

Successful ablation of refractory neonatal atrial flutter



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Introduction

Atrial flutter is an uncommonly diagnosed arrhythmia in the neonatal period. The majority of cases can be successfully converted to sinus rhythm by electrical cardioversion or atrial overdrive pacing. Sinus rhythm usually is maintained without the need for ongoing arrhythmic medication.¹ Ablation of atrial flutter in neonates for arrhythmia control has yet to be reported.

Case report

The patient was a male infant born at 38 weeks' gestation. Atrial flutter was diagnosed *in utero* at 35 weeks of gestation, at which time no hydrops fetalis was present and no treatment was commenced. At 6 hours of life, the infant developed tachycardia. The electrocardiogram (ECG) showed atrial flutter with 2:1 atrioventricular conduction (Figure 1). Initially the rhythm could be reverted to sinus by biphasic direct current (DC) cardioversion. However, atrial flutter recurred despite intravenous amiodarone infusion. The infant developed heart failure on day 3 of life. An echocardiogram demonstrated a structurally normal heart with significant biatrial dilation and moderately impaired biventricular function. No intracardiac thrombus was evident. Transesophageal overdrive pacing was attempted but failed. DC cardioversion (at 1 J/kg) could revert the rhythm to sinus, but atrial flutter recurred within 12 hours. In total, DC cardioversion was attempted 6 times with varying combinations of intravenous amiodarone (15 µg/kg/min), intravenous esmolol (up to 175 µg/kg/min), oral digoxin (8 µg/kg/day), sotalol (4.5 mg/kg/day), and flecainide (4 mg/kg/day), but none of them could maintain the rhythm in sinus, and atrial flutter recurred within 12 hours. In view of the persistent ventricular dysfunction with biatrial dilation, electrophysiologic study with potential radiofrequency ablation of the atrial flutter was performed on day 14 of life.

KEYWORDS Neonatal atrial flutter; Radiofrequency ablation; Infant

ABBREVIATIONS DC = direct current; ECG = electrocardiogram (Heart Rhythm Case Reports 2015;1:245–248)

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A 6Fr transesophageal electrode was placed to record left atrial activation and allow pacing. A 5Fr Therapy™ ablation catheter (St. Jude, Saint Paul, MN) (nonirrigated 4-mm tip) was placed via right femoral venous access for mapping in the right atrium. The EnSite™ 3-dimensional cardiac mapping system (St. Jude, Saint Paul, MN) was used for electroanatomic mapping. Activation sequence confirmed a typical counterclockwise macroreentrant atrial flutter (Figure 2). Entrainment pacing confirmed the cavotricuspid isthmus was in the primary tachycardia circuit. Linear lesions were created using the point-to-point technique through radiofrequency with gradual withdrawal of the catheter from the tricuspid ring to the inferior vena cava. Four applications (mean energy delivery of 8 W, mean temperature 58°C) were given before atrial flutter was terminated (Figure 3). Further ablation was performed to complete the ablation line along the cavotricuspid isthmus. Total ablation time was 3 minutes 49 seconds. Double potentials and unidirectional block along the ablation line were demonstrated (Figure 4).

Atrial flutter recurred 12 hours after ablation. Electrophysiologic study was repeated on day 16 of life. Reinforcement of the ablation line was performed using a different catheter positioning, and slightly higher energy levels up to 13 W were achieved. Bidirectional isthmus block was confirmed by differential pacing from a 5Fr, 20-pole diagnostic catheter placed along the tricuspid ring from the right lateral atrial wall over the isthmus to the septal wall. There was a greater than 50-ms time difference in both directions. Neither inducible atrial flutter nor recurrence was observed after this ablation. Average radiation dose was 0.7 µGm² per procedure. Overall radiation time for both procedures was less than 5 minutes of fluoroscopy.

Ventricular function improved significantly within days of conversion to sinus rhythm. A small pericardial effusion developed after the procedure but resolved within 1 week. A 24-hour ambulatory ECG excluded further recurrence of atrial flutter. No further recurrence was evident 6 months after the procedure without antiarrhythmic medication.

Discussion

To our knowledge, this is the first report of successful treatment of neonatal refractory atrial flutter using

KEY TEACHING POINTS

- Radiofrequency ablation of neonatal flutter after failure of all other treatment options is an option but rarely necessary. Use of a 5Fr ablation catheter and a diagnostic transesophageal catheter limits vascular damage.
- Use of a 3-dimensional system keeps radiation to a minimum and allows an approach using only 1 intravascular catheter. System resolution is just adequate for neonatal body size.
- Bidirectional block over the cavotricuspid isthmus is the gold standard for proving successful ablation and a line without gaps. In the neonatal setting, the transesophageal catheter did not reliably exclude a gap. A multipolar 5Fr catheter properly placed along the tricuspid valve can be used instead.

radiofrequency ablation. There is a single case report of radiofrequency used to ablate the atrioventricular node in a 7-week-old infant who was reported to have refractory atrial

flutter requiring extracorporeal membrane oxygenation support.² A pacemaker was implanted, but atrial flutter could not be reverted.

Most often neonatal atrial flutters can be easily converted to sinus rhythm. Various antiarrhythmic medications, such as digoxin, quinidine, procainamide, flecainide, sotalol, propranolol, and amiodarone, all have been used alone or in combination, with varying reported success rates. DC cardioversion usually is extremely effective, with a success rate greater than 85%, whereas transesophageal pacing seems to have a lower success rate. Sometimes a wait-and-see approach can be adopted until possible spontaneous resolution in hemodynamically stable infants. Recurrence is uncommon.¹

The indication for ablation in children weighing less than 15 kg or younger than 4 years should be limited to patients with incessant tachycardia or tachycardia-induced cardiomyopathy unresponsive to a multiagent antiarrhythmic regimen.³ An underlying abnormality causing therapy-refractory flutter was considered, although there was no evidence of channelopathy in any ECG of the child or his family members. Genetic testing has not been performed. Intracardiac signals and conduction time recorded after ablation were normal.

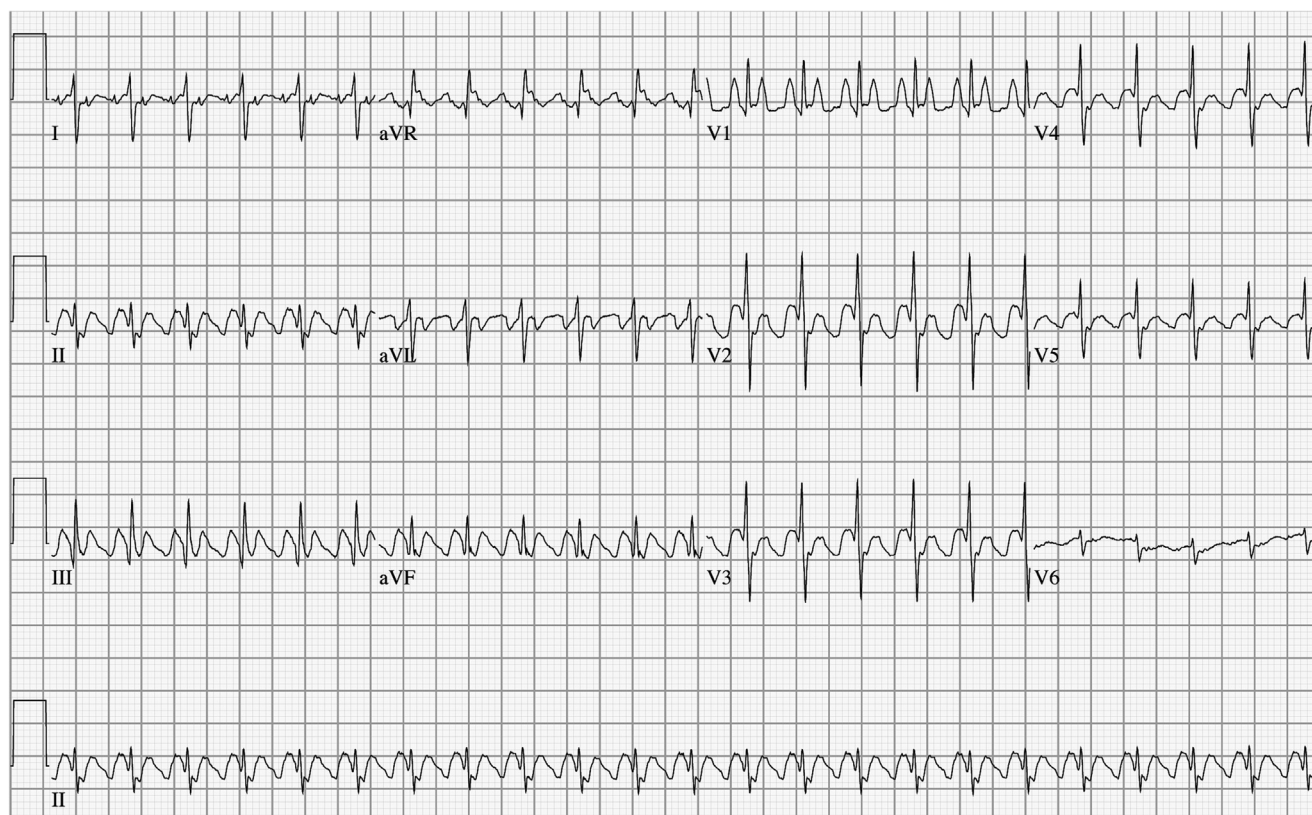


Figure 1 Electrocardiogram showing atrial flutter with atrioventricular 2:1 block and ventricular rate of 170 bpm.

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