Incessant Monomorphic Ventricular Tachycardia Associated With Pneumococcal Meningitis: A Case Report

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Abstract

A 22-year-old man with incessant ventricular tachycardia(VT)associated with pneumococcal meningitis without obvious heart disease manifesting as febrile sensation and severe headache visited our emergency department. Initial electrocardiography showed ventricular premature couplets, but the rhythm grew more serious and developed into incessant monomorphic VT resulting in an electrical storm. After examining the cerebrospinal fluid, bacterial meningitis was suspected. The electrical storm ended 21 hr after he had received conservative treatment for meningitis. *Streptococcus pneumonia* was cultured from the cerebrospinal fluid. No VT was observed during the remainder of the hospital stay and could not be induced in the electrophysiological study.

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INTRODUCTION

The interconnection between the central nervous system(CNS)and heart was first described in 1903.[1] Electrocardiographic(ECG)changes and arrhythmias are frequent complications in patients with cerebral injury.[2] Increased plasma concentrations of catecholamines may result in tachyarrhythmias, whereas enhanced vagal activity can cause sinus bradycardia and atrioventricular block. Among the cardiac arrhythmias, ventricular tachycardia(VT)accounts for 2% of the rhythm disturbances in patients with a subarachnoid hemorrhage.[3] The vasocardiac reflexes from the circle of Willis and diffuse cerebral dysfunction may be possible factors causing ECG changes in patients with pyogenic meningitis.[4] We report a case of an incessant monomorphic VT in a patient with pneumococcal meningitis associated with a marked increase in the intracranial pressure.

CASE REPORT

A 22-year-old man visited the emergency room because of a fever and severe headache lasting for 6 hr. He had no cardiovascular risk factors. His vital signs were blood pressure of 130/80 mmHg, respiratory rate of 30/min, pulse rate of 100 beats/min, and body temperature of $39.0\,^{\circ}\mathrm{C}$. The initial ECG showed frequent couplets of ventricular premature beats(**Fig. 1**). The initial laboratory findings were as follows: white blood cell count 25,980/mm³ (neutrophil count 93%), serum potassium level 3.7 mEq/*l*, serum calcium level 9.3 mg/d*l*, and the others were within normal limits. Transthoracic
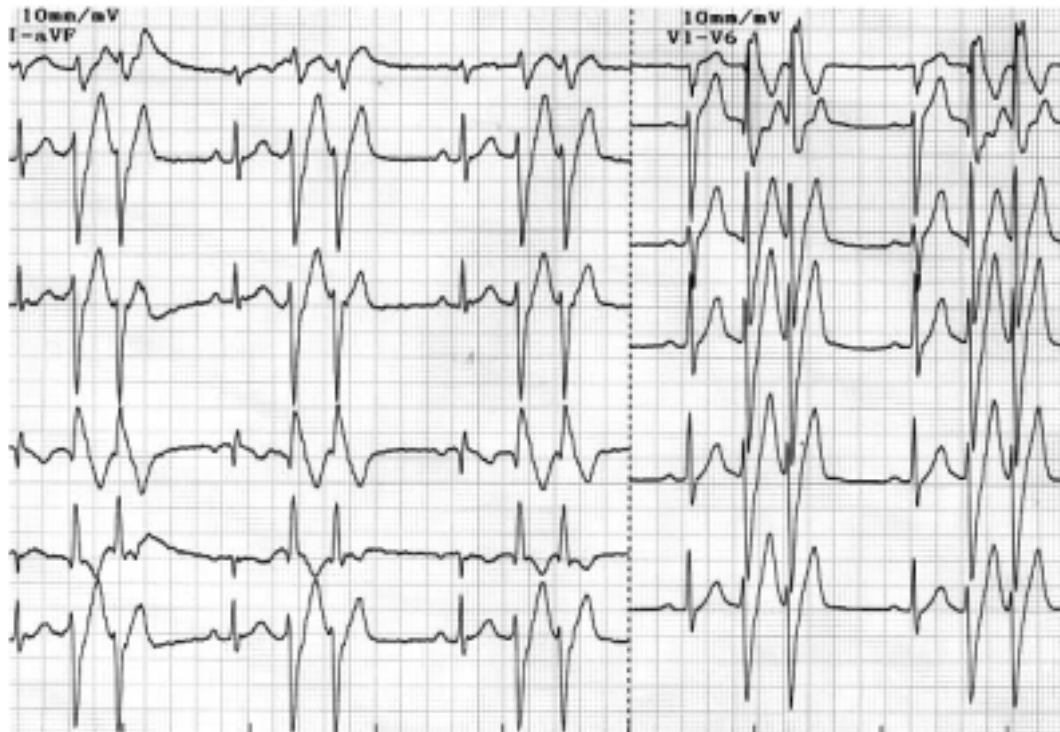
Fig. 1   Initial 12-lead electrocardiogram showing couplets of ventricular premature beats with right bundle branch block morphology and superior axis

echocardiography revealed normal systolic function and chamber size.

Two hr later, monomorphic VT spontaneously developed with a right bundle branch block morphology and superior axis(**Fig. 2－A**), and was terminated by electrical cardioversion. Continuous infusion of amiodarone was started. After 12 hr, a cerebrospinal fluid study was performed, which revealed that the intracranial pressure was 550 mmH$_2$O, red blood cell 1,400/high power field (HPF), white blood cell 18,000/HPF(neutrophil 90％), protein 269 mg/dl, and glucose 10 mg/dl. Following that, intravenous antibiotics(vancomycin and ceftria-xone)were started as an empirical treatment for a suspected bacterial meningitis. After 16 hr, monomorphic VT with the same morphology as the previous event occurred(**Fig. 2－B**). However, his blood pressure was stable. Attempts were made to terminate the VT with electrical cardioversion and continuous infusions of anti-arrhythmic agents such as amiodarone, lidocaine, procainamide, and esmolol, were administered, but none worked and the VT continued. The potassium level decreased to 3.0 mEq/l even with a continuous infusion of potassium(**Fig. 3**). Fortunately, the VT spontaneously

ended 10 hr after the continuous administration of large amounts of potassium, and agents used to lower the intracranial pressure such as mannitol and corticosteroids. *Streptococcus pneumoniae* was cultured from the cerebrospinal fluid several days later. An electrophysiologic study was performed 2 weeks later. No ventricular tachyarrhythmia could be induced by the programmed electrical stimulation. The patient was discharged without sequelae.

DISCUSSION

Subarachnoid hemorrhages and strokes are the most common CNS causes related to ventricular tachyarrhythmias. The present case is a unique report of incessant VT associated with a CNS infection.

Incessant VT is an uncommon arrhythmia and is refractory to anti-arrhythmic drugs and repeated electrical cardioversions.[5] Incessant monomorphic VT has been reported in a variety of clinical settings, including coronary artery disease,[6] Brugada syndrome,[7] and dilated cardiomyopathy.[8]

Morphological electrocardiographic changes have mostly been reported in patients with subarachnoid and intracerebral hemorrhages, but could
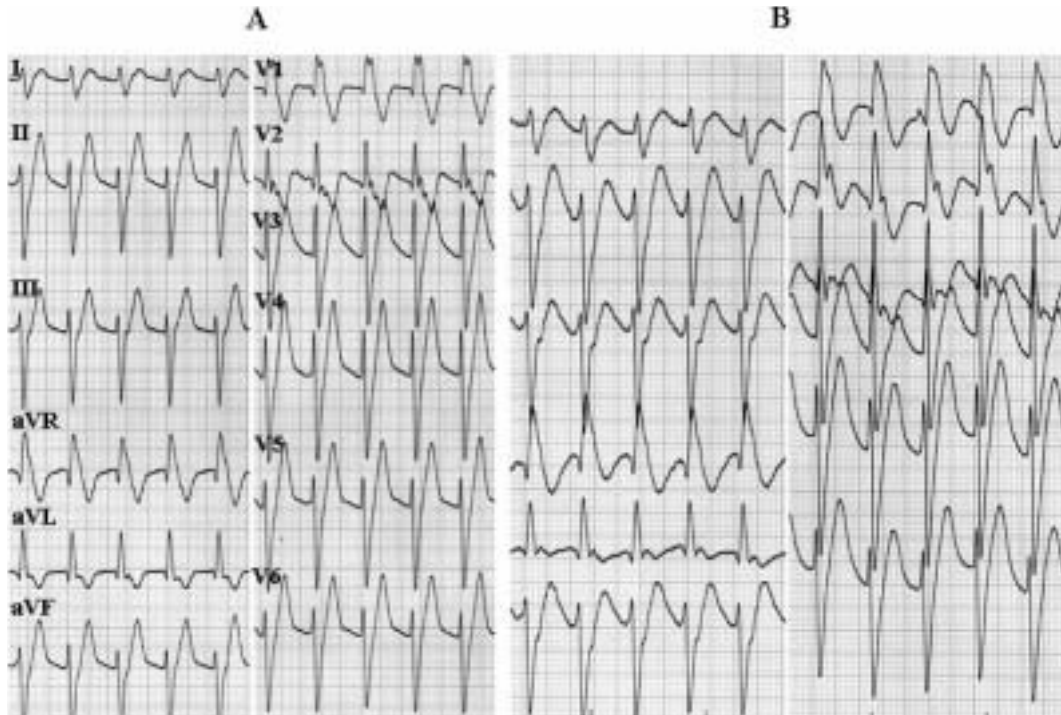
Fig. 2 Twelve-lead electrocardiograms showing monomorphic ventricular tachycardia with right bundle branch block morphology and superior axis
Panel A shows the initial monomorphic ventricular tachycardia.
Panel B shows the second monomorphic ventricular tachycardia which was the incessant type and occurred 16 hr later.
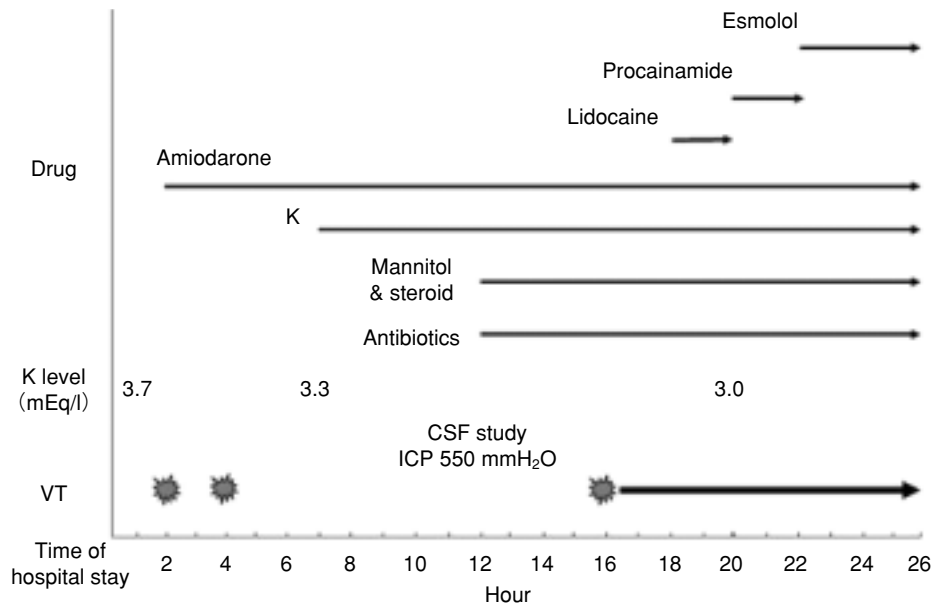

Fig. 3 Time course of the ventricular tachycardia attack, potassium level and administration of various types of medications
VT = ventricular tachycardia; CSF = cerebrospinal fluid; ICP = intracranial pressure.

occur in a variety of CNS injuries including infections, tumors or neurosurgical procedures.[9] The most commonly reported cause of ventricular arrhythmias associated with CNS disease such as sustained VT and torsade de pointes is subarachnoid hemorrhage. Other causes have rarely been reported.

Several studies have suggested possible mechanisms of the ECG alterations in patients with cerebral injuries. First, a sympathetic storm which causes inhomogeneity in the ventricular repolarization. The level of the norepinephrine metabolism is correlated with the prolongation of the QTc interval, predisposing to substrate to ventricular tachyarrhythmias. Cerebral injury causes a stress response resulting in increased concentrations of plasma catecholamines. However, increased intracranial pressure (ICP) can cause a marked reflex sympathetic discharge. The increased ICP may be important in mediating the observed ECG changes.[10] Second, hypokalemia may precipitate ventricular arrhythmias by prolonging the action potential duration. Vomiting, which increases the circulating catecholamines and rennin, or hypercortisolism are obvious causative factors.[11] In particular, excessive activation of the renin-angiotensin system (RAS) can lead to hypokalemia. It is unclear whether the activation of the RAS results from the direct effect of hypothalamus stimulation or an overactivity of the sympathetic system.[12] Third, stimulation of the hypothalamus has been shown to cause frequent ventricular arrhythmias as well as changes in the ECG morphology.

In this case, the initial ICP was 550 mmH$_2$O and was sufficient to cause a sympathetic overflow, but was not confirmed by the blood test. The potassium

level was 3.7 mEq/*l*. Even though the potassium levels continued to decrease whether this was the effect of using the steroids or an overactive state of the RAS remained unclear. Because the initial potassium level could have predisposed the patient to ventricular arrhythmias, it can be assumed that the effect of the hypokalemia on the VT was a result of the meningitis. Antibiotics including macrolides, quinolones, and amphotericin B can induce fatal cardiac arrhythmias.[13,14] In this case, we gave vancomycin and ceftriaxone, which have a low possibility of causing cardiac arrhythmias. The QT interval was 320 msec at the time of the initial ECG. The proarrhythmic effect of amiodarone is associated with QT interval prolongation.[15] Even though we infused amiodarone continuously after the termination of the VT, no arrhythmias were noted, but a mild prolongation of the QT interval to 480 msec was observed.

In the case of intracranial or subarachnoid hemorrhage, cardiac arrhythmias could be directly induced by space-occupying lesions such as hemorrhage-associated transtentorial herniations, which could cause autonomic stimulation over a wide area. On the other hand, cardiac arrhythmias associated with meningitis are caused only by secondary changes such as an elevated ICP or excessive activation of the RAS. Vasocardiac reflexes in the circle of Willis and diffuse cerebral dysfunction may be factors causing ECG changes in patients with pyogenic meningitis.[4]

The main cause of the VT in this case appeared to be a marked increase in the ICP which was facilitated by the hypokalemia. The tachycardia was terminated by decreasing the elevated ICP and correcting the hypokalemia.

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