Case Report

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

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S U M M A R Y

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.>

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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) for 12 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 × 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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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 1. Electrocardiographic monitoring showing recurrent episodes of polymorphic ventricular tachycardia. Periods of accelerated idioventricular rhythm are also observed.

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.
was not completely denied as the cause of PVT, but it was discontinued soon after because it was not effective for the suppression of PVT. We treated hyperkalemia with glucose-insulin solution (14 units of regular insulin in 500 mL of 10% glucose), intravenous sodium bicarbonate (24 mEq), and K⁺-exchange resin (calcium polystyrene sulfonate) administered orally (3 g) and rectally (30 g). Four hours later, the patient’s serum potassium level decreased to 6.3 mEq/L, and her PVT resolved. On the next day, her serum potassium and digoxin levels decreased to 5.3 mEq/L and 2.3 ng/mL, respectively, and PVT did not recur.

ECG performed on the fifth day after admission showed atrial fibrillation and negative T waves in I, aVL, and V4-6 leads (Fig. 2c). Echocardiography showed asymmetric septal hypertrophy with no evidence of left ventricular outflow tract obstruction; however, apical hypertrophy could not be confirmed. Cardiac magnetic resonance imaging showed left ventricular hypertrophy predominantly in the apex (Fig. 3a–d). Coronary angiography did not show organic stenosis, and left ventriculography showed a spade-like configuration, suggesting apical HCM (Fig. 3e and f).

Discussion

We report a case of PVT with a normal QT interval caused by digitalis intoxication with severe hyperkalemia in a patient with HCM. Digitalis intoxication causes hyperkalemia by inhibiting Na⁺/K⁺-ATPase, thereby shifting potassium to the extracellular compartment [8,9]. Potassium replacement is effective for the treatment of digitalis intoxication with hypokalemia. In contrast, digitalis intoxication with hyperkalemia or life-threatening arrhythmia can be treated with digoxin-specific Fab antibody fragments [11]. Because digoxin-specific Fab antibody fragments are not available in Japan, we treated our patient with lidocaine and by lowering serum potassium levels. Recent guidelines indicate that lidocaine has a class IIb recommendation for the treatment of PVT associated with acute myocardial ischemia or infarction and a class III recommendation for digitalis toxicity [1]. Because acute myocardial ischemia cannot be excluded in this patient, we used lidocaine, which was not effective for the suppression of PVT. Dyckner et al. reported a necropsy case of fatal digitalis intoxication in which an intracellular potassium deficit in the cardiac muscle was confirmed [12]. Therefore, we speculated that shifting potassium to the intracellular compartment with glucose-insulin therapy would potentially be effective for the suppression of PVT in this patient.

Digitalis intoxication causes various ventricular arrhythmias such as ventricular premature complexes, ventricular tachycardia, and ventricular fibrillation; moreover, bidirectional ventricular tachycardia is particularly characteristic of digitalis intoxication [6,7]. To the best of our knowledge, this is the first report of PVT complicated with digitalis intoxication in a patient with HCM. Although digitalis intoxication causes various arrhythmias, ventricular tachyarrhythmias are uncommon in patients with a healthy heart and are usually observed in older patients with underlying heart disease [6,7]. This observation may be due to the exacerbation of enhanced Purkinje fiber automaticity by ischemia, fiber stretch, or other injuries [6]. Watson et al. speculated that inducible PVT in patients with HCM might be related to the underlying myocardial architecture, characterized by myocardial cellular disarray and fibrosis [13]. Therefore, we suppose that the occurrence of PVT in this patient was due to the underlying HCM. In addition to PVT, AIVR was observed in this patient. When AIVR is associated with digitalis intoxication, the main mechanism involved is triggered activity [14]. Although ventricular arrhythmias of digitalis intoxication are primarily due to enhanced automaticity and triggered activity [6], digitalis can also predispose reentry by shortening the effective refractory period of the ventricular muscle [15]. We speculate that reentry with an arrhythmogenic substrate in HCM was the mechanism of PVT in this patient; however, enhanced automaticity and triggered activity are other possible mechanisms.

Although spironolactone can decrease digoxin clearance and increase serum digoxin concentration, eplerenone has been
reported to have no significant pharmacokinetic interaction with digoxin [16,17]. However, combination therapy with digoxin and an aldosterone receptor antagonist may predispose the patient to life-threatening hyperkalemia. Although our patient’s serum creatinine and potassium levels were within normal limits 4 months before admission, digitalis intoxication with life-threatening hyperkalemia developed during combination therapy with digoxin and eplerenone. We speculate that the maintenance dose of digoxin (0.25 mg/day) may have been too high for this patient because of her mild renal dysfunction and advanced age.

Conclusion

We have reported a case of PVT complicated with digitalis intoxication and severe hyperkalemia during combination therapy with digoxin and eplerenone in a patient with HCM. This patient was successfully treated by lowering serum potassium levels. Careful monitoring of serum potassium levels is highly recommended in elderly patients treated with digoxin and eplerenone, especially in patients with an underlying myocardial disease such as HCM.

References


