Ventricular Tachycardia with Two Distinct Morphologies Caused by Herbal Medicine Containing *Ephedra sinica*

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**ABSTRACT**

A 77-year-old man presented to the emergency department with dizziness and a fluttering sensation in the chest. Electrocardiography demonstrated sustained monomorphic ventricular tachycardia with two different morphologies (right bundle branch block, left bundle branch block). The patient had taken herbal medicine containing *Ephedra sinica* for two days. Based on the findings of a thorough work up, I concluded that both of the ventricular tachycardias might have been caused by this herbal medicine.

**Key Words:** Tachycardia, ventricular, Herbal medicine, *Ephedra sinica*

**Introduction**

Ephedra alkaloids are a group of sympathomimetic compounds derived from the shrubs of the *Ephedra* genus. These alkaloids are structurally similar to amphetamines and catecholamines, and possess both α- and β-adrenergic activity. Ephedra alkaloids have been used in traditional folk remedies, particularly in China, for millennia. The principle alkaloid ephedrine was first isolated in 1885, and has been used in modern medicine as a bronchodilator, decongestant, and vasopressor. Ephedra extracts are also commonly taken to enhance weight loss and athletic performance.

Ephedra-containing dietary supplements were banned by the American Food and Drug Administration in 2004 because of significant adverse effects. Between 1995 and 1997, 37 cases of adverse effects were reported and predominantly involved serious cardiovascular events (e.g., stroke, myocardial infarction, sudden cardiac death), despite no underlying cardiovascular disease.

Here, I report a case of ventricular tachycardia (VT) with two distinct morphologies caused by the consumption of herbal medicine containing *Ephedra sinica* (Ma Huang).

**Case**

A 77-year-old man presented to the emergency department with a fluttering sensation in the chest and sudden onset dyspnea. Electrocardiography showed sustained monomorphic VT (208 bpm), a right bundle branch block (RBBB) pattern in the precordial leads, and left axis deviation. Biphasic electrical cardioversion (200 J) successfully terminated the VT through conversion to atrial fibrillation.
Figure 1. Twelve-lead electrocardiograms (ECG).
(A) The initial ECG shows fast monomorphic ventricular tachycardia (208 bpm), a right bundle branch block in the precordial leads, and left axis deviation. VT was inferred based on the interpretation of the ECG with the captured beat (arrow).
(B) Atrial fibrillation is observed following electrical cardioversion.
(C) The ECG of the second VT (210 bpm) shows a left bundle branch block pattern and superior axis deviation.
(D) The ECG shows restored sinus rhythm after additional electrical cardioversion.
(210 bpm) developed spontaneously, with a QRS morphology that was indicative of left BBB (LBBB) and superior axis deviation (Figure. 1C). Biphasic electrical cardioversion (200 J) was reattempted, and sinus rhythm was successfully restored (Figure. 1D).

After the treatment, the possible causes of the VT were considered, including anemia, electrolyte imbalance, hypoxemia, coronary artery disease, cardiomyopathy, congenital heart disease, or drug effects. Laboratory result revealed normal serum creatinine levels, electrolyte levels, and arterial blood gases. Similarly, ECG showed a normal left ventricular ejection fraction (65%) with no significant valve degeneration or structural abnormalities. Although coronary angiography revealed a stenosis of 70% in both the proximal part of the left circumflex coronary artery and the posterior descending artery, I decided to administer pharmacologic treatment based on the presence of normal blood flow and absence of angina.

The patient had been diagnosed with chronic obstructive pulmonary disease 30 years prior to admission. He had a history of smoking (one half-pack per day for 50 years) but had ceased this habit one year previously. The current medication included methylprednisolone (2 mg/day) and the β2-agonist fenoterol (2.5 mg/day). The patient reported no history of alcohol or intravenous drug abuse, and had no remarkable family history of cardiovascular disease or sudden cardiac death. He reported taking herbal medicine containing E. sinica over the course of two days (total four doses) for abdominal discomfort and indigestion. I concluded that the consumption of the herbal medicine was the cause of the VT with two distinct QRS morphologies.

The patient was prescribed clopidogrel, warfarin, an angiotensin-converting enzyme inhibitor, a β-blocker, and a statin, and was advised against resuming the herbal medication prior to discharge. No recurrences of VT occurred during more than 6 months of follow-up.

Discussion

Ephedrine and related alkaloids have been associated with numerous adverse cardiovascular events, including acute myocardial infarction, severe hypertension, myocarditis, and lethal cardiac arrhythmias. This development of adverse cardiovascular events is largely attributable to the sympathomimetic activity of these alkaloids. That is, these compounds directly stimulate α1, β1, and β2 adrenergic receptors and can thereby result in vasoconstriction, bronchodilation, increased heart rate, enhanced myocardial contractility, and increased automaticity. Such compounds also shorten cardiac refractory periods and can therefore facilitate the development of cardiac arrhythmogenesis. The long-term use of ephedrine may lead to the cardiomyopathy typically observed during catecholamine excess.

To date, there has been no report of VT with two distinct QRS morphologies (RBBB and LBBB) caused by E. sinica in patients. The present case is also distinct from previously reported cases of VT in several additional respects: 1) the sustained distinct monomorphic VTs originated from different ventricles; 2) the patient had taken unknown but small doses of E. sinica (four in total); and 3) the ingestion of the herbal medication had been of a relatively short duration (two days).

Recently, the molecular mechanisms underlying E. sinica activity have been demonstrated, in vivo, to involve the activation of the slowly activating KCNQ1 potassium channel and other members of the KCNE protein family. Although this activation could be expected to precipitate ventricular arrhythmia, further investigation is required to confirm this. Nonetheless, because even short-term, low-dose intake of E. sinica is associated with the risk of life-threatening ventricular arrhythmias, its use should be closely monitored and tightly regulated.

References


