sup>2</sup>), her renal dysfunction was advanced at the time of the arrhythmic event (serum creatinine was elevated to 1.57 mg/dL). Since this patient was a 74-year-old and had been treated by insulin for type 2 diabetes mellitus, it is highly possible that the patient had suffered from clinically un-manifested renal dysfunction.

In elderly patients suffering from cardiac diseases and/or diabetes mellitus, renal function may easily be altered by various factors. Physicians need to be aware of the possibility of potential renal dysfunction especially when drugs, which are primarily metabolized in the kidneys, are prescribed in such patients.

Hypertrophic cardiomyopathy could have worked as an arrhythmogenic substrate of the ventricular tachyarrhythmias in the patient reported by Mori et al. Since cardiac magnetic resonance imaging of this patient revealed a thick myocardium surrounding the entire left ventricle and a relatively preserved ventricular contraction, diastolic (rather than systolic) dysfunction of the left ventricle and presence of atrial fibrillation would seem to be a likely cause of her history of heart failure. Although a detailed clinical course of this patient was uncertain, beta-blocker or calcium antagonist might be suitable for the rate control of atrial fibrillation in this patient.

#### Summary

For the emergency treatment of incessant ventricular tachyarrhythmias, cardiologists and physicians should make their best effort to clarify the underlying mechanism of ventricular tachyarrhythmia so as to immediately apply the most appropriate treatment in order to save the life of their patients.

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