

# Bidirectional tachycardia in a patient with pulmonary embolism

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## Abstract

*We report a 55 year-old man with sudden cardiac arrest. Electrocardiography revealed runs of bidirectional ventricular tachycardia, and transthoracic echocardiography showed indirect findings of pulmonary embolism. (Cardiol J 2010; 17, 2: 194–195)*

**Key words: bidirectional tachycardia, pulmonary embolism**

## Introduction

Bidirectional ventricular tachycardia (BVT) is an uncommon type of ventricular tachycardia with atypical right bundle branch block pattern in right precordial leads and alternating polarity of the QRS complex in frontal plane leads [1].

Bidirectional ventricular tachycardia is most commonly linked to digitalis toxicity, aconite poisoning, familial hypokalemic periodic paralysis, catecholaminergic polymorphic ventricular tachycardia, ischemic heart disease, and myocarditis [1–4]. To the best of our knowledge, this case is the first report of the coexistence of BVT with acute pulmonary embolism.

## Case report

A 55 year-old man presented with sudden cardiac arrest. Upon arrival in the emergency room, his blood pressure was 90/60 mm Hg, his heart rate was 125/min, and his respiratory rate was 24/min. His serum electrolytes (sodium and potassium) were normal. He had no history of digitalis or other herbal poison ingestion or of a similar cardiac problem or sudden cardiac death in his family. The electrocardiography revealed tachycardia with a borderline narrow QRS complex of 115 ms, an alternating left-and right-axis deviation in the

frontal plane leading to the bidirectional appearance in the limb leads (Fig. 1). An emergency bedside echocardiogram showed right ventricle dilatation, mild tricuspid regurgitation and a peak tricuspid regurgitation pressure gradient of 40 mm Hg, with paradoxical motion of the interventricular septum. D-dimer level, from a blood sample taken during the arrest, was grossly elevated at 1092  $\mu\text{g/L}$  (normal < 200  $\mu\text{g/L}$ ). Thorax computerized tomography or pulmonary angiography for the diagnosis of pulmonary embolism was not done owing to the hemodynamic instability of the patient.

At this point, pulmonary embolism was suspected and emergency 20 mg boluses of recombinant tissue type plasminogen activator were administered via femoral vein, followed by an infusion of 90 mg over one hour. Serial troponin I measurements were within normal range. However, the patient died after two hours.

## Discussion

Bidirectional ventricular tachycardia has been described in a variety of clinical settings, including digitalis toxicity, herbal aconite poisoning, catecholaminergic polymorphic ventricular tachycardia, coronary artery disease, and structurally normal hearts [1–4]. A negative history for intake of digitalis and herbal poison ruled out drug toxicity as

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**Figure 1.** Bidirectional tachycardia at 125 bpm.

possible causes of BVT. Normokalemia and normal troponin I levels removed electrolyte abnormality and ischemic heart disease from the differential diagnosis. In the present case, BVT was most likely due to pulmonary embolism.

Bidirectional ventricular tachycardia is not a homogeneous clinical syndrome and therefore may have more than one mechanism. However, automaticity or triggered activity is more often implicated than reentry [2].

The correction of reversible factors (e.g. heart failure, digoxin excess, electrolyte abnormalities) is an important part of the treatment of BVT as in our patient.

As a temporary measure for patients without digoxin toxicity and with sustained hemodynamically compromising BVT, antiarrhythmic drugs may be used. Lidocaine is a reasonable drug as a first choice [5]. Quinidine and flecainide can be used, if lidocaine is ineffective in this patient [6]. Other treatments for tachyarrhythmias include antitachycardia pacing and electrical cardioversion.

Bidirectional ventricular tachycardia is commonly described as a tachycardia with alternating QRS axis in the frontal plane on a beat-to-beat basis. However, there are at least three cases in which the bidirectional morphology of the tachycardia was caused by alternating right and left bundle branch block type pattern [7].

To our knowledge, this is the first report of BVT most likely due to pulmonary embolism. Our case strengthens the need for a thorough search for

pulmonary embolism, if there is no other obvious cause of ventricular arrhythmias.

The author of this manuscript has certified that he complies with the Principles of Ethical Publishing in the International Journal of Cardiology [8].

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