CASE REPORT

Catheter ablation of sinoatrial re-entry tachycardia in a 2 month old infant

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Successful catheter ablation of sinoatrial re-entry tachycardia in an infant has not been previously reported. This procedure is described in a 2 month old boy with tachycardia induced cardiomyopathy.

Sinoatrial re-entry tachycardia (SART) has been estimated to account for anywhere between 2–17% of all arrhythmias^{1,2}; nonetheless, the literature on ablation is limited to small numbers of cases^{3–6} and only two series of more than five patients.^{7,8} SART is characterised by paroxysmal tachycardia with a P wave morphology identical to sinus rhythm, induction, and termination with atrial extrastimuli, and an atrial activation sequence from cranial to caudal as in normal sinus rhythm.⁹ This electrocardiographic similarity to sinus rhythm may be a cause of underdiagnosis. In the ablation cases reported to date, the youngest patient was 15 years of age.⁸ Most, however, were significantly older. We describe successful ablation of SART in a 2 month old infant who presented with heart failure.

CASE DESCRIPTION

The patient, a 2 month old boy, was born prematurely at 34 weeks after an otherwise uncomplicated pregnancy. The mother was in good health, was not on any medication, and was not a known substance abuser. The patient's weight at birth was 2140 g. His parents had noticed difficulty in completing feedings and had the impression that he was dyspnoeic during feeding and changing. He was referred to our institution by his paediatrician because of tachycardia at initial examination. When the patient was 2 months, we saw a 2780 g infant (weight below 0.6th centile) with a resting respiration rate of 35 breaths/min, pulse rate of 190 beats/min, and blood pressure 80/40 mm Hg. His temperature was 37°C. Cardiac ultrasound showed a dilated left ventricle (left ventricular end diastolic dimension above the 97th centile) with globally impaired systolic function but no other structural abnormalities. The resting ECG at admission showed apparent sinus tachycardia. During clinical observation, the heart rate varied between 110 and 235 beats/min. Resting heart rate was above 130 beats/min for most of the day, with sudden rate increases to above 200 beats/min, which lasted for up to two hours. P wave morphology during these episodes was indistinguishable from sinus rhythm. A diagnosis of SART was entertained based on these findings; no other cause for sinus tachycardia could be determined.

In light of the haemodynamic compromise to the patient, it was decided to withhold potentially negative inotropic antiarrhythmic drugs in favour of catheter ablation. At electrophysiological study, a 4 French quadripolar catheter (Bard, Lowell, Massachusetts, USA) and a 5 French quadripolar ablation catheter (Medtronic, Minneapolis, Minnesota, USA) were advanced to the right atrium under fluoroscopic

Figure 1 Endocardial recording at the successful ablation site during sinoatrial re-entry tachycardia at a cycle length of 310 ms. Note the fragmented local atrial electrogram (44 ms duration) 10 ms before onset of the P wave in the surface ECG, the negative deflection in the unipolar tip electrogram, and the craniocaudal activation pattern from maps 1 to 4. HRA, high right atrial reference catheter.

guidance through a right and left femoral venous approach. Sinus rhythm with a cycle length of 450 ms was present at baseline. Tachycardia with a cycle length of 300–320 ms was both inducible and could be terminated with atrial burst stimulation and atrial extrastimuli. P wave morphology and craniocaudal atrial activation were maintained during tachycardia, consistent with the diagnosis of SART. The endocardium was mapped during the arrhythmia. The site of earliest atrial activation relative to onset of the P wave in the surface lead, with an initially negative unipolar tip electrogram from the mapping catheter, was identified anterolaterally in the high right atrium, near the right atrial-superior vena cava junction. Before radiofrequency (RF) energy was applied, high output (10 mA) pacing was performed using the ablation electrode to exclude phrenic nerve stimulation and thus reduce the risk of thermal injury of the right phrenic nerve. RF energy was delivered during tachycardia for 60 s in the temperature controlled mode to a maximum of 55°C. Three pulses were required to abolish the arrhythmia.

At the successful ablation site, the atrial electrogram was 44 ms in duration and fragmented, initially negative in the unipolar tip electrogram, and 10 ms before onset of the P wave in the surface ECG (fig 1). After initial acceleration of the tachycardia to a cycle length of 265 ms at 8 s RF delivery, gradual deceleration was seen with termination of the tachycardia at 35 s (fig 2). An atrial escape rhythm with superior P

Abbreviations: RF, radiofrequency; sinoatrial re-entry tachycardia

Figure 2 Surface ECG during radiofrequency delivery showing termination of the tachycardia followed by a low atrial escape rhythm with cycle length 550 ms.

wave axis and cycle length 550 ms existed for the following six minutes, after which sinus rhythm recovered suddenly at a cycle length of 420 ms. SART was no longer inducible following this RF delivery. Despite the measures described, the patient was found to have paralysis of the right hemidiaphragm following the procedure. At eight weeks' follow up the infant was thriving, left ventricular end diastolic dimension had regressed to P50, and the ECG showed normal sinus arrhythmia. Moreover, the right hemidiaphragmatic paralysis had resolved spontaneously.

DISCUSSION

The patient described is the youngest reported with ablation for SART. While this is arguably an aggressive approach in such a young infant, the severely compromised left ventricular function was felt to warrant the choice of a primarily non-pharmacological treatment. Heart failure has been described in adults with SART,^{3,4} but concomitant hypertensive or ischaemic heart disease was always present. Data concerning children are lacking. In a group of 12 patients between 10 months and 19 years of age with ectopic atrial tachycardia, however, Walsh and colleagues¹⁰ did observe impaired systolic function, which normalised after successful treatment of the arrhythmia. We observed the same occurrence of a reversible tachycardiomyopathy in our patient with SART. Endocardial mapping of SART has invariably been directed towards pinpointing the site of earliest atrial activation relative to the P wave in the surface ECG. A wide range of timing has been reported, from -20 ms⁸ to -100 ms,⁴ on average between -35 and -45 ms relative to P wave onset. Timing in our patient was considerably shorter, with earliest atrial activation at the successful ablation site at only 10 ms before onset of the P wave. Given that all other reports concern adults, it is conceivable that this discrepancy arose because our patient was an infant weighing < 3000 g.

The atrial electrogram at the successful ablation site in SART has been described as prolonged and fragmented, with a duration of 50–125 ms.^{6–8} This morphology, also observed in our patient with a fragmented 44 ms electrogram, is presumably a reflection of an area of slow conduction in the re-entry circuit. In addition to early onset and fragmentation, Ivanov and colleagues⁸ have reported that acceleration of the tachycardia during RF is a marker for successful ablation sites. This also occurred in our patient, before deceleration and termination of the tachycardia. The subsequent low atrial escape rhythm followed by abrupt recovery of normal sinus rhythm was believed to indicate a transient period of complete sinoatrial exit block. Despite the measures taken to avoid injury to the right phrenic nerve, our patient was found to have transient paralysis of the right hemidiaphragm. This undoubtedly reflects the creation of a transmural lesion and underlines the need for extreme caution and minimum number of lesions if contemplating RF ablation in such small infants. Bearing this in mind, we conclude that RF ablation may be a feasible treatment option for highly symptomatic or haemodynamically threatening SART in infants.

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