



Flecainide Favors the Onset of Ventricular Tachycardia in GUCH Patient

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Abstract

Truncus arteriosus is a rare type of heart disease in which a single blood vessel (truncus arteriosus) comes out of the right and left ventricles, instead of the normal 2 vessels (pulmonary artery and aorta). It is present at birth (congenital heart disease). A case report describes a 33-year-old patient with a history of truncus arteriosus type 1 who underwent cardiac surgery correction in the neonatal age, bearer of a dual-chamber ICD due to a positive electrophysiological study for induction of ventricular tachycardia, presented in the ED for palpitations. In the clinical case described, the intravenous bolus administration of flecainide determined the transition from atrial flutter to wide complex tachycardia, posing a problem of differential diagnostics between aberration of conduction and ectopy.

Keywords: Flecainide; Tachycardia; Antiarrhythmic drugs; Truncus arteriosus

Introduction

Truncus arteriosus is a rare type of heart disease in which a single blood vessel (truncus arteriosus) comes out of the right and left ventricles, instead of the normal 2 vessels (pulmonary artery and aorta). It is present at birth (congenital heart disease) [1]. The repair of truncus arteriosus requires separation of the pulmonary artery bifurcation from the single arterial trunk, closure of the ventricular septal defect, and establishment of an unobstructed pathway from the right ventricle to the pulmonary arteries. These individuals are at risk of developing supraventricular and ventricular tachycardias not only in the immediate post-operative period, but also throughout their lives due to myocardial scarring following surgery [2].

Case Presentation

This case report describes a 33-year-old patient with a history of truncus arteriosus type 1 who underwent cardiac surgery correction in the neonatal age, bearer of a dual-chamber ICD due to a positive electrophysiological study for induction of ventricular tachycardia, presented in the ED for palpitations. The ECG at admission (Figure 1) showed an atypical atrial flutter/atrial tachycardia with variable AV conduction with right bundle branch block. The patient was hemodynamically stable. A bolus of IV Flecainide was administered, following which a wide QRS complex tachycardia appeared (Figure 2) with left bundle branch block morphology and lower axis.

The EGM during ICD interrogation showed a regular ventricular rhythm with ventricular-atrial

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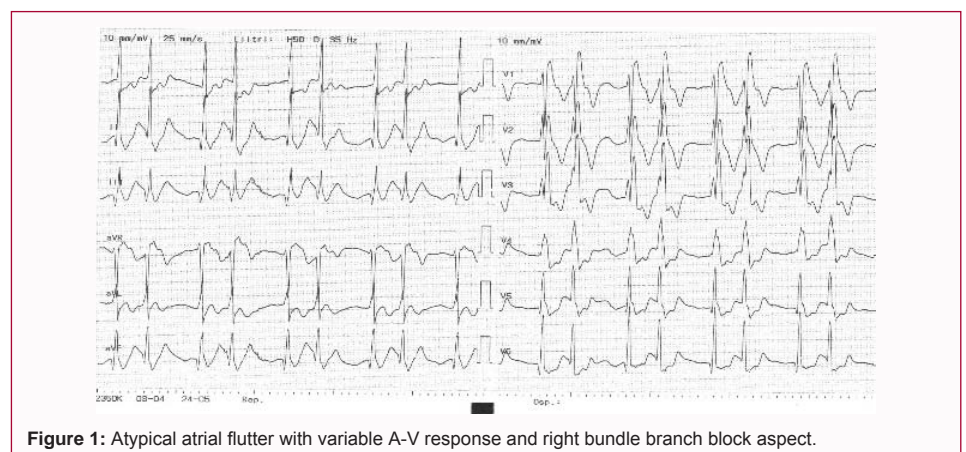


Figure 1: Atypical atrial flutter with variable A-V response and right bundle branch block aspect.

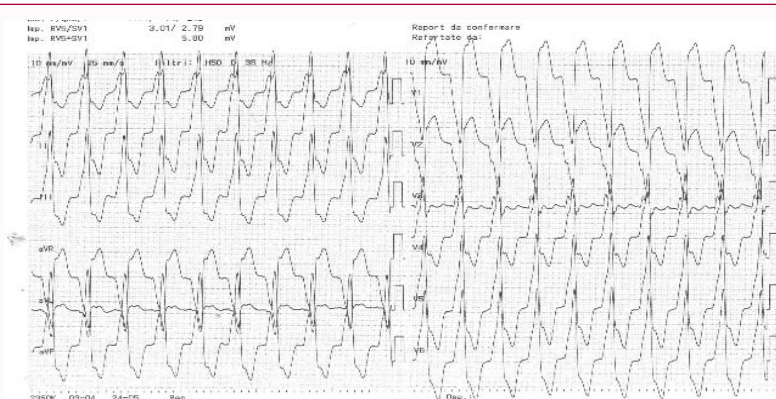


Figure 2: Wide QRS tachycardia after flecainide infusion.

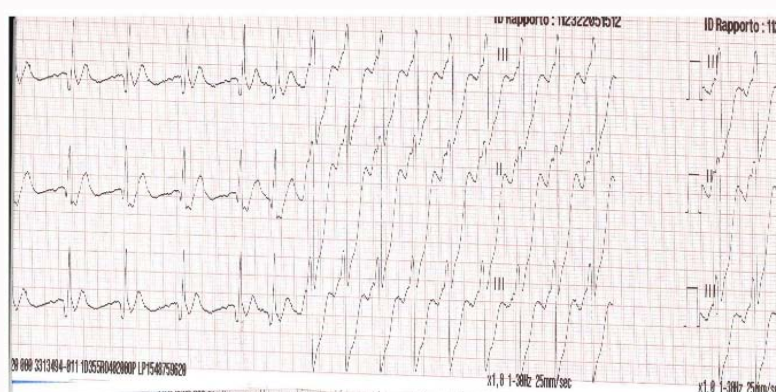


Figure 3: Wide complex tachycardia induced by premature atrial beat.

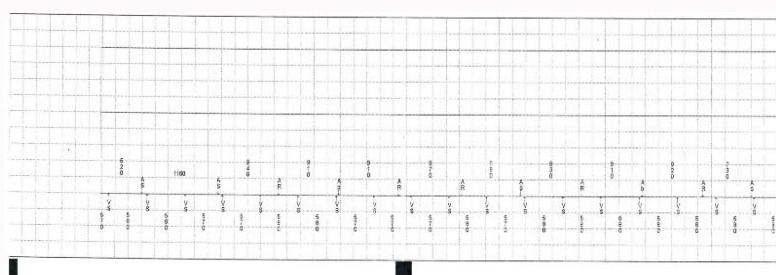


Figure 4: Ventricular-atrium dissociation on ICD mark channel.

dissociation (Figure 3).

The ATP pacing terminated the ventricular tachycardia.

Discussion

Type 1C antiarrhythmic drugs are approved as acute and chronic rhythm control therapy in patients with atrial fibrillation in the absence of structural heart disease as described by the ESC guidelines on supraventricular tachycardias [3]. On the other hand, these drugs are contraindicated in the treatment of atrial flutter (Class III) precisely because among their side effects there is the possible development of extremely wide QRS due to the aberrant conduction of the electrical impulse, they can determine the establishment of an AV relationship 1:1 and the possible triggering of ventricular reentrant tachycardias [4]. The latter occurrence is particularly true in patients with myocardial scars, such as patients undergoing cardiac surgical correction, in which these drugs, by inducing a marked slowing down

of electrical conduction, can determine the development of re-entry circuits and eventual ventricular tachycardias.

In the clinical case described, the intravenous bolus administration of flecainide determined the transition from atrial flutter to wide complex tachycardia, posing a problem of differential diagnostics between aberration of conduction and ectopy (Figure 3). Only the control of the device and the analysis of the endocardial EGMs (Figure 4) allowed to perform a correct diagnosis of ventricular tachycardia induced by flecainide. In these cases, it is not possible to apply the morphological criteria for differentiating a conduction disturbance from ventricular tachycardia and the EGMs analysis remains the only valid tool.

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