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Myopericarditis – a rare cause of bidirectional ventricular tachycardia

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Abstract

Bidirectional ventricular tachycardia is a rare dysrhythmia. It is a tachycardia with morphologically diverse presentations. We report a patient with bidirectional tachycardia secondary to fulminant myopericarditis.

Key words: bidirectional ventricular tachycardia, fulminant myopericarditis, ventricular tachycardia

Introduction

Bidirectional ventricular tachycardia is an unusual ventricular dysrhythmia with beat-to-beat fluctuation of the frontal QRS axis.[1] Severe digoxin toxicity is the most common cause identified for a bidirectional tachycardia.[2] Patients with familial catecholaminergic polymorphic ventricular tachycardia, a much less common disorder, can also present with bidirectional ventricular tachycardia.[2] Herbal aconite poisoning which occurs after ingestion of Chinese herbal medicine, acute ischemia, pulmonary embolism, familial hypokalemic periodic paralysis, fatty replacement in right ventricle, cardiac tumours, myocarditis, noncompaction cardiomyopathy, sarcoidosis, KCNJ2 gene mutation are also causes of bidirectional ventricular tachycardia.

Case history

A 19-year-old, previously healthy girl presented with shortness of breath associated with palpitations. She had an acute diarrhoeal illness associated with vomiting and fever for 2 days duration. She was not on any long-term medications. On examination, she had clinical features of congestive heart failure. Her blood pressure was 110/70 mmHg, pulse rate was 90 bpm with SpO2 of 98% on room air. A 12-lead electrocardiogram revealed a bidirectional ventricular tachycardia (Figure 1). Transthoracic echocardiography demonstrated global hypokinesia with moderate left ventricular dysfunction (ejection fraction of 35%), and a thin rim of pericardial effusion. Troponin I was elevated at 19.5 ng/mL. The clinical picture and the investigations pointed towards a diagnosis of myopericarditis. Limited laboratory facilities prevented us from obtaining a microbiological confirmation of the fastidious organism. Her full blood count showed a white cell count of 13.3 10^9/L with 78% of neutrophils, haemoglobin of 11.5 g/dL and platelet count of 230×10^9/L. C-reactive protein (CRP) was 45.8 mg/L and ESR was 44 mm/1st hour. Aspartate and alanine aminotransferase were 362 U/L and 424 U/L respectively. Renal functions and serum electrolytes were normal. Blood culture showed no growth. As the patient remained hemodynamically stable a decision was made to initially manage the arrhythmia conservatively by close monitoring and the rhythm spontaneously reverted to a sinus rhythm in 4 hours.

Echocardiography after a week showed mildly impaired left ventricular function with an ejection fraction of 45% and a repeated scan performed after a month showed preserved left ventricular function. Although an exact bacterial aetiology is not known, the atypical bacterial infection is suspected to be the culprit for this condition.

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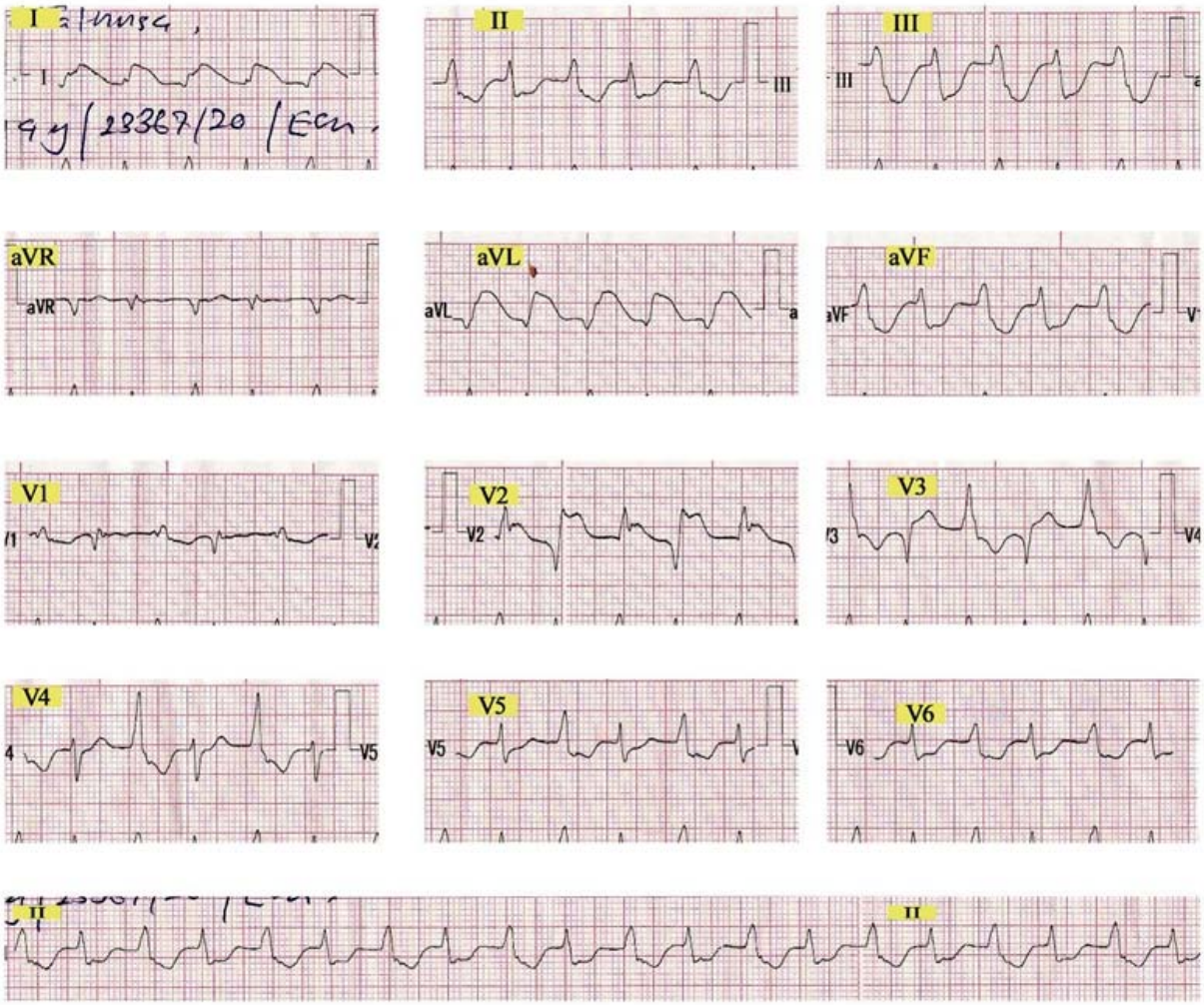

Figure 1. Bidirectional tachycardia.

Discussion

Ventricular tachycardia (VT) is a form of broad complex dysrhythmia originating from ventricles. Ventricular tachycardia is defined as a tachycardia of >100 beats per minute, with ≥ 3 successive beats originating from the ventricles. These beats are independent of atria or atrioventricular nodal conduction. In bidirectional VT there is an alternating frontal QRS axis in opposite direction.

Delayed after depolarization (DAD) developing at different heart rate thresholds is the mechanism described for bidirectional ventricular tachycardia.[3] It usually occurs in two different sites in the distal His-Purkinje system or ventricles. This leads to a ping-pong mechanism that causes reciprocating bigeminy between these two foci of ventricles.

Bidirectional ventricular tachycardia is mainly seen in digoxin toxicity. An inherited condition called catecholaminergic polymorphic ventricular tachycardia (CPVT) usually presents with bidirectional tachycardia.[2] It is a cardiac channelopathy inherited in an autosomal dominant fashion. Andersen-Tawil syndrome is an inherited condition responsible for bidirectional tachycardia.[4] The accelerated idioventricular rhythm which develops with the myocardial infarction, mainly in reperfusion, can cause bidirectional tachycardia.[4] Myocarditis is a rare cause of bidirectional ventricular tachycardia.

Treatment of bidirectional ventricular tachycardia consists of cardioversion and treating underlying causes such as reversing underlying causative agents (e.g.: digoxin toxicity). Among the antiarrhythmic agents, lidocaine is a superior antiarrhythmic treatment option for bidirectional ventricular tachycardia.[3] Lidocaine can inhibit DAD related mechanisms by blocking voltage-gated sodium channels.

Conclusion

Bidirectional ventricular tachycardia is a rare variety of VT. Myopericarditis can rarely be the cause of bidirectional ventricular tachycardia. If the patient is stable watchful waiting can be the initial strategy of management.

Consent

Informed written consent was obtained from the patient for publication of this case report.

Conflicts of interest

The authors declare that there are no conflicts of interest.

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