

Ablation of an idiopathic left ventricular tachycardia originating from the posterior mitral annulus in a toddler

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Ablation of a mitral annulus (MA)-ventricular tachycardia (VT), a rare form of idiopathic left VT, has not yet been described in patients <2 years of age. We describe a case of a toddler with an incessant, poorly tolerated idiopathic VT (190 bpm) refractory to medical therapy, which was successfully ablated in the left ventricle at the infero-posterior part of the MA. Different diagnostic and ablation steps are described. Mitral annulus-ventricular tachycardia, a rare form of idiopathic left VT, can safely and successfully be ablated in very young children.

Introduction

Idiopathic ventricular tachycardia (VT) is rare in the paediatric population. Recently, in adults idiopathic VT originating from the mitral annulus (MA) has been described.^{1,2} We describe a case of a toddler with an incessant, poorly tolerated idiopathic VT that was ablated successfully in the left ventricle (LV) at the infero-posterior part of the MA.

Case report

A previously healthy 21-month-old boy was referred to our institution for catheter ablation of a drug-resistant sustained VT resulting in a gradual decrease in echocardiographic left ventricular ejection fraction (LVEF) from 77 to 44%. The electrocardiogram (ECG) revealed ventricular-atrial dissociation during a V1 positive tachycardia (cycle length [CL] 316 ms) with relative narrow QRS complexes (QRS duration 110 ms) and left superior axis (*Figure 1*). Electrocardiogram during sinus rhythm revealed no abnormalities; no discrete inferior Q-waves suggesting an inferior aneurysm were present. Except for the impaired LVEF, echocardiography on referral was normal, excluding major structural heart disease.

The ablation procedure was guided by CARTO mapping and performed under general anaesthesia using the retrograde trans-aortic approach (non-irrigated Biosense Navistar Celcius 4 mm, B-curve mapping catheter). Limited voltage mapping of the LV during sinus rhythm showed an area of low voltage (<0.5 mV) at the infero-posterior wall of the LV adjacent to the MA (*Figure 2A*). During VT (CL 329 ms)—induced by programmed electrical stimulation after isoprenaline infusion (0.15 µg/kg/min)—earliest activation with centrifugal activation pattern was observed in the same area (*Figure 2B*). Pacing in that area resulted in a perfect pacemap. As delivery of radiofrequency (RF) energy in this region (20–35 W, 55°, 60 s) resulted in the termination of tachycardia with immediate re-occurrence, an irrigated-tip catheter (Biosense Navistar Thermocool 4 mm, B-curve) was used. Now RF delivery (30 W, 40°, 60 s) during VT at the site of earliest ventricular activation resulted in immediate and persisting termination of the tachycardia (*Figure 3*). Electrocardiogram monitoring during and after ablation demonstrated no ST segment changes. Heparin was given during 24 h (activated clotting time 150–180 s), and the patient was dismissed from the hospital 3 days later. After a follow-up period of 1 year, the patient remained free of arrhythmias and LV systolic function restored completely.

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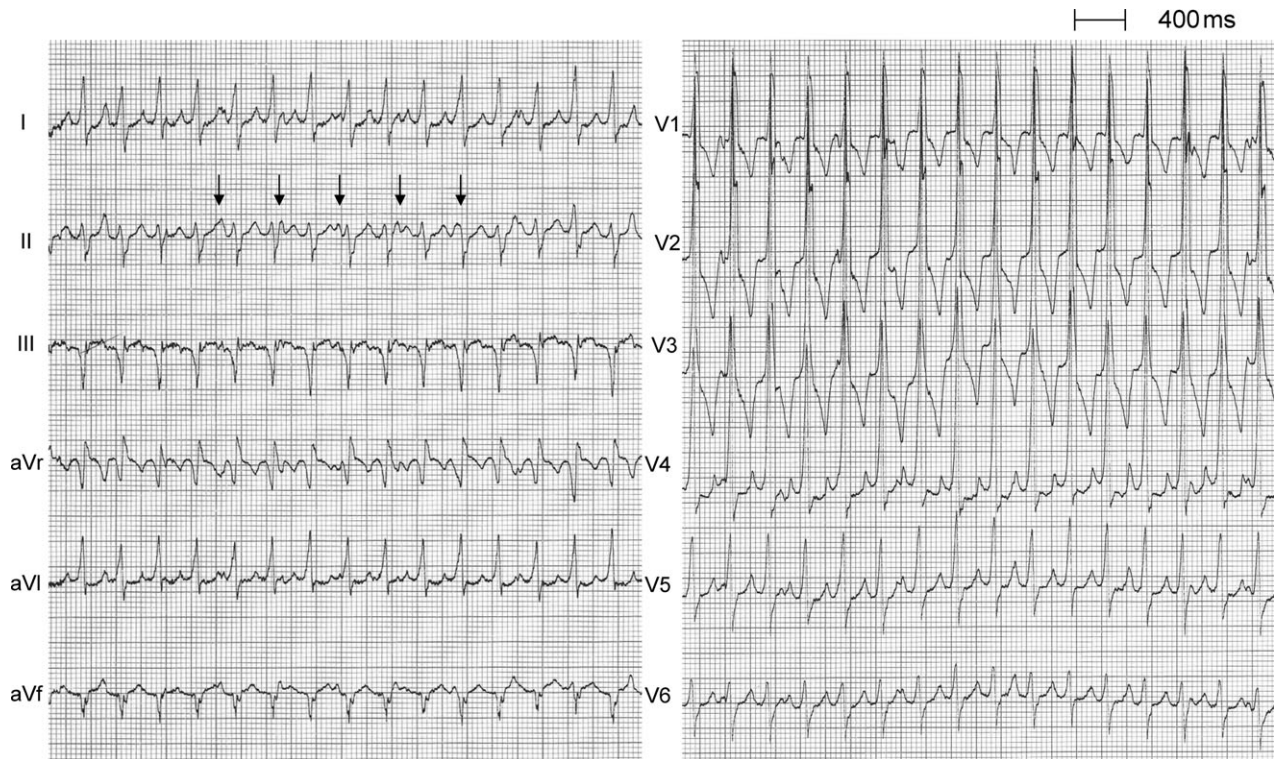


Figure 1 Twelve-lead electrocardiogram during ventricular tachycardia (190 bpm) with the presence of ventriculo-atrial dissociation (arrows demonstrate the P-waves).

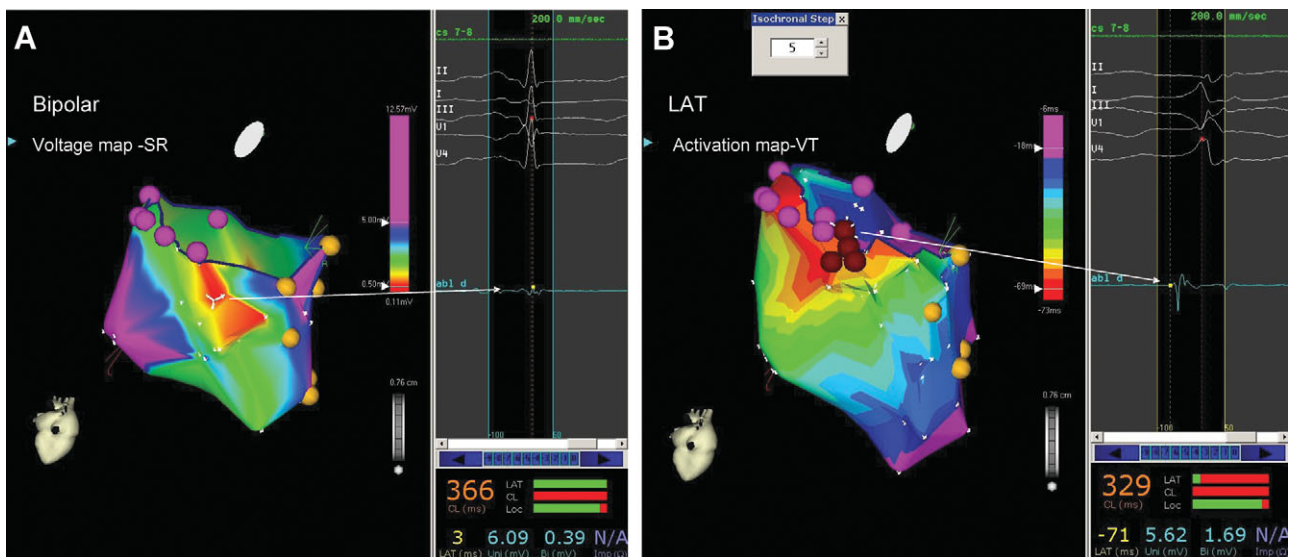


Figure 2 (A) Voltage map of the left ventricle during sinus rhythm (posterior view). Purple dots, mitral annulus; yellow dots, His and left bundle branch signals. Low voltage area $< 0.5\text{ mV}$ is marked in red. Arrow: low voltage bipolar signal of 0.39 mV . (B) Activation map of the left ventricle during ventricular tachycardia (posterior view). R-peak in V4 is used as a reference for the activation map. Isochrones of 5 ms demonstrate a centrifugal activation pattern with earliest activation at the infero-posterior part of the mitral annulus (red zone). Arrow: earliest ventricular activation -71 ms before the R-peak of V4. Red dots: sites of radiofrequency application.

Discussion

This patient presented with a VT originating from the posterior MA. Detailed analysis of the ECG during VT (negative QRS polarity and notching of the R-wave in the inferior leads, S-wave in V6, Rs-pattern in I, and positive QRS complex in V1) suggested a VT origin near the posterior part of the MA.¹

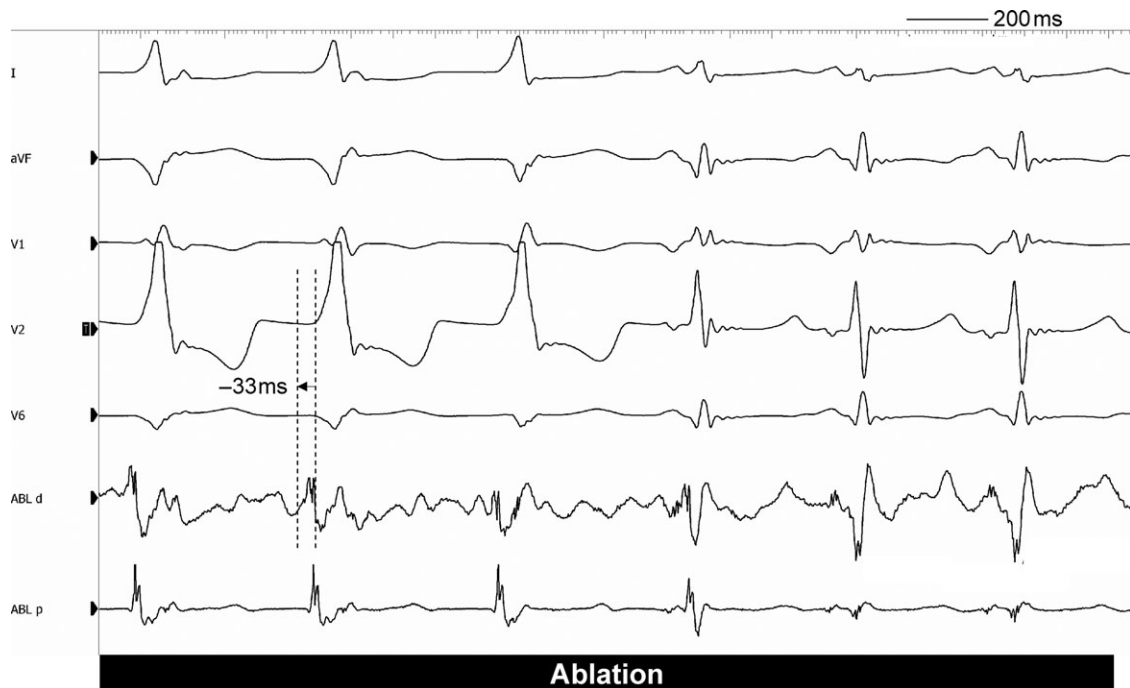


Figure 3 Bipolar electrogram at the site of successful ablation (ABL d). First component of the bipolar electrogram precedes the onset of the QRS complex by -33 ms. Radiofrequency application results in the termination of tachycardia.

Mitral annulus-ventricular tachycardia is a rare form of idiopathic LV tachycardia, accounting for 5% of all patients with idiopathic VT.¹ Idiopathic VT, especially in adults, most commonly arises from the right ventricular outflow tract and less often from the left ventricular outflow tract. Of all MA-VTs, only 11% have an origin at the posterior portion of the MA, whereas 58% originate from the antero-lateral portion and 31% from the postero-septal portion of the MA. Also in children, only 30% of all idiopathic VTs arise from the LV. To the best of our knowledge, no case of an LV MA-VT has been described in an infant <2 years of age.

In general, idiopathic VT in children has a good prognosis and can be treated pharmacologically. However, children with an idiopathic VT originating from the LV are more likely to undergo RF ablation, as they are more symptomatic (heart failure or syncope), have a lower chance for spontaneous resolution of VT, and are less likely to respond to medical treatment.³ The ablation technique itself is similar as in adults.^{4,5} However, because long-term effects of expansion of RF-scar lesions in the ventricles of a growing child are not well known, VT ablation in small children should only be considered in specific circumstances such as incessant, drug refractory VT.

The mechanism of VT in this patient and of idiopathic MA-VT, in general, is not clear. Several mechanisms such as triggered activity, re-entry, and abnormal automaticity have been suggested. In our patient, the area of low voltage at the posterior MA demonstrates subtle structural changes in an overall structurally normal heart and suggests a substrate for re-entry. Also centrifugal activation, as demonstrated by electro-anatomical mapping, is consistent with a possible small re-entrant circuit. In contrast, tachycardia in our patient occurred spontaneously or during isoproterenol infusion and not during programmed electrical stimulation alone, suggesting a possible non-re-entrant mechanism for tachycardia.

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