Ablation of atrial tachyarrhythmias late after surgical correction of tetralogy of Fallot: long-term follow-up

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Abstract

Background: After the surgical correction of tetralogy of Fallot, surgical scars and natural obstacles form pathways capable of supporting an atrial tachyarrhythmia (AT). Radiofrequency (RF) ablation is effective, although the few studies published on this topic had relatively short follow-up periods.

Aim: The aims of the study were to evaluate the acute and long-term effects of RF ablation of AT and examine the characteristics of arrhythmia recurrence.

Methods: Tetralogy of Fallot patients (n = 16, age 44.7 ± 10.7 years) referred for ablation of ATs, appearing 25.7 ± 9.6 years after repair, were studied.

Results: Twenty-five ATs were ablated, including 16 cavo-tricuspid isthmus atrial flutters (CTI-AFLs) and nine intraatrial reentrant tachycardia (IART). In one patient with paroxysmal atrial fibrillation (PAF), pulmonary vein isolation was also performed. Ten patients had permanent, and six had paroxysmal atrial fibrillation (PAF) prior to the first ablation. Four patients had PAF. Regardless of the type of the first ablated arrhythmia, all 16 patients required CTI-AFL ablation. The effectiveness of the first RF ablation reached 88%. The acute efficacy of RF ablation was 100% for CTI-AFL and 78% for IART. Long-term follow-up was possible in 15 out of 16 patients (mean follow-up 68.8 ± 36.6 months). Four patients were free of sustained arrhythmia, nine (60%) had AF. After the last RF ablation, an episode suggestive of CTI-AFL/IART was documented only in one patient.

Conclusions: Ablation of CTI-AFL/IART in tetralogy of Fallot patients is safe and effective. AF was observed in most patients during the long-term follow-up. Regardless of the type of the first ablated arrhythmia, all patients required CTI-AFL ablation.

Key words: tetralogy of Fallot, radiofrequency ablation, arrhythmia, atrial flutter

INTRODUCTION

Treatment of arrhythmia is not only a medical challenge but also one of the major economic issues in modern cardiology. Atrial arrhythmia is responsible for most arrhythmia-related hospitalisations [1]. Approximately 25% of adult hospital admissions due to congenital heart disease are associated with arrhythmia [2]. Tetralogy of Fallot (TOF) is the most common cyanotic congenital heart condition with an incidence of 1 in 2500; in patients with this defect, the presence of atrial arrhythmia is associated with higher mortality [3–5]. After the surgical correction of congenital or acquired heart disease, surgical scars and natural conduction obstacles form narrow pathways capable of supporting an intraatrial reentrant tachycardia (IART) [6, 7].
In younger patients with TOF, IART is more frequent than atrial fibrillation (AF). However, in older patients, the prevalence of AF exceeds that of IART [8].

Atrial flutter (AFL)/IART late after the surgical repair of TOF occurs in up to 30% of patients, with a higher prevalence in patients with significantly greater biatrial dilatation [9–11].

Most previous reports concerning patients after TOF surgery and radiofrequency (RF) ablation included small patient groups and reported relatively short follow-up times (23 ± 22 to 45 ± 24 months) [10–13].

The aims of the study were to evaluate the acute safety and efficacy as well as long-term outcomes of RF ablation of atrial arrhythmias and to examine the characteristics of arrhythmia recurrence in TOF patients.

METHODS

We retrospectively analysed the data of over 4000 RF catheter ablations performed at the Institute of Cardiology (2008–2016). Among the studied patients, 24 had a history of surgical correction of TOF.

Inclusion criteria were as follows: 1) history of surgical correction of TOF; 2) electrocardiographically (ECG) documented episodes of atrial tachyarrhythmia; and 3) history of RF catheter ablation of atrial tachyarrhythmia (AFL, atrial tachycardia). The criteria were fulfilled by 16 patients (men 56.25%; mean age 44.7 ± 10.7 years; age range 23–67 years) (Table 1).

We analysed the electrophysiological, echocardiographic, surgical, and clinical data to define the safety and long-term efficacy of RF ablation.

Follow-up

Patients were followed up in an outpatient clinic of our institute. All patients underwent 12-lead ECG, echocardiography, and Holter ECG monitoring at least three times during the follow-up period. Holter monitoring was performed at six-month intervals in the first year after the procedure and at 12-month intervals thereafter.

Patients were asked regularly about the symptoms of recurrent arrhythmia and were encouraged to notify the clinic if the symptoms recurred at any time during the follow-up period. In patients with implanted pacemakers, all recorded episodes of arrhythmia were analysed. Patients with electrocardiographically documented recurrence of arrhythmia were offered repeat ablation.

Statistical analysis

Categorical variables are expressed as numbers and percentages, while continuous variables are expressed as medians and interquartile ranges or means and standard deviations. Categorical variables were compared using the χ² test. P values < 0.05 were considered significant. Analyses were performed using SPSS software (IBM, Armonk, NY, USA).

Procedure

All antiarrhythmic agents were discontinued for > 72 h before the electrophysiological study and ablation. Prior to the procedure, all patients received full oral anticoagulation according to the current guidelines [14]. The electrophysiological study was performed under conscious sedation after fasting. The initial heparin bolus at a dose of 50–100 IU/kg (depending on international normalised ratio value) was administered at the beginning of the procedure, and subsequent 1000-IU boluses were given after each consecutive hour. Intracardiac bipolar electrograms and 12-lead ECG were digitally recorded. All measurements were performed using on-screen electronic callipers at a sweep speed of 100 to 200 mm/s and a 0.1 to 0.2 mV/cm gain.

We used a 7 F steerable, diagnostic quadripolar catheter with 2–5–2 mm interelectrode spacing (Marinr MCXL, Medtronic, Inc., Minneapolis, MN, USA) placed in the coronary sinus in order to perform electrophysiological study and as a reference for atrial tachyarrhythmia (cavo-tricuspid isthmus atrial flutters [CTI-AFLs], IART).

A system for three-dimensional (3D) imaging (CARTO XP or CARTO 3, Biosense Webster, Inc., Diamond Bar, CA, USA) was used in all patients.

A THERMOCOOL catheter was used 12 times, a NAVISTAR Dual Sensor (DS) ablation catheter (Biosense Webster, Inc.) was used twice, and a SMARTTOUCH (Biosense Webster, Inc.) ablation catheter (routinely since 2014) was applied during nine procedures.

Intracardiac mapping and entrainment procedures

Mapping procedures were guided by a 3D electroanatomical mapping system. Where possible, bipolar voltage maps in sinus rhythm and/or activation map of the present arrhythmia were created at the discretion of the operator [13]. Tachycardia mechanisms were diagnosed based on electrogram examination, activation, and entrainment mapping [11].

If atrial tachyarrhythmias were not present at the start of the procedure, induction was attempted with programmed stimulation that included up to two atrial extra-stimuli after a paced drive train of eight beats to a point of atrial refractoriness and burst pacing at the discretion of the operator [13]. During atrial tachycardia/AFL, ECG recordings were evaluated. The preprocedural evaluation included a careful review of the correction of the congenital anatomic defect and the reparative surgical operations undertaken. In addition, the most recently performed echocardiograms were reviewed.

In cases of sustained atrial arrhythmia, our aim was to identify an isthmus of slow conduction that was critical to the tachycardia circuit using electroanatomical mapping and entrainment techniques (Figs. 1, 2).

Entrainment was performed from the atrium during tachycardia at cycle lengths 10 to 40 ms shorter than that of the tachycardia [15].
<table>
<thead>
<tr>
<th>No.</th>
<th>Sex</th>
<th>Age during TOF correction</th>
<th>Number of surgical procedures</th>
<th>Type of surgical procedure</th>
<th>No. of RF ablations</th>
<th>Arrhythmia prior to RF</th>
<th>Age at first ablation of IART/CTI-AFL</th>
<th>Follow-up [months]</th>
<th>Arrhythmia during follow-up</th>
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<tbody>
<tr>
<td>1</td>
<td>F</td>
<td>16</td>
<td>2</td>
<td>TOF repair age 16; re-op + PVR + RVOT patch, VSD and ASD closure age 42</td>
<td>2 (1 VT)</td>
<td>1. VT 2. Paroxysmal CTI-AFL</td>
<td>51</td>
<td>96</td>
<td>Permanent AF</td>
</tr>
<tr>
<td>2</td>
<td>M</td>
<td>BT 7, 14</td>
<td>4</td>
<td>BT 7, TOF repair age 14; re-op VSD, closure and PVR, Sapien-Edwards 23 mm, tricuspid valve plasty age 36; re-op TVR Hancock II age 40, re-op VSD closure, PVR age 44, percutaneous pulmonary valve implantation age 48, re-op VSD, re-op pulmonary valve 52</td>
<td>1</td>
<td>1. Persistent CTI-AFL</td>
<td>45</td>
<td>91</td>
<td>Death due to renal failure and heart failure after cardiac surgery, PAF</td>
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<td>3</td>
<td>F</td>
<td>6</td>
<td>4</td>
<td>TOF repair age 6; re-op VSD closure, re-op VSD age 9, mitral valve plasty age 10, MVR SJM 29 mm, TVR (biological Biocor 30 mm), PVR (homograft), age 39</td>
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<td>1. Paroxysmal CTI-AFL, PAF — not targeted</td>
<td>32</td>
<td>80</td>
<td>Death due to car accident 7 years after RF ablation, PAF</td>
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<tr>
<td>4</td>
<td>F</td>
<td>BT 6, 36</td>
<td>4</td>
<td>BT age 6 and 13, TOF repair age 36; re-op PVR homograft 21 mm and tricuspid valve plasty (ring 32 mm), right ventricular plasty, VSD closure, pulmonary homograft age 54</td>
<td>1</td>
<td>1. Paroxysmal CTI-AFL, PAF — not targeted</td>
<td>47</td>
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<td>Permanent AF</td>
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<td>5</td>
<td>F</td>
<td>9</td>
<td>1</td>
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<td>1. Persistent CTI-AFL</td>
<td>41</td>
<td>38</td>
<td>Without arrhythmia</td>
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<tr>
<td>6</td>
<td>M</td>
<td>52</td>
<td>1</td>
<td>TOF repair age 52, AVR SJM Regent 25 mm, TVR Mosaic 31 mm age 52</td>
<td>2</td>
<td>1. Persistent CTI-AFL, 2. Persistent RA IART</td>
<td>53 and 57</td>
<td>54</td>
<td>Permanent AF</td>
</tr>
<tr>
<td>7</td>
<td>M</td>
<td>4</td>
<td>1</td>
<td>TOF repair age 4; percutaneous ASD II closure age 22</td>
<td>3</td>
<td>1. Persistent RA IART 2. Persistent RA IART 3. Persistent CTI-AFL</td>
<td>21, 22 and 23</td>
<td>37</td>
<td>Without arrhythmia</td>
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<td>8</td>
<td>F</td>
<td>BT 7, 30</td>
<td>4</td>
<td>BT age 7 and 13, TOF repair age 30, RVOT plasty, PVR homograft, tricuspid valve plasty (Edwards Lifesciences 32 mm) age 58</td>
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<td>1. Persistent CTI-AFL, PAF — not targeted</td>
<td>53</td>
<td>80</td>
<td>Permanent AF</td>
</tr>
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<td>9</td>
<td>F</td>
<td>7</td>
<td>2</td>
<td>TOF repair age 7, re VSD closure and RVOTO repair, tricuspid valve plasty, pulmonary valve valvulotomy age 43</td>
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<td>1. Paroxysmal CTI-AFL</td>
<td>46</td>
<td>105</td>
<td>Without arrhythmia</td>
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<td>No.</td>
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<td>Age during TOF correction</td>
<td>Number of surgical procedures</td>
<td>Type of surgical procedure</td>
<td>No. of RF ablations</td>
<td>Arrhythmia prior to RF</td>
<td>Age at first ablation of IART/CTI-AFL</td>
<td>Follow-up [months]</td>
<td>Arrhythmia during follow-up</td>
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<tr>
<td>10</td>
<td>M</td>
<td>13</td>
<td>3</td>
<td>TOF repair age 13 and 15, PVR, tricuspid valve plasty, pulmonary homograft, age 41</td>
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<td>1. Persistent CTI-AFL</td>
<td>38</td>
<td>40</td>
<td>Death due to post-surgery complications 3 years after ablation, 2 years after RF ablation permanent AF</td>
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<td>11</td>
<td>F</td>
<td>11</td>
<td>2</td>
<td>Brock correction age 11, VSD suture, RVOT plasty, pulmonary valvulotomy, age 35</td>
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<td>1. Persistent CTI-AFL</td>
<td>61</td>
<td>16</td>
<td>Without arrhythmia</td>
</tr>
<tr>
<td>12</td>
<td>M</td>
<td>7</td>
<td>2</td>
<td>TOF repair age 7, re VSD closure, pulmonary valvulotomy age 38</td>
<td>1</td>
<td>1. Persistent RA IART</td>
<td>39</td>
<td>92</td>
<td>RAF</td>
</tr>
<tr>
<td>13</td>
<td>M</td>
<td>BT 2, 3</td>
<td>3</td>
<td>BT age 2, TOF correction age 3, pulmonary homograft, age 11, pulmonary artery stenting age 14, percutaneous pulmonary valve implantation age 24</td>
<td>1</td>
<td>1. Paroxysmal CTI-AFL, RA IART (figure B phenomenon)</td>
<td>29</td>
<td>14</td>
<td>1 × wide QRS tachycardia</td>
</tr>
<tr>
<td>14</td>
<td>F</td>
<td>16</td>
<td>1</td>
<td>TOF correction age 16</td>
<td>1</td>
<td>1. Persistent CTI-AFL, RA IART (figure B phenomenon)</td>
<td>44</td>
<td>-</td>
<td>Lost during follow-up</td>
</tr>
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<td>15</td>
<td>F</td>
<td>41</td>
<td>1</td>
<td>TOF correction age 41</td>
<td>1</td>
<td>1. Persistent CTI-AFL</td>
<td>52</td>
<td>92</td>
<td>1 × ECV of AFL/IART</td>
</tr>
<tr>
<td>16</td>
<td>M</td>
<td>4</td>
<td>1</td>
<td>TOF correction age 4, percutaneous pulmonary valve implantation (Sapien XT) age 35</td>
<td>4</td>
<td>1. Persistent CTI-AFL and RA IART (figure B phenomenon) 2. RA IART 3. RA/LA AT (self-termination during procedure) 4. IART from septum</td>
<td>31, 33, 34, 35</td>
<td>45</td>
<td>RAF</td>
</tr>
</tbody>
</table>

AF — atrial fibrillation; AFL — atrial flutter; ASD — atrial septal defect; AT — atrial tachycardia; AVR — aortic valve replacement; BT — Blalock-Taussig; CTI-AFL — cavotricuspid isthmus atrial flutter; ECV — external cardioversion; F — female; IART — intraatrial reentrant tachycardia; LA — left atrium; LSPV — left superior pulmonary vein; M — male; MVR — mitral valve replacement; PA — pulmonary artery; PAF — paroxysmal atrial fibrillation; PVR — pulmonary valve replacement; RA — right atrium; RA IART — postincisional intraatrial reentrant tachycardia from lateral part of right atrium; re-op — reoperation; RF — radiofrequency; RSPV — right superior pulmonary vein; RVOT — right ventricular outflow tract; RVOTO — right ventricular outflow tract obstruction; SJM — St. Jude Medical; TOF — tetralogy of Fallot; TVR — tricuspid valve replacement; VSD — ventricular septal defect; VT — ventricular tachycardia
Figure 1. Activation (A) and bipolar voltage map (B) in a patient with counter clockwise cavo-tricuspid isthmus atrial flutter (patient no. 11, Table 1). During ablation along a line across the cavo-tricuspid isthmus (dark red dots), a sudden change in tachycardia cycle length from 280 to 260 ms was observed. Control entrainment mapping denotes a change in flutter mechanism to intraatrial reentrant tachycardia.

Figure 2. Activation (A) and bipolar voltage map (B) performed during intraatrial reentrant tachycardia (IART) (right lateral projection; patient no. 11, Table 1). The electrical wave front turns around the lateral scar (dark blue dot) in a counter clockwise direction. During ablation in this region, IART again transformed to another arrhythmia. Cavo-tricuspid isthmus (CTI)-dependency was proven by entrainment manoeuvres. CTI-line was performed with arrhythmia termination. Additional line was performed on the lateral side of the right atrium along fragmented potentials.

When entrainment with concealed fusion was demonstrated, the extent of the critical slow zone was carefully mapped to identify entrance sites, exit sites, and bystander regions. Critical isthmus sites were characterised by the presence of entrainment with concealed fusion and the postpacing interval-tachycardia cycle length of \( \leq 30 \) ms [15].
RF ablation

If possible, RF energy was delivered to locations meeting the electrophysiological criteria for critical conduction sites based on the entrainment techniques described above. In cases of IART, the ablation line was performed through the critical isthmus to bridge the two surgically or naturally occurring barriers. For patients in whom entrainment with concealed fusion could be demonstrated in more than one region, RF energy was first targeted to the area at which the isthmus was considered anatomically narrowest [15].

The maximum power output during ablation line formation did not exceed 40 W.

In patients without arrhythmia during the procedure, we performed a programmed stimulation protocol that included two atrial extra-stimuli after a paced drive train of eight beats to a point of atrial refractoriness. In cases of no induction of the arrhythmia and ECG documenting typical pattern of CTI-AFL, a CTI line was performed.

In cases where atrial tachyarrhythmias were multiple or frequently changing, a decision was made at the discretion of the operator to perform a substrate-based ablation, if appropriate [13].

A bidirectional block in the cavo-tricuspid isthmus was demonstrated both during proximal coronary sinus pacing and during lateral right atrial pacing manoeuvres [16]. Only the clinically documented arrhythmias were targeted for ablation. Successful ablation was defined as: (i) achievement of bidirectional conduction block for isthmus-dependent AFL; or (ii) successful termination of IART.

In patients in whom tachycardia was terminated during RF current application, after the ablation we evaluated whether the arrhythmia remained inducible by performing programmed stimulation (up to two extra-stimuli) to the point of atrial refractoriness. Isoproterenol was not routinely used during the procedure.

Definitions

Cavo-tricuspid isthmus-dependent AFL required that the CTI be involved in the propagation of macroreentrant right atrial tachycardia. Any other type of macroreentrant atrial arrhythmia not involving the right isthmus was defined as IART.

Arrhythmia was considered persistent if it lasted > seven days or lasted < seven days and required interruption with pharmacological or electrical cardioversion.

Significant adverse events attributed to catheter ablation were defined as: death, stroke, transient ischaemic attack, peripheral embolism, cardiac tamponade or perforation, valve damage, and arteriovenous fistula requiring surgical intervention.

Severe mitral, tricuspid, or pulmonary regurgitation was defined as previously described [17, 18].

RESULTS

Patient characteristics

In 16 patients with TOF, 23 RF ablations of atrial tachyarrhythmia (mean 1.4; range 1–4) were performed. Those patients underwent 36 cardiosurgical corrections of TOF (mean 2.25; range 1–4) prior to ablation. Six patients underwent a single surgical procedure, four underwent two, two underwent three, and four underwent four. Mean age during the first TOF correction was 18.7 ± 15.2 years, and the mean time between TOF correction and RF ablation was 25.7 ± 9.6 years (range 3–38 years). Four patients underwent a Blalock-Taussig procedure, while one underwent a Brock procedure.

In three patients a pulmonary valve was percutaneously implanted. Two patients underwent percutaneous closure of a type II atrial septal defect. One patient underwent pacemaker implantation prior to the RF ablation.

ECG data

Fifteen out of 16 patients presented right bundle branch block QRS pattern, and one patient had a paced rhythm. Mean cycle lengths of CTI-AFL and IART were 298 ± 65 ms and 276 ± 66 ms, respectively.

Echocardiographic data

All patients underwent echocardiographic assessment, and the results are presented in Table 2. None of the analysed echocardiographic parameters were predictors of arrhythmia recurrence during follow-up.

Clinical and electrophysiological data

Twenty-six arrhythmias were ablated. Sixteen patients had CTI-AFL and cavotricuspid isthmus ablation was performed. During the electrophysiological procedures, we recorded CTI-AFL in 12 patients. In four patients with a documented typical counter clockwise CTI-AFL ECG pattern and no ongoing arrhythmia during the procedure, CTI ablation was performed empirically during sinus rhythm.

During the electrophysiological studies, we recorded nine IARTs. The IART isthmus was located in the lateral wall of the right atrium in eight cases, and in one case the isthmus was located in the lower interatrial septum. In three patients we observed a switch from one type of tachycardia to another during RF delivery, suggesting the figure 8 phenomenon (Patients 13, 14, and 16, Table 1).

Ten patients were diagnosed with permanent and six with paroxysmal arrhythmia prior to the first ablation. Four (25%) patients had AF, but in three patients it was not targeted during ablation. One patient with AF underwent pulmonary vein isolation in addition to CTI-AFL. None of the recorded arrhythmias were focal.

The mean procedure duration was 85 ± 35 min and the mean fluoroscopy dose was 72.99 ± 63.94 mGy.

Acute efficacy of RF ablation of typical AFL was 100%, and 78% for IART. One IART required external cardioversion to terminate, and in one patient the IART terminated spontaneously. The acute effectiveness of the first RF ablation reached 88%, and no major early or late complications were reported.
Long-term follow-up was available in 15 out of 16 patients (mean follow-up duration 68.8 ± 36.6 months) undergoing RF ablation. One patient was lost to follow-up.

Four patients did not present any kind of sustained arrhythmia during the follow-up. Among them, three patients had single, early surgical correction of TOF (mean age 6.6 years). Interestingly, one patient without arrhythmia during follow-up had severe insufficiency of tricuspid valve and dilated right atrium (patient no. 7; Table 2).

Nine (60%) patients experienced AF during follow-up: five had permanent and four had paroxysmal AF. Among the five patients with permanent AF, three had normal or almost normal valve function and atrial volume (patients 1, 6, and 8; Table 2), but one underwent atrial septal defect closure at the age of 42 years and the second had late TOF correction at the age of 52 years. The third patient had TOF correction performed at the age of 30 years and was 58 years old during the follow-up. The fourth patient developed tricuspid and pulmonary valve insufficiency and permanent AF.

Despite pulmonary vein isolation, one patient still had episodes of AF.

After the last RF ablation, an episode suggestive of CTI-AFL/IART was documented in only one patient. Another patient had a single episode of wide QRS complex tachycardia.

Three patients died during follow-up: two due to complications after surgical procedures at 89 and 24 months after RF ablation; the other, who had episodes of AF, died following a car accident seven years after RF ablation of a paroxysmal AFL-CTI.

**DISCUSSION**

Publications reporting the long-term follow-up of patients with TOF and RF ablation of atrial tachyarrhythmia remain scarce. Moreover, previously published data reported relatively short follow-up periods (from 23 ± 22 to 45 ± 24 months) [8, 10, 11].

To our knowledge, no reports have ever been published on a Polish population of patients after TOF correction and RF ablation of atrial tachyarrhythmia. Moreover, the current study reports the longest follow-up.

**Mechanism of atrial tachyarrhythmia in TOF patients**

In previous studies that included patients with TOF, the CTI-AFL mechanism of arrhythmia was more frequent than IART [10, 12, 13]. Our data support these findings; nevertheless, in our group, all patients underwent CTI-AFL ablation, regardless of the type of the first ablation arrhythmia. Such findings have not been reported until now.

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**Table 2. Echocardiographic parameters**

<table>
<thead>
<tr>
<th>Patient</th>
<th>EF [%]</th>
<th>LVDD [mm]</th>
<th>LA [cm²]</th>
<th>RA [cm²]</th>
<th>Severe MR [yes/no]</th>
<th>Severe TR [yes/no]</th>
<th>RVOT [mm]</th>
<th>TAPSE [mm]</th>
<th>RVSP [mmHg]</th>
<th>Severe PR [yes/no]</th>
<th>PV peak gradient [mmHg]</th>
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<tr>
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<td>45</td>
<td>20</td>
<td>26</td>
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EF — ejection fraction; LA — left atrium; LVDD — left ventricular diastolic diameter; NA — not available; MR — mitral regurgitation; RA — right atrium; PR — pulmonary regurgitation; PV — pulmonary valve; RVOT — right ventricular outflow tract; RVSP — right ventricular systolic pressure; TAPSE — tricuspid annular plane systolic excursion; TR — tricuspid regurgitation.

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Moreover, none of our patients had focal mechanism of arrhythmias. This finding has never been reported to date; however, our group was small. The acute efficacy of CTI-AFL and IART RF ablation noted in our group was similar to that reported by other authors [10, 12, 13].

All of our patients were treated with the help of a 3D navigation system.

The use of 3D navigation for IART is reimbursable (listed as simple ablation), but in patients with TOF and CTI-AFL prior to ablation, figure-eight phenomenon occurs in a significant percentage of patients. Our data can contribute to the ongoing discussions of the reimbursement of RF ablations in Poland.

**AF in TOF patients**

Interestingly, AF was documented in 25% of patients in our group prior to ablation, whereas other studies reported a prevalence of 6% to 11% [10, 12, 13]. Similarly, in our patients, AF occurred 3 to 5 times more frequently during the follow-up period (reaching 60%) than in the reports of other authors [10, 12, 13].

The length of follow-up in the current study was among the factors that may have influenced the high frequency of AF in the study group.

Older age at the time of TOF correction is a known risk factor for arrhythmia; nevertheless, age at the time of corrective surgery in our group was almost identical to that reported in the largest multicentre study published to date. The time from surgery to the first ablation and the mean age at the first ablation were also virtually the same [12].

In contrast to other studies, number of cardiac surgeries and patient age were not predictors of arrhythmia recurrence in this study [12, 13]. As reported by Ezzat et al. [13], the presence of AF prior to ablation was a predictor of future AF.

We were unable to directly compare surgical techniques and the medical histories of our patients to previously described groups. However, TOF patients do not constitute a homogenous group because their arrhythmic risks and burdens can vary.

Tricuspid, mitral, and pulmonary regurgitation are known risk factors for AF in TOF patients; nevertheless, our group was heterogenous and too small to allow us to draw reliable conclusions.

**Isolation of pulmonary veins in TOF patients**

The decisions not to perform pulmonary vein isolation in three patients with AF were made personally by the operator. It must be mentioned that those patients were treated in 2008, but the knowledge of pulmonary vein isolation efficacy in TOF patients remains unclear today. Thus, further studies are required to examine the mechanism of AF in patients with TOF.

This is a retrospective analysis, so several limitations must be taken into account. Firstly, important limitations of the present study are the relatively small number of enrolled patients and the non-homogenous nature of the group of patients. Secondly, patients who presented typical F-wave AFL on the ECG might have been more willingly referred to our centre by their physicians.

In conclusion, the ablation of macroreentrant atrial tachyarrhythmia in TOF patients is highly successful, safe, and effective. Nevertheless, AF is often observed during long-term follow-up. Thus, further studies are required to examine the mechanism of AF in patients with TOF. Regardless of the first ablated arrhythmia type, all patients in the current study required CTI-AFL ablation.

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**References**


