Original Article

The prognostic value of transesophageal echocardiography after transvenous lead extraction: landscape after battle

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Background: In patients undergoing transvenous lead extraction (TLE) transesophageal echocardiography (TEE) provide valuable information after procedure.

Methods: We analyzed data from 936 TEE performed in patients undergoing TLE between 2015 and 2019 (mean follow-up 566.23±224.47 days) and assessed the role of echocardiographic phenomena after procedure.

Results: Increment in tricuspid regurgitation (TR) was observed in 9% of patients after TLE. Factors increasing the risk of TR were: binding sites between lead and right ventricle (RV) (OR: 5.429), tricuspid valve (TV) (OR: 3.42), superior vena cava (SVC) (OR: 3.30) and lead-to-lead adhesions (OR: 2.88). Predisposing factors of residual structures after TLE were: asymptomatic masses on the leads (AMEL) (OR: 1.68), binding sites between SVC and cardiac structures (OR: 1.72), and multiple leads (OR: 1.30). Probability of vegetation remnants increased in the presence of abandoned leads (OR: 7.91). The risk factors of tamponade were: dwell time of the oldest lead (OR: 1.17), lead-to-lead adhesion (OR: 22.47), binding sites between lead and TV (OR: 6.08), RA (OR: 11.50), SVC (OR: 4.47), higher LVEF (OR: 2.35; P=0.006), female gender (OR: 5.43), multiple leads (OR: 2.11), looped leads (OR: 4.90) and AMEL (OR: 6.42). The risk of lead fracture was increased by: lead-to-lead adhesion (OR: 5.69), fibrosis binding the lead to RV (OR: 5.16), RA (OR: 2.39) and dwell time of the oldest lead (OR: 1.068). The mortality rate was 11.97% during follow-up. The risk of death was increased by: severe TR and vegetation remnants.

Conclusions: The most important phenomena evaluated after TLE are: tricuspid valve function, residual fibrosis and vegetation remnants, progression of pericardial effusion and retained lead fragments. Postoperative TEE provides information about the results of TLE and helps establish further management.

Keywords: Transvenous lead extraction (TLE); transesophageal echocardiography (TEE); lead extraction-related tricuspid valve damage; vegetation remnants; retained lead fragments; connective tissue scar

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Introduction

Transvenous lead extraction (TLE) is a first-line strategy for the treatment of complications related to cardiac implantable electronic devices (CIED). The procedure, performed in accordance with the commonly accepted standards of clinical practice is relatively safe; the rate of major complications ranges from 0.9% to 4.0%, whereas a death rate is low i.e., 0.2–0.4% (1-3). The European Heart Rhythm Association (EHRA) and the Heart Rhythm Society (HRS) advocate continuous transesophageal echocardiography (TEE) monitoring or intracardiac echocardiography (ICE) to improve procedural safety (1-3). The available evidence shows the increasing role of echocardiography during TLE. Apart from evaluating changes related to chronically indwelling endocardial leads and their impact on the course of TLE, monitoring of lead extraction and the extractor's manipulations within the heart walls to explain possible hemodynamic instability (4-9), echocardiographic assessment after the procedure ("landscape after battle") is a very important component of effective care management.

We present the following article in accordance with the MDAR reporting checklist (available at http://dx.doi.org/10.21037/cdt-20-871).

Methods

Study group

In this study, a total of 1,026 lead extractions were performed at a single high volume center from June 2015 to October 2019. Of these, 936 procedures were guided by TEE before, during and after the extraction. The indications for TLE included infectious and non-infectious complications associated with cardiac implantable electronic devices (CIED). The infectious complications were pocket infection and/or lead-related infective endocarditis (LRIE). Non-infectious indications included mechanical lead damage (electrical failure), lead dysfunction (exit/entry block, dislodgement, extracardiac pacing), perforation, the need to change the pacing mode, and the need to remove leads with immediate or potential threat if left in place.

The clinical characteristics of the patients before TLE were described in detail in previous publications (4,5).

Lead extraction procedure

All TLE procedures were performed in a hybrid operating room or an operating room using mechanical systems such as polypropylene Byrd dilators (Cook® Medical, Leechburg, PA, USA). The use of mechanical, cutting, and rotational force of the catheters and simple traction permitted complete extraction of a decided majority of leads. In case of difficulties second-line equipment was used i.e., Evolution (Cook), TightRail (Spectranetix) and lasso catheters, etc. Laser energy was not used. The organizational aspects of lead extraction procedure (venue, team) were described in detail in previous publications (4,5).

Echocardiographic study

TEE was performed using Philips iE33 or GE Vivid S 70 machines equipped with X7-2t Live 3D or 6VT-D probes with the recordings being archived. Leads were evaluated in the mid-esophageal, inferior esophageal and modified transgastric views to visualize the right heart chambers and the tricuspid valve. In order to obtain complete visualization of the anatomical structures and assessment of the course of the lead non-standard imaging planes were sometimes required. The projections and consecutive stages of echocardiographic monitoring were described in detail in previous publications (4,5).

Areas of interest

The relationship between CIED-associated clinical and procedural variables, and echocardiographic findings after TLE was analyzed. The following clinical factors were taken into account: demographics, left ventricular ejection fraction (LVEF), heart failure according to NYHA, renal failure, anticoagulation therapy and Charlson Comorbidity Index. Analysis of procedural factors included indications for TLE, lead implant duration, number and type of leads, procedure duration, TLE efficacy and periprocedural complications.

The analysis of some parameters after TLE required comparison with the phenomena assessed before and during the procedure. Echocardiographic phenomena assessed pre- and intra-procedure have already been presented by the author in previous publications (4,5).

Echocardiographic assessment after TLE included monitoring of the patient for signs and symptoms of cardiac tamponade, observation of vegetation dislodgement and visualization of their remnants in cardiac cavities, and detection of residual fibrosis after lead extraction. Fibrosis was defined as immobile masses binding the lead to the

vein or heart wall most frequently represent a sign of pre-existing asymptomatic inflammatory response triggered by the endocardial lead (foreign body reaction). The term encompassed also segmental lead-to-lead adhesion (two or three leads) moving along together with the cardiac walls.

Echocardiographic evaluation after TLE included also tricuspid valve function by comparing the degree of tricuspid regurgitation (TR) before and after TLE. Particular attention was focused on reduction in TR after TLE in patients with lead-dependent tricuspid dysfunction (LDTD).

We also searched for possible metal tips of the lead or silicon tube fragments left *in situ*.

Additionally, we evaluated the effect of all these factors on prognosis in 2-year follow-up. The exact date of death was obtained from the patient's medical records, relatives, or a national identity database.

Ethical statement

The study was conducted in accordance with the Declaration of Helsinki (as revised in 2013). The study was approved by institutional Bioethics Committee at Regional Physicians Chamber in Lublin (no. 288/2018/KB/VII). And informed consent was taken from all the patients

Statistical analysis

Despite nonparametric distribution of some of the continuous variables, for uniformity, they are presented as mean ± standard deviation and analyzed using the Wilcoxon signed rank test and the Mann-Whitney U test. The categorical variables are presented as number and percentage and compared using the Chi^2 test with Yates correction. The relationships between clinical and procedural factors, fibrous tissue changes, CIED-related variables, and the results of postoperative TEE with respect to TV function, vegetations, additional fibrous masses, retained lead fragments and fluid accumulation in the pericardial space were analyzed using univariate and multivariate regression analysis. All statistically significant (P<0.05) variables in univariate analysis were included into a multivariate model. Because of a small sample size, the incidence of tamponade was analyzed only in univariate analysis.

The impact of TEE findings on survival at 2-year follow-up was evaluated using univariate and multivariate Cox logistic regression. All statistically significant (P<0.05) variables in univariate analysis were included into a multivariate model. For selected parameters (vegetations, grade of TR after TLE: 0–II *vs.* III–IV) Kaplan-Meier survival curves were defined, and their course was evaluated with log rank test. A two-tailed P value <0.05 was considered statistically significant. The lack of statistical significance was indicated as NS (non-significant). Statistical analysis was performed with STATISTICA 13.0 (TIBCO Software Inc. Krakow, Poland).

Results

Clinical characteristics of the study group and procedure-related information (Table 1)

TEE after TLE was performed in 936 patients (including 355 women; 37.92%), with a mean age of 67.08±14.50. Patients in NYHA class III and IV were a minority (148; 15.81%). There were 230 (24.89%) patients with chronic renal failure, and 389 (41.56%) receiving chronic anticoagulation therapy. The mean Charlson Comorbidity Index was 4.886±3.76. Infection was an indication for TLE in 22.33% of patients, including 151 (16.13%) patients with LRIE, whereas 58 (6.20%) patients had pocket infection. Other indications, most common in this study population (77.67%) included lead dysfunction, the need to regain venous access and to change pacing mode, LDTD, need for MRI or radiotherapy. The number of leads in the patient before TLE was 1.83±0.63 on average, dwell time of the oldest lead in the patient 115.84±77.63 months. High voltage (HV) leads were inserted in 296 (31.62%) patients, coronary sinus (CS) lead in 153 (16.35%) patients. The mean duration of the procedure measured from dissection of the first lead to removal of the last one (sheath to sheath time) was 15.93±25.56 min on average. There were 18 (1.92%) major complications, including 12 (1.28%) cases of bleeding into the pericardial space with the signs of tamponade, 6 (0.64%) cases with severe tricuspid valve damage. There were no deaths related to TLE. Complete procedural success was achieved in 97.97%, complete clinical success in 97.86% (*Table 1*). Follow-up after TLE was 2 years, 556.20±224.50 days on average (min. 2, max, 700 days), there were 112 (11.97%) deaths.

A comparative analysis of echocardiographic phenomena occurring before and after TLE was presented in Appendix 1.

Table 1 Patient characteristics, system, and procedure information

Parameters	Values
Patient characteristics	
Number of patients, n (%)	936 (100.000)
Patient age during TLE (years), mean ± SD	67.081±14.500
Gender, Female patients, n (%)	355 (37.927)
NYHA III & IV (%), n (%)	148 (15.812)
LVEF [%], mean ± SD	47.885±15.557
LVEF normal ≥50%, n (%)	539 (57.585)
LVEF lowered <50%, n (%)	397 (42.415)
Renal failure (any), n (%)	230 (24.892)
Long-term anticoagulation, n (%)	389 (41.560)
Charlson's Comorbidity Index, mean ± SD	4.886±3.764
TLE indications	
LRIE with or without pocket infection n (%)	151 (16.132)
Local (pocket) infection (only) n (%)	58 (6.196)
Non-infectious indications n (%)	727 (77.671)
System and history of pacing	
Dwell time of the oldest lead (in months) in the patient before TLE, mean ± SD	115.843±77.633
Mean implant duration (in months) before TLE, mean ± SD	108.215±69.660
Cumulative dwell time of extracted lead (in years) before TLE, mean ± SD	17.843±14.530
Number of leads in the system before TLE, mean ± SD	1.834±0.629
Abandoned leads before TLE, n (%)	86 (9.188)
HV lead before TLE, n (%)	296 (31.624)
CS lead before TLE, n (%)	153 (16.346)
Number of procedures before lead extraction (SD)	1.837±0.990
TLE procedure efficacy and outcomes	
Procedure duration (in minutes) (sheath to sheath), mean ± SD	15.931±25.558
Technical problems during TLE (any), n (%)	231 (24.679)
TLE efficacy and complications	
Major complications (any), n (%)	18 (1.923)
Complete clinical success, n (%)	916 (97.863)
Complete procedural success, n (%)	917 (97.970)

Factors influencing functional and morphological changes occurring during TLE

Results of univariate and multivariable regression analysis *(Table 2)*

Factors influencing function of the tricuspid valve

Fibrosis binding leads to tricuspid apparatus (OR: 0.33; P<0.001), lead colliding with the tricuspid valve apparatus (OR: 0.39; P=0.04) and fibrosis binding leads to the RV wall (OR: 0.49; P=0.014) were the factors that determined valve function after TLE. The likelihood of TR worsening by ≥2 grades was significantly higher in patients with adhesive interaction between leads and endocardial surface of the RV wall (OR: 5.43; P<0.001), tricuspid valve apparatus (OR: 3.42; P=0.009), superior vena cava (SVC) (OR: 3.30; P=0.017) as well as lead-to-lead adhesions (OR: 2.88; P=0.025). TR worsening by one grade was significantly related only to binding sites between lead and RV (OR: 4.45; P=0.001). The factors that decreased the likelihood of valve damage were excessive lead loops (OR: 0.07; P=0.041) and chronic atrial fibrillation (OR: 0.22; P=0.046). Improvement of TV function was more frequently observed in women (OR: 1.66; P=0.027), in patients with noninfectious indications for TLE (OR: 2.40; P=0.008) and when the lead was adherent to the RA wall (OR: 2.08; P=0.028). Chances for TV improvement were smaller (OR: 0.85; P=0.031) in patients with low LVEF (*Table 2*).

Factors influencing the ultimate fate of vegetations

The only factor that influenced the fate of vegetations were redundant leads. In these patients, vegetation remnants were significantly more frequent (OR: 7.91; P=0.011) and consequently, the vegetations less often "disappeared" (broke into pieces and migrated to the pulmonary circulation) during TLE (OR: 0.13; P=0.012) (*Table 2*).

Factors influencing the incidence of residual fibrosis

The factors that increased the likelihood of residual fibrosis after TLE included asymptomatic masses on the leads (AMEL) (connective tissue surrounding the lead, lead thickening, thrombus attached to the lead, vegetation-like masses) (OR: 1.68; P=0.001), binding sites between leads and SVC and cardiac anatomical structures (OR: 1.72; P=0.001), and multiple extracted leads (OR: 1.30; P=0.034).

The study did not identify the factors that would decrease the likelihood of residual fibrosis after TLE (*Table 2*).

Factors influencing bleeding into the pericardial space

The most significant risk factors for acute tamponade were dwell time of the oldest lead (each year) (OR: 1.17;

Table 2 Factors influencing functional and morphological changes during TLE: results of univariate and multivariate regression analysis

Parameters	OR	95% CI	P
Factors influencing any TV function change in the course of TLE			
Lead collision with tricuspid apparatus	0.390	0.159–0.958	0.040
Fibrous tissue binding the lead to the tricuspid apparatus	0.328	0.203–0.529	0.000
Fibrous tissue binding the lead to the RV wall	0.487	0.274–0.865	0.014
Factors influencing increment in TR by 1 in the course of TLE			
Fibrous tissue binding the lead to the RV wall	4.448	2.104–7.787	0.001
Factors influencing increment in TR by ≥2 in the course of TLE			
Permanent AF	0.215	0.047–0.973	0.046
Excessive lead loops in the heart	0.067	0.005–0.903	0.041
Lead-to-lead adhesion	2.876	1.142–7.240	0.025
Fibrous tissue binding the lead to the tricuspid apparatus	3.423	1.356–8.639	0.009
Fibrous tissue binding the lead to the SVC	3.302	1.241–8.787	0.017
Fibrous tissue binding the lead to the RV wall	5.429	2.252–13.086	0.000
Factors influencing reduction in TR by any grade after TLE			
Female	1.664	1.060–2.613	0.027
Higher LVEF (by 10% p)	0.854	0.740–0.986	0.031
Non-infectious indications for TLE	2.403	1.252–4.614	0.008
Fibrous tissue binding the lead to the RA wall n (%)	2.082	1.080–4.013	0.028
Factors influencing vegetation remnants after TLE			
Abandoned leads before TLE	7.905	1.593–39.233	0.011
Factors influencing floating connective tissue scars after TLE			
Number of extracted leads	1.303	1.020–1.664	0.034
Strong connective tissue scar binding the lead to heart structures (any)	1.723	1.237–2.399	0.001
AMEL before TLE	1.676	1.265–2.222	0.001
Risk factors for cardiac tamponade * (univariate regression analysis)			
Age of patients at first CIED implantation	0.962	0.937–0.988	0.005
Female	5.432	1.482–19.905	0.011
Higher LVEF (by 10%)	2.349	1.284–4.298	0.006
Charlson's Comorbidity Index	0.790	0.630–0.991	0.041
Excessive lead loops in the heart before TLE	4.904	1.043–23.060	0.044
Lead-to-lead adhesion	22.473	6.766–74.647	0.000
Number of extracted leads	2.114	1.146–3.901	0.016
Dwell time of the oldest lead (by 1 year)	1.173	1.100–1.252	0.000
Fibrous tissue binding the lead to the SVC	4.474	1.196–16.731	0.026
Fibrous tissue binding the lead to the RA wall	11.504	3.750–35.295	0.001
Fibrous tissue binding the lead to the tricuspid apparatus	6.083	1.944–19.031	0.002
AMEL	6.417	1.412–29.168	0.016

Table 2 (*continued*)

Table 2 (*continued*)

Parameters	OR	95% CI	P
Pericardium: fluid without hemodynamic consequences			
Dwell time of the oldest lead (by 1 year)	1.087	1.017–1.162	0.014
Factors affecting lead fracture			
Lead-to-lead adhesion	5.686	2.635–12.271	0.000
Dwell time of the oldest lead (by 1 year)	1.068	1.014–1.125	0.013
Fibrous tissue binding the lead to the RA wall	2.393	1.031–5.556	0.042
Fibrous tissue binding the lead to the RV wall	5.157	2.613–10.176	0.000

P<0.001), lead-to-lead adhesion (OR: 22.47; P<0.001), binding sites between leads and TV (OR: 6.08; P=0.002), RA (OR: 11.50; P=0.001), SVC (OR: 4.47; P=0.026) and higher LVEF (for each 10%) (OR: 2.35; P=0.006). Moreover, the risk of tamponade increased in relation with female gender (OR: 5.43; P=0.011), multiple extracted leads (OR: 2.11; P=0.016), excessive lead loops (OR: 4.90; P=0.044) and AMEL (OR: 6.417; P=0.016). Older age at CIED implantation (OR: 0.96; P=0.005) and accompanying diseases expressed as higher Charlson Comorbidity Index (OR: 0.79; P=0.041) were found to decrease the probability of bleeding into the pericardial space.

The sole factor increasing the risk of asymptomatic/hemodynamically stable hemopericardium was dwell time of the oldest lead (OR: 1.09; P=0.014) (*Table 2*).

Factors influencing lead fracture during TLE

The risk factors for lead fracture included lead-to-lead adhesion (OR: 5.69; P<0.001), binding sites between leads and RV (OR: 5.16; P<0.001), RA (OR: 2.39; P=0.042) and dwell time of the oldest lead (OR: 1.07; P=0.013). The study did not identify the factors that would decrease the risk for lead fracture (*Table 2*).

Effect of postoperative functional and morphological findings within the heart on 2-year survival (Table 3)

During the mean follow-up period of 566.229±224.468 (2 to 730) days 112 deaths were observed in the study population.

The effect of TEE variables on survival at 2-year follow-up was evaluated using univariate and multivariate Cox regression, of which only the presence of significant TR and vegetation remnants were of prognostic value. Mortality risk was a 45.6% (HR 1.46; 95% CI: 1.23–1.73; P=0.001) higher for each grade of regurgitation. The presence of vegetation remnants after TLE was associated with a 75.9% (HR 1.76; 95% CI: 1.35–2.30; P=0.001) higher risk of death at 2-year follow-up (*Table 3*).

Discussion

TEE is a very useful tool in patients undergoing transvenous leads extraction. TEE can detect adverse consequences of procedure and helps in making postoperative clinical decisions (5-9). The present study describes "landscape after battle" with TEE results being grouped into the following five areas: tricuspid valve function, residual fibrosis, the ultimate fate of vegetations, accumulation of fluid in the pericardial space and lead remnants inadvertently left in the heart.

The assessment of TV function before and after TLE is very important as the available evidence shows that severe tricuspid regurgitation is associated with a poor prognosis (10-12). Additionally, damage to the tricuspid apparatus can occur during extraction of the RV lead, most frequently due to adhesion of chronically indwelling leads to cardiac anatomical structures (including TV) (13-19) (*Figure 1*).

Damage to the tricuspid valve during extraction is estimated to range from 3.5% to 15%, and even to 19% (1,13-18). In this study clinically insignificant valve dysfunction was detected in about 9% of cases, whereas significant TV damage that caused TR worsening by 2 or 3 grades as compared to baseline (before TLE) occurred in 3.5% of patients, which is less than previously reported (1,13-18). The need for surgical intervention in such cases is rare (17). Initially, 1.4% of patients were evaluated as potentially requiring surgical repair, finally valvuloplasty was performed in 8 (0.9%) patients. The relatively good results in this study may be explained by a vast practical experience and continuous TEE monitoring helping warn the operator about potentially harmful situations leading to TV damage (5). The

Table 3 The impact of functional (grade of tricuspid regurgitation or its change after TLE) and morphological changes, diagnosed by TEE during TLE on survival after TLE at 2-year follow-up: results of univariate and multivariate Cox regression analysis

Parameters	Univariate Cox regression analysis			Multivariate Cox regression analysis		
	HR	95% CI	P	HR	95% CI	P
Tricuspid regurgitation (after TLE) (yes/no)	1.500	1.270–1.772	0.001	1.456	1.227–1.727	0.001
Tricuspid regurgitation without change after TLE (yes/no)	0.956	0.604–1.512	0.847			
Tricuspid regurgitation change after TLE (+1) degree	0.863	0.379–1.965	0.726			
Tricuspid regurgitation change after TLE (+ 2 or 3) grade	1.097	0.404–2.976	0.856			
Tricuspid regurgitation change after TLE (–1 or 2 or 3) grade	1.133	0.635–2.020	0.672			
Floating connective tissue scars (yes/no)	0.776	0.515–1.169	0.225			
Appearance of fluid during TLE causing cardiac tamponade	0.691	0.097–4.950	0.713			
Appearance of fluid during TLE without cardiac tamponade (yes/no)	0.968	0.239–3.921	0.964			
Presence of vegetations after TLE (yes/no)	1.963	1.523–2.529	0.001	1.759	1.348–2.295	0.001
Lead fracture during extraction (yes/no)	1.776	0.927–3.402	0.083			

TEE, transesophageal echocardiography; TLE, transvenous lead extraction.

available evidence shows the following factors predisposing to TV damage: longer implant duration, pacemaker leads, patient age ≥75 years, removal of ≥2 leads (13) as well as female gender, use of laser catheters, the need for additional tools during TLE, vegetations attached to TV and/or leads (15,18-20). This study demonstrated that the probability of TV damage was related mainly to adhesion of leads to tricuspid valve. Binding sites between leads and RV endocardium and lead-to-lead adhesions may play an indirect role through the impact on the incompletely controlled traction during lead extraction. It may be concluded that a long lead dwell time creates conditions for the development of connective tissue and adhesion of leads to tricuspid leaflets or tendinous chords. In consequence, the technically difficult procedure of lead removal requiring the use of additional, more aggressive tools and stronger traction during the extraction increases the risk of damage. It is interesting to note that in this study chronic atrial fibrillation was associated with a lower incidence of significant TV damage. A possible explanation might be dilatation of the TV annulus due to enlargement of right heart chambers in which lead mobility is preserved, thus reducing chances for adhesion. In turn, the effect of excessive lead loops on tricuspid valve function depended on their location. Our previous study demonstrated that excessive lead loops in the TV orifice were found only in 3.7%, in the RV in 2.99%, but most often in the RA i.e., in 14.7% of cases, which usually did not affect tricuspid function (5). In this study, generally there was no significant difference in the incidence of tricuspid regurgitation before and after the procedure. Lead-unrelated TR was found in almost 42% of patients (significant TR in over 23%), and the rates were comparable before and after TLE. At 2-year follow-up TR was found to be a significant factor worsening prognosis and causing a 45.6% increase in the risk of death for each grade of TV regurgitation (HR 1.456, 95% CI: 1.227–1.727, P=0.001) (*Figure 2*).

Another important issue addressed in the present study is tricuspid regurgitation induced by a right ventricular lead. LDTD is a well-known complication of CIED implantation occurring at a rate of 25% to 35% (21-26), not infrequently being a direct indication for TLE (25-27). LDTD contributes to RA and RV enlargement and increases pulmonary arterial pressure, and what is important, it is associated with higher mortality in patients having CIED. Significant TR associated with the presence

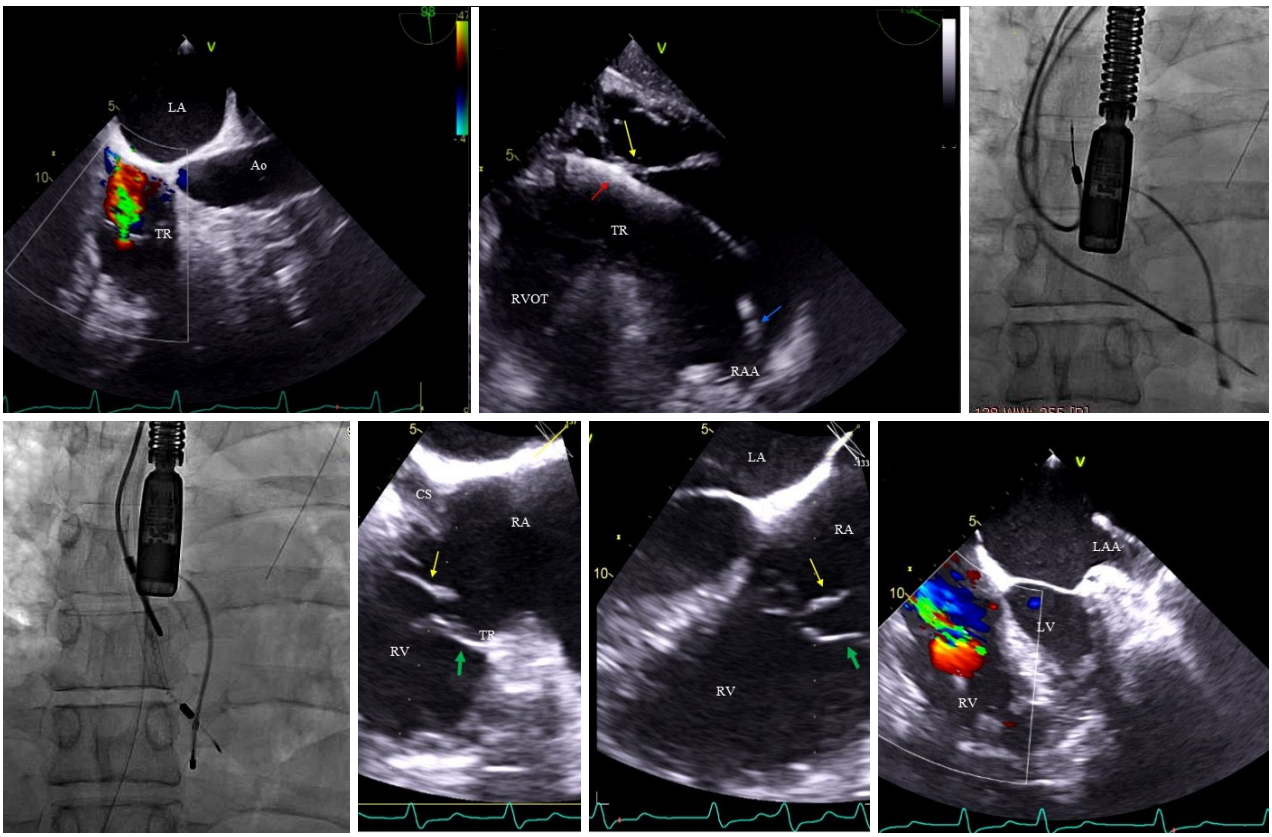

Figure 1 Damage to the tricuspid valve during TLE demonstrable by TEE. Red arrow: adhesion of the lead to the posterior leaflet (yellow arrow); green arrow: anterior leaflet; blue arrow: lead in the right atrium.

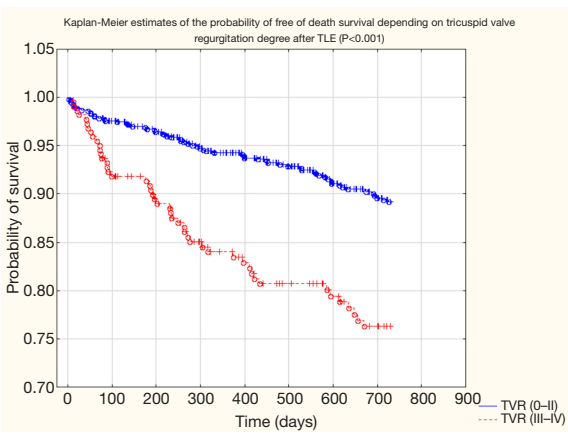

Figure 2 Kaplan-Meier estimates of the probability of free of death survival depending on the grade of tricuspid valve regurgitation after TLE. P<0.001.

of right ventricular leads worsens long-term prognosis and is an independent risk factor for premature death (22-24). On the other hand, timely removal of the lead inducing valve dysfunction may result in valve improvement (*Figure 3*).

Our understanding of improvement in TR after lead removal is limited to a few reports of other investigators (28,29) and our previous research (27). In this study tricuspid function improved in 100 patients, including significant improvement in only 10 cases, whereas severe TR was found in 96.66% of patients with LDTD. A possible cause of low rate of improvement is too late referral for TLE in the situation when RA enlargement, TV annulus dilatation and RV remodeling in severe regurgitation (28) are too large.

Another important TEE finding that deserves attention after TLE is dislodgement of vegetations and residual fibrosis. Previous research in preoperative TTE, TEE and ICE has found movable masses attached to the leads, representing vegetations or connective tissue both in asymptomatic and symptomatic patients (30-34). Some of them may remain in cardiac cavities after TLE whereas others may emerge during lead dissection. In this study they were detected in a total of 360 (38.461% of all) patients,

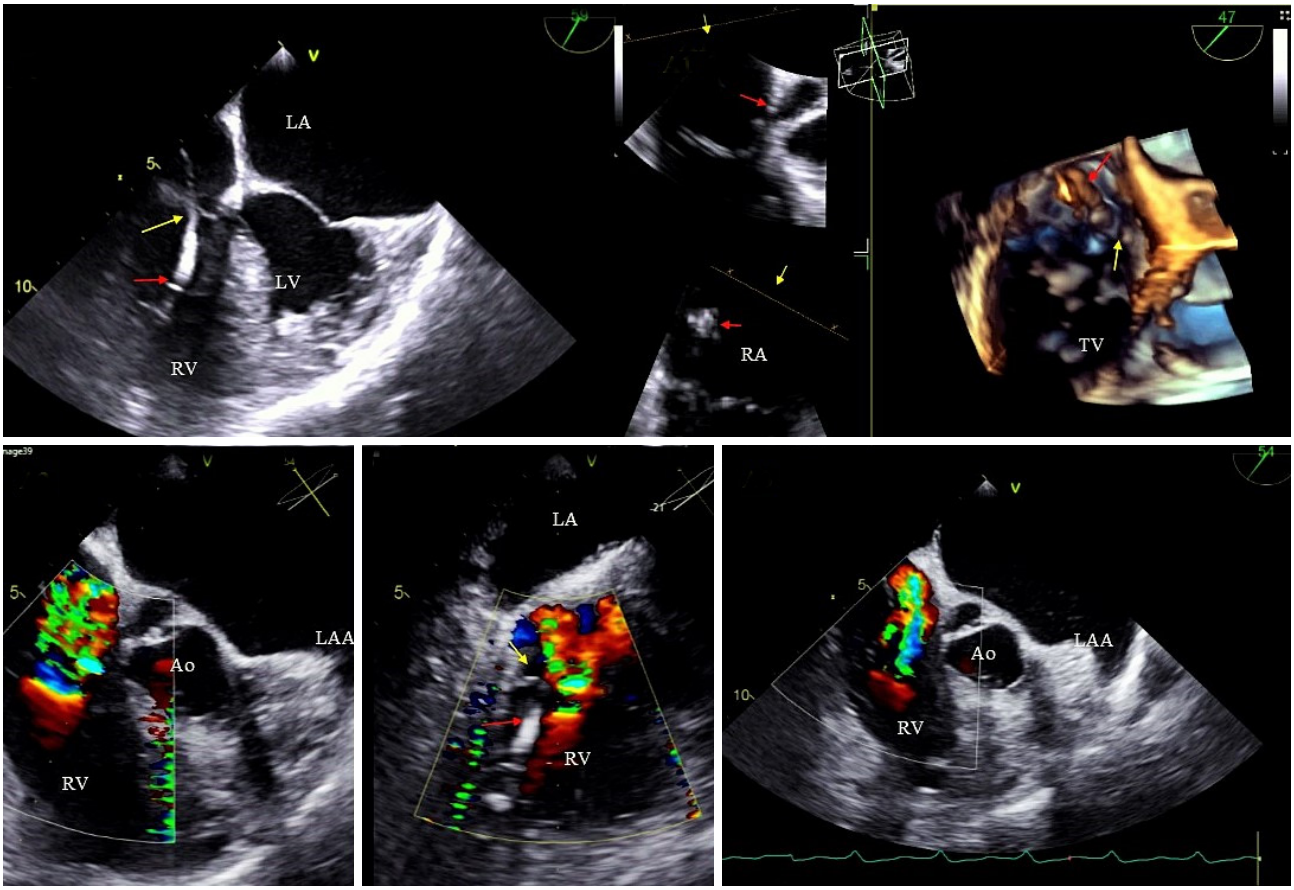

Figure 3 Improvement in TR after removal of the lead impinging on the leaflet. The arrows show the course of the lead colliding with the tricuspid valve leaflet.

including residual fibrosis in 310 patients (33.119% of all) and vegetation remnants in 50 (5.341% of all) patients. These additional masses detected after TLE have been referred to as ghosts (1,35-37), however the definition is not precise, as the term encompasses fragments both of connective tissue and vegetations. This phenomenon was first described in the patient undergoing TLE under ICE guidance because of local device infection in whom connective tissue masses were detected in the RA/SVC immediately after the procedure. What is important, TEE at 3 months after anticoagulation and antibiotic therapy revealed that the masses remained unchanged and asymptomatic (38). The term "ghost" was introduced by Le Dolley *et al.* and defined as a new tubular mobile mass detected by echocardiography on the path of the lead immediately after its removal (36). The masses were found in 8% of patients undergoing TLE for infectious indications. By contrast, in the present study "ghosts" were demonstrated in patients with and without infections. A possible explanation may be that the available evidence has been obtained from TEE examinations in patients undergoing TLE due to infection. TEE in patients with noninfectious indications for lead extraction is performed only if TTE result is inconclusive, i.e., in rare cases.

Up to now, a number of studies have shown that "ghosts" have an incidence ranging from 14% to 19% (35,39). In this study, additional masses after TLE were found within the RA (19.44%) and SVC (9.94%). In these locations the flow of blood slows down, which is one of the conditions of the deposition of connective tissue on the leads, and promotes thrombosis due to endothelial dysregulation. Thrombi formed soon after lead implantation may undergo lysis or reorganization, causing the collagenous capsule to grow around the leads (40-42). Preoperatively, accretions (mobile connective tissue masses) were detected in 160 (17.09%) cases, whereas after lead extraction there was a statistically significant increase in the incidence of intracardiac lead masses up to 33.12% (P<0.001). This

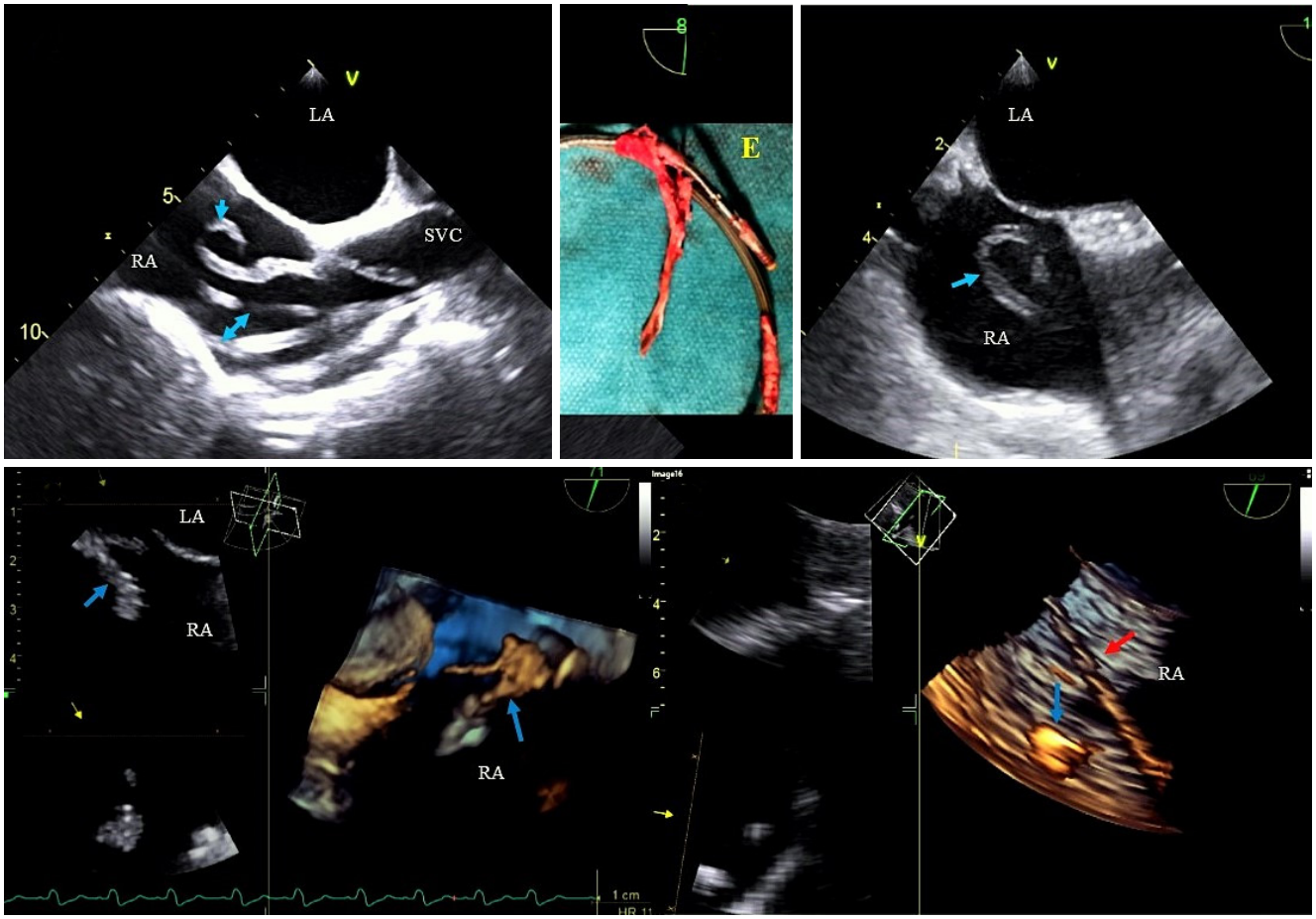

Figure 4 Residual fibrosis demonstrated in postoperative TEE (blue arrows). Red arrow: lead during extraction.

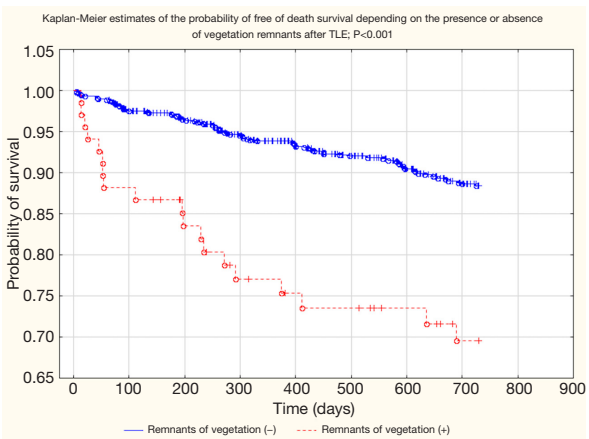

Figure 5 Kaplan-Meier estimates of the probability of free of death survival depending on the presence or absence of vegetation remnants after TLE. P<0.001.

result may be explained by partial dissection of leads from connective tissue which is also found in SVC (limitations of TEE) and the presence of so-called lead thickening and hyperechogenicity representing foci of immobile encapsulation sheaths (31) (*Figure 4*).

The factors that significantly increased the likelihood of residual fibrosis after TLE included multiple leads, binding sites between leads and cardiac anatomical structures and presence of asymptomatic masses on endocardial leads (AMEL) before TLE. In contrast to earlier findings (39), however, no evidence was found that residual fibrosis was significantly more frequently associated with ICD than pacemaker leads.

Clinical relevance of "ghosts" (necessity to differentiate from vegetation remnants) and their impact on long-term prognosis merits consideration. In this study "ghosts"

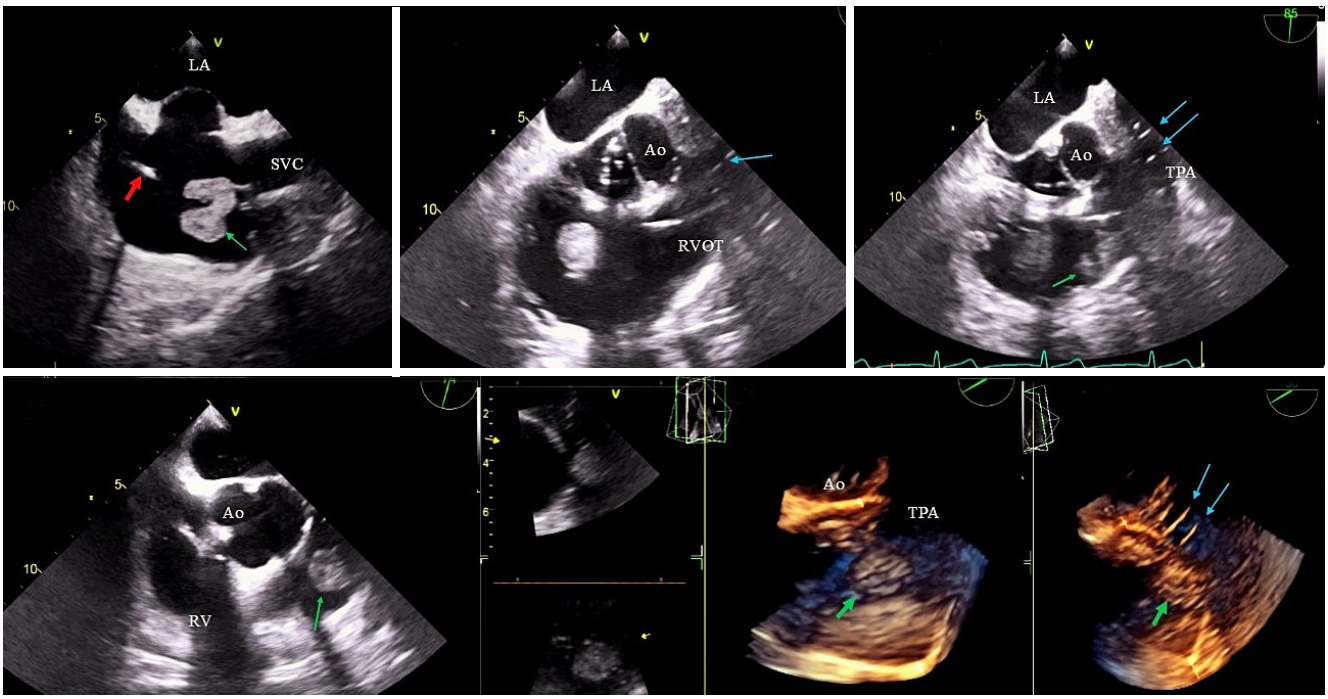

Figure 6 Embolic protection during TLE in patients at high risk of large vegetation dislodgement. Green arrows: large vegetation; red arrow: lead; blue arrows: basket in pulmonary trunk. TLE, transvenous lead extraction.

found in noninfectious patients did not affect long-term survival, whereas persistent vegetations or their fragments significantly worsened 2-year prognosis (HR 1.76, 95% CI: 1.35–2.30; P=0.001) (*Figure 5*).

Data from several studies suggest that vegetation remnants after TLE are associated with poor long-term survival (35,43). In this study 58% of vegetations disappeared but 42% of them remained within the heart after the procedure. In most cases the freed pieces of vegetations dislodged into the pulmonary circulation without producing the signs of pulmonary embolism. In patients at high risk of embolization (large vegetation size) embolic protection systems were used during TLE (*Figure 6*).

One interesting finding in this study—reported for the first time—was that a remnant was bigger than the initial vegetation (P<0.001). A possible explanation for this might be that some of the vegetations on the intravenous lead route were not visualized in TEE and were displaced with a dissecting catheter towards the SVC orifice; additionally, infection that increases connective tissue buildup on the leads results in the formation of a conglomerate composed of vegetations and fibrous tissue peeled off during lead dissection (*Figure 7*).

From a clinical perspective it was very important to confirm the risk associated with lead abandonment detected in our previous study (44). The abandoned nonfunctional leads increased the risk of infective endocarditis (44,45) and caused as high as 8-fold increase of risk of vegetation remnants after lead extraction, thus worsening prognosis and reducing chances of long-term survival.

Another very important finding in TEE after TLE is accumulation of fluid in the pericardial space. Hemorrhage to the pericardium in the course of heart wall injury during TLE is the most severe complication of the procedure occurring at a rate of 2% to 4% (1-3). TEE is a tool that precisely and immediately visualizes small amounts of fluid, changes in fluid volume and signs of tamponade that may lead to hemodynamic instability. Furthermore, this examination facilitates rapid therapeutic decision making (diagnosis of blood clotting excluding pericardiocentesis) (4-9). Risk factors for heart and wall injury during TLE are directly related to increased connective tissue proliferation. Most dangerous is fibrosis binding leads to a thin-walled RA, which is at greatest risk of injury. Other important factors are: longer lead dwell time, lead-to-lead adhesion, AMEL and higher LVEF. The higher LVEF characterizes healthier patients with potentially longer pacing time and simultaneously longer lead implant duration, which also

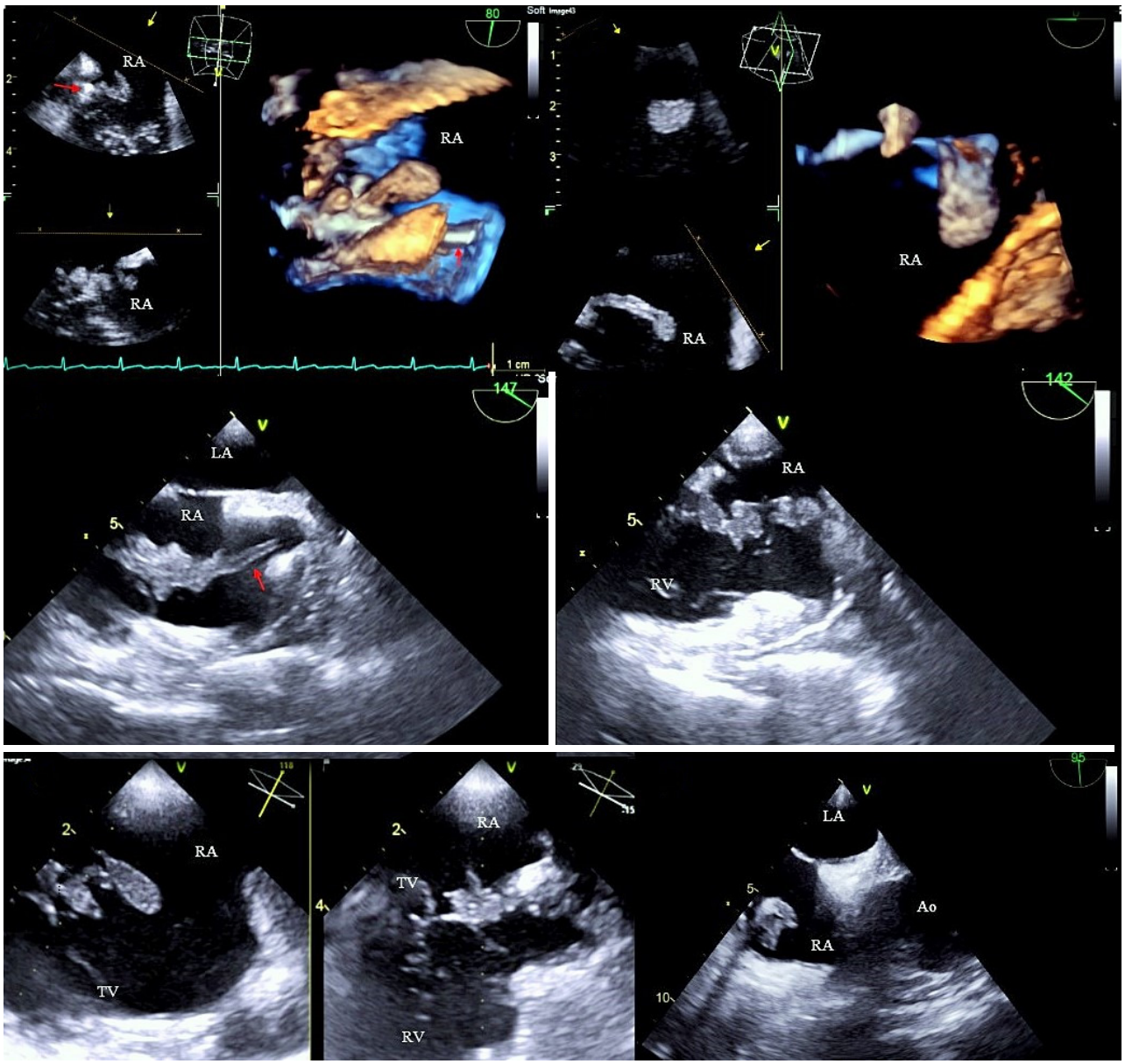

Figure 7 Vegetation remnants in postoperative TEE. Red arrows show lead before extraction. TEE, transesophageal echocardiography.

predisposes to progressive development of connective tissue. On the other hand advanced age and accompanying diseases may be protective factors in tamponade. This result is likely to be related to a shorter lead dwell time and weaker connective tissue reaction to chronically implanted leads (*Figure 8*).

Echocardiographic assessment after TLE includes confirmation of procedure efficacy. In this study complete procedural success meaning complete removal of the leads from cardiac cavities was achieved in 98% of patients. The complete lead removal is especially important in case of infectious indications for TLE, as retained lead fragments cause a persistent infection, thus increasing the risk of death (1-3). Fragments of broken leads or silicon tubes are relatively rarely left in the heart i.e., from 2% to 3% (46,47) (*Figure 9*).

In this study, 5% of leads were broken during the extraction procedure, but lead fragments were non-removable only in 0.6% of cases (TEE confirmed no possibility of capturing the proximal lead tip embedded

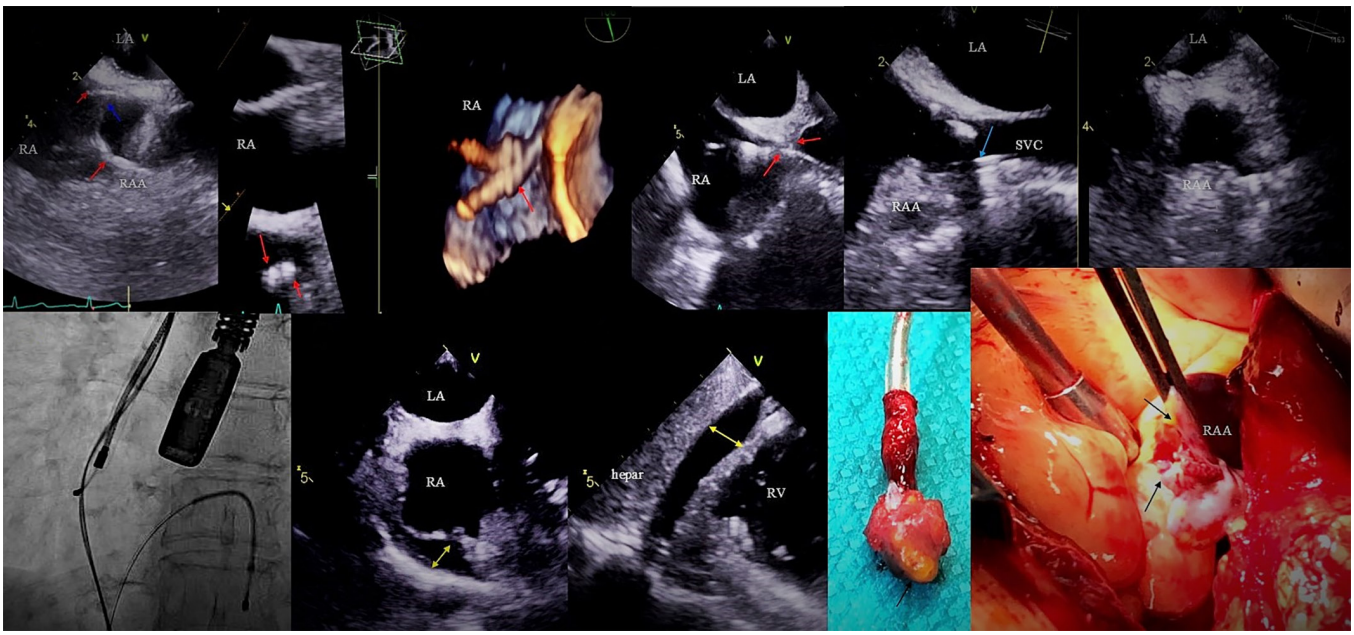

Figure 8 Extraction of 26-year-old DDD leads in a 58-year-old woman complicated with pericardial tamponade as a result of RAA perforation. Red arrows: leads; blue arrow: lead-to-lead adhesion; yellow arrow: fluid (blood) in pericardium.

in the endocardium). The strongest risk factors for lead fracture were lead-to-lead adhesions and binding sites between leads and RV (P<0.000) and RA (P=0.042) as well as age of extracted leads (P=0.013). Several investigators have emphasized the contribution of connective tissue to increased risk of procedure difficulty and complications but so far no clear relationships have been identified (48,49). The remnants of silicon tube surrounding the lead (when the lead is broken) was found incidentally i.e., in 0.7% of cases. The tube, invisible in radiological examination is detectable in TEE and for this reason it is possible to remove it and achieve complete procedural success (50).

Limitations

This is a single center observational study based on experience. The technique of TLE did not include laser energy.

Conclusions

Postoperative TEE provides additional information which is invaluable for the management of patients after TLE. Postoperative TEE results can be interpreted correctly only if compared with preoperative and intraoperative findings. The most important variables evaluated after TLE is tricuspid valve function, residual fibrosis and vegetation remnants, progression of pericardial effusion and retained lead fragments. Increment in TR after TLE occurs in 9.0% of patients, however severe TR requiring surgical repair is found only in 0.64% of patients. The likelihood of increment in TR is higher in the presence of binding sites between leads and RV, tricuspid apparatus and SVC as well as lead-to-lead adhesions before TLE. After TLE new strips of connective tissue appear along the course of the extracted lead. The predisposing factors are encapsulation sheaths before TLE and multiple extracted leads. Vegetation remnants are a frequent finding after TLE in patients with LRIE, their average size is larger as compared with initial measurements and have a negative prognostic value. Pericardial effusion occurs during or immediately after the procedure in several percent of cases, and progresses to acute cardiac tamponade in 1.3%. The risk factors for acute tamponade are associated with massive proliferation of connective tissue. Lead fracture occurs during 5.3% of extraction procedures, but lead fragments cannot be removed only in 0.6%. In 0.7% silicon tube fragments are left behind but they can be removed with a lasso catheter under TEE guidance. The risk factors for lead fracture are associated with preexisting binding sites and lead-to-lead-adhesions.

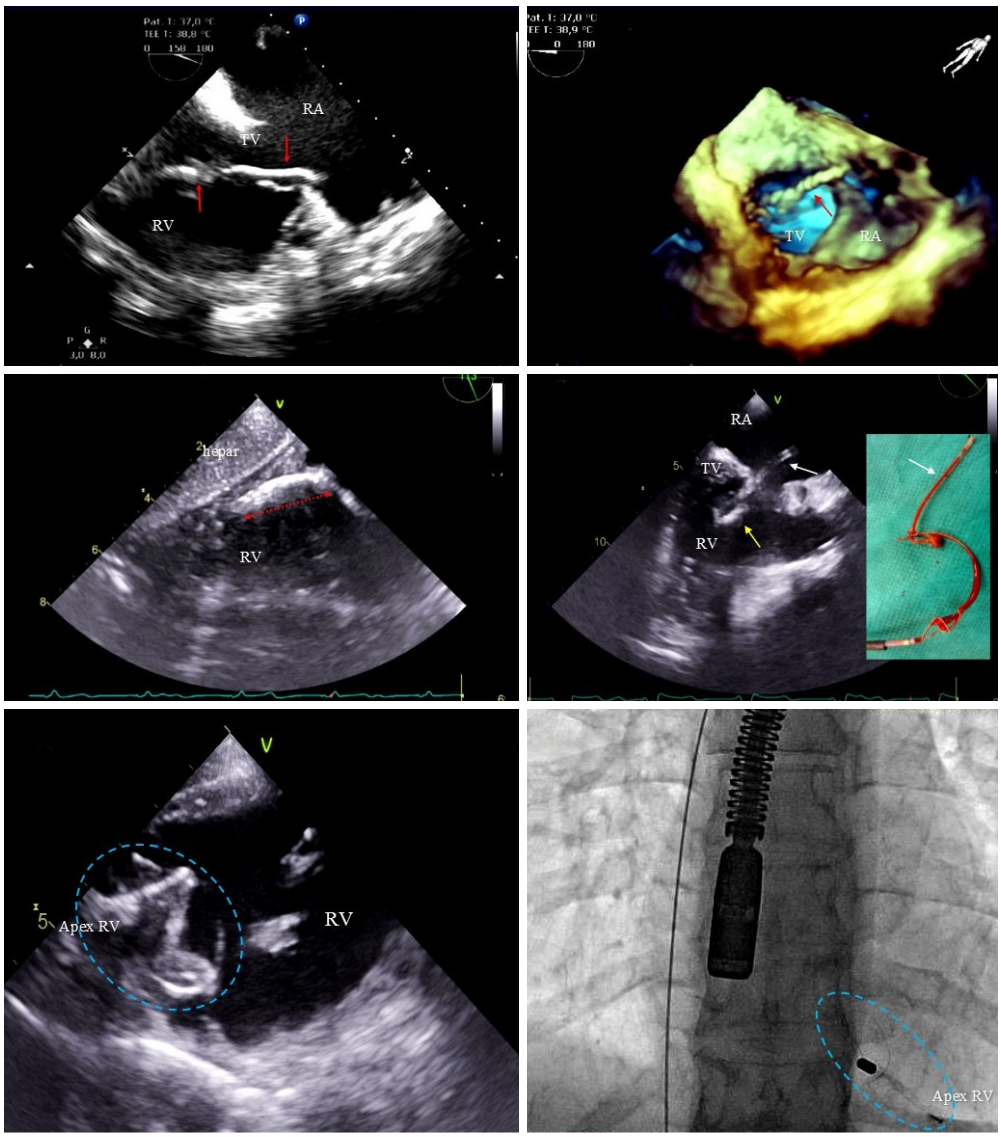

Figure 9 Fragments of leads broken during TLE and demonstrated in TEE (arrows and circles). TLE, transvenous lead extraction; TEE, transesophageal echocardiography.

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Footnote

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Ethical Statement: The authors are accountable for all aspects of the work in ensuring that questions related to the accuracy or integrity of any part of the work are appropriately investigated and resolved. The study was conducted in accordance with the Declaration of Helsinki (as revised in 2013). The study was approved by institutional

Bioethics Committee at Regional Physicians Chamber in Lublin (No. 288/2018/KB/VII). and informed consent was taken from all the patients

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Postprocedural echocardiographic data—comparison with preprocedural findings (*Table S1*)

Tricuspid valve function

Significant/severe TR was detected in 220 (23.504%) patients before TLE, and in 222 (23.718%) patients (P=0.957) after the procedure. Similarly, there were no differences in mild and moderate TR before and after the procedure. Severe LDTD was confirmed in 58 patients.

After TLE the severity of regurgitation remained unaltered in 80.235% of patients. Exacerbation of TV dysfunction was observed in 85 (9.081%) patients, in most cases (n=52) it was a mild increment in TR. TV function improved after TLE in 100 patients (P=0.278 as compared to increased TR by the same grade). After the procedure increment in TR by one grade was seen in 52 patients, and improvement by one grade in 90 patients (P<0.001). Increment in TR by 2 or 3 grades occurred in 33 patients, improvement by 2 or 3 grades in 10 patients (P<0.001). Severe tricuspid valve damage was detected in 6 (0.641%) patients after TLE. TEE revealed that tricuspid valve damage in 12 (1.389%) individuals may require surgical repair, 8 patients were selected for tricuspid valvuloplasty, including one urgent (during the same procedure). Rupture of the tendinous chords was found during 29 (3.098%) TLE procedures (*Table 2*).

Vegetations

Out of 151 (16.132%) patients with LRIE detected in preprocedural TEE 119 (78.80% of LRIE) were diagnosed with having vegetations. The average size of the vegetation was 1.533±1.137 cm. Lead-associated vegetations were most common being detected in 114 (75.496% of LRIE) patients. Multiple vegetations were seen in more than half of LRIE patients (82; 54.304%), in most cases they were below 2 cm in size (62.913%). There were 10 (6.622%) large (2.1–3.0 cm in size) and 14 (9.271%) very large (>3 cm in size) vegetations. In the latter case special devices were used during TLE for prevention of pulmonary embolism. In case of 3 vegetations >4 cm in size it was decided to carry out surgical treatment (hybrid). After lead extraction vegetation remnants were seen in cardiac cavities in 50 (33.112% of LRIE) patients, the average size of remnants was 2.520±1.703 cm and was larger than at baseline (P<0.001). A total of 69 (45.695% of LRIE) patients had no vegetation remnants after lead removal (*Table 2*).

Residual fibrosis

Overall, 549 (there were multiple manifestations in single patients) fibrous encapsulation sheaths were detected in 437 (46.688%) patients before TLE. Adhesive interactions between leads and various anatomical structures were diagnosed in 236 (25.213%) patients, binding to right atrial (RA) wall in 65 (6.944%), to superior vena cava (SVC) in 56 (5.983%), and lead-to-lead adhesions in 172 (18.376%) patients (*Table 2*).

After TLE residual fibrosis was found in 310 patients, being almost twice as frequent as before TLE. Floating connecting tissue scar was detected as a single focus in 219 (23.39%) patients, multiple foci in 91 (9.722%). Their average length and width were 21.015±15.446 and 4.411±1.590 mm, respectively. They were most often found in RA (182; 19.444%) and SVC (93; 9.935%), less often in RV (61; 6.517%), on the tricuspid apparatus (46; 4.914%), incidentally in CS. Floating connecting tissue scar was found at more than one site in 8.653% of patients (*Table 2*).

Pericardial effusion

Preoperatively, pericardial effusion was found in 54 (5.770%) patients, including 40 (4.274%) with "wet" perforation, mainly of the RV wall. In 151 (16.132%) patients perforation was "dry" i.e., the tip of the lead was beyond the RV wall contour without signs of fluid in the pericardium, sometimes with a pericardial reaction and a drop of fluid near the lead tip. A total of 34 (3.632%) patients were monitored for the presence of effusion, including 12 (1.282%) with signs of tamponade during or after TLE (*Table 2*).

Retained lead fragments

The lead was broken during 50 (5.342%) extraction procedures, in 6 (0.641%) cases the lead fragment could not be removed, in the remaining 44 (4.701%) procedures pieces of the lead were successfully extracted using additional tools (lassos and sheaths). In 7 (0.0749%) cases there were fragments of silicon tube demonstrable only in TEE (invisible under fluoroscopy), which in 5 patients were removed transvenously, in the remaining two patients during operations for other reasons (*Table 2*).

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Table S1 TEE findings before and after TLE

TEE findings	*Before TLE	*After TLE	Wilcoxon paired test, χ^2 test
TR (0–IV), mean ± SD	*1.689±1.009	*1.708±0.991	0.959
TR absent/ mild (0–I), n (%)	*543 (58.013)	*541 (57.799)	0.963
TR moderate (II), n (%)/LDTD, n	*173 (18.483)/2	*173 (18.438)	1.000
TR significant/severe (III–IV), n (%)/LDTD, n (%)	*220 (23.504)/58	*222 (23.718)	0.957
Tricuspid regurgitation change after TLE			
TR without change after TLE, n (%)		751 (80.235)	
TR change after TLE (+1) *vs.* (–1), n (%)	52 (5.556)	90 (9.615)	0.001
TR change after TLE (+2 or 3) *vs.* (–2 or 3), n (%)	33 (3.526)	10 (1.068)	0.001
TR change (all) (+) *vs.* (–), n (%)	85 (9.081)	100 (10.684)	0.278
TLE-associated damage to tricuspid valve apparatus			
Increment in TR after TLE by 2 degrees, n (%)	NA	33 (3.526)	
Increment in TR after TLE by 2 degrees to IV degree, n (%)	NA	6 (0.640)	
Damage to tendinous chords, n (%)	NA	29 (3.098)	
Flail tricuspid leaflet initially requiring intervention after TLE, n (%)	NA	12 (1.389)	
Vegetations			
Presence of vegetations (TTE or and TEE), n (%)	*119 (12.714)	*50 (5.342)	P<0.001
Max. diameter of vegetation (if present) mean ± SD	*1.533±1.137	*2.520±1.703	P<0.001
Presence of vegetations <2 cm, n (%)	*95 (10.150)	*37 (3.953)	0.261
Presence of vegetations >2 cm, n (%)	*24 (2.564)	*13 (1.389)	0.261
Single vegetation, n (%)	*37 (3.953)	*45 (4.808)	P<0.001
Multiple vegetations, n (%)	*82 (8.761)	*5 (0.534)	P<0.001
Vegetations associated with leads, n (%)	*114 (12.179)	NA	
Vegetations not associated with leads, n (%)	*3 (0.321)	*4 (0.427)	1.000
Vegetations associated with leads and heart structures, n (%)	*2 (0.214)	NA	
Tissue scars—AMEL, 549 (58.654%) in 437 patients			
Lead thickening, n (%)	277(29.594)	NA	
Clot on the lead, n (%)	75 (8.013)	NA	
Vegetation-like masses, n (%)	37 (3.953)	NA	
Fibrous tissue encasing the lead/floating connecting tissue scars, n (%)	160 (17.094)	310 (33.120)	P<0.001
Single floating connective tissue scars after TLE, n (%)	NA	219 (23.397)	
Multiple floating connective tissue scars after TLE, n (%)	NA	91 (9.722)	
Average length of floating connective tissue scars after TLE, mean ± SD	NA	21.015±15.446	
Average width of floating connective tissue scars after TLE, mean ± SD	NA	4.411±1.590	
Fibrous tissue binding the lead to the superior vena cava and heart structures			
Fibrous tissue binding the lead to the SVC, n (%)	56 (5.983)	NA	
Fibrous tissue binding the lead to the RA wall, n (%)	65 (6.944)	NA	
Fibrous tissue binding the lead to the heart structures (all), n (%)	90 (9.615)	NA	
Fibrous tissue binding the lead to the RV wall, n (%)	106 (11.325)	NA	
Lead–to–lead adhesion	172 (18.377)	NA	
Pericardial fluid after or during TLE			
Appearance of fluid during TLE causing cardiac tamponade (complication), n (%)	NA	12 (1.282)	
Appearance of fluid during TLE without hemodynamic disturbances (clinically asymptomatic), n (%)	NA	22 (2.350)	
Pericardial fluid surrounding the tip as a symptom of “wet” cardiac wall perforation during TLE, n (%)	40 (4.274)	NA	
Asymptomatic pericardial fluid before and after TLE not caused by TLE or lead perforation, n (%)	14 (1.496)	NA	
Lead fracture			
Lead fracture during extraction	NA	50 (5.342)	
Broken lead insulation with successful extraction	NA	7 (0.749)	
Broken lead with unsuccessful extraction of metal remnant	NA	6 (0.641)	

AMEL, asymptomatic masses on endocardial leads; LDTD, lead-dependent tricuspid dysfunction; RA, right atrium; RV, right ventricle; SVC, superior vena cava; TEE, transesophageal echocardiography; TLE, transvenous lead extraction; TR, tricuspid regurgitation; TTE, transthoracic echocardiography.

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