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Case Report

Polymorphic ventricular tachycardia in a patient with hypertrophic cardiomyopathy and digitalis intoxication

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SUMMARY

We report the case of a 74-year-old woman who presented with recurrent episodes of polymorphic ventricular tachycardia (PVT) with a normal QT interval due to digitalis intoxication (serum digoxin concentration, 5.0 ng/mL) and severe hyperkalemia (serum potassium level, 8.3 mEq/L). In addition, laboratory data showed elevated levels of blood urea nitrogen (54 mg/dL) and serum creatinine (1.57 mg/dL), suggesting dehydration. She had been treated with a combination of digoxin and eplerenone for atrial fibrillation and heart failure. The PVT resolved after treatment for hyperkalemia. Cardiac magnetic resonance imaging and left ventriculography showed left ventricular hypertrophy predominantly in the apex, suggesting apical hypertrophic cardiomyopathy (HCM). We presume that the presence of HCM was related to the occurrence of PVT in this patient with digitalis intoxication and hyperkalemia.
<**Learning objective:** PVT with a normal QT interval caused by digitalis intoxication with hyperkalemia was observed in a patient with HCM treated with digoxin and eplerenone for atrial fibrillation and heart failure. The presence of HCM may be related to the occurrence of PVT. Combination therapy with digoxin and aldosterone receptor antagonist may predispose severe hyperkalemia, and monitoring of serum digitalis concentration and potassium level should be done strictly.>

Introduction

Polymorphic ventricular tachycardia (PVT) is a life-threatening form of arrhythmia that may be associated with prolonged or normal QT intervals. PVT with a normal QT interval is most frequently observed in acute myocardial ischemia or infarction but may also be seen in other structural heart diseases such as cardiomyopathy [1,2] or in the absence of organic heart disease (e.g. Brugada syndrome [3], catecholaminergic PVT [4], or the short-coupled variant of torsade de pointes [5]). Severe digitalis intoxication causes hyperkalemia and malignant ventricular arrhythmia, necessitating prompt diagnosis and treatment [6–9]. Selective aldosterone antagonism with eplerenone in patients with heart failure is clearly beneficial but is associated with an increased risk of hyperkalemia [10]. Here, we present a case of PVT with a normal QT interval in the setting of digitalis intoxication and hyperkalemia during combination therapy with digoxin and eplerenone in a patient with hypertrophic cardiomyopathy (HCM).

Case report

A 74-year-old woman with a history of type 2 diabetes mellitus treated with insulin presented to our hospital with complaints of fainting and vomiting after a 2-day history of nausea and anorexia. She had been treated for permanent atrial fibrillation and heart failure with digoxin (0.25 mg/day) and furosemide (40 mg/day) for12 years. Because her heart failure had not been well controlled and she had frequently needed hospitalization due to the exacerbation of symptoms, eplerenone (25 mg/day) was added to the treatment regimen 16 months before admission. Four months before admission, her serum creatinine level was within normal limits (0.76 mg/dL), and her estimated glomerular filtration rate (eGFR) was mildly decreased (56.1 mL $min^{-1} \times 1.73\ m^{-2}$). Her serum potassium level was normal (4.8 mEq/L) at that time. The patient had no family history of heart disease or sudden cardiac death.

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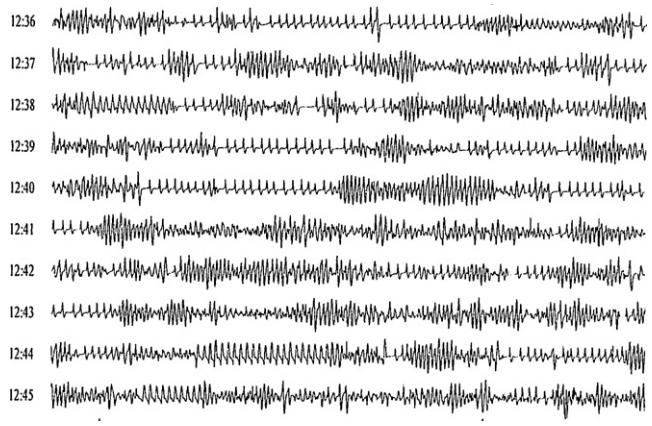

Figure 1. Electrocardiographic monitoring showing recurrent episodes of polymorphic ventricular tachycardia. Periods of accelerated idioventricular rhythm are also observed.

On admission, the patient's height was 142 cm and her body weight was 50 kg. Her pulse was weak and irregular, and her blood pressure could not be measured. Electrocardiographic (ECG) monitoring showed recurrent episodes of PVT; periods of accelerated idioventricular rhythm (AIVR) were also observed (Figs. 1 and 2a). Twelve-lead ECG performed between episodes of tachycardia showed that the QRS complex was widened, but the QT interval was not prolonged (Fig. 2b). Laboratory assessment showed marked hyperkalemia (serum potassium level, 8.3 mEq/L) and elevated levels of blood urea nitrogen (54 mg/dL) and serum creatinine (1.57 mg/dL). The calculated transtubular potassium gradient was low (3.5; normal range, 8–9), suggesting hypoaldosteronism. The patient was diagnosed with PVT complicated with digitalis intoxication and hyperkalemia induced by dehydration; high serum digoxin concentration was confirmed later (5.0 ng/mL; therapeutic range, 0.8–2.0). Digoxin and eplerenone were discontinued, and intravenous administration of lidocaine was initially used because myocardial ischemia

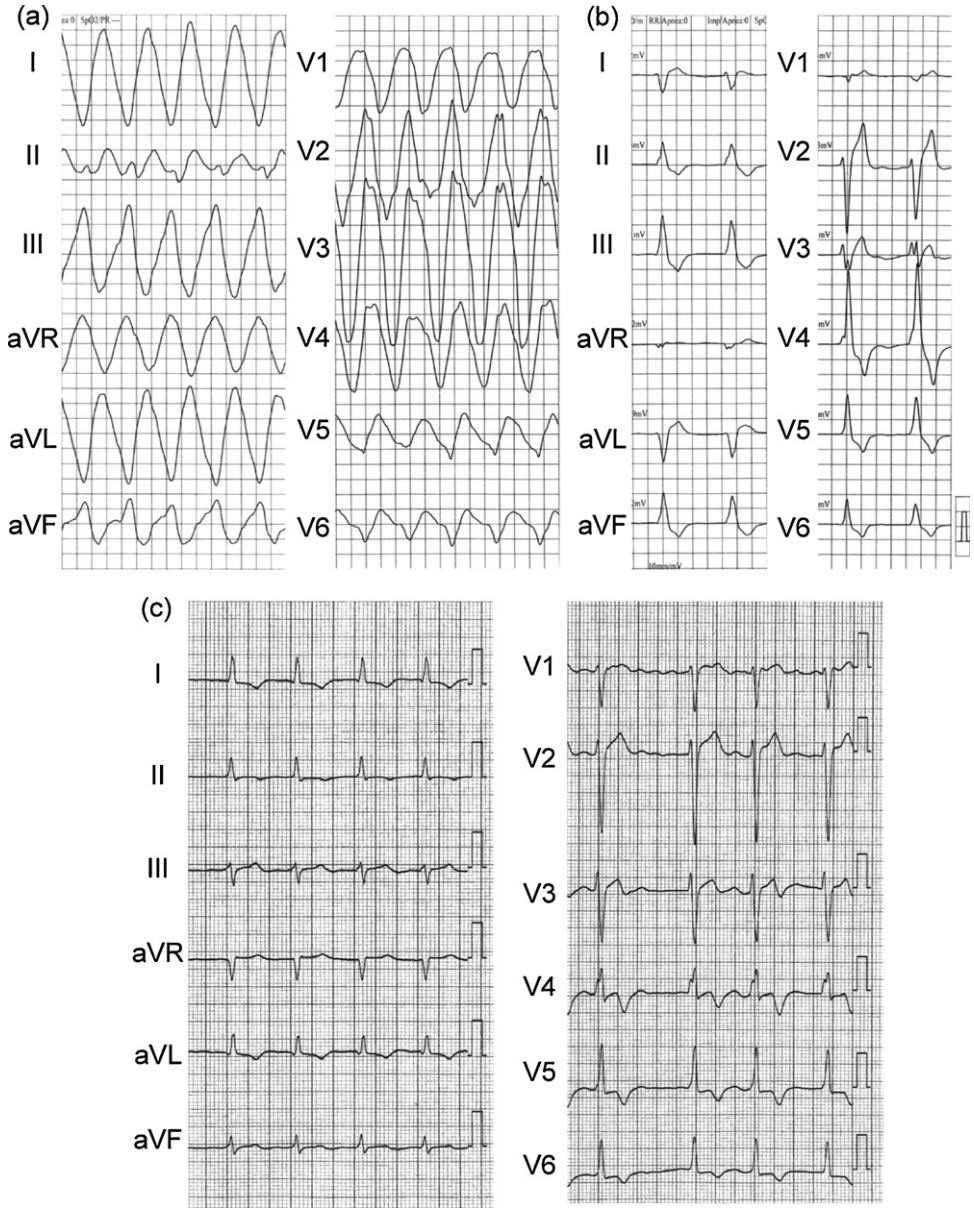

Figure 2. (a) Electrocardiographic monitoring (ECG) showing accelerated idioventricular rhythm. (b) ECG performed between episodes of ventricular tachycardia showing prolonged QRS complexes and normal QT intervals. (c) ECG performed on the fifth day after admission showing atrial fibrillation and negative T waves in I, aVL, and V4-6 leads.