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Case Report

Atropine-induced monomorphic ventricular tachycardia in an adolescent: A rare case

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ABSTRACT

A 13-year-old girl had a history of episodic palpitations lasting for approximately 5 min since the first grade of junior high school. She was noticed to have tachycardia during auscultation at a school-based health screening program. Since non-sustained ventricular tachycardia of the left bundle branch block type was induced by a triple master exercise load at a local doctor's clinic, she was referred to our pediatric cardiology department for further management. Tachycardia could not be induced by programmed stimulation in an electrophysiological study, although ventricular tachycardia was induced by atrial high frequency pacing with intravenous injection of atropine under continuous isoproterenol infusion.

<**Learning objective:** Monomorphic ventricular tachycardia probably developed due to increased sympathetic drive caused by atropine injection in the present case. The mechanism of atropine-induced monomorphic ventricular tachycardia is considered related to decreased antagonist effect on the sympathetic nervous system.>

Introduction

Exercise-induced ventricular tachycardia in children is an indication for radiofrequency ablation because of the likelihood of sudden death. This therapy requires the induction of tachycardia, which is often difficult due to the patient being sedated during the procedure; isoproterenol loading and programmed stimulation, the usual methods of inducing tachycardia in such cases, are often not successful, which makes therapy difficult. Atropine, which is used for the treatment of bradyarrhythmias, may induce ventricular tachycardia by inhibiting parasympathetic nerve activity.

Case report

A 13-year-old girl presented with occasional palpitations since the first grade of junior high school. Although the palpitations appeared predominantly with exercise, they sometimes occurred at rest, and lasted for up to 5 min. Since they terminated spontaneously, they were left untreated. In a school-based health screening program conducted when she was in the second grade at junior high school, tachycardia was identified by auscultation, for which she consulted a local doctor. Non-sustained ventricular tachycardia with a heart rate of 208 beats per minute that lasted for seven seconds was induced by application of a triple master exercise load. An electrocardiogram performed at that time showed ventricular tachycardia with left bundle branch block with inferior axis morphology (Fig. 1). She was therefore referred to our department for further evaluation and treatment. Echocardiography showed normal left ventricular function. Hematological investigations showed a slight increase in B-type natriuretic peptide levels to 26.8 pg/ml. We explained to the patient and parents about therapy for arrhythmia. They preferred radiofrequency ablation to medication, and furthermore, chose physical limitation until ablation. Thus, electrophysiological studies were performed with the patient sedated by propofol loading because ventricular tachycardia was easily induced by exercise load and ventricular tachycardia rate was high rate. As pacing catheter, Iuquiry catheter (Abbott Medical Global, Abbott Park, IL, USA) was used. However, ventricular tachycardia could not be induced by either programmed stimulation with atrial and right ventricular burst pacing or the same protocol on isoproterenol load of 0.01 μg/kg/min, although premature ventricular

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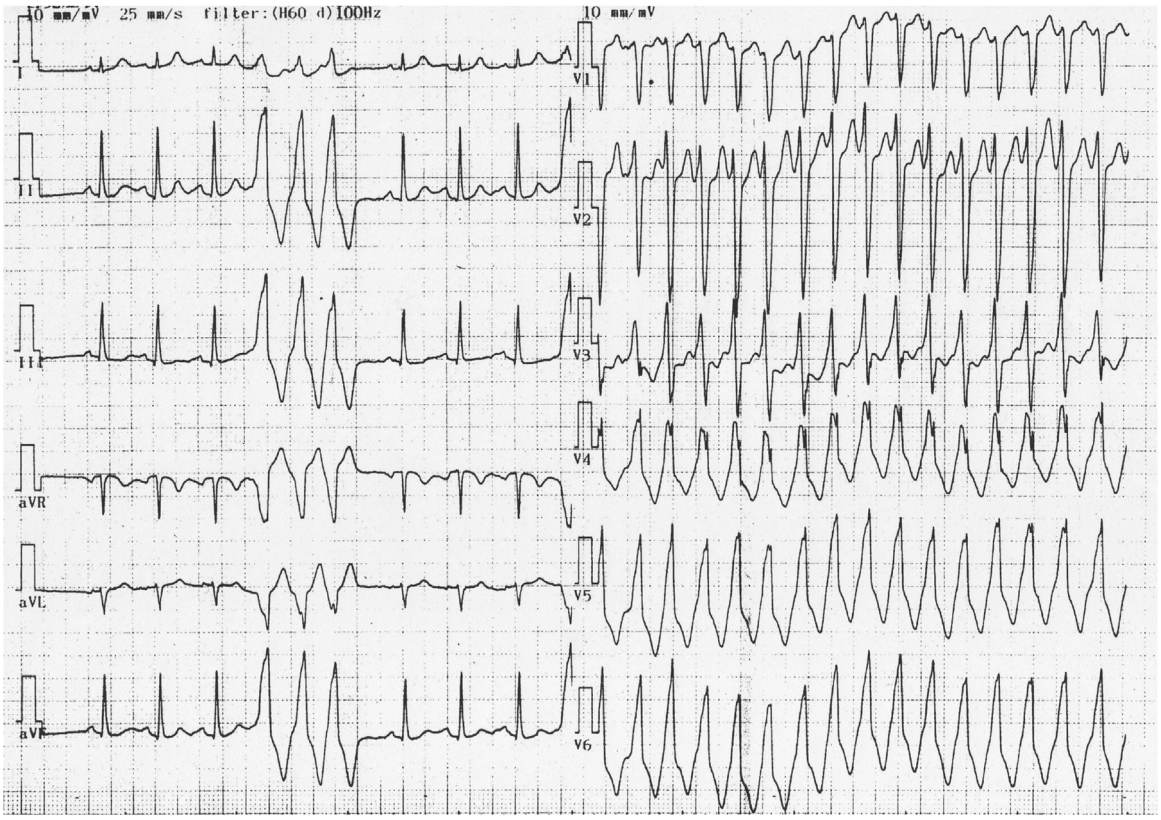

Fig. 1. Electrocardiography with monomorphic ventricular tachycardia. The type is left bundle branch block and inferior axis.

contractions occurred when 0.5 mg atropine sulfate was injected intravenously. Furthermore, non-sustained ventricular tachycardia was induced by atrial high-frequency pacing of rate 160 beats per minute combined with intravenous injection of atropine sulfate under continuous isoproterenol infusion. Finally, sustained ventricular tachycardia was induced (Fig. 2). A noncontact balloon catheter of EnSite system (St. Jude Medical, St Paul, MN, USA) was inflated in the right ventricular outflow tract. The EnSite system incorporates a multielectrode array for noncontact mapping. Then, an activation map of premature ventricular contractions that were triggered by the atropine sulfate load was created, which indicated that maximum pre-excitation occurred at the posterior wall of the right ventricular outflow tract. As for the electrical potential of the ablation catheter for which was used EPT typeTK1 catheter (Boston Scientific, Natick, MA, USA) in the posterior wall of the right ventricular outflow tract, it was 38 msec earlier than the beginning of the body surface electrocardiogram, and the fourth beat of pace-mapping resembled the induced ventricular tachycardia very well at that site (Fig. 3A). Ablation was performed during ventricular tachycardia (Fig. 3B), setting during ablation was 50 W and 55 °C, with a resultant acceleration of the tachycardia for 4.1 s and it was then terminated. After the ablation, it was confirmed that ventricular tachycardia could no longer be induced by programmed stimulation under loading with atropine sulfate injected intravenously under continuous isoproterenol infusion.

Discussion

Monomorphic ventricular tachycardia originating from the ventricular outflow tract can be induced by an isoproterenol and exercise load. Atrial high frequency stimulation also induces monomorphic idiopathic left ventricular tachycardia. Atropine has recently been reported to induce polymorphic ventricular tachycardia, similar to catecholamine- induced polymorphic ventricular tachycardia. As the mechanism of atropine-induced polymorphic ventricular tachycardia, the autonomic nervous system plays an important role in ischemic myocardium [1]. However, in the present case, monomorphic ventricular tachycardia was induced by atropine, probably because, given the patient's age, myocardial ischemia was unlikely. In the present case, the cardiac sympathetic nerve was initially stimulated by the isoproterenol load, followed by inhibition of the cardiac vagus nerve by administration of atropine. Furthermore, atrial high-frequency stimulation resulted in repeated induction of ventricular tachycardia. Thus, ventricular tachycardia was induced by both stimulation of the sympathetic nerve and inhibition of the vagus nerve, whereas, premature ventricular contractions were not observed during sleep and in the sedated state, at which time her vagus nerves were stable and dominant, and hence, tachycardia could not be induced by the programmed stimulation. However, when her sympathetic nerves were active, the vagal output change allowed induction of ventricular tachycardia. This is called accentuated antagonism [2]. After all, the vagus nerve is regarded as dominant in ventricular tachycardia [3]. The mechanism of ventricular tachycardia in this report is accentuated antagonism, where ventricular tachycardia was induced by atropine under continuous isoproterenol infusion, which is different from atropine-induced ventricular tachycardia reported by Tsou et al. [4].

The morphology of the first ventricular premature contraction at the onset in this case was different from that of ventricular tachycardia. Thus, the mechanism of ventricular tachycardia was considered related to cAMP-mediated triggered activity [5].

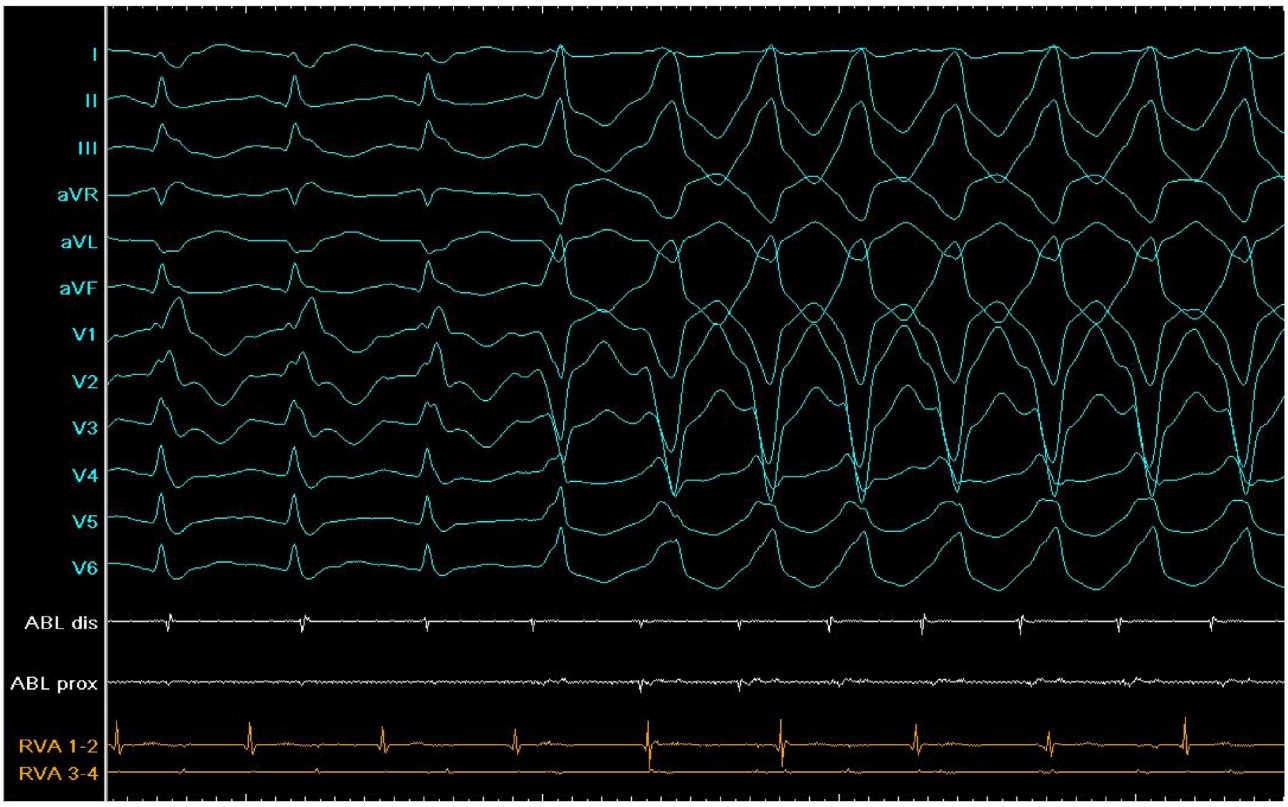

Fig. 2. Electrocardiography (50 mm/sec) with sustained ventricular tachycardia induced spontaneously by intravenous injection of atropine (0.5 mg) under continuous isoproterenol infusion (0.01 μg/kg/min).

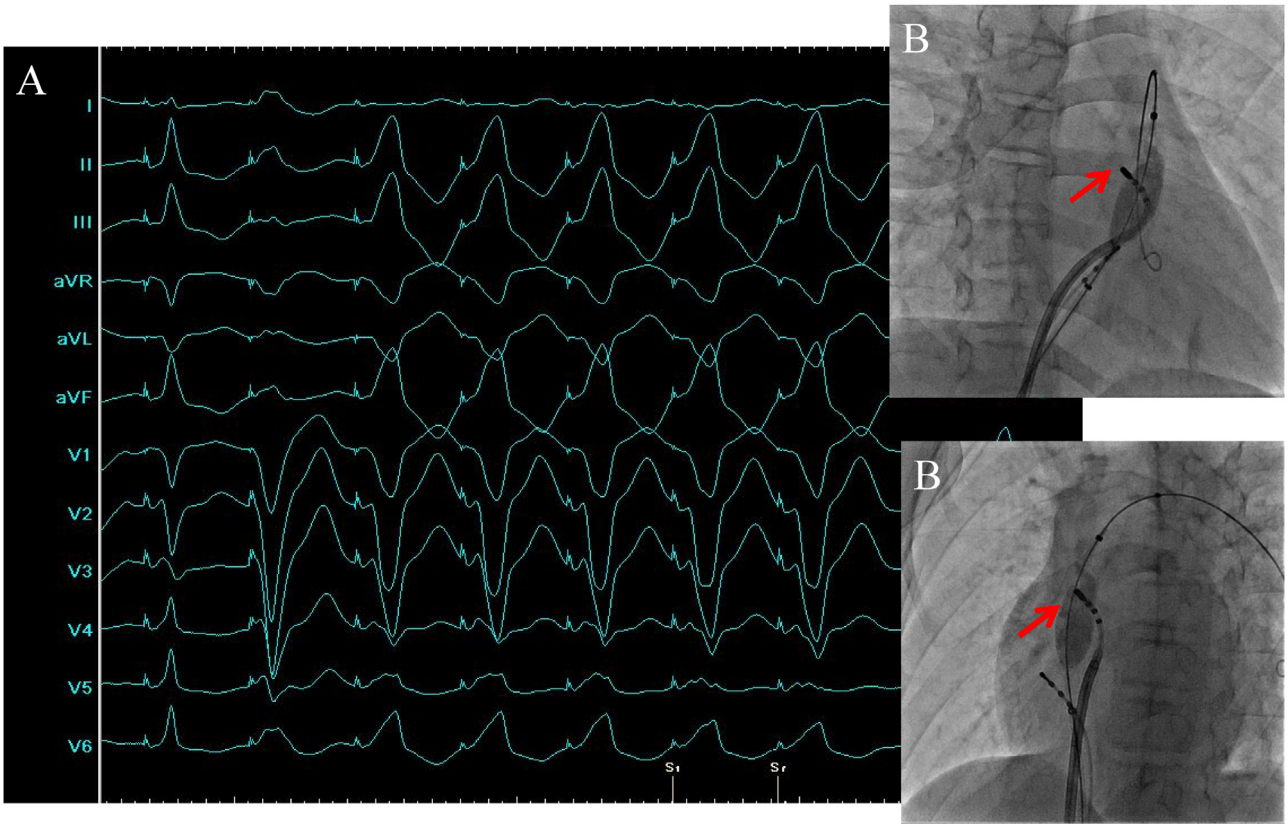

Fig. 3. (A) Pace-mapping at ablation site. (B) Ablation site (arrow) in right anterior oblique (upper) and left anterior oblique (lower).

Acetylcholine-sensitive potassium channels associated with muscarinic receptors in which acetylcholine is released by vagus nerve contacts do not exist in ventricular cardiac muscle cells. Thus, atropine has a dominant antagonist effect on sympathetic nerves, and intracellular cAMP levels increase in ventricular cardiac muscle cells. Furthermore atropine administration would have blocked the muscarinic receptors, thereby resulting in increased adrenalin from the ends of sympathetic nerves. As a result, induction of ventricular tachycardia was considered possible.

In young people, vagus nerve activity is dominant in the autonomic nervous system. The site of origin of ventricular premature contractions in this patient was the posterior free wall of the right ventricular outflow tract. Parasympathetic nerve fibers are reportedly not found in the interventricular septum [6], however they are present in the posterior free wall of the right ventricular outflow tract. These factors could together explain the observed atropine effect in this case.

Whereas, as the choice of the medical treatment, from the mechanism and the origin of the tachycardia, β blocker is considered to be the first line, which inhibits the sympathetic nerve. However, the patients are usually required to continue medication for longer periods of time. In the present case, the patient and parents preferred radiofrequency ablation to medication, therefore ablation was performed.

In conclusion, monomorphic ventricular tachycardia probably developed due to increased sympathetic drive caused by atropine injection in the present case. It is essential to emphasize that atropine decreased the antagonist effect on the sympathetic nervous system.

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