

## An incessant atrial tachycardia originating from epicardial left atrial appendage in a 12-year-old girl: ablation or excision?

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*To the Editor:* Left atrial appendage (LAA) is one of the major sources of atrial arrhythmias in children.<sup>[1]</sup> But atrial tachycardia (AT) with an epicardial LAA origin is uncommon and usually arises from the base.<sup>[2]</sup> Catheter ablation (CA) may be an important strategy for patients with atrial arrhythmia,<sup>[3]</sup> especially due to the LAA firing trigger.<sup>[4]</sup> Due to a large number of trabeculations and true epicardial location, endocardial CA in LAA may fail or even cause fatal tamponade.<sup>[2,5]</sup> The epicardial CA may be an alternative approach.<sup>[6]</sup> Because the LAA has a very thin wall and may be prone to perforation, caution should be taken when LAA ablation is performed,<sup>[4]</sup> even during an epicardial ablation. There may be recurrence of AT, and surgical left atrial appendectomy (SAE) may be required.

We report one child with incessant AT that was found to be arising from epicardial LAA, in whom CA was successfully done through the epicardial approach with subsequent surgical appendectomy without any complications.

A 12-year-old girl weighing 46 kg and 165 cm in height (with a body surface area of 1.45 m<sup>2</sup>) presented with almost incessant drug-refractory AT was referred for radiofrequency CA. She had a 5-month history of palpitations and the electrocardiogram (ECG) showed persistent AT with variable (3–1:1) conduction, and a maximum heart rate of 150 beats/min [Figure 1A].

The echocardiogram revealed enlarged left atrium diameter (LAD, 37 mm; normal range <35 mm). The left ventricular end-diastolic and end-systolic diameters (left ventricular end-diastolic diameter [LVEDD] and left ventricular end-systolic diameter [LVESD]) were 48 (normal range <48 mm) and 30 mm, respectively, with a normal left ventricular ejection fraction (LVEF) of 67% [Supplementary Table 1, <http://links.lww.com/CM9/B127>]. The markers of myocardial injury were normal and no inflammatory edema of myocardial tissue was found by echocardiography. The ECG was repeated and

the results showed that the AT was in the range of 140 to 180 beats/min without dynamic ST change. P-wave morphology was negative in leads I and aVL and positive in the inferior leads and lead V<sub>1</sub>, suggestive of the origin of left atrial localization.<sup>[1]</sup> The AT lasted for >2 days with elevated N-terminal pro-B-type natriuretic peptide (NT-proBNP; 2586 pg/mL, normal range <285 pg/mL), elevated alanine amino transferase (ALT) (109 U/L, normal range <75 U/L), and elevated aspartate amino transferase (AST) (43 U/L, normal range <38 U/L) level.

After ruling out reversibility and other causes, the patient was admitted to the hospital for electrophysiology examination and ablation. Transesophageal echocardiography was performed before ablation to verify the absence of a left atrial thrombus. Patients had been treated with anticoagulants before ablation.

As previously described,<sup>[2]</sup> an electrophysiological procedure was performed with standard catheter positions in the coronary sinus, His bundle region, and high anterolateral right atrial wall after informed consent was obtained. Following a single transeptal puncture by use of the modified Brockenbrough technique and an 8 Fr Swartz SL transeptal sheath (St. Jude Medical, St. Paul, MN, USA), systemic anticoagulation was made with administering intravenous heparin (50 U/kg), with additional 1000 U boluses every hour after the first infusion. An ablation catheter was positioned at the left atrium through a transeptal puncture. Then activation mapping with the Ensite Precision (St. Jude Medical, Inc.) cardiac mapping system was performed at the left atrium.

Activation mapping revealed a distal-to-proximal atrial activation sequence in coronary sinus (CS). Further endocardial mapping demonstrated activation time of –112 and –105 ms (relative to the onset of the A wave at CS<sub>9-10</sub>) in the right ventricular outflow tract and LAA, respectively.

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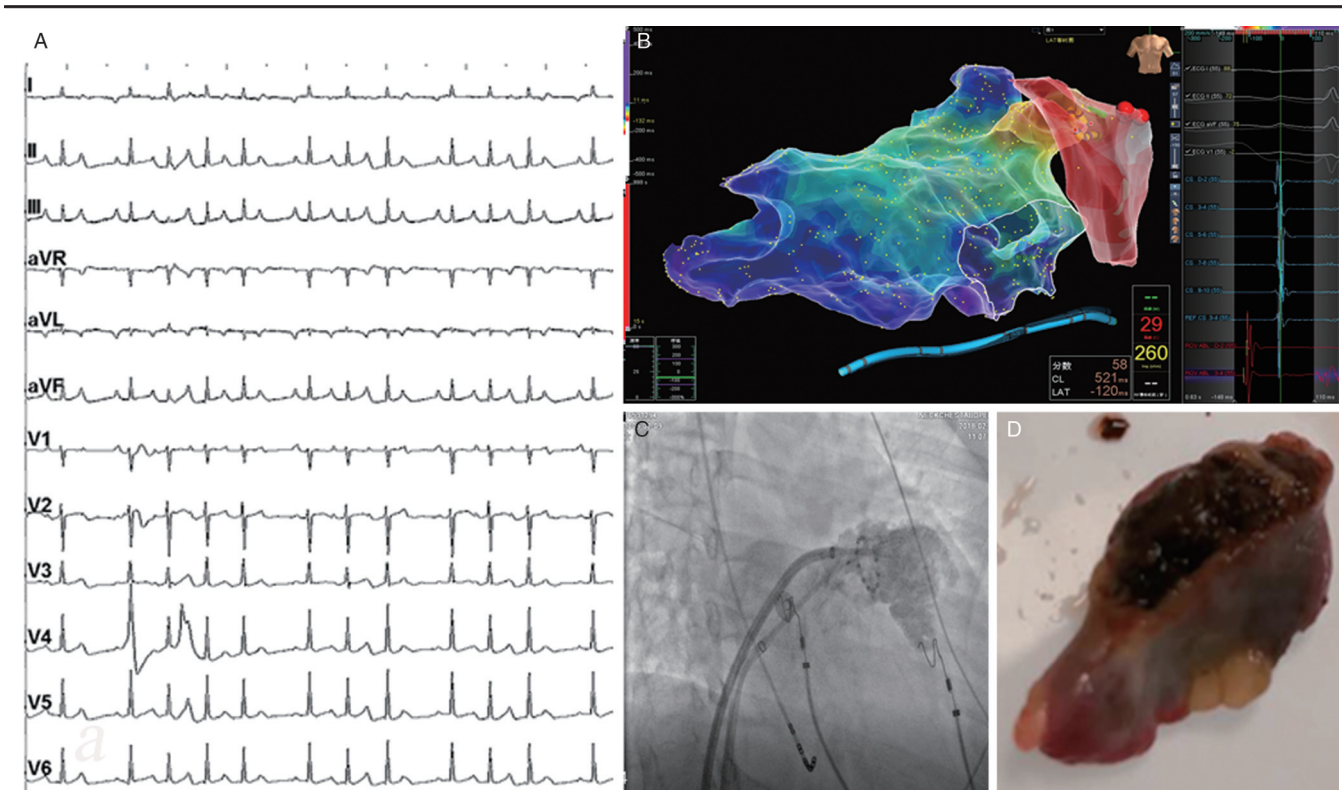
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**Figure 1:** Twelve-lead ECG of tachycardia (A), electroanatomic maps (B), angiograms (C), and a macroscoping findings (D) of the LAA. (A) From ECG, the P-wave morphology showed a negative P-wave in leads I and aVL and a positive P-wave in the inferior leads and lead V<sub>1</sub>. (B) The earliest activation at the epicardial apex of the LAA was 120 ms ahead of CS<sub>9-10</sub>. The maroon tags represent radiofrequency applications at the earliest activation sites which terminated the AT. (C) The Lasso catheter was placed at base of LAA. (D) The middle sites of LAA epicardial surface had lesions from CA. AT: Atrial tachycardia; CA: Catheter ablation; ECG: Electrocardiogram; LAA: Left atrial appendage.

A TactiCath Quartz ablation catheter (St. Jude Medical) was used for ablation. Radiofrequency energy applications at multiple sites with early endocardial activation in LAA ostium did not terminate the tachycardia using a temperature of 43°C and power settings of 17 to 25 W.

The patient was referred for surgery. As previously described,<sup>[2,7]</sup> the patient underwent general anesthesia with a double lumen endotracheal tube for selective single-lung ventilation, and then was placed in the supine position with bilateral forearms alongside the body and slightly below the table to expose the axillary regions. After right single-lung ventilation, the procedure was begun on the left side in the fourth intercostal space on the midaxillary line. The left-side pericardium was opened at 2 cm posterior to the phrenic nerve to expose LAA. After the left-side pericardium opened, LAA with multiple lobes was seen.

Then activation mapping was performed on the epicardial surface of LAA and demonstrated that the earliest activation (-120 ms) during tachycardia was found at the apex of upper lobe [Figure 1B]. LAA angiography demonstrated that this site was adjacent to the endocardial site of earliest activation [Figure 1C]. At this site, radiofrequency application using a FlexAbility irrigated ablation catheter (St. Jude Medical) eliminated the tachycardia within 5 s of onset of energy (a temperature of 43°C, irrigation rate of 30 mL/min, and power settings

of 30 W). After adequate power was achieved, the AT could not be induced.

After ablation, retraction sutures were used to facilitate the exposure. The LAA excision was performed. From macroscoping findings, the middle sites of LAA epicardial surface had lesions from CA [Figure 1D]. The surgical procedure took 90 min without any complications.

Seven days after the procedure, the echocardiogram showed that LAD, LVEDD, and LVESD decreased to 29, 48, and 32 mm, respectively, and the LVEF increased to 61%. The blood chemistry data were improved with normal NT-proBNP (133 pg/mL), GPT (47 U/L), and GOT (28 U/L) level. During the 12-month follow-up, the patient has remained free of symptomatic arrhythmias.

This is a rare report on one child illustrating an AT with an epicardial LAA origin that was successfully ablated through epicardial approach with subsequent surgical appendectomy. The free of AT and a decrease in LAD and NT-proBNP were achieved during the follow-up. On the whole, the preliminary results were inspiring.

The LAA is the remnant of the original embryonic left atrium that develops during the third week of gestation,<sup>[8]</sup> so it is a structure anatomically appended to the main body of the LA, which is located between the left upper pulmonary vein and the left ventricle. The LAA has a wide

perimeter that interfaces with the atrial musculature.<sup>[4]</sup> In the LAA, thick cords of pectinate muscle intervene with thin-walled tissue.<sup>[8]</sup> The anisotropic junctional tissue, with a complex fiber orientation, results in electrophysiological properties that may predispose this region to be the source of the arrhythmia.<sup>[3]</sup>

In children, left atrium in size is small and increase as ages. The LAA lies within the confines of the pericardium, and thus its emptying and filling may be affected by left ventricular function. Currently, no relevant guideline exists for the epicardial LAA AT treatment in children. Although, endocardial CA was considered to be the best strategy for the child in the present study if AT could be terminated without any complications. The complex anatomy of the LAA makes it a difficult intracardiac structure in which to manipulate catheters safely. In one report, firm forward pressure was exerted on the ablating catheter, causing the appendage to straighten. However, this maneuver could result in LAA perforation and thus was not attempted in the reported case.

Thus, the epicardial CA may be an alternative approach to endocardial CA. CA using an epicardial access by a pericardial puncture may be necessary and is a proven feasible route for the management of a variety of arrhythmias.<sup>[6]</sup> But the response to radiofrequency applications in epicardial areas differs markedly from the response in endocardial areas because incomplete ablation might result from a lack of contact with the ablation electrode and the thermal homeostatic effect of cavitory saline infusion on the LAA muscles. And the limited space of pericardial cavity makes it still difficult to manipulate catheter safely.

The LAA is one of the major sources of ATs in children.<sup>[1]</sup> The mechanism of AT originating in the LAA is unknown. Intracellular recordings demonstrated that the mechanism was abnormal automaticity.<sup>[2]</sup> Histology showed that spontaneous activity arose in an area with abnormal cells.<sup>[2]</sup> However, CA in an atrial appendage may fail or there may be recurrence of AT, and surgical appendectomy may be required.<sup>[2,5]</sup> Because the LAA has a very thin wall and may be prone to perforation, caution should be taken when LAA ablation is performed,<sup>[4]</sup> even if epicardial ablation.

In the current case, because of its favorable risk-benefit ratio rather than a second attempt at CA, we chose

epicardial CA with subsequent SAE. The patient's heart function improved with significantly decreased LVEDD, LAD, and enhanced New York Heart Association functional class.

The excision of LAA may be a useful strategy for children with incessant AT originating from epicardial LAA. However, the long-term safety and efficacy of SAE in children should be further estimated.

### Conflicts of interest

None.

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