CLINICAL NOTE

Wide QRS-complex tachycardia following administration of adenosine

Joaquín VELILLA MOLINER[1], José Javier SALAZAR GONZÁLEZ[2], Antonio ASSO ABADÍA[2], Antonio GIMÉNEZ VALVERDE[1], María Isabel MARQUINA LACUEVA[3], Luis Javier PLACER PERALTA[2]

[1]Emergency Department. [2]Cardiology Department. Unit of Electrophysiology and Arrhythmias. Hospital Miguel Servet de Zaragoza, Spain. [3]Mobile Emergency Unit 061 Aragón, Spain.

Correspondence:
Joaquín Velilla Moliner
Avda. de la Ilustración 39,
esc. 4ª - 1º A
50012 Zaragoza, Spain
E-mail: jovelilla@terra.es

Received:
8-5-2007

Accepted:
6-5-2008

Conflict of interest:
None

Regular tachycardia with a narrow QRS complex is a common presenting complaint in emergency departments. The differential diagnosis includes atrioventricular reentrant tachycardia (intranodal or accessory pathway-mediated) and atrial tachycardia (atrial flutter). These types of tachycardia are commonly treated using vagal manoenvures and adenosine. We describe a patient with a history of paroxysmal atrial fibrillation receiving regular treatment with flecainide who presented with a regular narrow QRS-complex tachycardia (205 beats/min). After administration of adenosine the heart rate slowed briefly, followed by regular wide QRS-complex tachycardia at the same rate as before and apparently ventricular in origin. The patient was in class 1C atrial flutter with 1:1 atrioventricular conduction. Severe conduction disturbance due to flecainide treatment was intermittently exacerbated by the adenosine-induced atrioventricular block. [Emergencias 2008;20:359-362]

Key words: Tachycardia. Flecainide. Adenosine.

Introduction

Regular tachycardia with narrow QRS is a frequent disease in hospital emergency departments (HED). This type of tachycardia was denominated supraventricular tachycardia which was inadequate for auricular-ventricular re-entry through an accessory route because of the involvement of the ventricular myocardium. At present, it is preferentially called narrow QRS–complex tachycardias, although this terminology also presents limitations, such as branch aberrance and antidromic tachycardias. Differential diagnosis fundamentally includes intranodal tachycardia, middle tachycardias by accessory vias, auricular tachycardia and auricular flutter. The initial approach in this type of tachycardia is to carry out vagal manoeuvres and/or the administration of adenosine, or, sometimes, the performance of urgent electric cardioversion, if the patient presents haemodynamic instability. In many cases these treatments interrupt the tachycardia and in others they help to achieve the diagnosis on increasing the degree of auricular-ventricular blockade which allows a better visualisation of auricular arrhythmia. We present a case in which the administration of adenosine in regular narrow QRS tachycardia produced the appearance of regular wide QRS tachycardia.

Clinical case

A 58-year-old patient with a history of paroxysmal auricular fibrillation for many years, habitually receiving flecainide 100 mg every 8 hours and warfarin arrived at the emergency department. Since 3 hours before the patient presented profuse sweating and a presyncopal picture. Neither palpitations or chest pain nor dyspnoea were reported. An electrocardiogram (ECG) was performed which showed regular narrow QRS at 205 bpm (Figure 1) with good haemodynamic tolerance at rest. Ineffective vagal manoeuvres were initiated followed by the administration of 6 mg of adenosine in a rapid intravenous bolus and a second administration of 12 mg after which cardiac frequency transitorily slowed to, in scarce seconds, present regular wide QRS

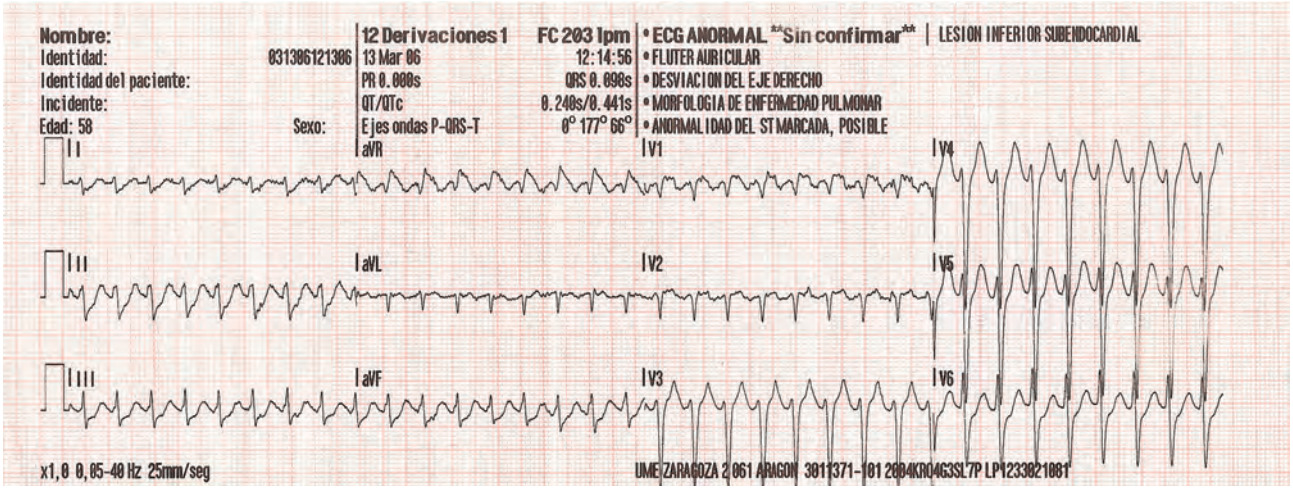

Figure 1. Baseline ECG in which regular narrow QRS tachycardia is observed.

tachycardia at the same frequency (Figure 2). The tachycardia showed a blockade configuration of the left branch (morphology in V1 r6), QRS width of 165 ms, rS in V6, and a concordant negative pattern in the precordia and upper right axis. The rhythm changed to atypical auricular flutter on intravenous amiodarone administration with a variable ventricular response and narrow QRS. Cardiac frequency was controlled on the association of beta blockers. The echocardiogram was normal as was the blood analysis and thyroid hormones. An electrophysiological study was performed showing normal auriculoventricular conduction intervals and ventricular pacing programmed from the apex of the right ventricle with 2 longitudes of cycle and 2 extra-pacings without inducing ventricular arrhythmias.

This was therefore a patient with a persistent paroxysmal auricular fibrillation crisis of several years of evolution who, after treatment with flecainide, developed a 1C flutter with 1:1 conduction to the ventricle which generated a great aberrance in intraventricular conduction. He was advised to avoid group 1C drugs in the future.

Discussion

Flecainide is a group 1C drug which acts by blocking the sodium (Na) channel, slightly prolongs the duration of the action potential and fundamentally reduces the velocity of conduction at the myocardial level and of the specific conduction system. Flecainide has dependency in use, that is, it presents a greater effect on rapid car-

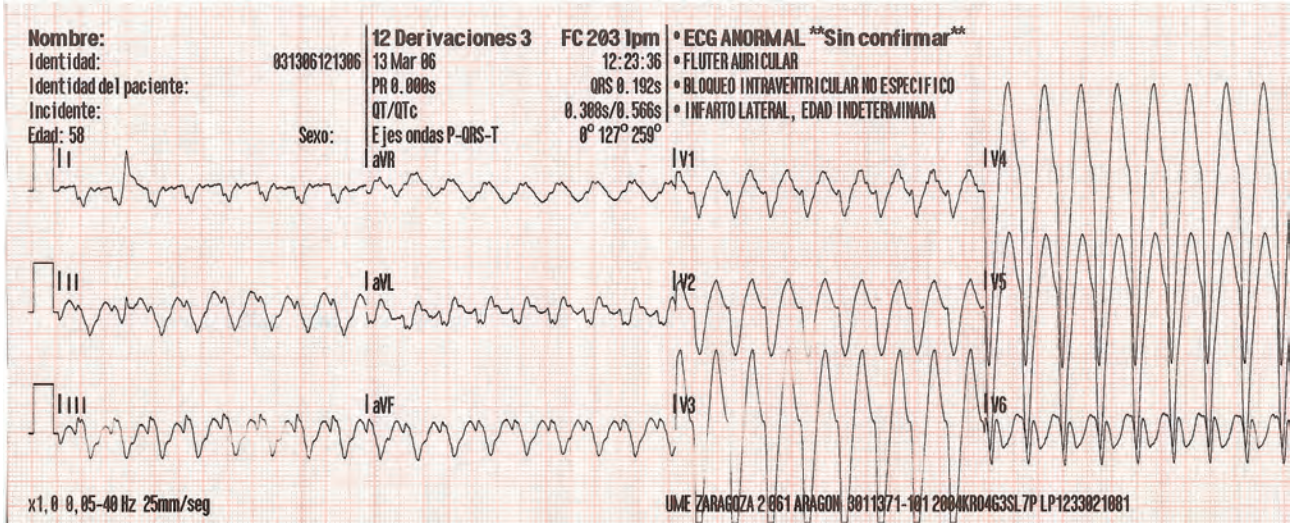

Figure 2. ECG one minute after the administration of 6 + 12 mg of adenosine IV showing a regular wide QRS tachycardia.

diac frequencies, due to its joining to the active state of the canal and the slow separation kinetics. Thus, the more activations the channel has (rapid cardiac frequency), the greater the amount of drug joined at the Na channels and the greater the effect. This explains why flecainide is more effective in acute auricular fibrillation reversion (rapid cardiac frequency) than in the prevention of recurrence and also explains the greater pro-arrhythmia of flecainide associated with elevated cardiac frequencies such as in auricular flutter with rapid ventricular response or after sinusal tachycardia associated with physical exercise[1].

Flecainide is therefore a group 1C anti-arrhythmia drug which has shown to be effective in acute cardioversion of auricular fibrillation as well as in the prevention of recurrence[2]. Its use is not advisable in the presence of structural cardiopathy, significant ventricular hypertrophy, branch blockade or conduction disorders because of the possibility of generating ventricular arrhythmia[3].

Occasionally, as in the case presented herein, flecainide is able to convert auricular fibrillation into an auricular flutter (denominated 1C flutter) and/or slow the frequency of a pre-existing auricular flutter due to its refractoriness effect and conduction velocity. Thus, an auricular flutter with a slower frequency is generated (in our case 205 bpm) which, in this form, may be conducted 1:1 to the ventricle which generated an elevated ventricular frequency since flecainide has a very scarce effect on nodal conduction. Likewise, this elevated frequency makes the effect of flecainide at a ventricular level rise greatly thereby generating a very marked and diffuse slowing at the level of the His-Purkinje system and ventricle producing very manifest, atypical conduction aberrance. This conduction aberrance is very atypical and with classical electrocardiographic criteria it is practically indistinguishable from ventricular tachycardia[4]. In the present case, the tachycardia demonstrated a QRS width of 165 ms, left branch blockade configuration, a pattern concordant with the precordial leads and an upper right axis. This type of tachycardia was indistinguishable with only electrocardiographic criteria of ventricular tachycardia. Thus, the diagnosis is fundamentally based on clinical suspicion in a patient receiving treatment with 1C drugs due to previous episodes of auricular fibrillation and the absence of significant structural cardiopathy. The incidence of 1C flutter in patients with auricular fibrillation treated with flecainide is low, being around 3%, and 1:1 conduction is even more exceptional (0.6%)[5]. The acute or chronic association of auriculoventricular node blockers (betablockers or calcioantagonists) may significantly reduce the probability of auricular flutter 1:1[6].

Another aspect to consider is that amiodarone should not be associated with 1C drugs because of metabolic interactions (amiodarone reduces the hepatic metabolism of flecainide) and the sum of electrophysiological effects (prolongation of the refractoriness and slowing of conduction velocity) with a consequent greater risk of proarrhythmia[1].

On the other hand, in the present case, the conduction aberrance (produced by flecainide) was provoked after the administration of adenosine. The high efficacy together with the pharmacokinetics of adenosine, with a half life of less than 10 sec. and the scarce incidence of secondary effects has led this drug to be a first line drug after vagal manoeuvres in the management of regular narrow QRS tachycardia. Its main effect consists in the transitory blockade of nodal conduction and, to a lesser extent, the depression of sinusal automatism, a reduction in auricular refractoriness and a depression in pacemarker activity of the Purkinje fibres. The administration of adenosine usually provokes transitory secondary effects such as prolonged sinusal pauses and/or high grade auriculoventricular blockade in addition to the induction of fibrillation and auricular flutter as an infrequent phenomenon[7].

In the present case, adenosine produced a transitory slowing in nodal conduction followed by the reappearance of rapid auriculoventricular conduction with ventricular response at 205 bpm. It is of note that the conduction aberrance occurred after adenosine. The transitory slowing in ventricular response produced by adenosine most probably induced prolongation of the refractory ventricular period thereby favouring the aberrance on recovering the 1:1 conduction after the effect of adenosine remitted.

References

1 Fármacos antiarrítmicos. García-Tejada J, Salguero R, Sánchez I, Arribas F, López Gil M. En: José Luis Merino Llorens. Arritmología clínica Madrid: Momento Médico Iberoamericana s.l.; 2003:233-254.

2 Almendral Garrote J, Marín Huerta E, Medina Moreno O, Peinado Peinado R, Pérez Álvarez L, Ruiz Granell R, et al. Guías de práctica clínica de la Sociedad Española de Cardiología en arritmias cardíacas. Rev Esp Cardiol 2001;54:307-67.

3 The Cardiac Arrhythmia Supresión Trial (CAST) Investigators preliminary report: effect of encainide and flecainide on mortality in a randomized trial of arrhythmia suppression after myocardial infarction. N Engl J Med 1989;321:406-12.

4 García Cosío F, Pastor A, Núñez A, Magalhaes AP, Awamleh P. Flutter auricular: perspectiva clínica actual. Rev Esp Cardiol 2006;59:816-31.
5 Alboni P, Botto GL, Baldi N, Luzi M, Russo V, Gianfranchi L, et al. Outpatient treatment of recent-onset atrial fibrillation with the "pill in the pocket" approach. N Engl J Med 2004;351:2384-91.
6 Coll-Vinent Puig B, Sánchez Sánchez M, Mont Girbau L. Nuevos conceptos en el tratamiento de la fibrilación auricular. Med Clin 2001;117:427-37.
7 Belardinelli L, Lerman BB. Electrophysiological basis for the use of adenosine in the diagnosis and treatment of cardiac arrhythmias. BR Heart J 1990;63:3-4.
8 Josephson ME. Electrophysiologic investigation: general concepts. En: Josephson ME. Clinical Cardiac Electrophysiology. Philadelphia: Lippincott Williams & Williams; 2002. Pags 19-67.

Taquicardia de QRS ancho tras administración de adenosina

Velilla Moliner J, Salazar González JJ, Asso Abadía A, Giménez Valverde A, Marquina Lacueva MI, Placer Peralta LJ

Las taquicardias regulares de QRS estrecho constituyen una patología frecuente en la práctica de los servicios de urgencia. El diagnóstico diferencial incluye taquicardias por reentrada aurículo-ventricular (intranodal o mediada por vía accesoria) y taquicardia auricular/*flutter* auricular. El tratamiento con maniobras vagales y adenosina es una práctica habitual ante este tipo de taquicardias. Presentamos el caso de un paciente con antecedentes de fibrilación auricular paroxística, en tratamiento habitual con flecainida, que presentó taquicardia regular de QRS estrecho a 205 lpm. Tras administrar adenosina se enlenteció transitoriamente la frecuencia cardiaca, para posteriormente presentar una taquicardia regular de QRS ancho a la misma frecuencia cardiaca de morfología aparentemente ventricular. Se trataba de un *flutter* auricular 1C con conducción aurículo-ventricular 1:1 con gran aberrancia de conducción debido al tratamiento con flecainida y favorecida puntualmente por el bloqueo aurículo-ventricular transitorio generado por la adenosina. [Emergencias 2008;20:359-362]

Palabras clave: Taquicardia de QRS estrecho. Flecainida. Adenosina.