Clinical Case Report

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Aggravation of atrial arrhythmia by amiodarone during the perinatal period

A case report

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Abstract

Rationale: Amiodarone, a broad-spectrum antiarrhythmic drug, is widely used for the clinical treatment of tachyarrhythmias because of its safety and efficacy.

Patient concerns: A 30-year-old woman presented with known paroxysmal atrial tachycardia and severe preeclampsia. Two days before admission, she had given birth to twins. She described her symptoms as a sudden palpitation at 10:20 accompanied by chest tightness and shortness of breath. Diagnosis: Cardiac arrhythmia and acute left heart failure.

Interventions: Furosemide and sodium nitroprusside were administered to control the heart failure. At 16:20, 150 mg amiodarone (15 mg/min) was injected intravenously and continued at 1 mg/min. At 16:50, her electrocardiogram showed possible atrial tachycardia or atrial flutter with a ventricular rate of 206 beats/min. Administration of amiodarone was stopped at 17:23, and the medication was changed to esmolol.

Outcomes: After 3 minutes, the palpitations stopped, the heart rate changed to a sinus rhythm, and the ventricular rate was 100 beats/min. Four days later, the patient underwent an electrophysiologic study and radiofrequency ablation.

Lessons: When amiodarone is used to treat atrial arrhythmia, the ventricular rate may accelerate, which can cause patients with borderline heart failure to develop acute heart failure or further deterioration of acute heart failure. For heart failure induced or mediated by atrial arrhythmias, short-term β-blockers may be used to control the ventricular rate more quickly and effectively and to prevent the progression of heart failure.

Abbreviations: AAD = antiarrhythmic drug, ECG = electrocardiogram, EPS = electrophysiologic study, HD-HDP = heart disease related to hypertensive disorders in pregnancy, HF = heart failure, LVEDD = left ventricular end-diastolic dimension, LVEF = left ventricular ejection fraction, LVESD = left ventricular end-systolic dimension, TTE = transthoracic echocardiography.

Keywords: amiodarone, atrial arrhythmia, electrophysiologic study, heart failure

1. Introduction

Amiodarone, a broad-spectrum antiarrhythmic drug (AAD), is widely used in the clinical treatment of tachyarrhythmias because of its safety and efficacy.[1] Its clinical efficacy in patients with acute multifocal atrial tachycardia, atrial fibrillation, and atrial flutter has been confirmed.[2–5] Regarding atrial arrhythmias, there has been no report of amiodarone accelerating the ventricular rate. We report a case of failed amiodarone treatment of perinatal atrial arrhythmia in a postpartum woman, resulting in an accelerated ventricular rate and further aggravation of acute heart failure (HF). We discuss the mechanisms by which ventricular rate changes may occur.

1.1. Ethical statement and consent

This study was approved by the Ethics Committee of Wenzhou People's Hospital. The clinical and imaging data were obtained with the patient's consent for the publication of this report.

2. Case report

A 30-year-old woman presented with known paroxysmal atrial tachycardia and severe preeclampsia. Two days before admission, she had given birth to twins. Her prenatal electrocardiogram (ECG) revealed a sinus rhythm, and transthoracic echocardiography (TTE) revealed a left ventricular end-diastolic dimension (LVEDD) of 53 mm, a left ventricular end-systolic dimension (LVESD) of 35 mm, and a left ventricular ejection fraction (LVEF) of 62%. She described her symptoms as a sudden palpitation at 10:20, accompanied by chest tightness and shortness of breath. On admission examination, her heart rate was 150 beats/min, her blood pressure was 161/113 mm Hg, and her SpO_2 was 90% (nasal tube 5 L/min oxygen inhalation). Patches of moist rales were heard over her lung fields. The heart sounds were regular, and no murmur was detected. At 16:02, the ECG showed possible atrial tachycardia or atrial flutter with a ventricular rate of 150 beats/min (Fig. 1). She was diagnosed with

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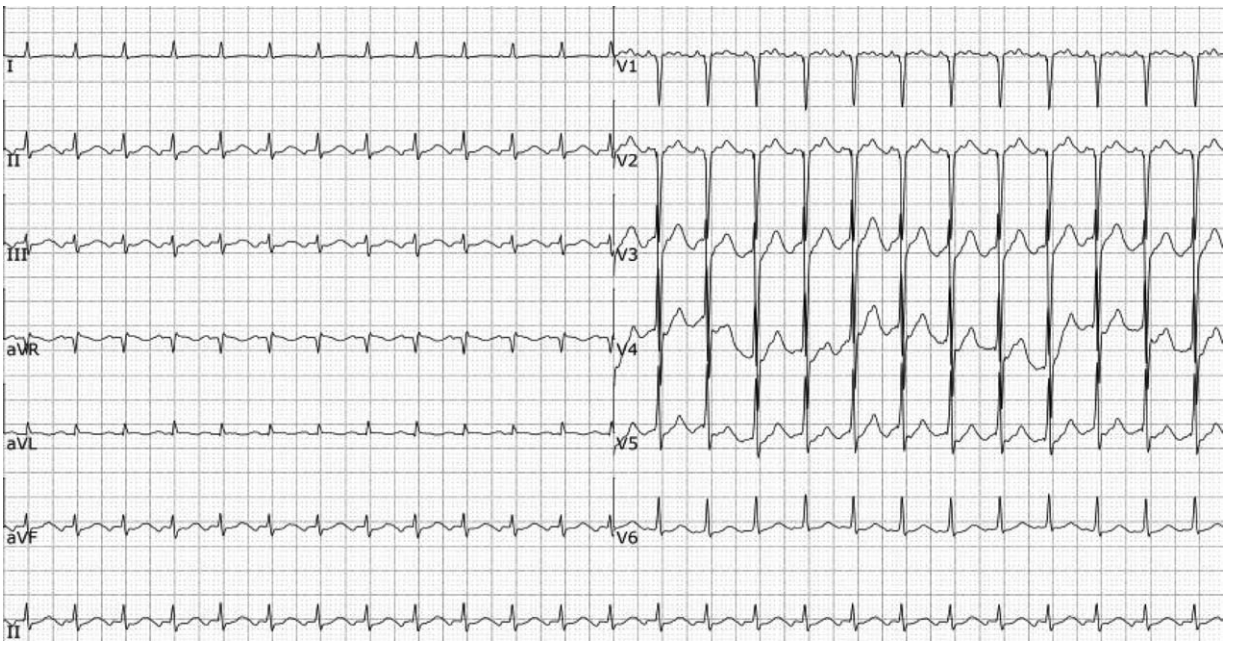

Figure 1. Electrocardiogram at 16:02, ventricular rate 150 bpm.

cardiac arrhythmia and acute left HF. Furosemide and sodium nitroprusside were used to control the HF. At 16:20, amiodarone 150 mg (15 mg/min) was injected intravenously and was continued at 1 mg/min. At 16:50, her palpitations became aggravated, and her heart rate was 206 beats/min. The ECG showed possible atrial tachycardia or atrial flutter with a ventricular rate of 206 beats/min (Fig. 2). Administration of amiodarone was stopped at 17:23, and the medication was changed to esmolol. After 3 minutes, the palpitations stopped, her heart rate changed to sinus rhythm, and her ventricular rate was 100 beats/min. Treatment was continued with furosemide, sodium nitroprusside, and esmolol.

Four days later, the patient underwent an electrophysiologic study (EPS) and radiofrequency ablation. Preoperative TTE showed an LVEDD of 61 mm, an LVESD of 43 mm, and an LVEF of 55%. The EPS confirmed that S1S1 and S1S2 had induced focal atrial tachycardia (130–146 beats/min) in the heart vein in the right atrium. The S1S2S3 450–300–250 ms stimulation in the right atrium can induce focal atrial tachycardia (206 bpm) in the right posterior wall of the right atrium.

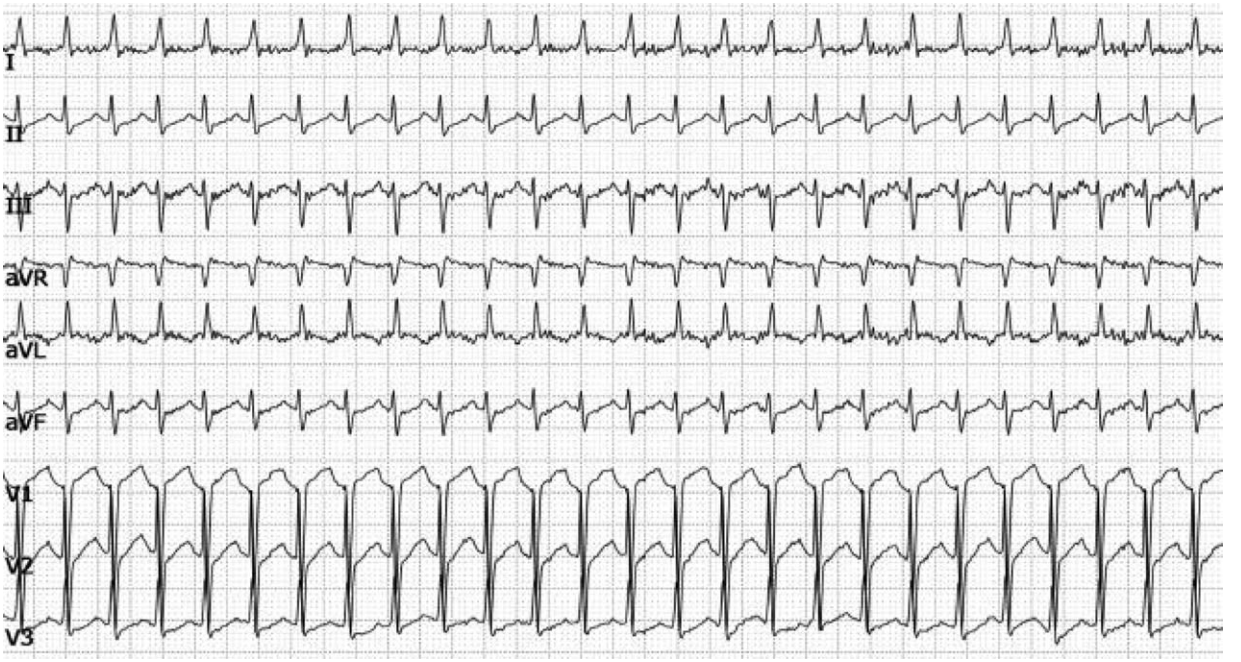

Figure 2. Electrocardiogram at 16:50, ventricular rate 206 bpm.

Three days after surgery, a 24-hour dynamic ECG showed a sinus rhythm (84 beats/min on average), single atrial premature beats (13 times), short atrial tachycardia (1 time), and single ventricular premature beats (2942 times). Fourteen days after surgery, the TTE showed an LVEDD of 57 mm, an LVESD of 41 mm, and an LVEF of 49%. Five months after surgery, the TTE showed an LVEDD of 47 mm, and LVESD of 32 mm, and an LVEF of 61%. The patient had no palpitations or chest tightness.

3. Discussion

Our patient had a history of preeclampsia. Heart structure changes were considered to be a hypertensive disorder complicating pregnancy before admission. Perinatal cardiomyopathy refers to: Firstly, symptomatic HF occurring from the last month of pregnancy to 5 months after birth. Secondly, HF with no other definite cause. Thirdly, contractile HF confirmed by echocardiography.[6] The diagnostic criteria for HF on echocardiography are as follows: left ventricular ejection fraction $<$ 45%, shortening fraction $<$ 30%, and/or left ventricular end diastolic diameter $>$ 2.7 cm/m^2.[7] To make a diagnosis of heart disease related to hypertensive disorders in pregnancy (HD-HDP), there is no definite requirement on echocardiography. Arrhythmia caused our patient's HF. Arrhythmia-induced cardiomyopathy refers to atrial and/or ventricular dysfunction secondary to rapid and/or asynchronous/irregular myocardial contractions that are partially or completely reversed after treatment of the causative arrhythmia. Two categories of the condition exist: in 1, arrhythmia is the only reason for ventricular dysfunction (arrhythmia-induced), and in the other, the arrhythmia exacerbates ventricular dysfunction and/or worsens HF in a patient with concomitant heart disease (arrhythmia-mediated).[8] Therefore, our postpartum patient was considered to have atrial tachycardia-mediated acute HF based on HD-HDP.

Amiodarone is a broad-spectrum antiarrhythmic drug with the electrophysiological effects of class III antiarrhythmic drugs. The main electrophysiological function of amiodarone is to prolong the action potential and effective refractory period of cardiac myocytes, inhibit the function of the sinus node and atrioventricular node, and prolong atrioventricular conduction.[1] In our patient with palpitations, the ventricular rate increased from 150 to 200 beats/min after the amiodarone treatment. There are 2 possible ways to explain this phenomenon:

1. According to the electrocardiogram, she was considered to have atrial tachycardia. We believe that a possible explanation is that 30 minutes after loading with amiodarone, the concentration of amiodarone reached a peak and was able to control the focal atrial velocity at the source of the heart vein. At this point, the central posterior wall of the right atrium was dominated by focal atrial tachycardia, but the amiodarone could not control the focal atrial tachycardia from the middle posterior wall of the right atrium. Therefore, we refer to it ass opportunistic atrial tachycardia. After administering esmolol, the central atrial tachycardia in the mid-posterior wall of the right atrium was controlled and converted to a sinus rhythm. The half-life of esmolol is about 2 minutes, and its elimination half-life is about 9 minutes.[9] After loading at a suitable dose of 0.05 to 0.3 mg/kg/min, the product can reach a steady state of blood concentration within 5 minutes.

2. Alternatively, the electrocardiogram revealed atrial flutter: after the use of amiodarone, a slight beta blockage reduced the frequency of the flutter waves, and the flutter waves decreased from 300 beats/min to 206 beats/min. The regular atrioventricular 2:1 conduction ratio was converted to 1:1, and the ventricular rate increased from 150 beats/min to 206 beats/min. This situation aggravated the patient's HF. Kauffmann et al reported that 1 patient had atrial flutter associated with 1:1 atrioventricular conduction due to a propafenone intravenous injection.[10] El-Harari and others reported that oral propafenone leads to atrial flutter associated with 1:1 atrioventricular conduction and that the cause of this phenomenon is due to the β blocking effect of propafenone.[11] The anticholinergic effects of quinidine and disopyramide are believed to promote atrioventricular node conduction, which can also cause atrial flutter associated with 1:1 atrioventricular conduction.

With the development of catheter mapping technology, electrophysiologic physicians have reached a consensus that atrial flutter is considered to be a type of auricular tachycardia caused by a large reentry ring formed by a fixed and/or functional barrier. Although the EPS did not induce atrial flutter, it was not clear whether atrial flutter was present in this patient. Aouate et al believe that the most probable explanation for the physiopathology of 1:1 AV conduction flutter related to class I AAD is twofold: Firstly, a decrease conduction velocity in the atrium and subsequently, a reduction in the atrial flutter rate. Secondly, a block in the fast AV pathway, therefore facilitating transmission of the flutter waves over the slow pathway in which refractoriness is short and insensitive to these pharmacodynamic agents.[12] The EPS suggests that esmolol hydrochloride injection has a typical beta adrenergic receptor blocker, which can prolong the AH interval in atrial rhythm and successfully reduce the frequency of F waves after use.

It is difficult to directly identify patients with atrial flutter or atrial tachycardia by electrocardiogram. Vagus nerve stimulation can be used.[13] If it is atrial flutter, the ventricular rate may be slowed down to 3:1, 4:1, or even higher proportions of atrioventricular conduction without affecting the frequency of the F wave, and the wave of the flutter can also be clearly revealed.

In conclusion, when amiodarone is used to treat atrial arrhythmia, the ventricular rate may accelerate, which causes patients with borderline heart failure to develop acute heart failure or further deterioration of acute HF. For HF induced or mediated by atrial arrhythmias, short-term β-blockers may be used to control the ventricular rate more quickly and effectively and to prevent progressive HF.[14]

Author contributions

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Supervision: Wenbing Jiang.
Writing – original draft: Fanhao Ye, Yi Wang.
Writing – review & editing: Fanhao Ye, Yi Wang.

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