Journal of Cardiology Cases xxx (2017) xxx–xxx

Contents lists available at ScienceDirect

Journal of Cardiology Cases

journal homepage: www.elsevier.com/locate/jccase

Case Report

A case of life-threatening supraventricular tachycardia storm associated with theophylline toxicity

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ARTICLE INFO

Article history:
Received 4 November 2016
Received in revised form 6 December 2016
Accepted 11 December 2016

Keywords:
Supraventricular tachycardia storm
Theophylline toxicity
Mechanical circulatory support

ABSTRACT

A 76-year-old man taking theophylline was admitted to our hospital with congestive heart failure and supraventricular tachycardia (SVT). After admission, he developed cardiogenic shock as a result of SVT storm, which was refractory to medical treatment including adenosine and electrical cardioversion. The serum theophylline concentration at admission was identified as toxic. Therefore, theophylline toxicity was considered as a major cause of the SVT storm. Hemodynamic stability was achieved by using mechanical circulatory support. Additionally, continuous hemodiafiltration was performed to remove theophylline, and it was effective for suppression of SVT. The patient was successfully weaned off mechanical circulatory support. After the patient's general status had improved, an electrophysiological study was performed, and it showed orthodromic atrioventricular reentrant tachycardia with a right free wall accessory pathway. Radiofrequency catheter ablation was successfully performed.

<**Learning objective:** SVT is often hemodynamically stable and medically well-controllable with adenosine. However, SVT is occasionally refractory or life-threatening under specific conditions such as theophylline toxicity, since theophylline has an inhibitor effect on adenosine. Mechanical circulatory support should be used in case of life-threatening SVT storm associated with theophylline toxicity.>

Introduction

Theophylline is widely used for management of patients with chronic obstructive pulmonary disease and bronchial asthma. However, in some cases, theophylline toxicity causes major complications because of its narrow therapeutic window [1]. Cardiac arrhythmia, including supraventricular tachycardia (SVT) and multifocal atrial tachycardia, is one of the complications of theophylline toxicity [2]. In most cases, supraventricular arrhythmias are hemodynamically stable and well-controllable with medical treatment. However, in few cases, mechanical circulatory support was needed to control such arrhythmias, which are refractory to medical therapy [3,4]. We describe here a case of life-threatening SVT storm associated with theophylline toxicity, in which mechanical support, as well as continuous hemodiafiltration (CHDF), was effective.

Case report

A 76-year-old man with a history of smoking was referred to our hospital with worsening dyspnea. He had been taking medication for chronic obstructive pulmonary disease for 3 months, including theophylline (200 mg twice a day), long-acting muscarinic antagonists, and a long-acting beta-agonist inhaler. On arrival at hospital, his blood pressure was 121/63 mmHg, pulse was 192 bpm, breathing rate was 30/min, and body temperature was 36.3 °C. An electrocardiogram showed SVT with a ventricular rate of 211 bpm (Fig. 1). In the past, he had never experienced heart palpitation. A laboratory examination showed an elevation of N-terminal pro-brain natriuretic peptide concentration (17,310 pg/mL). A chest X-ray demonstrated acute pulmonary

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http://dx.doi.org/10.1016/j.jccase.2016.12.004
1878-5409/

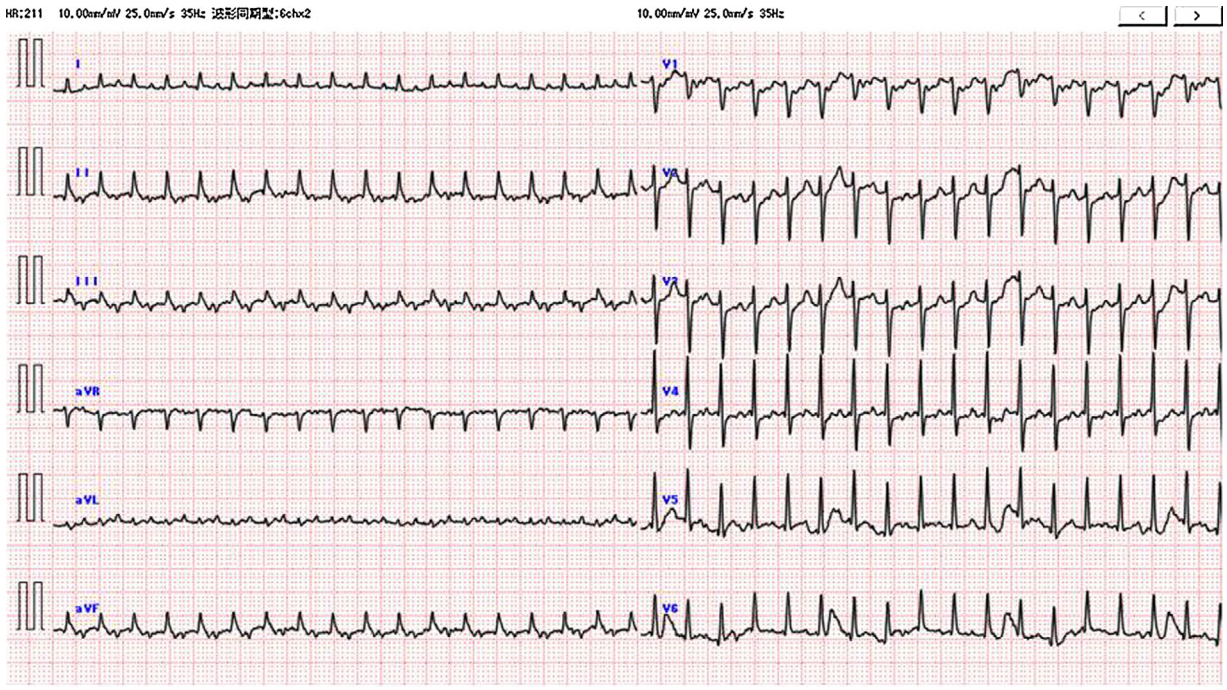

Fig. 1. Electrocardiogram on admission. An electrocardiogram shows regular tachycardia of a narrow QRS complex at 211 bpm.

edema with cardiac enlargement (Fig. 2A). A transthoracic echocardiogram revealed global severe hypokinesis of the left ventricle (LV) with a reduced left ventricular ejection fraction (LVEF) of 20%. The LV end-diastolic diameter was 54 mm and neither major valvular disease nor pericardial effusion was found. At the emergency room, intravenous adenosine (20 mg) was repetitively administered to terminate his regular, short RP' tachycardia (Fig. 1). However, there was no response to adenosine, and SVT was not terminated.

After admission, SVT continued, and his systolic blood pressure gradually decreased to 60 mmHg. Electrocardiographic monitoring showed that there were frequent, sporadic atrial extra-systoles and SVT was initiated just after these atrial extra-systoles (Fig. 2B). Tachyarrhythmia was temporarily converted to sinus rhythm by electrical cardioversion. However, SVT recurred soon after electrical cardioversion. Even with intravenous infusion of amiodarone, sinus rhythm was difficult to maintain after electrical cardioversion, and SVT restarted immediately after sporadic atrial extra-systoles. The serum theophylline concentration at admission was 38.5 μg/mL, which was toxic (normal therapeutic range: 10–20 μg/mL). Therefore, theophylline toxicity was considered as a major cause of SVT storm.

A blood test at 15 h after admission showed multi-organ dysfunction with acute renal failure (serum creatinine level, 1.55 mg/dL) and acute hepatic dysfunction (aspartate aminotransferase, 8106 U/L; alanine aminotransferase, 1626 U/L). Arterial blood gas analysis under 10 L/min oxygen by mask showed metabolic acidosis with a pH of 6.977, PCO_2 of 40.6 mmHg, PO_2 of 85.5 mmHg, and lactic acid level of 18 mmol/L. We decided to use mechanical circulatory support to control the life-threatening SVT storm. Right heart catheterization prior to mechanical circulatory support showed right atrial pressure of 12 mmHg, mean pulmonary artery pressure of 40 mmHg, pulmonary capillary wedge pressure of 30 mmHg, cardiac output 2.4 L/min, and cardiac index 1.4 L/min per m^2. Percutaneous cardiopulmonary support (PCPS) was initiated with percutaneous cannulation of the left femoral artery and vein, and an intra-aortic balloon pump was also placed via the right femoral artery. CHDF was subsequently initiated to remove the theophylline. Metabolic abnormalities and theophylline toxicity improved once PCPS flow and CHDF were established. A total of 20 h after CHDF initiation, the theophylline concentration decreased to the normal therapeutic range (8.2 μg/mL). After achieving hemodynamic stability, an intravenous verapamil infusion could be initiated, which was also effective for suppression of SVT. Under mechanical circulatory support, we could maintain sinus rhythm, and sporadic atrial extra-systoles disappeared (Fig. 3). It was a possible option to perform catheter ablation at the time of PCPS introduction. However, the SVT could be controlled by combined modality therapy fortunately, we decided to perform elective catheter ablation. PCPS and CHDF were successfully removed after 3 days. On the 16th day, echocardiography showed that LVEF had improved to 41%. This finding suggested that his reduced LVEF was tachycardia-induced cardiomyopathy, and he had asymptomatic tachyarrhythmia associated with theophylline toxicity in a chronic course.

On the 36th day, cardiac catheterization, including coronary angiography and an electrophysiological study, was performed after the patient's general status had improved. Coronary angiography showed no significant stenosis in major coronary arteries. An electrophysiological study (EPS) showed retrograde ventriculoatrial conduction via the accessory pathway in the lateral tricuspid annulus and provoked atrioventricular reentrant tachycardia with this accessory pathway (Fig. 3). During the procedure, the SVT was terminated with a bolus of intravenous adenosine 10 mg. Radiofrequency catheter ablation for this accessory pathway was successfully performed, and any tachycardia could not be induced. His general status fully recovered, and he was then discharged on day 46. After discharge, sinus rhythm was maintained for 5 months. Finally, LVEF improved to 63% and the NT-pro BNP level decreased to 99 pg/mL.

Discussion

We describe here a rare case of life-threatening SVT storm caused by theophylline toxicity that resulted in tachycardia-induced cardiomyopathy with cardiovascular collapse. Mechanical

Please cite this article in press as: Ichikawa K, et al. A case of life-threatening supraventricular tachycardia storm associated with theophylline toxicity. J Cardiol Cases (2017), http://dx.doi.org/10.1016/j.jccase.2016.12.004