

Percutaneous catheter ablation of malignant, recurrent ventricular arrhythmia in a 10-month-old toddler



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Introduction

Catheter ablation is a safe and effective treatment modality for ventricular arrhythmia. The procedure is routine at many electrophysiology centers and is being undertaken ever earlier in patient care.¹ However, the great majority of these procedures involve adults suffering from ischemic heart disease. Only 3 cases of transapical access catheter ablation of ventricular tachycardia (VT) have been reported in children of under 15 months of age.^{2–4} Such procedures are technically difficult because the catheters available are ill-suited to the small-sized heart of a child. We report a case of a 10-month-old toddler in whom life-saving ablation of VT was performed via a percutaneous femoral approach with transseptal access to the left ventricle (LV).

Case report

A 10-month-old baby boy weighing 9 kg with unremarkable past medical history was admitted into our establishment following recovery from sudden cardiac arrest. He had been at the house of his grandmother, who is a nurse. About 30 minutes after bedtime, at about 9 PM, she noticed abnormal breathing sounds. In the bedroom, she found him cyanotic and appearing lifeless. In the absence of a pulse, she started cardiac massage and called an ambulance. When the emergency services arrived, they administered care with external cardiac massage and ventilation. Electrocardiography (ECG) showed ventricular fibrillation. Defibrillation was carried out and 1 shock restored sinus rhythm. En route to the hospital, 3 further episodes of ventricular

arrhythmia occurred, each requiring electrical cardioversion. On admission into Intensive Care, the child was intubated and given a general anesthetic. The child was afebrile and hemodynamically stable. Echocardiography showed a normal-sized LV with an ejection fraction of 35%. The remainder of the evaluation was unremarkable. The coronary arteries were visible and in a normal configuration. ECG findings were normal between the episodes of ventricular arrhythmia. Tests showed normal blood counts, moderately elevated C-reactive protein (CRP) at 14.1 mg/L (<5.0 mg/L), hepatic cytolysis (aspartate aminotransferase 222 IU/L [<50 IU/L] and 70 IU/L [<50 IU/L]), very high brain natriuretic peptide at 3143 ng/L (<100.0 ng/L), and troponin Ic at 2.651 ng/mL (<0.35 ng/mL). Electrolytes were normal.

In the hour following admission, he experienced a series of rapid VT episodes, relatively monomorphic at 280 beats per minute, with most episodes progressing to ventricular fibrillation (Figures 1 and 2). About 20 electric shocks had to be delivered despite the intravenous administration of amiodarone (5 mg/kg over 30 minutes, then 20 mg/kg/day) and a beta-blocker (esmolol, 200 µg/kg/min). Lidocaine was transiently used without effect. All episodes began with monomorphic ventricular extrasystole, identical to the VT, with an origin at the median posterolateral part of the LV (Figures 1 and 2).

Over the following few hours, there were numerous recurrences of ventricular arrhythmia. The baby's hemodynamic condition deteriorated and his left ventricular ejection fraction dropped further, down to 25%. The clinical picture incited emergency circulatory assistance with extracorporeal membrane oxygenation (ECMO) via right jugulocarotid cannulation. Although hemodynamics improved and brain natriuretic peptide and troponin levels returned to normal on sustained intravenous amiodarone plus esmolol, recurrent episodes of VT persisted. These were monomorphic and slower but were practically incessant. Some of them continued to

KEYWORDS Catheter ablation; Electrical storm; Extracorporeal membrane oxygenation; Pediatric; Ventricular arrhythmias
(Heart Rhythm Case Reports 2019;5:299–303)

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KEY TEACHING POINTS

- Percutaneous ventricular tachycardia ablation under 1 year of age is feasible.
- It is technically difficult because of the mismatch between the size of the catheters and the size of the heart.
- The cause of sudden death at this age may be ventricular arrhythmias.

progress to ventricular fibrillation, requiring electrical cardioversion (Figure 2). Several electric shocks were required every day. In all, more than 50 shocks were delivered over 7 days in Intensive Care on ECMO, despite intensive pharmacologic treatment. Given the incessant recalcitrant nature of the ventricular arrhythmia without any clear etiology, it was decided to attempt life-saving catheter ablation.

The approach strategy chosen to access the LV was the right femoral vein combined with transeptal puncture guided by a transesophageal echocardiography (Figure 3). We mapped left ventricular activation using a three-dimensional Velocity navigation system (St. Jude Medical, Saint Paul, MN). We first used a standard 4 mm radiofrequency ablation catheter (5F, small curve, St. Jude Medical). This catheter was advanced through a short, small-curve Agilis sheath (St. Jude Medical). Manipulating the catheter was difficult because the

small curve was large relative to the small heart. The Agilis sheath proved ineffective and was therefore left in the right atrium. When the catheter was in place in the LV, amiodarone and esmolol were stopped. Subsequently, the ventricular arrhythmia got worse very quickly. The voltage map did not show any scar. The tachycardia activation map showed primary activation in the posterolateral wall of the middle part of the LV (maximum precocity 19 ms). Pace mapping at this site revealed a near-identical QRS morphology to the clinical arrhythmia (Figure 3). Radiofrequency application was limited at 5 W because maximal temperature was reached, without any effect on the arrhythmia. Then we decided to change to an irrigated ablation catheter with contact force measurement (Tacticath, 4 mm, 8F, small curve, St. Jude Medical). As soon as radiofrequency application was begun at this site with the irrigated catheter, the ventricular arrhythmia slowed down and then stopped entirely after 11 seconds (Figure 3). In all, 4 60-second, 25-W applications were administered with a contact force of between 15 and 20 grams. The whole procedure took 188 minutes with 12 minutes of fluoroscopy.

No recurrence occurred after the procedure despite termination of all antiarrhythmic treatment. Postablation echocardiography showed moderate, noncompressive pericardial effusion opposite the LV. Although this was noncompressive, this serosanguinous fluid was drained because of the need for intensive anticoagulant therapy related to the ECMO. The origin of the pericardial effusion remains unknown.

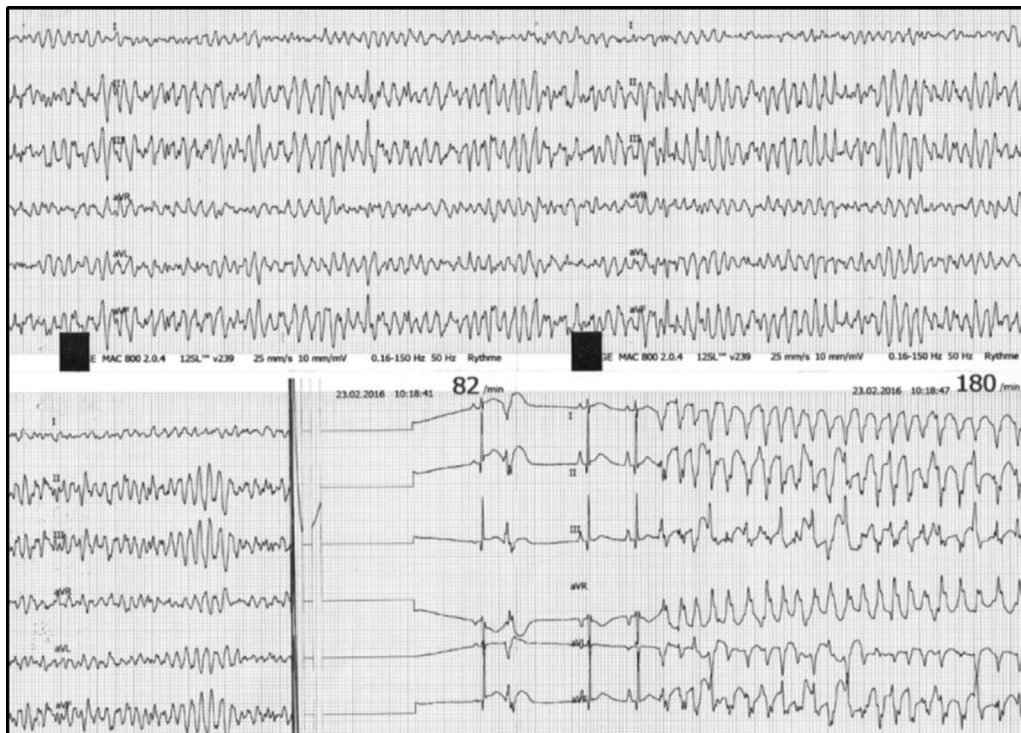


Figure 1 External defibrillation of a ventricular fibrillation (VF) episode. Six-lead electrocardiogram recorded at the patient's arrival in our institution. The VF was converted to sinus rhythm for only 3 beats, interrupted by a premature ventricular contraction (PVC). Then a very rapid ventricular tachycardia started with the same morphology compared to the PVC.

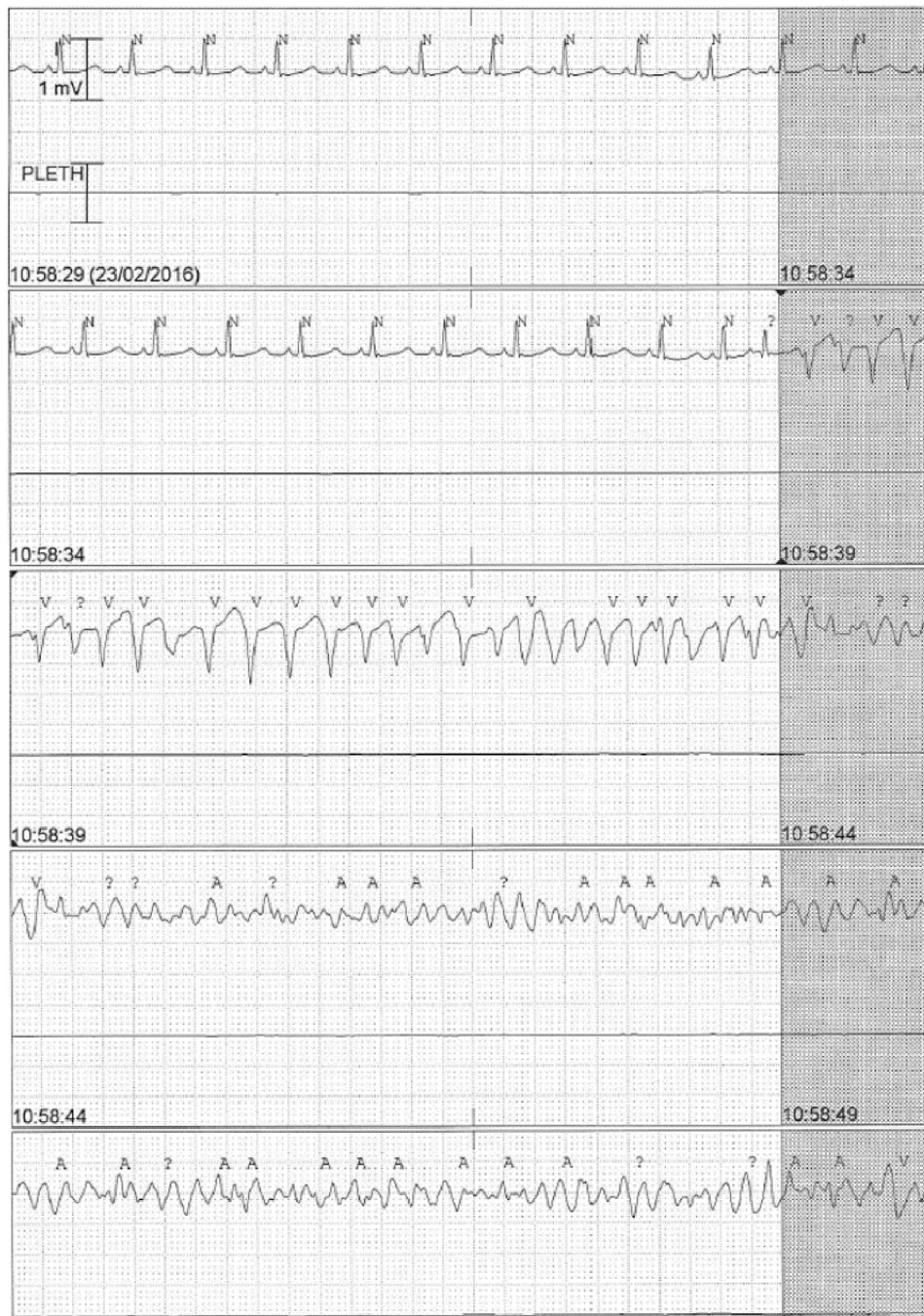


Figure 2 Beginning of a ventricular arrhythmia episode. This episode happened despite a maximal medical treatment, under extracorporeal membrane oxygenation. The first line is a normal sinus rhythm. At the end of the second line, a rapid ventricular tachycardia started, converting into ventricular fibrillation few seconds later at the end of the third line.

ECMO could be discontinued 48 hours after the ablation procedure.

Cardiac magnetic resonance imaging (MRI) a week after the procedure showed a left ventricular ejection fraction of 50% with hypokinesia of the posterolateral wall of the LV, a T2 hyposignal, and contrast medium uptake after injection of gadolinium into the subendocardial portion of the middle, inferolateral segment. It was concluded that this was probably related to the delivery of radiofrequency energy.

The child's recovery was complicated by symptoms of withdrawal from the sedative regimen. A physical examination showed left hemiparesis. The stroke may have been owing to the ablation procedure itself being on the left side of the heart or to the jugulocarotid circulatory assistance.

Before discharge, a Linq ECG monitor (Medtronic, Minneapolis, MN) was implanted for remote monitoring.

After 30 months of follow-up, no recurrence of the arrhythmia has been observed. Global LV systolic function

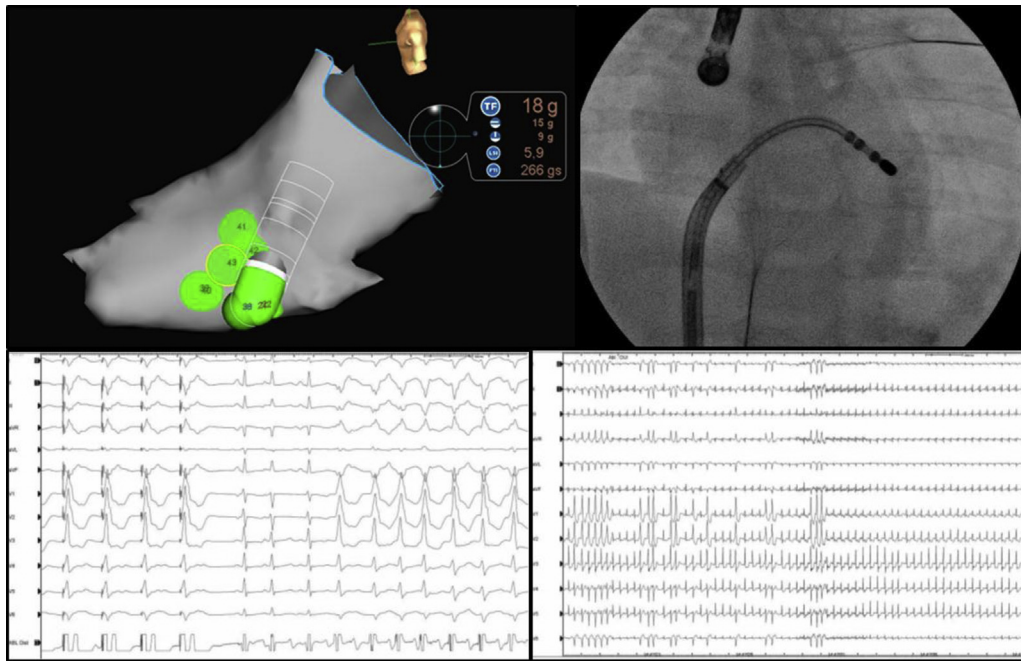


Figure 3 Catheter ablation procedure. The upper left panel is a left lateral view of the left ventricle using a three-dimensional system (Velocity, St. Jude Medical, Saint Paul, MN). The ablation points are represented in *green*; the mitral annulus is represented in *blue*. The upper right panel is an anteroposterior fluoroscopic view. The Agilis sheath (St. Jude Medical) is in the right atrium. The ablation catheter crosses the interatrial septum and is located at the successful site in the left ventricle. The lower left panel shows a 12-lead electrocardiogram (ECG) representing a 12/12 pace map followed by 3 beats of normal sinus rhythm and the spontaneous ventricular tachycardia (VT). The lower right panel shows a 12-lead ECG at the beginning of the successful radiofrequency application. The disappearance of the VT is observed 11 seconds after the radiofrequency was turned on. ST-segment changes can be observed. These could be due to ischemic phenomenon relative to incessant ventricular arrhythmia. The ischemia may have been worsened by the radiofrequency current application itself on the lateral wall of the left ventricle.

has normalized, although some highly localized segmental hypokinesia persists in the ablated region. Psychomotor progress has been satisfactory, with full recovery of motor function on the left side of the body. Motor and intellectual development have been normal and the child goes to regular school.

ECG in sinus rhythm has never detected any abnormality, in particular no abnormality suggesting channelopathy. A family investigation including ECG and echocardiography on siblings and parents revealed nothing. Nor did a genetic analysis focusing on all the genes known to be associated with channelopathy reveal any abnormality.

In the end, the etiology of this malignant arrhythmia remains unknown.

Discussion

To our knowledge, this is the first report of successful catheter ablation of left VT achieved by transseptal access in a child of under 1 year of age.

The incidence of VT is low in pediatric patients without any underlying heart disease.⁵ Most cases concern idiopathic VT, commonly arising from the right ventricular outflow tract.^{5–8} Three cases of VT ablation via a transapical approach have been reported in the literature.^{2–4} In all 3 of these cases, the VT involved the posterior mitral annulus. The children's ages were 10, 11, and 14 months. It should be noted that in 2 of the 3 cases, transapical access was

only attempted secondarily, after failure of transseptal and retroaortic approach.

In our procedure, the main technical difficulties were associated with the transseptal approach and manipulation of the ablation catheter. Despite the use of the smallest curve available, it was still far too big for use in such a small heart. The steerable sheath proved entirely ineffective.

For the mapping, primary activation was localized fairly quickly, even though it was limited (–19 ms) compared with what is usually seen in adults. This was probably not a reentry because no mid-diastolic potential was recorded and the tachycardia did not respond to ventricular stimulation.

Despite decreasing prevalence, sudden infant death syndrome remains the leading cause of death in babies aged 1–12 months in the developed world, affecting some 1 in 2000 births in the United States.⁹ Various pathogenic mechanisms are suspected and certain risk factors have been identified. The role of arrhythmia may be underestimated because autopsies often find nothing and genetic analyses are uninformative.

In the case reported here, the initial cardiologic investigations did not find any abnormality or establish the etiology. In particular, ECG findings did not point to channelopathy. Explorations in first-degree relatives found nothing: all findings were perfectly normal.

The overall clinical picture did not indicate myocarditis. Moderate rises in CRP and troponin Ic were attributed to the ventricular arrhythmia as well as repeated shocks. There

was never any fever and both CRP and troponin levels soon returned to normal. ECG findings remained normal between episodes of ventricular arrhythmia. Unfortunately, cardiac MRI was not performed before the ablation procedure. The MRI abnormalities observed following ablation were probably associated with the delivery of radiofrequency energy. No subepicardial uptake of contrast medium—classically seen in myocarditis—was observed. In contrast, there was delayed subendocardial enhancement associated with hypokinesia, corresponding to the tissue targeted for ablation.

Conclusion

We have reported the first catheter ablation of left VT via a percutaneous, transseptal approach in a 10-month-old baby on ECMO suffering from malignant arrhythmia refractory to pharmacologic treatment. The cause of this malignant arrhythmia was not determined. After 30 months of follow-up, no recurrence of the arrhythmia has been observed.

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