

Accelerated idioventricular rhythm associated with propranolol treatment in a child

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Abstract

Accelerated idioventricular rhythm (AIVR) is a ventricular arrhythmia most commonly seen in adults with underlying cardiac disease. It is important to establish the diagnosis when it occurs to differentiate this benign phenomenon from dangerous ventricular tachycardia.

We present the case of a healthy child who developed episodes of AIVR associated with propranolol treatment. Her 24-hour electrocardiography recording showed AIVR with difference between sinus and ventricular beats. The arrhythmia resolved with the discontinuation of propranolol, and eventually the case was in sinus rhythm. This patient is the first case of AIVR associated with propranolol treatment in the literature. (Cardiol J 2011; 18, X: xx–xx)

Key words: accelerated idioventricular rhythm, children, propranolol

Introduction

Accelerated idioventricular rhythm (AIVR) was first defined by Harris [1] in an experimental study as having about the same rate as the underlying sinus rhythm, being thus faster than the expected rate of an idioventricular focus, but slower than the usual rate of ventricular tachycardia. The first pediatric series was reported by Gaum et al. [2]. Rarely, AIVR can be detected in children without any structural heart disease [3]. When AIVR presents in individuals with no structural heart disease, the mechanism involved is usually an increased vagal tone and decreased sympathetic tone [4]. AIVR is a benign ventricular arrhythmia, does not cause acute or progressive disorder, requires no specific treatment, and resolves over the course of time [5, 6]. We report the case of an asymptomatic child with AIVR related to propranolol treatment used for prophylaxis of supraventricular tachycardia (SVT).

Case report

A seven year-old girl was referred to the pediatric emergency service with palpitations. She had no known disease or drug use. There was no family history of sudden death. The electrocardiogram (ECG) showed a non-sinus rhythm (P wave was absent) of 250 bpm (Figs. 1A, B). Her examination and laboratory tests revealed normal findings except tachycardia. SVT was diagnosed and verapamil was administered intravenously. Subsequently, the ECG showed a sinus rhythm of 120 bpm (Fig. 1C). Echocardiography performed 12 hours later showed no evidence of heart disease. Two months after the first episode of SVT, SVT reappeared. It was treated with adenosine and then she was started on propranolol for prophylaxis. During the following 24-hour period, continuous ECG monitoring showed no cardiac rhythm disorder. Repeated echocardiography was done and was normal.

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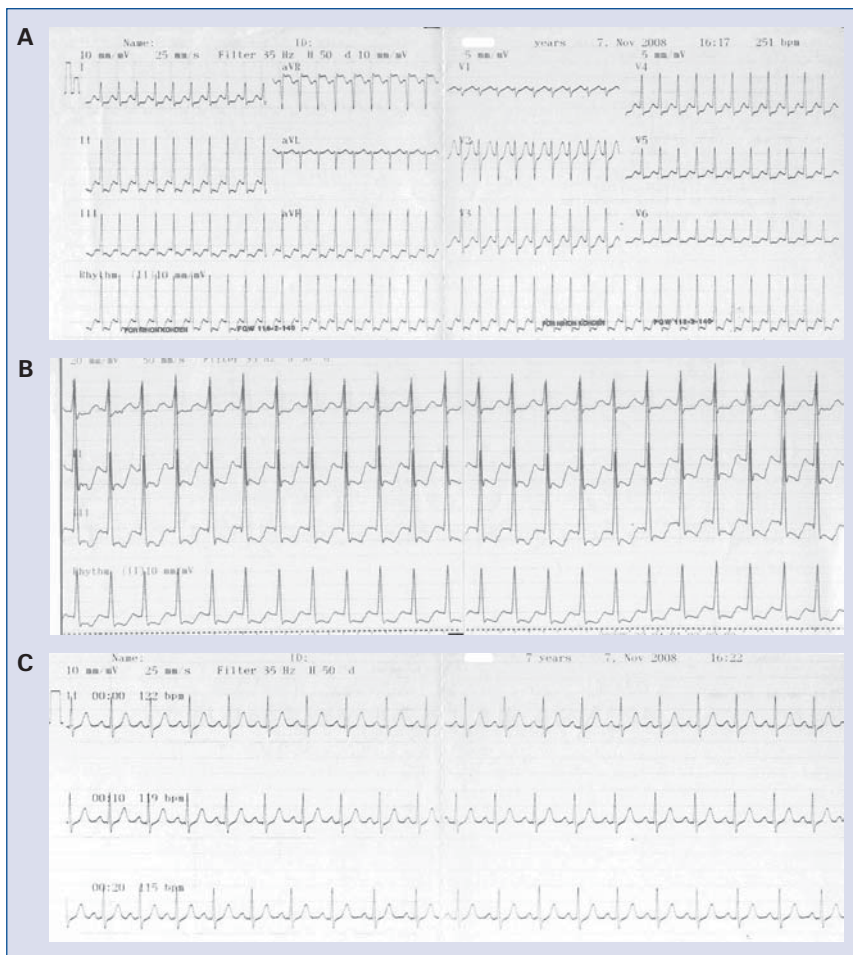


Figure 1. A. A 12-lead electrocardiogram (10 mm/mV; 25 mm/s) showing narrow QRS tachycardia with no P wave at 250 bpm; **B.** A rhythm strip (20 mm/mV; 50 mm/s) recording with the same findings; **C.** A rhythm electrocardiogram (10 mm/mV; 25 mm/s) showing a sinus rhythm at 120 bpm after verapamil administered intravenously.

One year after the first SVT episode, when she was asymptomatic, routine 24-hour continuous ECG monitoring showed short PR intervals (< 0.06 s) and wide QRS complexes (0.14 s) especially during sleep/night hours. Then episodes of ventricular arrhythmia occurred with absent P waves and wide QRS complexes indicative of AIVR alternating with sinus rhythm at every 10–20 beats (Fig. 2A) or persisting for a few hours (Fig. 2B). The ventricular rhythm at this time was 70–80 bpm. The daytime record of the same ECG monitoring showed normal sinus rhythm with a rate of 85–125 bpm (Fig. 2C). Accelerated idioventricular rhythm was seen in the nighttime record of the 12-lead electrocardiogram (Fig. 3). We thought that the propranolol treatment might be causing AIVR by bradycardia. Therefore, propranolol was stopped, and 24-hour continuous ECG monitoring was recorded at the end of a week, a month, three months, six months,

and a year. In the recordings, neither symptomatic nor asymptomatic arrhythmia was found.

Discussion

In this case, an asymptomatic child was documented with arrhythmia characterized by consecutive monomorphic ventricular beats alternating with sinus rhythm. There was a rhythm with all the ECG characteristics of ventricular tachycardia, but with a rate approximately the same as the prevailing sinus rhythm, usually less than 100 bpm. Therefore, AIVR, very rarely seen in childhood and individuals with completely normal hearts, was diagnosed in this patient. She used propranolol because of SVT prophylaxis. Propranolol, a beta-blocker drug, decreases sympathetic tone and heart rate and thus can cause AIVR. The treatment was stopped in this case because AIVR was diagnosed, and the

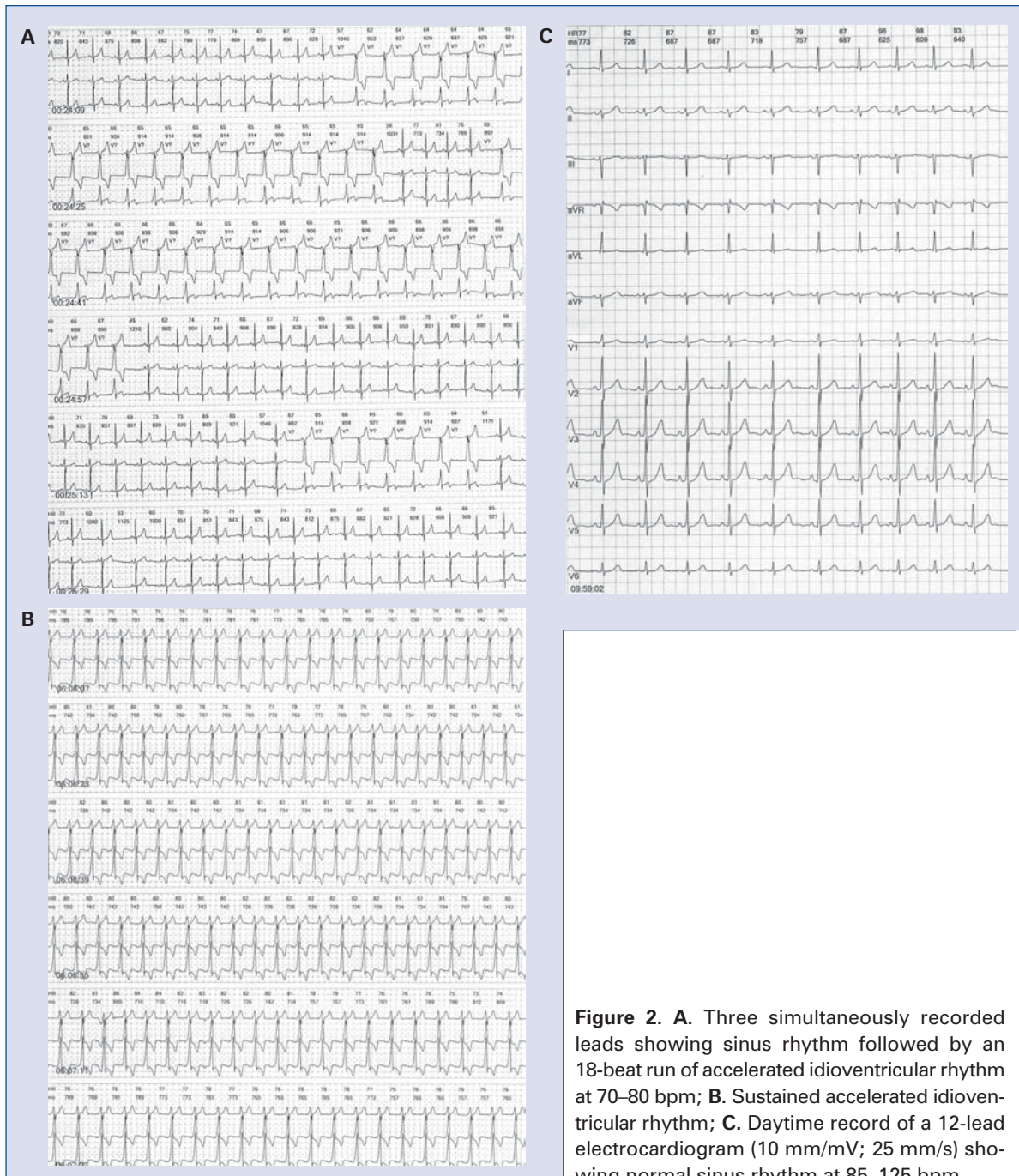


Figure 2. A. Three simultaneously recorded leads showing sinus rhythm followed by an 18-beat run of accelerated idioventricular rhythm at 70–80 bpm; B. Sustained accelerated idioventricular rhythm; C. Daytime record of a 12-lead electrocardiogram (10 mm/mV; 25 mm/s) showing normal sinus rhythm at 85–125 bpm.

arrhythmia resolved spontaneously. To the best of our knowledge, this is the first case report of AIVR associated with propranolol treatment.

Accelerated idioventricular rhythm is characterized by an ectopic ventricular rhythm with three or more consecutive ventricular premature beats at a rate faster than ventricular escape, but slower than 100 to 125 bpm. AIVR may persist for a few minutes, or alternate with sinus rhythm at every 6–10 beats [6]. The main electrophysiological mech-

anism involved in AIVR is an abnormal calcium-dependent automatism (ectopic automaticity) that affects phase 4 of the action potential (diastolic depolarization) [7]. Fusion and capture beats can be seen at onset or termination due to slower rate. AIVR manifests gradually with deceleration of sinus rhythm or acceleration of premature ventricular contractions over sinus rhythm due to sino-atrial/atrio-ventricular block. The AIVR terminates gradually with acceleration of prevailing sinus

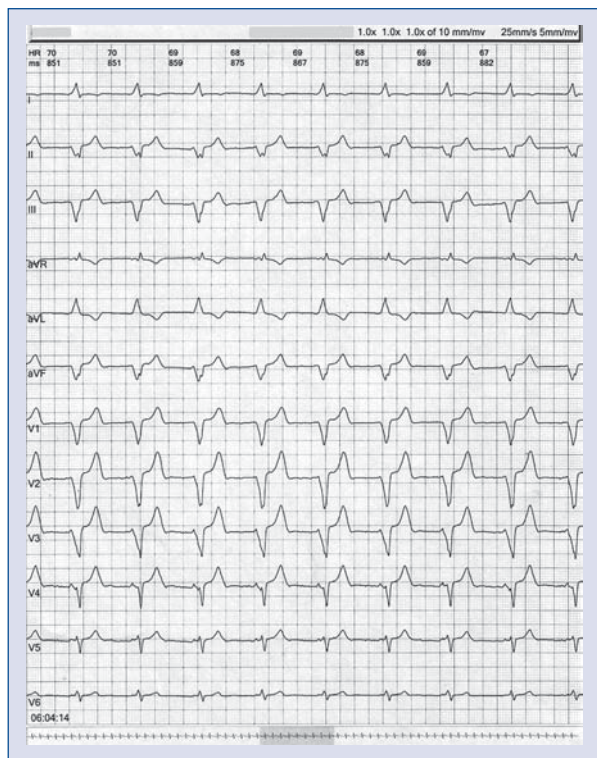


Figure 3. Nighttime record of a 12-lead electrocardiogram showing accelerated idioventricular rhythm.

rhythm or deceleration of ectopic rhythm [3]. The importance of AIVR lies in its potential confusion with other, potentially serious, rhythm disorders that have, as AIVR almost invariably does, a left bundle branch block pattern by ECG: ventricular tachycardia, SVT such as those with aberration or involving Mahaim fibers, and antidromic supraventricular tachyarrhythmia using a standard right-sided accessory pathway for antegrade conduction [6].

Accelerated idioventricular rhythm often occurs in adult patients with acute myocardial infarction, especially when thrombolysis results in reperfusion [4, 8]. AIVR has been also described associated with several drugs, myocarditis, electrolyte imbalance, dilated cardiomyopathy, hypertrophic cardiomyopathy, and arrhythmogenic right ventricular dysplasia [5, 6, 9]. In children, it is rarely seen in normal hearts. AIVR has been seen in patients with congenital heart diseases (i.e. tetralogy of Fallot and subaortic VSD), in children with Fontan, and in transplanted subjects [5, 6].

Accelerated idioventricular rhythm is usually well tolerated and no specific treatment is recommended apart from removing the suspected cause. Several medications have been tried in the past, including mexiletine, digoxin, and quinidine [6]. Beta-blockers and calcium channel blockers such as verapamil are irrelevant in treatment because

they may induce symptoms by decreasing the heart rate and blood pressure. Atropine and atrial pacing help to control AIVR by accelerating the sinus rhythm [5, 6]. Although development of a faster ventricular arrhythmia is very rare, it requires differentiation from ventricular tachycardia.

Nine clinical features are helpful in distinguishing the two: chance discovery; no symptoms; no hemodynamic effects; less than 10% of sinus isochronicity; heart rate of less than 120 bpm; simply converts to sinus rhythm; short bursts; no effective drug treatment; and left bundle branch block [6]. Although AIVR tends to persist, there is no report in post-neonatal children of AIVR degenerating into ventricular tachycardia. The most important reasons for diagnosing AIVR are to avoid unnecessary and potentially harmful treatment, and to allay parental concern [5].

Conclusions

The relation of AIVR to propranolol and the longer duration of AIVR in our case are noteworthy. We conclude that propranolol treatment may precipitate ventricular arrhythmias such as AIVR.

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