CJC Pediatric and Congenital Heart Disease 3 (2024) 272–284

Review

Tachycardia-Induced Cardiomyopathy: A Case Series and a Literature Review

Wisam Abozaid, MD,[a,b] Samantha Wong,[a,b] Marc W. Deyell, MD, FRCPC,[a,c] Shubhayan Sanatani, MD, FRCPC, FHRS,[a,b] and Sakethram Saravu Vijayashankar, MD, MRCPCH[a,b]

[a] *Department of Pediatrics, the University of British Columbia, Vancouver, British Columbia, Canada*

[b] *Children's Heart Center, British Columbia Children's Hospital, Vancouver, British Columbia, Canada*

[c] *Department of medicine, St. Paul's Hospital, Vancouver, British Columbia, Canada*

ABSTRACT

Tachycardia-induced cardiomyopathy (TIC), also known as arrhythmia-induced cardiomyopathy or tachycardiomyopathy, is a reversible form of heart failure characterized by persistent tachyarrhythmias and associated ventricular dysfunction. TIC is characterized by the reversal of myocardial damage with resolution of the arrhythmia. Early diagnosis of TIC is imperative, as the treatment course is distinct from cardiomyopathy of other or unknown causes. However, distinguishing TIC from tachycardia secondary to increased catecholamines due to congestive heart failure can be very challenging. There are relatively few paediatric reports, and herein we present a case series of 48 paediatric patients with TIC from literature (2014-2024). We also present 4 illustrative cases with TIC seen at our site (BC Children's Hospital, Vancouver, Canada). The mean age in this case series was 6.98 ± 4.9 years. The majority of patients had ectopic atrial tachycardia (41.7%), followed by permanent junctional reciprocating

RÉSUMÉ

La cardiomyopathie induite par une tachycardie (CIT), aussi appelée cardiomyopathie induite par l'arythmie ou tachycardiomyopathie, est une forme réversible d'insuffisance cardiaque caractérisée par des tachyarythmies persistantes et qui est associée à une dysfonction ventriculaire. La CIT se caractérise par la réversibilité des lésions myocardiques lors de la résolution de l'arythmie. Le diagnostic précoce de CIT est essentiel, car son traitement diffère de celui des cardiomyopathies d'autres causes ou de cause inconnue. Il peut toutefois être ardu de distinguer la CIT de la tachycardie secondaire à l'élévation du taux de catécholamines attribuable à une insuffisance cardiaque congestive. Il existe relativement peu de rapports de cas pédiatriques. Nous présentons ici une série de cas issus de la littérature (entre 2014 et 2024) englobant 48 enfants atteints de CIT, de même que quatre études de cas de CIT traités dans notre établissement (BC Children's Hospital, Vancouver, Canada). Dans cette série de cas, l'âge moyen

Dilated cardiomyopathy (DCM) can be life-threatening and is a leading indication for heart transplantation in children.[1] Approximately 40% of children diagnosed with cardiomyopathy either succumb to the condition or require heart transplantation within 2 years of experiencing symptoms.[1] DCM can be caused by a variety of conditions, including genetic variants, congenital structural heart disease, and inflammatory and infectious causes. Sixty-six percent of DCM cases are idiopathic, followed by myocarditis and neuromuscular diseases as the next most common causes, with a prevalence of 46% and 26%, respectively.[2] Tachycardia-induced cardiomyopathy (TIC), also known as arrhythmia-induced cardiomyopathy or tachycardiomyopathy, is a reversible form of heart failure (HF) characterized by persistent tachyarrhythmias and associated ventricular dysfunction.[3–6] A hallmark of TIC is the recovery in cardiac function when the arrhythmia is treated.[4,6] Therefore, it is essential to consider TIC in the differential diagnosis and recognize it to enable appropriate treatment.[3,4,6–8] The prognosis of TIC is favourable compared with other causes of DCM, with a greater improvement of left ventricular ejection fraction (LVEF) and lower frequency of hospitalizations and cardiac death during follow-up.[9,10] Unfortunately, misdiagnosis or delayed diagnosis is frequent for TIC as it can be difficult to recognize that the rapid rhythm is not secondary to the adrenergic effects of HF.[3,4,6–8,11] A standardized diagnostic approach has not yet been established in practice, particularly for children, and the final confirmation of TIC occurs after treatment of tachycardia when ventricular function improvement is observed.[12]

Received for publication September 14, 2024. Accepted October 28, 2024.

Corresponding author: Dr Sakethram Saravu Vijayashankar, BC Children's Hospital, 4480 Oak St, 1F9, Vancouver, British Columbia V6H 3V4, Canada. Tel.: +1-604-875-2295.

E-mail: saketh.saravu@cw.bc.ca

Twitter: @MarcDeyell (M.W. Deyell), @DrSanatani (S. Sanatani), @Saravuram (S.S. Vijayashankar)

https://doi.org/10.1016/j.cjcpc.2024.10.007

2772-8129/© 2024 Published by Elsevier Inc. on behalf of the Canadian Cardiovascular Society. This is an open access article under the CC BY-NC-ND license (http://creativecommons.org/licenses/by-nc-nd/4.0/).

tachycardia (20.8%), ventricular tachycardia (16.7%), and atrioventricular re-entrant tachycardia or atrioventricular nodal re-entrant tachycardia (10.4%). Pharmacologic treatment was the predominant therapy, but 70.8% of patients needed at least 1 ablation procedure. All patients demonstrated significant improvement in left ventricular ejection fraction after treatment, with most achieving at least near-normal ejection fractions in 80 days on average since presentation. In conclusion, TIC is overall a treatable condition with challenging diagnosis but generally has a favourable prognosis when diagnosed and treated appropriately. This article emphasizes the importance of considering TIC in the differential diagnosis of tachycardia in the context of reduced ventricular function, to recognize it and to enable targeted treatment initiation as soon as possible.

des patients était de 6,98 ± 4,9 ans. La majorité des patients présentait respectivement une tachycardie auriculaire ectopique (41,7 %), une tachycardie jonctionnelle permanente par rythme réciproque (20,8 %), une tachycardie ventriculaire (16,7 %) et une tachycardie supraventriculaire réentrante ou une tachycardie auriculo-ventriculaire par réentrée intranodale (10,4 %). Les patients ont pour la plupart reçu un traitement pharmacologique, mais 70,8 % d'entre eux ont également été soumis à au moins une ablation. Après le traitement, on a observé une amélioration significative de la fraction d'éjection ventriculaire gauche (FEVG) chez tous les patients, la plupart ayant affiché une fraction d'éjection normale ou quasi normale dans un délai moyen de 80 jours depuis leur présentation initiale. En conclusion, la CIT est généralement une maladie traitable, qui peut être difficile à diagnostiquer, mais dont le pronostic est généralement favorable lorsqu'elle est diagnostiquée et traitée adéquatement. Cet article souligne l'importance d'envisager la CIT lors du diagnostic différentiel des tachycardies en présence d'une diminution de la fonction ventriculaire, car la détection de la CIT en temps opportun permet d'instaurer un traitement ciblé dans les meilleurs délais.

Although paroxysmal arrhythmias can be associated with TIC, incessant arrhythmias are more likely to be causative.[12,13] Atrial tachycardia (AT) and permanent junctional reciprocating tachycardia (PJRT) are the most common associated arrhythmias in children.[3,4] Treatment for TIC is centred on rhythm or rate control, which allows for the reversal of cardiac remodelling and cardiomyopathy.[4,7,8,11,13,14] Active interventions such as medication and catheter ablation are needed as spontaneous resolution of tachycardia is unlikely.[3,5,8,13–15] There is growing support for ablation to be considered earlier in the treatment process to avoid progressive worsening of cardiomyopathy and HF, which can result in worse clinical outcomes.[4,8,13] This must be balanced with the risks of sedation, the procedure itself, and the availability of extracorporeal membrane oxygenation (ECMO) support for children after ablation.[6]

The etiology and pathophysiology of TIC in paediatric populations remain poorly understood. It remains unclear why some people develop TIC. The paediatric literature consists of case reports and small case series, with the last international multicentre study on paediatric TIC conducted over a decade ago.[3] Because of the advancement of catheter ablation techniques and technology, it was important to update the evidence base to determine if risk factors can be identified. Also, outcomes and course of recovery remain poorly described.

Methods

The cases included in the literature review were retrieved from PubMed based on a search of articles published between January 2014 and July 2024. The following search combinations were used: ("Tachycardia-induced cardiomyopathy" OR "tachycardia induced cardiomyopathy" OR "tachycardiomyopathy") AND ("pediatrics" OR "pediatric" OR "children" OR "child") with or without ("ablation"). Only articles in English with detailed information for each patient younger than 18 years were included in our case summary. The articles were reviewed by 2 separate authors (W.A. and S.W.), and the articles with the same authors or from the same institution were compared. When overlapping patient descriptions were identified, it was ensured that no duplicate patients were included in our statistical analysis. For the cases gathered from existing literature, descriptive statistics were performed for both continuous and categorical variables.

We also present 4 illustrative cases with TIC seen at our site (BC Children's Hospital, Vancouver, Canada). This is not a comprehensive list of cases but were chosen to describe the challenges seen in patients with TIC. Institutional ethics review was conducted, and the need for consent and approval was waived as this was a chart review and deidentified data were used.

Results

Illustrative case series

Case 1. A previously healthy 7-year-old girl presented to the emergency department with a history of chest pain, exercise intolerance for 2-3 weeks, and respiratory distress. On examination, she was tachycardic with a heart rate (HR) of 200 beats per minute (BPM) along with tachypnea and hepatomegaly. Electrocardiogram (ECG) indicated long RP, narrow QRS tachycardia with a rate of 200 BPM, with a negative P-wave axis in inferior leads. Adenosine terminated the tachycardia with a QRS complex. Differential diagnosis included PJRT and ectopic AT. Adenosine conversion was short-lived with recurrence of tachycardia. An echocardiogram revealed severely depressed left ventricular (LV) function and severe LV dilatation, leading to her transfer to the cardiac intensive care unit (CICU). Antifailure management was started along with intravenous (IV) loading of procainamide and digoxin, which terminated the tachycardia, with sustained sinus rhythm (SR). This was done under extracorporeal life support backup due to the cardiac dysfunction and concern for hypotension and negative inotropy with the procainamide. The patient was converted to oral flecainide and sotalol. Frequent breakthroughs were noted (Fig. 1A). The patient was discharged home on antifailure and antiarrhythmic medications and was followed as an outpatient. Serial improvement in function was

seen on repeated echocardiograms, with Holter monitoring demonstrating brief self-limited runs of tachycardia with and without aberrancy. After improvement in cardiac function and stabilization of HF, she was taken to the electrophysiology (EP) lab. Tachycardia was easily inducible in the lab. It was a narrow complex tachycardia, with a cycle length of 400 milliseconds (ms) and a 1:1 A:V ratio, and at times 2:1 relationship to the ventricle, earliest atrial activation was noted in the proximal coronary sinus. His refractory premature ventricular contractions (PVCs) did not advance the tachycardia, and right ventricle (RV) entrainment showed a V-A-A-V response. Thus, an AT was diagnosed. This AT was successfully ablated at the 6 o'clock position on the tricuspid annulus. During postablation testing, with isoproterenol infusion, a second tachycardia was noted. This was a wide complex tachycardia with a left bundle branch block pattern and inferior axis; the tachycardia cycle length was similar at 400 ms, with VA dissociation. Thus, a secondary ventricular tachycardia (VT) was diagnosed. However, as this was only inducible with isoproterenol, we deemed it to be nonclinical and did not ablate. During her outpatient follow-up after her first ablation, she was found to have a significant burden of the wide complex tachycardia on repeated Holter studies. Her function remained mildly depressed on repeated echocardiography; therefore, she was taken again to the EP lab. We mapped an automatic VT focus site in the posterior right ventricular outflow tract-pulmonary valve interface, which was successfully ablated (Fig. 1C). After the second procedure, all antiarrhythmic medications were discontinued. She made a full recovery of function and LV dimensions, with return to her baseline level of energy and activity without exercise intolerance after almost 7 months of follow-up.

Case 2. A previously healthy 9-year-old girl presented to medical care with a 5-week history of cough, decreased appetite resulting in weight loss (approximately 4 kg), and 1 day of fever and vomiting. On assessment, she appeared diaphoretic and had tachypnea and mild respiratory distress, and her cardiac examination revealed an active precordium with the leftward and inferiorly displaced apex. A gallop rhythm and a III/VI systolic murmur were heard. She had a rapid HR in the 130s, blood pressure (BP) of 101/66 mm Hg, and a respiratory rate of 48 breaths per minute. A chest radiograph showed cardiomegaly. Her ECG demonstrated a long RP narrow complex tachycardia at 139 BPM, 1:1 A:V ratio, with P mitrale and voltage criteria for LV hypertrophy as well as nonspecific ST segment changes (Fig. 2A). Her P-wave had a normal axis and had a predominantly negative component in V1 and was positive in V2-6. She also had an unusual sharp deflection in the P-wave noted in the frontal leads. Given the normal axis of the P-wave, no abrupt changes were observed and there was a diurnal variation in rate. She was initially diagnosed with sinus tachycardia. Her echocardiogram showed normal segmental anatomy, a dilated left atrium (LA) and LV with moderately reduced LV systolic function with ejection fraction (EF) of 30%-35%, and preserved RV systolic function. She was admitted to CICU and was started on antifailure therapies including IV milrinone infusion and diuretics. Her cardiac magnetic resonance imaging (C-MRI) showed oedema within the septum in the T1 map with nonuniform distribution, consistent with inflammation. With a presumptive diagnosis of myocarditis, she received IV immunoglobulin once and a pulse of IV methylprednisolone for 3 days. During her admission, her LV systolic function worsened, her EF decreased to 20%-25%, and her B-type natriuretic peptide peaked at 23,000 ng/L. Her metabolic and genetic screens were normal. Metoprolol was started for rate control. While on metoprolol, she was noted to have a brief period at a lower rate of 90 BPM with a normal biphasic P-wave in V1. Due to detection of 2 different rates with differing P-wave morphologies, we made a diagnosis of AT-induced TIC. Several antiarrhythmic medications including ivabradine, propranolol, metoprolol, sotalol, and flecainide were tried. She started to have symptomatic low BP, presyncope events, and severe weakness while on antiarrhythmics; hence, she was brought to the EP lab for ablation with ECMO standby. During the EP study, the earliest activation was in the LA. RV entrainment showed a V-A-A-V pattern, hence confirming an AT. Transseptal puncture was complex due to high LA pressures of 25 mm Hg. After transseptal access was gained, a high-definition catheter showed that the earliest signals in the LA were from the carina underneath the left superior pulmonary vein. Radiofrequency ablation (RFA) was applied in this region with termination of tachycardia within 3 seconds of RFA (Fig. 2B). No further arrhythmia was noted. Follow-up Holter showed a low atrial ectopy burden of 4%; ivabradine was initiated and was effective in terminating this ectopic burden. She made a complete clinical recovery with recovery of LV function. Repeated C-MRI showed normal thickness of the myocardium, with no oedema and no late gadolinium enhancement.

Case 3. A previously healthy 13-year-old boy presented to the emergency department with a 4-week history of abdominal pain, increasing exercise intolerance, and shortness of breath. His personal and family histories were unremarkable. His physical examination revealed normal HR with preserved BP. On examination, he had a gallop rhythm with increased work of breathing and hepatomegaly. His ECG demonstrated an accelerated idioventricular rhythm with short runs of nonsustained VT, capture beats, and fusion beats. The ventricular beats had a right bundle branch block-type pattern along with a superior axis (Fig. 3A). His echocardiogram demonstrated normal segmental anatomy with a severely dilated LV and poor biventricular systolic and diastolic function. The EF was approximately 20% with an abnormal ventricular strain pattern. Right ventricular systolic pressure was elevated at 50 mm Hg. He was admitted to CICU for respiratory support and cardiac antifailure management. Antiarrhythmics including amiodarone, β-blockers, sotalol, flecainide, and mexiletine were tried. They reduced the arrhythmia burden, but the patient continued to have frequent PVCs. A full cardiomyopathy workup including infectious, metabolic, and genetic evaluation was negative. Because of his severe symptoms, cardiac transplant assessment was initiated. C-MRI showed no evidence of scarring. While he was under the effect of general anaesthesia (propofol) for a cardiac catheterization, he spontaneously converted to SR. An echocardiogram at that time confirmed that he had improvement in LV function while in SR. He converted back to his previous rhythm of

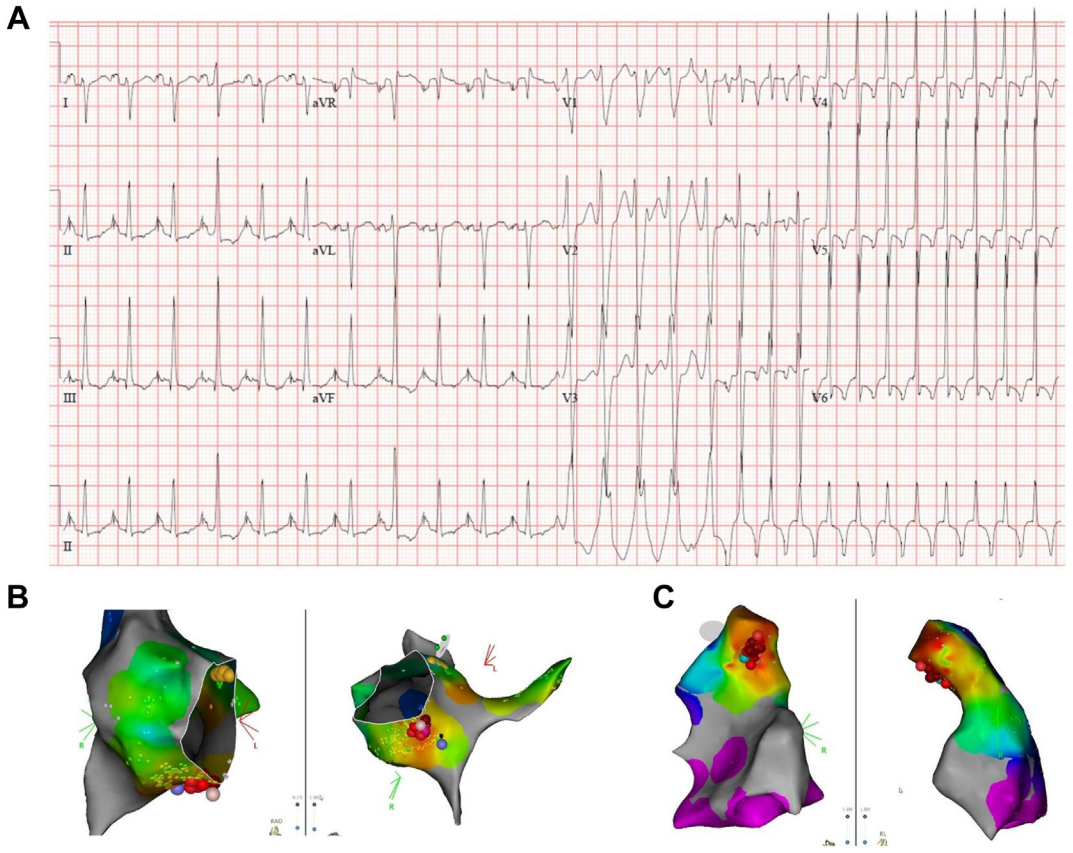

Fig 1. (**A**) Non-sustained ventricular tachycardia seen to initiate the narrow complex tachycardia. (**B**) Right anterior and left anterior oblique images depicting the site of successful EAT ablation. (**C**) Posterior-anterior and right lateral images depicting site of successful VT ablation align the posterior aspect of the pulmonary artery-right ventricular outflow tract interface.

frequent PVCs after emergence from general anaesthesia. Because of failure of antiarrhythmic medications, he was taken to the EP lab. Using a mapping catheter in a retrograde fashion, ventricular ectopy was mapped. Early activation was identified near the anterolateral annular region of the basal LV. We were not able to get ahead of surface QRS endocardially, and ablations at the site only transiently suppressed the ventricular ectopy. We concluded that we likely had an epicardial source for VT. Epicardial access was then obtained via a subxiphoid approach. PVCs had stopped, and we continued with epicardial pace mapping. Both endocardial and epicardial voltage maps showed healthy myocardium with no scar. Optimal pace mapping was adjacent to the endocardial site on the surface epicardium at the anterolateral aspect of basal LV. A coronary angiogram was performed, which showed that the site of interest was proximal to a marginal artery bifurcation off the left circumflex. After this, epicardial ablations with caution were undertaken at this site (Fig. 3B). A limited area could be ablated due to the proximity of the coronary bifurcation. The ventricular ectopy burden diminished to minimal after ablation. Although the process was successful, after the procedure, he still had symptomatic PVCs. Therefore, he was started on oral sotalol and flecainide, which were now enough to keep the PVC burden at 1%. At his follow-up assessment, he had made a complete recovery with ECG showing SR, echocardiogram showing normal function, and Holter ECG showing PVC burden less than 1%. His repeated echocardiogram showed normal EF and LV dimensions.

Case 4. A 9-year-old boy presented with complex congenital heart disease, including right atrial isomerism, unbalanced atrioventricular septal defect, a dominant RV, malposed great arteries with pulmonary atresia, and pulmonary blood flow via 4 major aortopulmonary collateral arteries. In addition, he had a right aortic arch with mirror image branching and left pulmonary vein stenosis. He had previously undergone major aortopulmonary collateral artery angioplasty and stenting, as well as left pulmonary vein stenting. Because of complex anatomy, the patient was not suited for either a biventricular repair or Fontan procedure and was managed palliatively. He also had a history of having SVT as an infant. He presented with 6 days of feeling unwell, experiencing lethargy, decreased appetite, left ear pain, vertigo, and personality changes. He was diagnosed to have a cerebral abscess with an associated midline shift and left-sided uncal herniation on his head imaging, for which he underwent a craniotomy and drainage procedure. Culture of the abscess fluid yielded a positive result for *Aggregatibacter aphrophilus*. During the period of this illness, he began experiencing episodes of palpitations with tachycardia detection. ECG showed both wide and narrow complex tachycardia with abrupt shifts between each other.

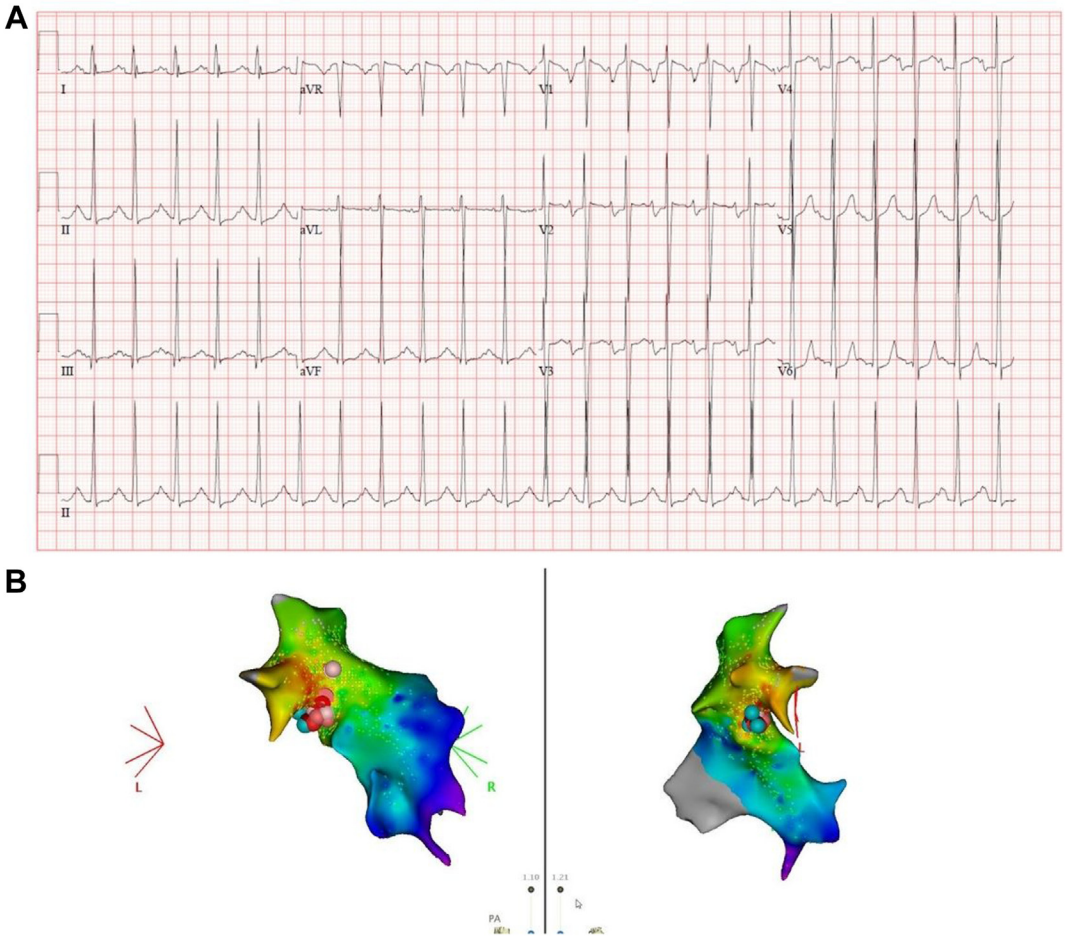

Fig 2. (**A**) Atrial tachycardia. Note the fast heart rate with a predominantly negative P wave in V1 and normal axis. (**B**) Images in posterio-anterior and left lateral showing site of successful ablation along the carinal aspect of the left upper pulmonary vein.

The wide complexes appeared pre-excited with a delta wave and shorter PR intervals (Fig. 4). There was a burst-on and burst-off pattern with rates often reaching 130-150 BPM. The rhythm was diagnosed as AT with intermittent pre-excitation manifesting as narrow and wide complexes. We also considered that dual AV node physiology was a possibility given the right atrial isomerism. Treatment was initiated with flecainide with the subsequent addition of atenolol. With the arrhythmia controlled, he was discharged for home IV antibiotic treatment for his brain abscess. A week after discharge, he represented with symptoms of abdominal pain, ascites, and elevated liver enzymes, indicative of congestive HF. His HR was 140 BPM, and a 12-lead ECG showed usual complex tachycardia with a short RP interval. Notably, pre-excitation was no longer visible during the tachycardia episodes. A negative P-wave was observed on lead AVF, suggestive of low to high atrial activation. Tachycardia was often noted to self-terminate with an atrial impulse. Given the change in QRS morphology to the usual complex during tachycardia, recent diagnosis of intermittent pre-excitation and termination with atrial impulses, we diagnosed him to have likely a second atrioventricular re-entrant tachycardia (AVRT). His echocardiogram exhibited a hyperechogenic finding on his right atrioventricular valve not previously observed. Mild to moderate right atrioventricular valve regurgitation was also noted, along with moderately decreased univentricular systolic function. The clinical presentation was most consistent with TIC along with infective endocarditis. During his admission, antibiotic therapy and cardiac antifailure management were continued. Flecainide and atenolol doses were increased to gain arrhythmia control. He received overall a 12-week course of broad-spectrum antibiotics for suspected culture-negative infective endocarditis and cerebral abscess. His arrhythmia was effectively controlled, and there was a significant improvement in his heart function, which nearly normalized. After a few months, he was taken to the EP lab for the diagnosis of tachycardia and ablation of the accessory pathway. Unexpectedly, there was no inducible clinical tachycardia or evidence of an accessory pathway. No ablation was performed. Weaning of medications was attempted, but the patient was noted to have frequent symptomatic premature atrial contractions; therefore, he was kept on both atenolol and flecainide. We hypothesize that the inflammatory state may led to the incessant ectopic atrial rhythm, and the pre-excitation was also only manifest in this inflammatory state. We believe that the negative EP study was due to the

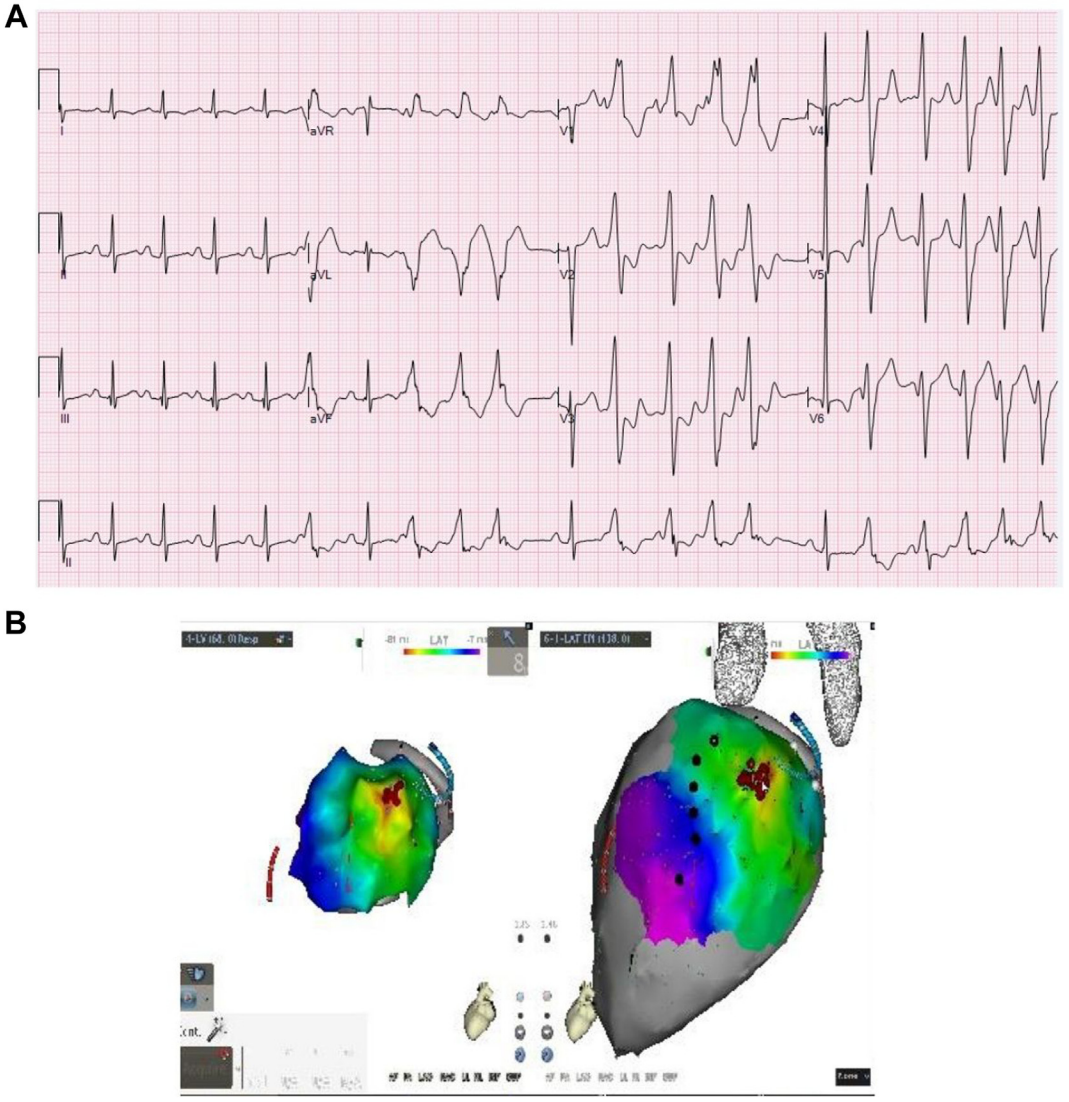

Fig 3. (**A**) ECG at presentation with the patient in an accelerated ventricular rhythm with a right bundle branch block type pattern and an inferior axis. Note the frequent fusion beats. (**B**) Epicardial map showing site of successful ablation and termination of PVC's (**red balls**). The **black balls** indicate phrenic nerve stimulation sites.

resolution of this inflammatory state, and effective antiarrhythmic medications have led to control of TIC.

Literature review results

This retrospective review analyses patients with TIC published in case series and case reports in the past 10 years (January 2014 to July 2024). We identified 48 patients. The most common etiologies were AT and PJRT accounting for 41.7% and 20.8%, respectively. The average age at TIC presentation was 6.98 ± 4.9 years (range: day 1 of life to 15.8 years). Patients with AT had a mean age of 7.4 ± 4.69 years at presentation (range: 1 month to 13 years; n = 20), patients presenting with PJRT had a mean age of 5.53 ± 4.56 years (range: day 1 of life to 14.5 years; n = 10), and patients presenting with VT had a mean age of 9.98 ± 4.35 years (range: 5-15.83 years; n = 8). The mean duration of symptoms before diagnosis was 109.3 days (range: 1 day to 2 years; n = 16). The average HR at presentation was 182 BPM (range: 110-280 BPM; n =23) and the presenting LVEF mean was 35.1% ± 12.8% (range: 10%-57%; n = 45). Patients with AT presented with a mean LVEF of 34.58% ± 14.2% (range: 17%-56%; n = 19), patients with PJRT had a mean LVEF of 30.9% ± 14.1% (range: 10%-57%; n = 10), and patients with VT presented with a mean LVEF of 41.75% ± 9.6% (range: 25%-54%; n = 8). Just over two-thirds (70.8%) of 48 patients underwent RFA and 7% required ECMO support during their EP lab course (one had AT and one had PJRT). Patient demographics and tachycardia characteristics are summarized in Table 1. Ninety-seven percent of patients had normalization of their LVEF after treatment (LVEF ≥55%). The time for recovery was 80.5 ± 110.3 days on average and varied widely between 3 days and 1 year (n = 32). In patients with AT, time to normalization of LVEF ranged from 3 days to 1 year with a mean of 87.8 ± 121.25 days (n = 15), patients with PJRT needed an average of 93.5 ± 121.61 days (n = 10) for recovery

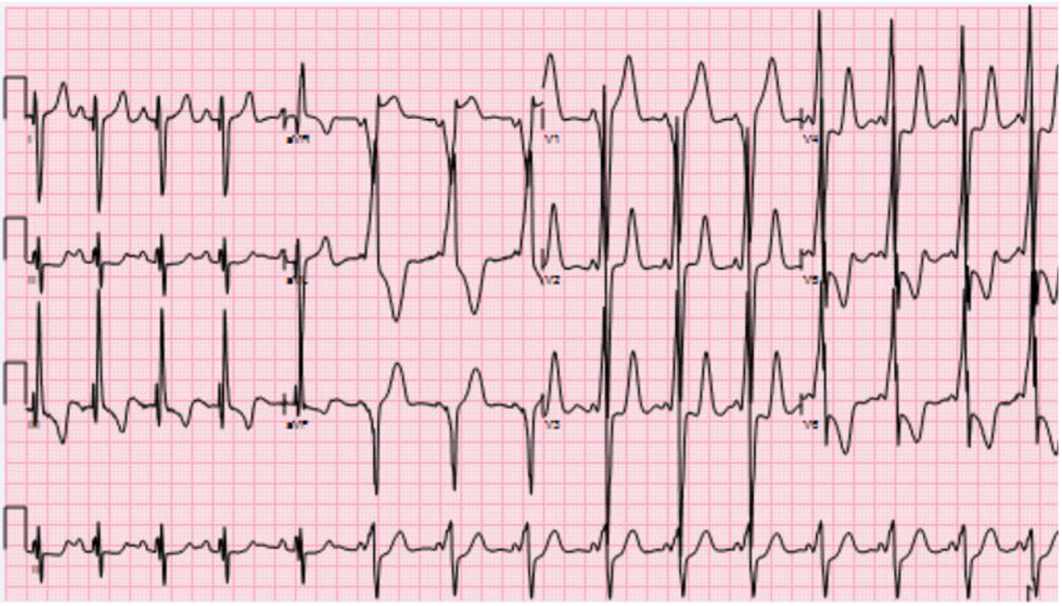

Fig 4. Intermittent pre-excitation seen on ECG for patient Case: 4.

of the LVEF with a range of 1 month to 1 year, and patients with VT had a recovery time of 5-180 days with a mean of 51.25 ± 85.9 days (n = 4).

In addition, we found that 91% of patients were started initially on antiarrhythmic medications, whereas 9% underwent direct ablation. Three patients with AT needed additional atrial appendectomy to control the arrhythmia.

Discussion

The incidence of AT among patients in our review presenting with TIC was lower than the known published data (41.7% vs 59%).[4] Our results showed that PJRT as the causative arrhythmia was more common than VT, at 20.8% and 16.7%, respectively, compared with 23% and 7% reported by Moore et al.[4] The mean time for recovery overall was 80.47 days and varied widely between 3 days and 1 year. This period was longer than what is reported in literature.[4,7,12] In addition, we found that 9% of the patients we analysed underwent direct ablation, considerably less than those (35%) from the study conducted by Moore et al.[4]

Arrhythmia-induced HF was first documented in a case series published in 1913 when Gossage and Hicks[16] introduced the concept that auricular fibrillation (atrial fibrillation) can begin in a healthy heart, instead of being secondary to cardiomyopathy, and result in subsequent dilatation and reduced ventricular function. In the following years, reversibility of HF was reported with the first case published by Parkinson and Campbell[17] in 1930 where a patient with persistent atrial fibrillation that developed into HF was able to regain all functions after normal SR was restored. The reversibility of HF and the notion that early recognition can lead to a better prognosis were postulated in a case study by Brill in 1937.[18] Whipple et al.[20] reported the usage of animal models in 1962,[19] which has allowed for the controlled study of TIC.

Studies in animal models posit that the development of TIC is due to a remodelling process that is powered by neurohormonal activation, intracellular calcium handling defects, and extracellular matrix alterations.[11] Abnormal cardiac contractions seen in tachyarrhythmias contribute to excessive neurohormonal activation, which results in fibrosis, increasing the risk of TIC development.[11,12] Defects in calcium handling affect the excitation-contraction coupling process, resulting in myocyte contractility impairments that decrease LVEF.[12] All of this culminates in a cellular remodelling process that causes dilatation and elevated filling pressures in the ventricles, along with a decrease in cardiac contractility and reduced diastolic time resulting in TIC.[12,21]

Diagnosis of TIC is challenging, as it can be difficult to distinguish between DCM with secondary tachycardia and TIC. Multiple studies have been performed in attempts to elucidate their differences in the adult population. Vera et al.[10] focused on ECG and cardiac magnetic resonance parameters, and categorized patients with TIC as those with a recovered LVEF of over 50% at follow-up after treatment and patients with DCM as those with an LVEF below 50%. Patients with TIC were seen to have a narrower QRS, an absence of late gadolinium enhancement on cardiac magnetic resonance, and a higher degree of LVEF recovery and better clinical outcomes, represented by lower hospitalization rates for HF, compared with those diagnosed with DCM.[10] Jeong et al.[22] found that patients with TIC had smaller LV dimensions, mass and volume indices, and a normal LV end-diastolic diameter (≤55 mm) in the background of LV dysfunction unlike patients with DCM. Gelb et al.[23] reported that patients with TIC had a higher shortening fraction, less LV dilatation, inverted and notched P-wave in lead V_1, second-degree AV block on ECG or Holter monitor, and more rapid atrial rates (>150% of predicted rate for age).

Causative arrhythmias

AT, also known as ectopic AT or focal AT, can be defined as SVT that originates from and is sustained by an automatic pacemaker located outside of the sinus node.[24] It is known to

Table 1. Patient demographics, tachycardia characteristics, and treatment

Patient number	Sex, age at TIC presentation	Symptoms at presentation	Mechanism of tachycardia	Heart rate (BPM)	Duration since symptom onset to diagnosis	LVEF as estimated by echocardiogram at presentation (%)	Time to recovery (d)	LVEF as estimated by echocardiogram after treatment (%)	Pharmacologic treatment	Nonpharmacologic therapy	Reference
1	M, 3 mo	Grunting, respiratory distress, and decreased feeding for 2 d	PJRT	150	Known SVT since fetal life with HR 180-200 BPM (at 32 wk)	22	270	70	During fetal life: D, F After delivery: A, B, F, P, S After presentation: A, B, D, E, Fu, M	RFA of epicardial lesion placed in a diverticulum of the coronary sinus	42
2	F, 13 y	Heart failure	AT	140	Unknown	18	Unknown	Normalized	Unknown	RFA anterior and inferior to the right inferior pulmonary vein	43
3	M, 11 y	Rapid palpitations associated with dyspnea	Para-Hisian AT—originating from RSPV or LAA	Unknown	5 mo	29	90	78	PP	Successful ablation from the right para-Hisian area	44
4	M, 10 y	Effort intolerance (NYHA III) and palpitations	Fascicular VT	160	4 mo	32	5	50	Ad, V	Electrical cardioversion, RFA	45
5	F, 7 y	Progressive dyspnea	Para-Hisian region PJRT	116	3 mo	25	90	Normalized	None	RFA in NCS of the aorta	46
6	M, 5 mo	FTT, cough, and wheezing. Weight loss for 2 mo	AFL	200	2 mo	14	98	59	Adr, Dob, Dop and O	Intubation, ventilation, synchronized electrical cardioversion	47
7	F, 3.5 y	Tachycardia	Sustained AT	200	5 d	42	365	65.	A	RFA	48
8	F, 5.75 y	Syncope 3 d before presentation	Sustained AT	160	3 d	29	365	60	A, C	RFA combined with LAA resection	48
9	M, 12.9 y	Palpitations	Sustained AT	145	2 y	45	30	56	A, Be, D, PF	RFA combined with thoracoscopic RAA resection	48
10	Unknown, 1 mo	Asymptomatic	Idiopathic AFL	216	Unknown	42-45	10	Normalized	Ad, D, PP	Successful synchronized electrical cardioversion	49
11	M, 4 y	Prolonged abdominal pain and lethargy	AT	208	Unknown	33	7	60	A, D, E	RFA (tachycardia recurred). Surgical thoracoscopic atrial appendectomy	50
12	M, 60 d	Query dilated cardiomyopathy	Incessant AT	250	Unknown	17	7	58	Ad, A, D, I, Me, PP, S, En, Fu, M	Unsuccessful synchronized cardioversion ×2. Unsuccessful unsynchronized cardioversion ×3	51
13	M, 30 d	Respiratory distress and cardiogenic shock	Incessant AT	200	Unknown	19	3	55	Ad, D, I, M, Fu	None	51
14	F, 11.8 y	Abdominal pain, cough, and exercise intolerance	Mahaim accessory pathway + AVNRT	Unknown	Unknown	42	Unknown	78	A, Me	Synchronized cardioversion, cryoablation for typical AVNRT, and RFA for Mahaim pathway	6
15	F, 14.5 y	Query myocarditis	Left posterior fascicular VT	Unknown	Unknown	44	Unknown	62	A, Me, V	Unsuccessful electrical cardioversion and RFA	6
16	M, 13 y	Severe congestive heart failure (NYHA class IV)	AT	Unknown	Unknown	17	180	56	None	RFA	6
17	M, 12.3 y	Dyspnea	AT	Unknown	3 d	18	30	56	A	ECMO, HVAD, and RFA ×2	6
18	M, 14.5 y	Query myocarditis	PJRT	Unknown	Unknown	10	365	55	Adr, Dob, S	ECMO and RFA ×2	6
19	M, 15.8 y	Chest pain, fever, and sweating, upper respiratory infection symptoms	Left posterior fascicular VT	Unknown	3 d	47	6	68	Me	Synchronized cardioversion, and RFA	6
20	M, 7.9 y	Tachycardia	AVRT	Unknown	Unknown	38	Unknown	69	Ad, A	None	6
21	Unknown, 7 y	Unexplained systolic dysfunction during postoperative follow-up (ventricular septal defect repair)	AT	Unknown	Unknown	32	Unknown	60	A	None	6
22	Unknown, 4 y	Unexplained systolic dysfunction during postoperative follow-up (Senning procedure)	AT	Unknown	Unknown	48	Unknown	62	S	None	6
23	Unknown, 5 y	Unexplained systolic dysfunction during postoperative follow-up (total anomalous pulmonary venous connection repair)	AT	Unknown	Unknown	48	Unknown	64	S	RFA	6

Continued

Table 1. Continued.

Patient number	Sex, age at TIC presentation	Symptoms at presentation	Mechanism of tachycardia	Heart rate (BPM)	Duration since symptom onset to diagnosis	LVEF as estimated by echocardiogram at presentation (%)	Time to recovery (d)	LVEF as estimated by echocardiogram after treatment (%)	Pharmacologic treatment	Nonpharmacologic therapy	Reference
24	F, 14 y	Intermittent palpitations and chest discomfort	Ventricular ectopy and Bidirectional VT	Unknown	2 mo	48	14	72	A, ACEi, F, PP	None	[34]
25	F, 5 y	Vomiting and tachypnea	Incessant idiopathic right VT	216	Unknown	37	Unknown	Unknown	A, Me	RFA	[52]
26	M, 5.5 y	Vomiting and syncope	Incessant idiopathic right VT	166	Unknown	47	Unknown	Unknown	A, Me	RFA	[52]
27	F, 6 y	Progressive palpitations	Incessant idiopathic right VT	142	Unknown	54	Unknown	Unknown	PF, V	RFA	[52]
28	F, 9 y	Palpitation, dyspnea, and heart failure (NYHA II)	AVRT	Unknown	Unknown	35	Unknown	58	Ad, B	RFA	[53]
29	F, 4 y	Heart failure and pounding of the precordium	Incessant AT (originating from RAA)	280	6 mo	Unknown	30	Normal	Ad, D, B, diuretics	RFA	[54]
30	M, 12 y	Respiratory distress and hypotension	Accelerated idioventricular rhythm	110	Unknown	25	180	Normal	A	Electrical cardioversion and RFA	[55]
31	M, 5 y	Palpitation, dyspnea, and heart failure	AT	170	3 mo	22-30	90	57	D, captopril, L-carnitine, carvedilol, F	None	[56]
32	F, 15 y	Palpitations, shortness of breath, early fatigue, and HF	Atypical (incessant fast-slow) AVNRT	176	3 mo	Unknown	14	Unknown	M, PF	Cryoablation	[57]
33	M, 2 mo	Drug resistant supraventricular tachycardia	Congenital JET	220	2 wk	Unknown	Unknown	Unknown	M, Adr, F, A, PP, I	Transesophageal electrophysiology study—overdrive pacing	[58]
34	F, 10 mo	Unknown	Congenital JET	170-190	Unknown	40	Unknown	Normal	Low-molecular-weight heparin, M, PP, A, PF, I	Transesophageal electrophysiology study	[58]
35	M, 52 d	Restlessness, poor nutrition, poor general condition, hypotonic, and intubated	Congenital JET	220	Unknown	25-30	10	70	Ad, A, F, D, M, I	Cardioversion, resuscitation, and peritoneal dialysis	[58]
36	M, 24 d	Poor feeding, lethargy, and pallor	AVRT	128	1 d	35	Unknown	Unknown	Ad, E, PP, M, Ep, hydrocortisone, ampicillin, cefotaxime, acyclovir, En	None	[59]
37	F, 11 y	Unknown	AT	Unknown	Unknown	55	30	67	Unknown	RFA	[60]
38	F, 5 y	Unknown	PJRT	Unknown	Unknown	44	30	65	Unknown	RFA ×2	[60]
39	F, 6 y	Unknown	PJRT	Unknown	Unknown	57	30	63	Unknown	RFA	[60]
40	M, 11 y	Unknown	Focal AT initially, AVRT (recurrent)	Unknown	Unknown	54	30	56	Unknown	RFA ×2	[60]
41	M, 2.5 y	Unknown	PJRT	Unknown	Unknown	34	30	61	Unknown	RFA	[60]
42	M, 3 y	Unknown	PJRT	Unknown	Unknown	46	30	63	Unknown	RFA	[60]
43	M, In utero	Unknown	PJRT	Unknown	Unknown	24	30	65	Unknown	RFA	[60]
44	M, 13 y	Unknown	AT	Unknown	Unknown	47	30	65	Unknown	RFA	[60]
45	F, 11 y	Unknown	PJRT	Unknown	Unknown	23	30	69	Corticosteroid, IVIG	RFA	[60]
46	F, 9 mo	Unknown	AT	Unknown	Unknown	56	30	58	Unknown	RFA	[60]
47	M, 6 y	Unknown	PJRT	Unknown	Unknown	24	30	62	Unknown	RFA	[60]
48	F, 11.5 y	Unknown	AT	Unknown	Unknown	28	Unknown	71	Unknown	RFA ×2	[60]
49	F, 7 y	Chest pain and fatigue for 2-3 wk	AT (primary diagnosis) and VT	180	2-3 wk	18	210	57	D, P, S, Sp, enoxaparin, lasix, M	RFA for PJRT and later for PVC from RVOT origin	Case 1
50	F, 9 y	Cough, decreased appetite, and weight loss for 5 wk	AT	130	5 wk	30	180	54	F, I, Me, S, Sp, lasix, M, methyl prednisolone, IVIG	RFA for left upper pulmonary vein origin of AT	Case 2
51	M, 13 y	Persistent abdominal pain and exercise intolerance for 4 wk	Frequent PVCs and short runs of VT	170	1 mo	20	Unknown	50	A, E, F, mexiletine, S, ASA, En, Sp, lasix	Epicardial RFA	Case 3
52	M, 9 y	Feeling unwell, lethargic, decreased appetite, ascites, and signs of heart failure for 6 d	AVRT with intermittent pre-excitation (primary diagnosis) and AT	140	6 d	Moderately decreased univentricular systolic function	Unknown	Nearly normalized	Atenolol and F	EP study—nonclinical cavotricuspid isthmus line–dependent AFL and no ablation performed	Case 4

A, amiodarone; ACEi, angiotensin-converting enzyme inhibitors; Ad, adenosine; Adr, adrenaline; AFL, atrial flutter; AT, atrial tachycardia; ASA, acetylsalicylic acid; AVNRT, atrioventricular nodal re-entrant tachycardia; AVRT, atrioventricular re-entrant tachycardia; B, β-blockers; Be, betaloc; BPM, beats per minute; C, cedilanid; D, digoxin; Dob, dobutamine; Dop, dopamine; E, esmolol; ECMO, extracorporeal membrane oxygenation; En, enalapril; Ep, epinephrine; F, flecainide; FTT, failure to thrive; Fu, furosemide; HR, heart rate; HVAD, HeartWare Ventricular Assist Device; I, ivabradine; IVIG, intravenous immunoglobulin; JET, junctional ectopic tachycardia; LAA, left atrial appendage; LVEF, left ventricular ejection fraction; M, milrinone; Me, metoprolol; NCS, non coronary sinus; NYHA, New York Heart Association; O, olprinone; P, procainamide; PF, propafenone; PJRT, permanent junctional reciprocating tachycardia; PP, propranalol; PVC, premature ventricular contraction; RAA, right atrial appendage; RFA, radiofrequency ablation; S, sotalol; Sp, spironolactone; TIC, tachycardia-induced cardiomyopathy; V, verapamil; VT, ventricular tachycardia.

be partially resistant to conventional antiarrhythmic medications and tends to cause TIC if left untreated. It is one of the leading causes of TIC in children, with 59% of paediatric TIC cases in the largest international multicentre study conducted by Moore et al.[4] having AT as the underlying arrhythmia.[4,13] This was slightly different from our review where 41.7% of patients presented with AT. AT in paediatric populations is likely to have a different mechanism than that of adults because children possess immature myocardium, whereas AT in adults is caused by diseased atrial myocardium and accompanying local degenerative processes, which is not reversible.[25] In a paediatric multicentre retrospective review by Kang et al.,[26] 28% of patients diagnosed with AT had progression to cardiomyopathy, whereas the incidence rate was 22.6% in another paediatric study conducted by Ge et al.[27] In Kang et al.'s study,[26] spontaneous resolution was seen in 74% of patients presenting with AT below 3 years of age, whereas it was seen in only 13% of patients presenting at age 3 years or above. Successful tachycardia control by antiarrhythmic medication was seen in 72% of patients; despite this, RFA was often elected as the success rate was slightly higher than that of medication treatment at 81%.[26] Fifteen of the 20 patients with AT in our review had an ablation, and 3 of these 15 patients had an additional atrial appendectomy to control the arrhythmia. One patient with AT required ECMO support after ablation.[6] Other treatments such as the use of ivabradine, a selective inhibitor of hyperpolarization-activated cyclic nucleotide-gated channels that lowers HR as it mediates the pacemaker funny current in the sinoatrial node, have also been explored.[28] In a study conducted by Xu et al.,[29] ivabradine was used as monotherapy for the treatment of AT, and 50% of patients had effective suppression of tachycardia. In our review, ivabradine was used in 5 patients who did not require RFA. Moreover, patients with AT had a mean age of 7.4 ± 4.69 years at presentation (range: 1 month to 13 years; n = 20) and the mean LVEF was 34.58% ± 14.2% (range: 17%-56%; n = 19). In patients with AT, time to normalization of LVEF was widely distributed, ranging from 3 days to 1 year with a mean of 87.8 ± 121.25 days (n = 15). The recovery period for AT was the second longest period among the tachyarrhythmias after PJRT.

PJRT is the second most common cause of TIC in the paediatric population. In 1967, Coumel et al.[30] published the first description of PJRT, a rare and incessant SVT that predominantly affects infants and children, is unlikely to resolve spontaneously, often refractory to treatment, and is characterized by a prolonged RP interval.[31] Atrioventricular re-entry is the cause of PJRT, with the AV node responsible for anterograde conduction and the decremental and slow conducting accessory pathway often located in the tricuspid posteroseptal location, for retrograde conduction.[31–33] PJRT is associated with a high risk of TIC and is one of the most common types of arrhythmias seen in TIC paediatric cases.[4,15] In our review, PJRT accounted for 20.8% of the patients who presented with TIC, which was comparable to 23% as reported by Moore et al.[4] in their international multicentre study.[4] In our data, patients presenting with PJRT had a mean age of 5.53 ± 4.56 years (range: day 1 of life to 14.5 years; n = 10). At presentation, the mean LVEF was 30.9% ± 14.1% (range: 10%-57%; n = 10). Time to normalized LVEF ranged from 1 month to 1 year with a mean of 93.5 ± 121.61 days (n = 10).

VT is the third most common cause for TIC according to our study. Sixteen percent of the patients in our case series presented with VT preceding TIC diagnosis, while it was diagnosed only in 7% of patients with TIC from Moore et al.'s international multicentre study.[4] In Moore et al.'s study,[4] 50% of patients with VT achieved partial control or full reversion to SR with only medication treatment and the rest underwent successful ablation. Only 1 case in our review was successfully treated pharmacologically, and 7 of 8 patients were successfully treated with RFA.[34] The most common documented type of VT in our review is fascicular VT, found in 6 of 8 patients. Fascicular VT was first described by Cohen et al.[35] in the 1970s. In 90%-95% of patients with fascicular VT, right bundle branch block and left superior axis morphology can be identified, with the arrhythmia often arising from the left fascicles.[36] Arrhythmia can technically originate from any part of the fascicular system in both structurally normal and abnormal hearts.[37] In our cases, patients presenting with VT had a mean age of 9.98 ± 4.35 years (range: 5-15.83 years; n = 8). At presentation, the mean LVEF was 41.75% ± 9.6% (range: 25%-54%; n = 8). Time to normalized LVEF ranged from 5 to 180 days with a mean of 51.25 ± 85.9 days (n = 4).

Frequent PVCs are asynchronous myocardial contractions that can contribute to the development of TIC as well.[11] A swine model was designed by Walters et al.[38] to replicate conditions of frequent PVCs; they found that PVC-induced cardiomyopathy was mechanistically different from that of TIC and that more severe LV systolic dysfunction and calcium mishandling was observed.

Not all cases of cardiomyopathy are tachyarrhythmia-induced; therefore, distinguishing the underlying etiology is crucial for selecting the most effective treatment. Pre-excitation-induced cardiomyopathy is another form of cardiomyopathy seen in the paediatric population.[39–41] It is characterized by LV remodelling, dilation, and dysfunction combined with persistent ventricular pre-excitation on the ECG instead of persistent tachycardia.[39] Dyssynchrony caused by pre-excitation has been identified as the mechanism leading to cardiomyopathy in pre-excitation-induced cardiomyopathy cases.[39–41] In a case series of 12 paediatric patients by Dai et al.,[40] the mean LVEF before RFCA was 42.63% ± 4.34%. AVRT and atrioventricular nodal re-entrant tachycardia are rare but possible causes of TIC with 5% of paediatric TIC patients having AVRT as the causative arrhythmia in Moore et al.'s study.[4,12] In our case series, 3 patients presented with AVRT and 2 patients with atrioventricular nodal re-entrant tachycardia, accounting for 10.4% of total patients with TIC. Pacing-induced cardiomyopathy occurs when chronic RV pacing and LV systolic dysfunction occur in conjunction.[1] In children who require RV pacing, 5%-10% will be diagnosed with pacing-induced cardiomyopathy, and the development of the cardiomyopathy is slow and typically occurs 15 years after pacemaker implantation if it were to develop.[1] In these cases, switching to single site pacing of the LV or a biventricular system could lead to improvement and reverse the cardiomyopathy.[1]

Study limitations

This study has some important limitations, primarily due to its retrospective nature. The study includes a relatively small population size, which we believe is a result of underpublication in this field in recent years. Because the review is composed of case reports and series from different centres, some parameters were reported inconsistently. It is also likely that case reports usually delve into more rare entities than case series or larger studies. In addition, our focus was on LV systolic function, as there was insufficient information available on LV diastolic function assessment across the different cases. Furthermore, this study features a wide range of follow-up timeframes, predominantly involving short-term follow-up, while the reporting of long-term follow-up data, which is critically important in these cases, was lacking. There is a need for larger retrospective and prospective studies with standardized protocols and extended follow-up periods to further explore the relationship between different types of arrhythmias associated with TIC and to determine the optimal treatment strategies, outcomes, and recovery times.

Conclusions

TIC is not uncommon in the paediatric population. Diagnosing TIC and differentiating it from mimicking etiologies can be challenging and requires a high index of suspicion whenever incessant tachyarrhythmias are identified. This review supported the previous finding of AT being the most common etiology of TIC in paediatrics and confirmed that PJRT and VT are the second and third most common etiologies, respectively, in keeping to what was reported in the existing literature.[4,7] There was no difference in age between the different etiologies at presentation. In our review, 70.8% of patients underwent RFA, which supports the importance of invasive intervention for the rescue of these patients. The average time to recovery was 80.47 days, which was longer than reported periods in previous studies. This finding is significant, as it suggests that patients with TIC require long-term treatment and follow-up before normalization occurs.

Acknowledgement

The authors acknowledge Ye Shen for his contributions towards statistical analysis.

Ethics Statement

This article adheres to the relevant ethical guidelines and permission was granted for this study from the University of British Columbia's review and ethics board.

Patient Consent

The authors confirm that a patient consent form is not applicable to this article as this is a retrospective case report and literature review article that uses deidentified data; therefore, the institutional review board did not require consent from the patients.

Funding Sources

No funding was received for this study.

Disclosures

The authors have no conflicts of interest to disclose.

References

1. Lipshultz SE, Law YM, Asante-Korang A, et al. Cardiomyopathy in children: classification and diagnosis: a scientific statement from the American Heart Association. *Circulation*. 2019;140:e9–e68.
2. Towbin JA, Lowe AM, Colan SD, et al. Incidence, causes, and outcomes of dilated cardiomyopathy in children. *JAMA*. 2006;296:1867–1876.
3. Gopinathannair R, Etheridge SP, Marchlinski FE, et al. Arrhythmia-induced cardiomyopathies: mechanisms, recognition, and management. *J Am Coll Cardiol*. 2015;66:1714–1728.
4. Moore JP, Patel PA, Shannon KM, et al. Predictors of myocardial recovery in pediatric tachycardia-induced cardiomyopathy. *Heart Rhythm*. 2014;11:1163–1169.
5. Ponnusamy SS, Muthu G, Kumar M, et al. Safety, efficacy, and intermediate-term outcomes of radiofrequency catheter ablation for paediatric arrhythmias. *Cureus*. 2020;12:e10488.
6. Arslan A, Erdoğan İ, Varan B, et al. Reversible cardiomyopathy-tachycardiomyopathy in children. *Turk J Pediatr*. 2019;61:552–559.
7. Kafalı HC, Öztürk E, Özgür S, et al. Electrophysiologic characteristics and catheter ablation results of tachycardia-induced cardiomyopathy in children with structurally normal heart. *Anatol J Cardiol*. 2020;24:370–376.
8. Kylat RI, Samson RA. Permanent junctional reciprocating tachycardia in infants and Children. *J Arrhythm*. 2019;35:494–498.
9. Spahic A, Chen TH, Geller JC, Saenger J, Ohlow MA. Life in the fast lane: clinical and immunohistological characteristics of tachycardia-induced cardiomyopathy-a retrospective study in 684 patients. *Herzschrittmacherther Elektrophysiol*. 2020;31:292–300.
10. Vera A, Cecconi A, Martínez-Vives P, et al. Electrocardiogram and CMR to differentiate tachycardia-induced cardiomyopathy from dilated cardiomyopathy in patients admitted for heart failure. *Heart Vessels*. 2022;37:1850–1858.
11. Báez Cabanillas MV, Colque R, Tibaldi M, et al. Emerging concepts in heart failure management and treatment: focus on tachycardia-induced cardiomyopathy. *Drugs Context*. 2023;12:2022–2028, 4.
12. Shoureshi P, Tan AY, Koneru J, et al. Arrhythmia-induced cardiomyopathy: JACC state-of-the-art review. *J Am Coll Cardiol*. 2024;83:2214–2232.
13. Kang KT, Potts JE, Radbill AE, et al. Permanent junctional reciprocating tachycardia in children: a multicenter experience. *Heart Rhythm*. 2014;11:1426–1432.
14. Sahin GT, Kafali HC, Ozturk E, Guzeltas A, Ergul Y. Catheter ablation of focal atrial tachycardia in children using three-dimensional electroanatomic mapping system: a 6-year single-centre experience. *Cardiol Young*. 2021;31:744–750.
15. Velázquez-Rodríguez E, García-Hernández N, Martínez-Sánchez A, et al. Coumel tachycardia in children. *Bol Med Hosp Infant Mex*. 2023;80:69–76.
16. Gossage A, Braxton Hicks J. On auricular fibrillation. *QJM*. 1913;6:435–440.
17. Parkinson J, Campbell M. Paroxysmal auricular fibrillation. A record of two hundred patients. *QJM*. 1930;24:67–100.
18. Brill IC. Auricular fibrillation with congestive failure and no other evidence of organic heart disease. *Am Heart J*. 1937;13:175–182.

19. Shinbane JS, Wood MA, Jensen DN, et al. Tachycardia-induced cardiomyopathy: a review of animal models and clinical studies. *J Am Coll Cardiol*. 1997;29:709–715.

20. Whipple GH, Sheffield LT, Woodman EG, Theophilis C, Friedman S. Reversible congestive heart failure due to chronic rapid stimulation of the normal heart. *Proc N Engl Cardiovasc Soc*. 1962;20:39–40.

21. Bozkurt B, Colvin M, Cook J, et al. Current diagnostic and treatment strategies for specific dilated cardiomyopathies: a scientific statement from the American Heart Association. *Circulation*. 2016;134:e579–e646.

22. Jeong YH, Choi KJ, Song JM, et al. Diagnostic approach and treatment strategy in tachycardia-induced cardiomyopathy. *Clin Cardiol*. 2008;31:172–178.

23. Gelb BD, Garson A. Noninvasive discrimination of right atrial ectopic tachycardia from sinus tachycardia in "dilated cardiomyopathy". *Am Heart J*. 1990;120:886–891.

24. Naheed ZJ, Strasburger JF, Benson DW, Deal BJ. Natural history and management strategies of automatic atrial tachycardia in children. *Am J Cardiol*. 1995;75:405–407.

25. Wang JN, Wu JM, Tsai YC, Lin CS. Ectopic atrial tachycardia in children. *J Formos Med Assoc*. 2000;99:766–770.

26. Kang KT, Etheridge SP, Kantoch MJ, et al. Current management of focal atrial tachycardia in children: a multicenter experience. *Circ Arrhythm Electrophysiol*. 2014;7:664–670.

27. Ge H, Li X, Liu H, Jiang H. Predictors of pharmacological therapy of ectopic atrial tachycardia in children. *Pediatr Cardiol*. 2017;38:289–295.

28. Banavalikar B, Shenthar J, Padmanabhan D, et al. Clinical and electrophysiological correlates of incessant ivabradine-sensitive atrial tachycardia. *Circ Arrhythm Electrophysiol*. 2019;12:e007387.

29. Xu X, Guo Y, Gao W, et al. Ivabradine monotherapy in pediatric patients with focal atrial tachycardia: a single-center study. *Eur J Pediatr*. 2023;182:2265–2271.

30. Coumel P, Cabrol C, Fabiato A, Gourgon R, Slama R. Tachycardie permanent par rhythm reciproque. *Arch Mal Coeur*. 1967;60:1830–1864.

31. Ergül Y, Sulu A, Çaran B, et al. Clinical course and electrophysiological characteristics of permanent junctional reciprocating tachycardia in children. *Anatol J Cardiol*. 2022;26:880–885.

32. Critelli G, Gallagher JJ, Monda V, et al. Anatomic and electrophysiologic substrate of the permanent form of junctional reciprocating tachycardia. *J Am Coll Cardiol*. 1984;4:601–610.

33. Vaksmann G, D'Hoinne C, Lucet V, et al. Permanent junctional reciprocating tachycardia in children: a multicentre study on clinical profile and outcome. *Heart*. 2006;92:101–104.

34. Ünal Yüksekgönül A, Azak E, Akalın A, et al. Efficacy of flecainide in bidirectional ventricular tachycardia and tachycardia-induced cardiomyopathy with Andersen-Tawil syndrome. *Eur J Med Genet*. 2022;65:104499.

35. Cohen HC, Gozo EG, Pick A. Ventricular tachycardia with narrow QRS complexes (left posterior fascicular tachycardia). *Circulation*. 1972;45:1035–1043.

36. Lerman BB, Stein KM, Markowitz SM. Mechanisms of idiopathic left ventricular tachycardia. *J Cardiovasc Electrophysiol*. 1997;8:571–583.

37. Kapa S, Gaba P, DeSimone CV, Asirvatham SJ. Fascicular ventricular arrhythmias: pathophysiologic mechanisms, anatomical constructs, and advances in approaches to management. *Circ Arrhythm Electrophysiol*. 2017;10:e002476.

38. Walters TE, Rahmutula D, Szilagyi J, et al. Left Ventricular dyssynchrony predicts the cardiomyopathy associated with premature ventricular contractions. *J Am Coll Cardiol*. 2018;72:2870–2882.

39. Zhang Y, Jiang H, Cui J, et al. Ablation of ventricular preexcitation to cure preexcitation-induced dilated cardiomyopathy in infants: diagnosis and outcome. *Circ Arrhythm Electrophysiol*. 2023;16:e011569.

40. Dai C, Guo B, Han L, et al. Dyssynchrony induced by ventricular preexcitation: a risk factor for the development of dilated cardiomyopathy. *Cardiovasc Innov Appl*. 2020;5:9–18.

41. Miyazaki A, Uemura H. Perspective of preexcitation induced cardiomyopathy; early septal contraction, and subsequent rebound stretch. *J Cardiol*. 2022;79:30–35.

42. McKenzie K, Ferns SJ. Epicardial ablation of persistent junctional reciprocating tachycardia in an infant with tachycardia-induced cardiomyopathy. *JACC Case Rep*. 2021;3:512–516.

43. Sullivan JS, Maitoza LA, Brysiewicz NR, Hall EK, Beach CM. Electromagnetic interference complicating Impella. *Ann Pediatr Cardiol*. 2022;15:533–535.

44. Tarek A, Ali W, Sayed M, Hammouda T, Kamel O. Para-Hisian atrial tachycardia: a neglected cause of severe heart failure in children. *Glob Cardiol Sci Pract*. 2022;2022:e202204.

45. Mukharjee SS, Mohapatra SK, Mukherji A, De A, Singhi AK. Radiofrequency ablation for fascicular ventricular tachycardia causing tachycardiomyopathy and brief literature review. *Ann Pediatr Cardiol*. 2021;14:99–104.

46. Vijay S, Bansal R, Kidambi BR, Naik N. A unique location of the accessory pathway in a child with permanent junctional reciprocating tachycardia. *Pacing Clin Electrophysiol*. 2024;47:925–928.

47. Sakai T, Tsuboi K, Takarada S, et al. Tachycardia-induced cardiomyopathy in an infant with atrial flutter and prolonged recovery of cardiac function. *J Clin Med*. 2024;13:3313.

48. Liu J, Cao X, Li C, et al. Case report: efficacy analysis of radiofrequency catheter ablation combined with atrial appendage resection for atrial tachycardia originating from the atrial appendage in children. *Front Cardiovasc Med*. 2022;9:990325.

49. Papadopoulou M, Tramma D, Thomaidis K, Papadopoulou-Legbelou K. Tachycardia induced cardiomyopathy in an infant with atrial flutter: a challenging but reversible cause of heart failure. *Pediatr Neonatol*. 2019;60:477–478.

50. Kwon HW, Bae EJ, Kwak JG, Seo JW. Successful video-assisted thoracoscopic atrial appendectomy in a 4-year-old child with intractable atrial tachycardia and tachycardia-induced cardiomyopathy. *Cardiol Young*. 2020;30:869–872.

51. Karmegaraj B, Balaji S, Raju PN, et al. Tachycardia-induced cardiomyopathy secondary to incessant ectopic atrial tachycardia in two infants: Potential new indication for early initiation of enteral ivabradine. *Ann Pediatr Cardiol*. 2021;14:422–427.

52. Wu L, Tian H, Wang F, Liang X, Chen G. Successful ablation of incessant idiopathic right ventricular tachycardia arising from unusual sites in children. *Cardiol Young*. 2016;26:764–771.

53. Poghosyan HR, Danoyan AB, Hovakimyan TB, Kartoyan ZE, Davtyan KV. An uncommon case of incessant tachycardia-induced cardiomyopathy in a child. *J Innov Card Rhythm Manag*. 2018;9:2982–2987.

54. Raja DC, Saravanan S, Sathishkumar AG, Pandurangi UM. Right atrial appendage tachycardia: a rare cause of tachycardia induced cardiomyopathy in a 4-year-old child. *Indian Pacing Electrophysiol J*. 2018;18:176–179.

55. Errahmouni A, Bun SS, Latcu DG, Tazi-Mezalek A, Saoudi N. Accelerated idioventricular rhythm requiring catheter ablation in a child: the dark side of a benign arrhythmia. *Ann Cardiol Angeiol (Paris)*. 2017;66:323–325.

56. Ahmadi A, Zolfi-Gol A, Arasteh M. Tachycardia-induced cardiomyopathy. *ARYA Atheroscler*. 2014;10:175–178.

57. Ergül Y, İrdem A, Esen O, Güzeltaş A. Successful cryoablation of incessant fast-slow atypical atrioventricular nodal reentrant tachycardia in a child with tachycardia-induced cardiomyopathy. *Turk Kardiyol Dern Ars*. 2018;46:406–410.

58. Ergul Y, Ozturk E, Ozgur S, et al. Ivabradine is an effective antiarrhythmic therapy for congenital junctional ectopic tachycardia-induced cardiomyopathy during infancy: case studies. *Pacing Clin Electrophysiol*. 2018;41:1372–1377.

59. Gardiner M, Ruttan TK, Kienstra AJ, Wilkinson M. Making the quick diagnosis: a case of neonatal shock. *J Emerg Med*. 2017;52:e139–e144.

60. Aykan HH, Karagöz T, Akın A, et al. Results of radiofrequency ablation in children with tachycardia-induced cardiomyopathy. *Anadolu Kardiyol Derg*. 2014;14:625–630.