Percutaneous transhepatic venous access for atrial tachyarrhythmia ablation in patients with single ventricle and interrupted inferior vena cava

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Introduction

Patients with congenital heart disease (CHD) who have undergone corrective and/or palliative surgery are at risk of developing different types of tachyarrhythmias, and their invasive management can represent a challenge to the electrophysiologist because of their complex anatomy, extensive surgical reconstructions, and use of intracardiac and extracardiac baffles and shunts, which limit conventional access to the cardiac chambers.[1] In cases in which there are interruptions or anomalies of the inferior vena cava (IVC), a superior approach or a retrograde aortic arterial access to gain entry into the cardiac chambers may be used, but catheter stability and manipulation can prove very difficult in comparison with a standard femoral venous approach.[2–7]

Percutaneous transhepatic venous access is an alternative in patients with no inferior venous access into the heart. Case studies of patients undergoing various cardiac interventions have reported complication rates of less than 5% using this approach.[8,9] Although most cases have been reported in the pediatric population, there is some published data in adult patients.[1–3]

We present the case of an adult woman with complex CHD who underwent ablation of atrial tachyarrhythmias using a transhepatic venous access after failed antiarrhythmic drug therapy.

KEYWORDS Atrial tachyarrhythmia; Congenital heart disease; Interrupted inferior vena cava; Single ventricle; Transhepatic percutaneous venous access

(Heart Rhythm Case Reports 2019;5:31–35)

Drs Garcia and Saenz received minor grants from Biosense Webster.

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KEY TEACHING POINTS

- Percutaneous transhepatic access is a safe and feasible method for atrial tachyarrhythmia ablation in patients with complex congenital heart disease and interrupted inferior vena cava.
- The use of long steerable sheaths via percutaneous transhepatic venous access greatly improves catheter stability and manipulation during mapping and ablation of atrial tachyarrhythmias.
- Closure of the hepatic parenchyma with vascular coils is a safe option to prevent hemorrhagic complications in patients undergoing percutaneous transhepatic access using large-caliber sheaths.

Case report

The patient was a 41-year-old woman with complex cyanotic CHD consisting of a single ventricle with left morphology and double inlet/outlet, transposition of the great vessels, left-sided interrupted IVC (with drainage via azygos system into the superior vena cava [SVC]), and suprahepatic veins draining directly into the right atrium (RA). She underwent pulmonary artery banding at the age of 1 month. Later, at 3 years of age, she underwent a Kawashima procedure. Because of the dilatation of persistent left SVC draining into the coronary sinus, she underwent percutaneous closure of the SVC with an Amplatz device at age 30.

At the age of 39 years, she began to experience paroxysmal palpitations associated with cyanosis and shortness of breath, with documentation of an atrial tachycardia. Despite treatment with amiodarone and beta-blockers, the patient continued to have palpitations and suffered a syncope, for which she was hospitalized. Twelve-lead

https://doi.org/10.1016/j.hrcr.2018.10.005

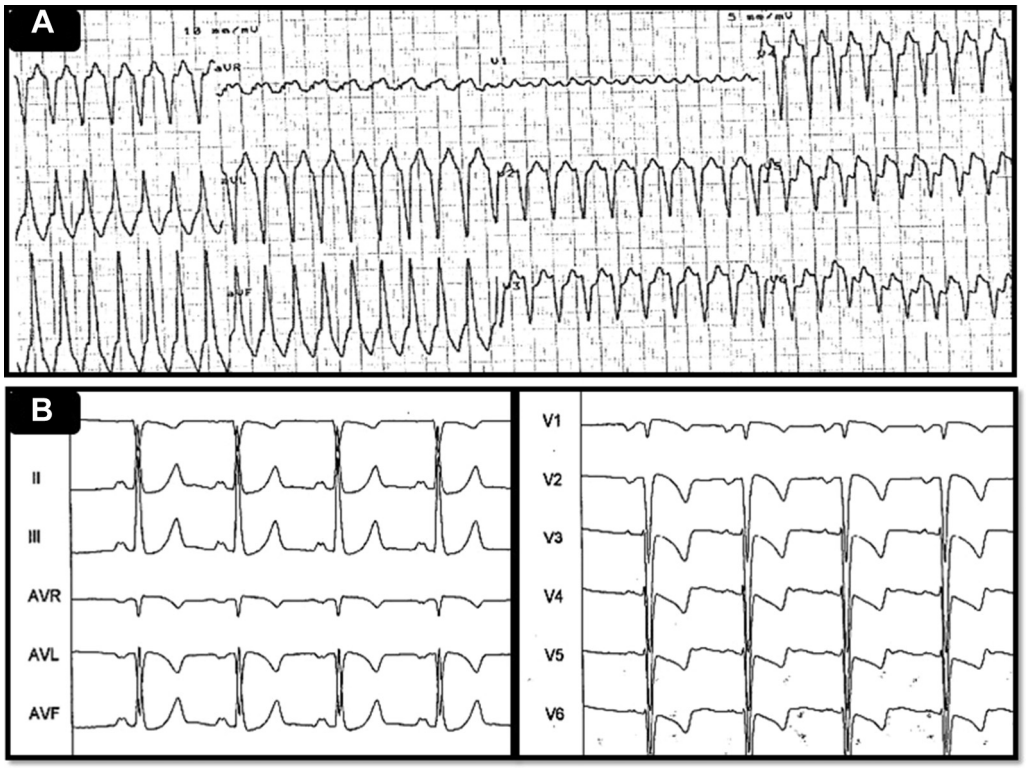

Figure 1 **A:** The 12-lead electrocardiogram (ECG) represents a regular usual complex tachycardia obtained in the emergency department. **B:** The 12-lead ECG in sinus rhythm.

electrocardiogram (ECG) showed a regular usual complex tachycardia. The patient underwent synchronized cardioversion with restoration of sinus rhythm (Figure 1).

We decided to proceed with an invasive strategy because the patient was refractory to antiarrhythmic drugs. The procedure was performed under general anesthesia, and the preoperative international normalized ratio was 1.29. Using bilateral femoral vein access, a 9F intracardiac ultrasound catheter (View Flex Xtra, Abbott, Lake Bluff, IL) and a 6F quadripolar diagnostic electrophysiology catheter (Inquiry, Abbott) were advanced via the azygos vein into a position posterior to the RA, to allow for visualization of intracardiac structures and to obtain far-field RA electrograms. Percutaneous transhepatic venous access was performed using a percutaneous 5F micropuncture kit (Neff Percutaneous Access Set, Cook, Bloomington, IN) with an 18 cm/21 gauge needle inserted along the right costochondral space with the anterior axillary line under sonographic and fluoroscopic visualization to guide passage of the needle through the liver parenchyma until the middle hepatic vein lumen was reached. Intravascular access was confirmed with injection of intravenous contrast reaching the suprahepatic vein and RA. A 0.018-inch microwire was advanced into the RA. A 5F long sheath dilator was then advanced, and the wire was exchanged for a 0.035-inch wire to allow insertion of an 8.5F bidirectional deflectable sheath (Agilis NxT, Abbott; Figure 2). Unfractionated heparin in boluses was administered throughout the case, with a goal activated clotting time between 300 and 350 seconds.

Using a multipolar circular mapping catheter (Inquiry AFocus II HD, Abbott), a high-density, 3-dimensional electroanatomical map of the RA was created using an EnSite velocity mapping system (Abbott). Areas of low bipolar voltage (<0.1 mV) consistent with scar were found on the posterolateral RA. The circular catheter was then exchanged for a 4-mm irrigated-tip ablation catheter (Therapy Cool Flex, Abbott). Burst pacing at 270 ms from the high RA induced intra-atrial reentrant tachycardia (IART) with a tachycardia cycle length (TCL) of 330 ms and atypical ECG characteristics. Atrial capture with concealed entrainment from the suprahepatic vein–RA isthmus showed a postpacing interval of 360 ms. A double line of ablation along the suprahepatic tricuspid isthmus terminated the arrhythmia. After we exchanged the ablation catheter for 2 4F diagnostic catheters advanced through the deflectable sheath and positioned on opposite sides of the ablation line, bidirectional block across the suprahepatic tricuspid isthmus was confirmed (Figure 3A). Postablation, burst pacing from the RA induced a second IART, with a TCL of 290 ms. Atrial capture with concealed entrainment and a postpacing interval between 290 and 320 ms was seen along the posterolateral RA scar (Figure 3B). During linear radiofrequency ablation along the longitudinal aspect of the scar connecting to the suprahepatic vein, the tachycardia changed, with a TCL of 600 ms. After we confirmed bidirectional block along the posterolateral RA with the double-catheter technique, the activation map of this third tachycardia showed a focal source with the earliest atrial activation near the ostium of the coronary sinus (–44 ms pre–P

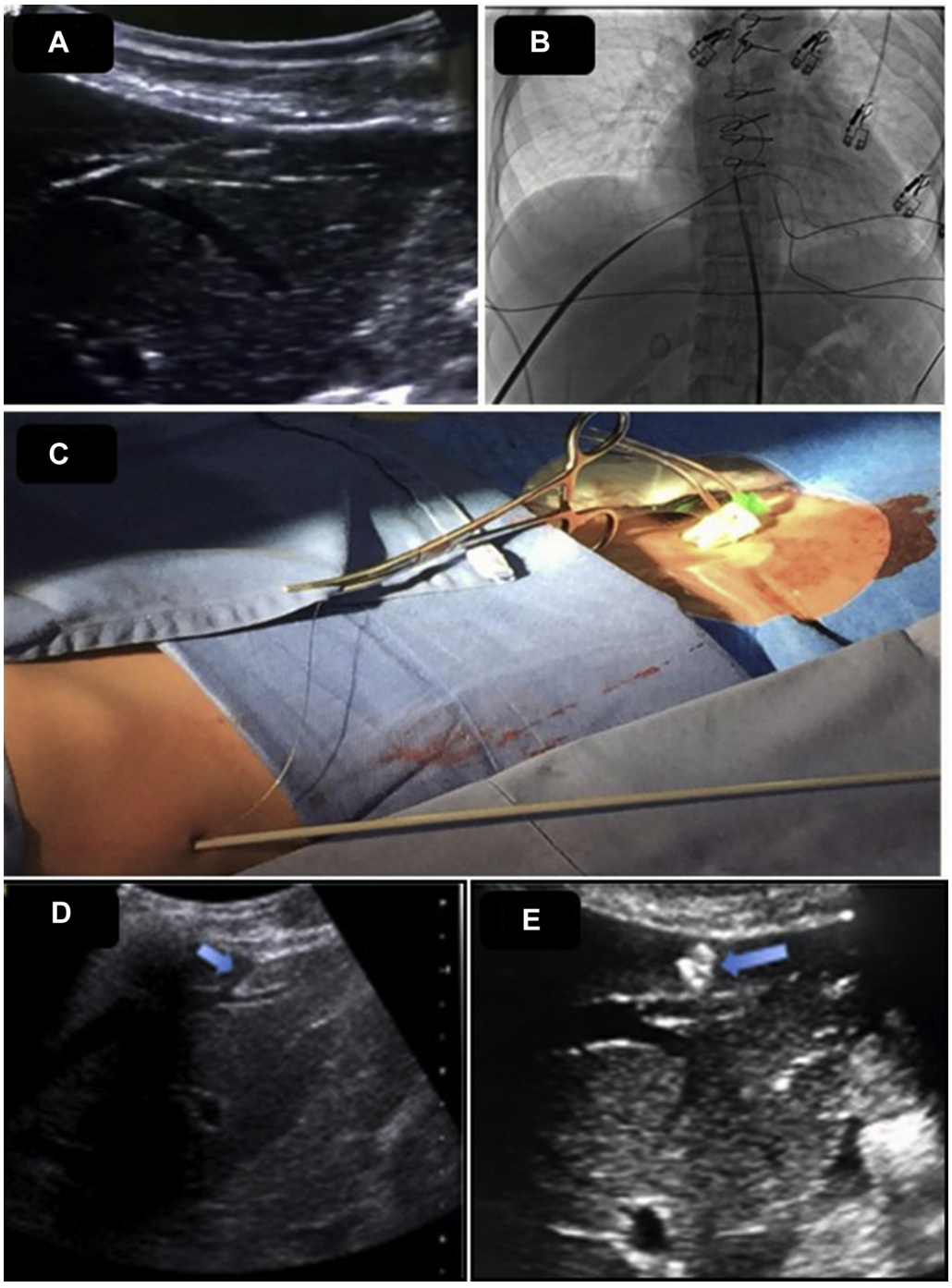

Figure 2 Percutaneous transhepatic venous access. **A:** Ultrasound-guided visualization of microneedle puncture to reach the middle hepatic vein. **B:** Access to the right atrium guided by fluoroscopy through a 5F long sheath dilator and the 0.035-inch wire. **C:** Once hepatic venous access is obtained, a large 8.5F deflectable sheath is advanced over a guidewire into the right atrium. **D:** After retrieval of the deflectable sheath, there is ultrasound evidence of an intraparenchymal tract (*blue arrow*). **E:** A vascular embolization coil is placed at the parenchymal-tract interface just outside of the vessel lumen, successfully closing the hepatic tract (*blue arrow*).

wave). Radiofrequency applications near the coronary sinus ostium terminated the tachycardia, and sinus rhythm was restored (Figure 3C). Postablation maneuvers, including burst pacing and programmed electrical stimulation from multiple RA sites, did not induce further arrhythmias.

Finally, intravenous protamine was administered to revert the heparin effect. The ablation catheter was removed, and the 0.035-inch × 150-cm guidewire was readvanced to allow exchange of the 8.5F steerable sheath for a long vascular introducer (8F, 11 cm), which was slowly withdrawn as contrast was injected until the interface between the hepatic veins and the liver parenchyma was identified within the residual tract left by the steerable sheath. The vascular introducer position was confirmed with ultrasound. The guidewire was slowly withdrawn, and a 0.038-inch, 3-cm × 5-mm vascular embolization

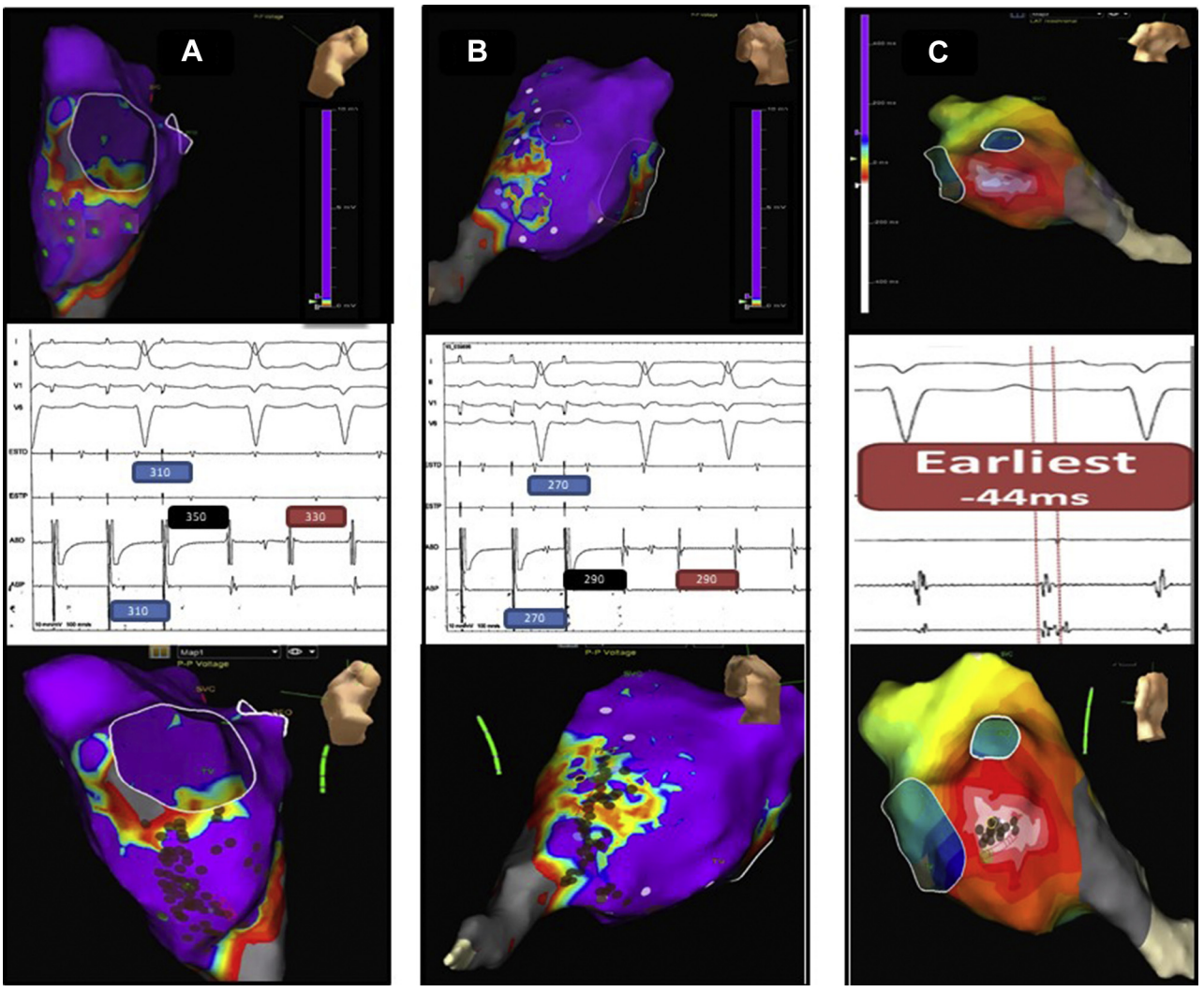

Figure 3 Atrial tachycardia ablation guided by a 3-dimensional mapping system. **A:** Intra-atrial reentrant tachycardia (IART) with a tachycardia cycle length of 330 ms and concealed entrainment from the suprahepatic vein–right atrium (RA) isthmus successfully terminated with linear ablation achieving bidirectional block. **B:** IART with a tachycardia cycle length of 290 ms and concealed entrainment along the posterolateral RA scar successfully terminated with linear ablation and bidirectional block. **C:** Focal atrial tachycardia with tachycardia cycle length of 600 ms with earliest centrifugal activation just below the coronary sinus os, successfully eliminated with focal ablation lesions.

coil (Cook) was advanced inside the tract and released under fluoroscopic and ultrasound visualization, achieving total occlusion of the aforementioned tract at the vascular-parenchymal interface without compromising hepatic vein flow. There was no ultrasound evidence of capsular hematoma or intra-abdominal bleeding afterward. The patient had an uncomplicated postoperative course and was discharged 2 days later with a stable hemoglobin level and a normal abdominal examination. Clinical, electrocardiographic, and Holter follow-up at 3, 6, and 12 months after the procedure confirmed no arrhythmia recurrence.

Discussion

Atrial tachyarrhythmias in adults with CHD are relatively common because of the presence of postsurgical scars and chamber dilatation, providing the ideal substrate for reentrant and focal arrhythmias. Refinement in surgical techniques and advances in medical therapy have led to improved survival of these patients, often well into adulthood. The presence of baffles and vascular shunts as well as venous repetitive trauma from multiple invasive procedures during their lifetime can limit their venous vascular access options. Finally, the complex anatomical variants imposed by the congenital defect and its subsequent surgical corrective repairs will inevitably lead to challenging access into the cardiac chambers.[1] Congenital absence or stenosis of the IVC has an incidence of approximately 0.15% in the general population[10] and is characterized by the presence of collateral venous flow from the lower extremities through the azygos and hemiazygos systems to the SVC. In patients with such anomalies undergoing catheter ablation, either a superior central venous or retrograde aortic approach can present challenges for catheter manipulation and stability.[2–7]

The transhepatic percutaneous venous approach has been described since 1995 and was initially used as a technique to gain vascular access for cardiac catheterization in pediatric patients with CHD and limited venous central access, because it allows for easier manipulation of catheters inside the heart.[8,9] The procedure has a complication rate of less than 5% and includes bleeding (subcapsular liver hematoma, hemoperitoneum, hemothorax), infectious (colangitis, liver abscess, sepsis), and other liver-related complications (transaminitis, hepatic vein thrombosis, portal vein thrombosis).[1,2,8–12] In our case, the lack of direct venous inferior access to the RA and the presence of IART refractory to medical therapy led to the decision to proceed with transhepatic access with ultrasound and fluoroscopic guidance. The ultrasound allowed for visualization of needle entry through the liver parenchyma into the hepatic vein, and fluoroscopy with contrast injection confirmed the intravascular location.[13]

A crucial step in our procedure was the placement of the intracardiac echocardiography catheter via a femoral vein and advancing it into the azygos vein just behind the RA, allowing for real-time comprehensive visualization of the patient's unique cardiac anatomy and guiding the intravascular catheters during mapping and ablation of the different tachycardias. Given the limitations of vascular access in this patient, the placement of a quadripolar catheter via the femoral vein into the azygos system behind the RA allowed for recordings of far-field electrical activity from the RA, which served as stable reference electrograms during activation mapping. Following successful ablation, the final step was the removal of the steerable sheath and management of the residual tract in the liver parenchyma. Several case reports on transhepatic percutaneous access have described different techniques for tract closure, including manual compression, right lateral decubitus position,[14,15] vascular coils, Amplatz closure devices, gel foam, and bipolar radiofrequency ablation. We elected to close the tract using a vascular coil to guarantee hemostasis owing to the large size of the sheath employed (8.5F). When using a closure method into the hepatic tract, it is very important to ensure delivery of the coil system at the parenchymal-tract interface outside of the vessel lumen to avoid embolization. Fluoroscopic and ultrasound visualization of the coil location during delivery is essential for safe closure of the tract, thus reducing the risk of hemorrhagic complications such as subcapsular hematoma. Our patient had no hemorrhagic sequelae following sheath removal and tract closure.

Conclusion

This case illustrates a safe endovascular alternative using percutaneous transhepatic access via the suprahepatic veins to allow ablation of multiple RA tachyarrhythmias in patients with complex CHD and interrupted IVC.

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