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Lead dependent tricuspid dysfunction: Analysis of the mechanism and management in patients referred for transvenous lead extraction

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Abstract

Background: *Lead-dependent tricuspid dysfunction (LDTD) is one of important complications in patients with cardiac implantable electronic devices. However, this phenomenon is probably underestimated because of an improper interpretation of its clinical symptoms. The aim of this study was to identify LDTD mechanisms and management in patients referred for transvenous lead extraction (TLE) due to lead-dependent complications.*

Methods: *Data of 940 patients undergoing TLE in a single center from 2009 to 2011 were assessed and 24 patients with LDTD were identified. The general indications for TLE, pacing system types and lead dwell time in both study groups were comparatively analyzed. The radiological and clinical efficacy of TLE procedure was also assessed in both groups with precision estimation of clinical status patients with LDTD (before and after TLE). Additionally, mechanisms, concomitant lead-dependent complications and degree (severity) of LDTD before and after the procedure were evaluated. Telephone follow-up of LDTD patients was performed at the mean time 1.5 years after TLE/replacement procedure.*

Results: *The main indications for TLE in both groups were similar (apart from isolated LDTD in 45.83% patients from group I). Patients with LDTD had more complex pacing systems with more leads (2.04 in the LDTD group vs. 1.69 in the control group; $p = 0.04$). There were more unnecessary loops of lead in LDTD patients than in the control group (41.7% vs. 5.24%; $p = 0.001$). There were no significant differences in average time from implantation to extraction and the number of preceding procedures. Significant tricuspid regurgitation (TR-grade III–IV) was found in 96% of LDTD patients, whereas stenosis with regurgitation in 4%. The 10% frequency of severe TR (not lead dependent) in the control group patients was observed. The main mechanism of LDTD was abnormal leaflet coaptation caused by: loop of the lead (42%), septal leaflet pulled toward the interventricular septum (37%) or too intensive lead impingement of the leaflets (21%). LDTD patients were treated with TLE and reimplantation of the lead to the right ventricle (87.5%) or to the cardiac vein (4.2%), or surgery procedure*

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with epicardial lead placement following ineffective TLE (8.3%). The radiological and clinical efficacy of TLE procedure was very high and comparable between the groups I and II (91.7% vs. 94.2%; p = 0.6 and 100% vs. 98.4%; p = 0.46, respectively). Repeated echocardiography showed reduced severity of tricuspid valve dysfunction in 62.5% of LDTD patients. The follow--up interview confirmed clinical improvement in 75% of patients (further improvement after cardiosurgery in 2 patients was observed).

Conclusions: *LDTD is a diagnostic and therapeutic challenge. The main reason for LDTD was abnormal leaflet coaptation caused by lead loop presence, or propping, or impingement the leaflets by the lead. Probably, TLE with lead reimplantation is a safe and effective option in LDTD management. An alternative option is TLE with omitted tricuspid valve reimplantation. Cardiac surgery with epicardial lead placement should be reserved for patients with ineffective previous procedures.* (Cardiol J 2013; 20, 4: 402–410)

Key words: tricuspid dysfunction, electronic devices

Introduction

Tricuspid valve (TV) dysfunction is one of more important complications in patients with cardiac implantable electronic devices due to the endocardial lead. The frequency of this complication is underestimated and its impact on the clinical symptoms is attributed to other illnesses. However, further increase in complications related to lead crossing the TV is expected with the increasing number of implanted pacemakers (PM), cardioverter-defibrillators (ICD) and cardiac resynchronization therapy devices [1]. The most important problem is a lack of criteria for identifying lead-dependent TV dysfunction. Available evidence shows diagnostic difficulties related to valve visualization using standard 2D echocardiography [2]. The hemodynamic significance of TV disorders, especially tricuspid regurgitation (TR) is difficult to assess due to high prevalence of this abnormality in general population, mainly in the elderly, as well as the need for proper evaluation of TR mechanism by echocardiography. Another important aspect is the management of patients with known severe TV dysfunction as a complication of permanent pacing, especially in the presence of other abnormalities.

The purpose of the present study was to analyze retrospectively the frequency of significant (grade III/IV) TR or stenosis (taking into account the pathomechanisms and predisposing factors) due to the presence of endocardial leads in the population of patients referred for lead removal for various reasons, as well as to analyze management and outcomes in these patients.

Methods

We analyzed data from 940 patients undergoing transvenous lead extraction (TLE) for various reasons in the Reference Center from 2009 to 2011. Based on medical data the patients were divided into two groups: group I consisting of 24 (2.6%) subjects with lead-dependent tricuspid dysfunction (LDTD) and group II consisting of the remaining 916 patients serving as a control group. The comparative analysis of indications for TLE in the study group was conducted with precision assessment of primary reasons to refer for TLE in LDTD population.

Patients were assigned to group I if they had LDTD documented by transthoracic echocardiography and/or transesophageal echocardiography. Echocardiograms were obtained by an experienced echocardiographer before and after TLE using an IE 33 Philips device. Patients with TR but not evidently connected to lead presence were not included in this group of patients. Echocardiography was repeated in all patients in 3–5 days after the procedure. LDTD was defined as moderate or severe TR or tricuspid stenosis (TS) due to the presence of endocardial leads in the right heart chambers. The primary criterion for eligibility was visualization of lead-dependent mechanisms of tricuspid dysfunction according to one of the possible reasons of leaflets coaptation disorders: 1. presence of the loop of lead, irritating the TV; 2. propping the leaflet by the lead; 3. impingement of the leaflet by the lead. Severity of TR was assessed as the extent of the regurgitant jet into the right atrium using Doppler color flow

imaging. Moderate TR (grade III) was defined as a regurgitant jet extending to less than a half of the right atrium, whereas severe TR (grade IV) as a jet extending to more than a half of the length of the right atrium. Pulsed wave and continuous wave Doppler ultrasound was performed to measure the spectrum of the regurgitant jet and to estimate the pulmonary artery systolic pressure (PASP). Lead--dependent TS was diagnosed based on measurements of tricuspid flow velocities with maximal and mean transvalvular gradients. TS was defined as an increase in transvalvular flow velocity > 1 m/s and mean transvalvular pressure gradient > 5 mm Hg. Patients with severe concomitant mitral valve dysfunction were excluded from the LDTD group.

Group II consisted of the remaining 916 patients who in 2009–2011 were undergoing TLE procedures. The assessment of TR frequency (not related to the lead presence) in those patients was conducted.

In order to identify factors that affect the development of LDTD we carried out a comparative analysis of the number, type and dwell time of the leads, as well as the type of pacing systems. We also analyzed the presence of complications related to TLE and radiological and clinical efficacy of the procedure in both groups. Additionally, in group I we evaluated the type and mechanisms of LDTD, echocardiographic parameters confirming the hemodynamic significance of tricuspid dysfunction, left ventricular ejection fraction (LVEF), clinical consequences of LDTD, treatment type, and severity of tricuspid dysfunction in 3–5 days after lead extraction. A telephone follow-up of the LDTD patients was performed to assess the clinical outcome and survival at mean time of 1.5 years after the procedure (local ethic committee approval number 2/2012).

Statistical analysis

Continuous variables were expressed as mean ± standard deviation. Student's t-test was used to test for the significance of differences between the means. Qualitative variables were compared using the χ^2 test. A P-value of ≤ 0.05 (two-sided) was considered statistically significant.

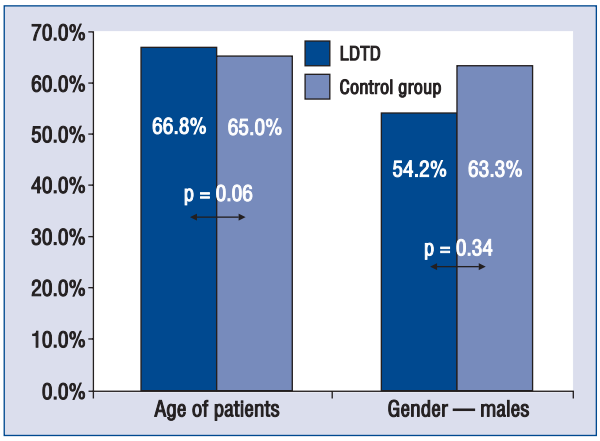

Figure 1. Demographic data; LDTD — lead-dependent tricuspid dysfunction.

Results

Demographic factors

The mean age of LDTD patients was 66.8 ± ± 16.0 years and did not differ from that of the remaining patients (65.04 ± 16.1 years) undergoing TLE for other complications of permanent pacing. In both groups TLE was performed more frequently in men with a trend of frequent prevalence of LDTD in women as compared with the control group (Fig. 1).

Indications for TLE

The most frequent indication for TLE in both groups of patients was lead's dysfunction or necessity to remove unnecessary leads. The second cause was infectious complications. There were no significant differences in the kind of indications for TLE between the groups (apart from LDTD presence in group I) (Table 1).

Patients in group I, apart from LDTD, had other complications of pacing therapy. Most frequently, LDTD was concomitant with the presence of loops of leads (10 patients; 41.7%) and infectious complications (7 patients; 29.2%). Additionally, 2 (8.3%) patients were found to have lead fractures and another 2 (8.3%) patients had lead-related dry right ventricular (RV) wall perforation up to the

Table 1. Indications for transvenous lead extraction.

		LDTD	Control group	P
Dysfunction of the lead/ /unnecessary lead		70.83% (including isolated LDTD in 45.73%)	58.91%	0.23
Infectious complications	Pocket infection	8.33%	23.06%	0.1
	LDIE	20.83%	18.03%	0.7

LDTD — lead-dependent tricuspid dysfunction; LDIE — lead dependent infective endocarditis

Table 2. Comparative analysis of the types of leads.

Types of leads	LDTD (total 58)	Control group (total 1,648)	P
Atrial	24 (41.4%)	718 (43.6%)	0.71
Right ventricular	30 (51.7%)	593 (36.0%)	0.02
Left ventricular	1 (1.7%)	79 (4.8%)	0.25
Defibrillator	3 (5.2%)	258 (15.7%)	0.04

LDTD — lead-dependent tricuspid dysfunction

epicardial fat. In 3 (12.5%) cases TLE was performed primarily in order to remove redundant leads. The complex coexistence of above mentioned complications in LDTD individuals was observed. Altogether, complications of permanent pacing concomitant with LDTD were found in 13 (54%) patients, in 11 (46%) subjects the sole reason for referral for lead extraction was LDTD.

Pacing systems

LDTD presence did not show simply a relation to complexity of pacing system. Comparative analysis of type of the leads indicated a higher number of RV leads in patients from group I but LDTD was unexpectedly frequently observed in patients having atrial leads. It seems to be caused by too long loops of atrial leads or by presence of abandoned leads (Table 2, Fig. 2).

In relation to the complexity of pacing systems (including abandoned leads) the number of extracted leads in LDTD patients was statistically higher than in the remaining ones (Fig. 3). In LDTD patients the mean time from lead placement to lead extraction was similar to that in the control group. There were no significant differences in the number of procedures preceding TLE, either. In LDTD patients a large loop of lead, crossing the TV (most often related to the dysfunction of lead fixing strip or lead fracture) was a significantly more frequent abnormality, probably affecting TV function. The most frequent presence of the atrial than ventricular lead loops was observed in 7 (29%) LDTD patients. There was also a slight trend towards frequent numbers of abandoned leads in these patients (Table 3).

The analysis of course of TLE and periprocedural events showed a statistically higher prevalence of technical complications in LDTD patients (33.3% vs. 17.1%; p = 0.04). Most frequently a breakdown (12.5%) or fragmentation of the lead (12.5%) were observed. In 8.3% of LDTD patients the advanced fibrosis reaction to lead was affirmed by failed TLE effort (Figs. 4, 5). The total number of major and minor complications related to TLE

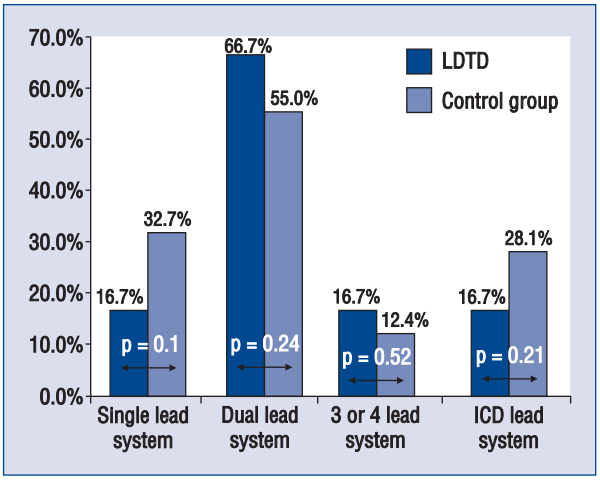

Figure 2. Comparison of pacemaker complexities; ICD — implantable cardioverter-defibrillator; LDTD — lead-dependent tricuspid dysfunction.

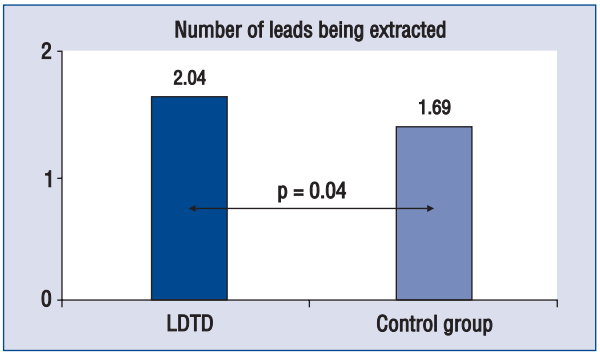

Figure 3. Comparison of numbers of lead removals; LDTD — lead-dependent tricuspid dysfunction.

(defined according to HRS Expert Consensus) and the radiological success were comparably frequent in both groups with a trend towards higher prevalence of these complications in the control group (Fig. 5) [3].

The clinical efficacy of TLE procedure was very high and comparable between the groups (100% vs. 98.4%; p = 0.46).

Table 3. Comparative analysis of mean dwell time and the commonest complications of pacing treatment in both study groups.

	LDTD (n = 24)	Control group (n = 916)	P
Mean lead dwell time before TLE [months]	91.0 ± 46.2	77.8 ± 57.8	0.27
Number of procedures before TLE	2.00 ± 1.53	1.92 ± 1.22	0.75
Unnecessary loops of lead in the tricuspid valve	10 (41.7%)	48 (5.24%)	0.0001
Atrial lead loops	7 (29.2%)	24 (2.62%)	0.0001
Ventricular lead loops	3 (12.5%)	24 (2.62%)	0.006
Number of abandoned leads	0.38 ± 0.71	0.21 ± 0.54	0.13

LDTD — lead-dependent tricuspid dysfunction; TLE — transvenous lead extraction

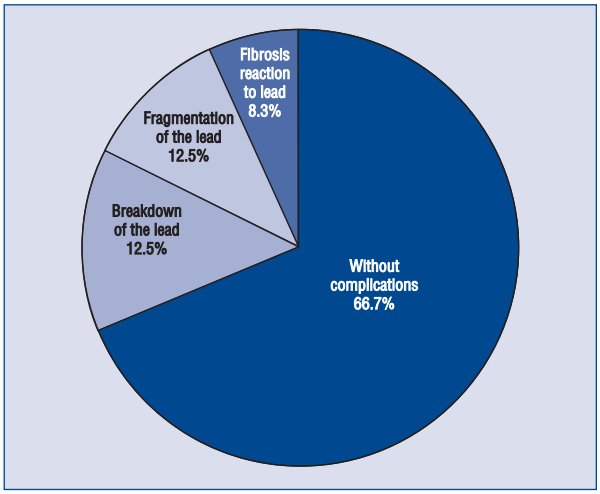

Figure 4. Transvenous lead extraction complications.

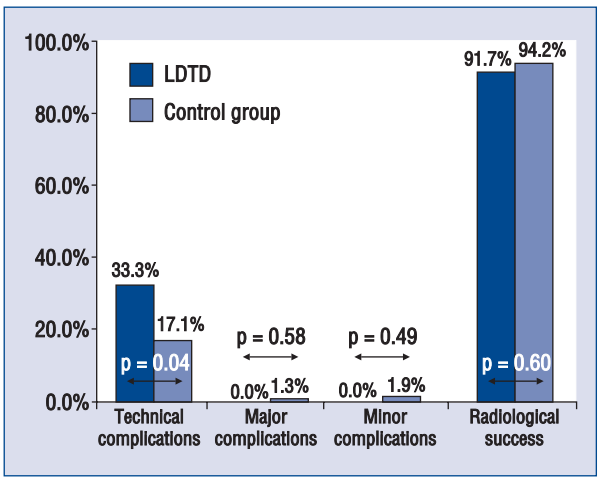

Figure 5. Comparative analysis of periprocedural complications and radiological efficacy of transvenous lead extraction; LDTD — lead-dependent tricuspid dysfunction.

Assessment of tricuspid valve dysfunction in group I

LDTD patients most frequently had severe TR, i.e. 20 (83%) patients. Four (16.7%) patients had TS with significant TR. The basic mechanism of LDTD was abnormal leaflet coaptation. In 10 (41.7%) patients this phenomenon was related to the presence of loops of leads in the tricuspid orifice, directly irritating the leaflets and causing their incomplete coaptation during ventricular systole. In 3 (12.5%) patients the lead loops were ingrown into the valve leaflets, which in 1 (4%) case did not allow for transvenous lead removal due to high risk of accidental leaflet damage. The second cause of abnormal coaptation was the lead propping on the leaflet — this mechanism was detected in 9 (37.5%) patients. In another 5 (20.8%) patients malcoaptation was a result of impingement on the leaflets by the lead causing valve leakage (Fig. 6).

In all 4 (16.7%) patients with TS coexisting with severe TR, valve dysfunction resulted from the lead loop ingrown into the leaflet with secondary valve stenosis and leakage.

The qualitative analysis of TR showed grade III and IV TR in 18 (75%) and 6 (25%) patients, respectively. The mean PASP was 42.1 ± 12.8 mm Hg with the PASP value not exceeding 40 mm Hg in 12 (50%) patients (Fig. 7).

The mean tricuspid flow velocity and gradient in patients with lead-dependent TS was 1.63 ± 0.25 m/s and 6 ± 0.82 mm Hg.

The mean LVEF was 44.8% ± 13.7 and severe systolic dysfunction (LVEF < 30%) was detected in 5 (20.8%) patients with LDTD (Fig. 8).

In 18 (75%) patients severe RV decompensation (NYHA class III/IV) was detected.

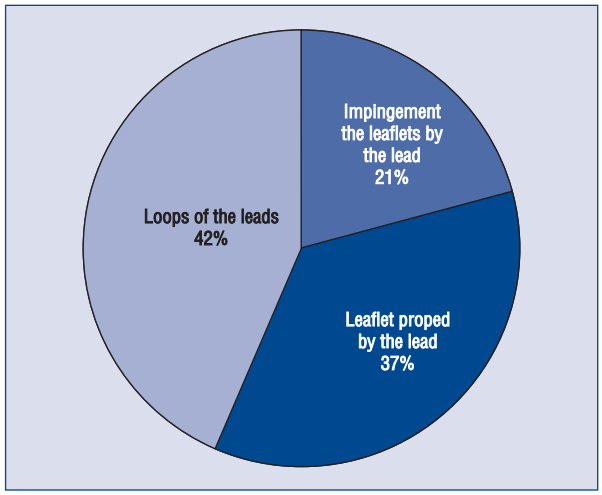

Figure 6. Mechanisms of lead-dependent tricuspid dysfunction.

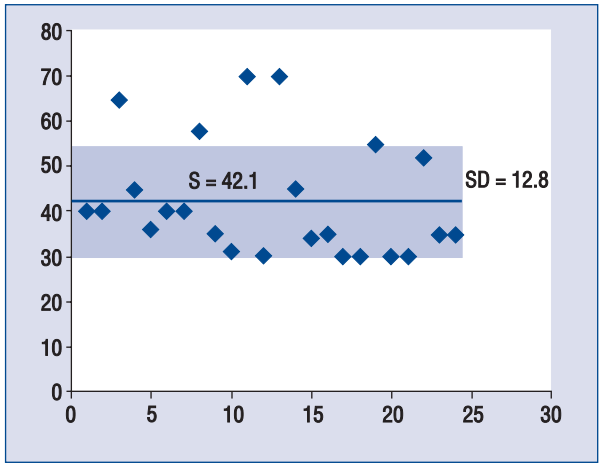

Figure 7. Estimation of mean pulmonary artery systolic pressure in lead-dependent tricuspid dysfunction patients.

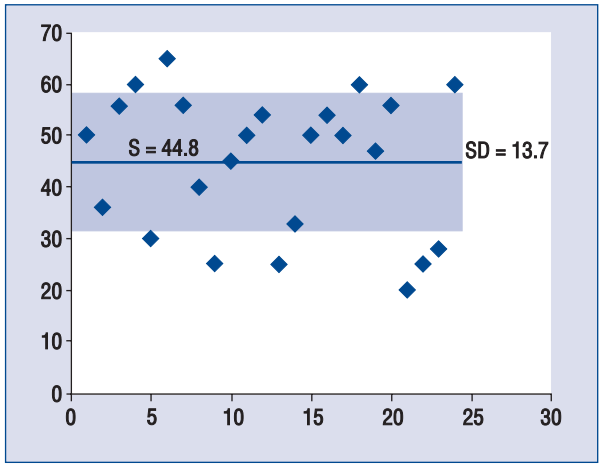

Figure 8. Measurement of left ventricular ejection fraction in lead-dependent tricuspid dysfunction patients.

Type and efficacy of LDTD management

In 21 (87.5%) patients TLE was performed with reimplantation of a RV lead, in 1 (4.2%) patient a LV lead was placed in the coronary sinus branches. Because of the TLE failure 2 (8.3%) patients were referred for surgical procedure of lead removal and positioning of epicardial leads directly on the myocardium of the LV. The procedures were withdrawn due to complications in 2 patients: in 1 patient occurred a following breakdown of ventricular lead, and in the next one because of strong adhesion of atrial lead loop with the TV leaflet.

Repeat echocardiograms showed improved tricuspid function in 15 (62.5%) patients, including 12 (50%) subjects in whom TLE with lead reimplantation diminished TR to grade II and in 3 out of 4 patients with TS it resulted in a reduction of transvalvular gradient (by 3/1.5 mm Hg on average). In the remaining 9 (37.5%) patients echocardiography did not show improvement, i.e. diminished severity of TV dysfunction, but in 2 patients further clinical improvement after cardiosurgery was observed.

Telephone follow-up (in the mean time 1.5 year after TLE) of patients with LDTD demonstrated marked clinical improvement in 18 (75%) patients. In most instances patients reported improved exercise tolerance and reduced peripheral edema or no edema at all. One patient selected for heart transplantation showed a spectacular improvement after lead reimplantation propped on the septal tricuspid leaflet, expressed as a 10-fold increase in 6-min walk distance, a significant increase in ergospirometric parameters and reduced edema with body mass reduction by 18 kg. The patient remains under careful observation with heart transplantation being postponed.

Within a year after TLE 2 patients underwent additional procedures to improve TV function: one subject with significant TS was submitted to percutaneous tricuspid valvuloplasty, and the other one with severe TR received a mechanical TV.

Long-term mortality was 4.1%: one death due to severe cardiac decompensation within a year after the procedure.

Discussion

LDTD has been rarely discussed in the literature, which is an indirect reflection of lower interest in the pathology and its treatment, compared to mitral valve anomalies. In clinical practice, however, there is an increasing number of patients with severe cardiac decompensation after permanent PM implantation without significant LV

impairment. The major issue is the visualization of the LDTD mechanism and identification of the direct relationship between LDTD and heart failure worsening that concerns also patients with LV damage. As it was already mentioned, the frequency of TV dysfunction in patients after permanent PM implantation has been rarely estimated. Furthermore, conflicting results have been obtained. Echocardiography in a group of 248 patients before and after PM and ICD implantation revealed that the severity of TR was significantly increased in 24.2% of patients after the procedure with the development of severe TR in 3.9% of patients. This phenomenon was more pronounced in patients with an ICD than a PM (32.4% vs. 20.1%; $p < 0.5$) [4]. In another study, patients with a PM/ICD did not show any significant increase in the prevalence and severity of TR; mild TR developed in 16% of patients after implantation, TR deteriorated from mild to moderate in 10% cases, there was no deterioration from moderate to severe TR. However, the investigators recommended longer patient monitoring in further studies [5]. Another long-term study in children with congenital heart defects confirmed a slight but significant deterioration of TR after PM implantation [6]. Reports of TS in patients with PMs are extremely rare. In most cases lead-dependent TS resulted from restricted leaflet mobility or perforation due to fibrous adhesion of PM lead [7, 8].

The present study was an attempt to evaluate the effect of PM-related factors on the development of LDTD. The complication tended to occur more frequently in women and in subjects with a larger number of implanted leads. More than 50% of LDTD patients were also found to have concomitant complications of PM therapy (unnecessary loops of leads, infectious complications, lead dysfunction and their mechanical damage and perforations).

Direct causes of valve dysfunction were also carefully analyzed. Mechanisms of LDTD ranged from abnormal coaptation as a result of lead impingement on the leaflets or lead adhesion to the leaflet (most frequently septal and posterior leaflets) or tendinous cords with progressive leaflet destruction, pulling the septal leaflet toward the interventricular septum, to functional TR due to RV asynchrony (altered propagation direction as a result of pacing) [9, 10]. The analysis of LDTD mechanisms in a group of 41 patients operated on for severe TR showed that the most common cause of valvular damage was mechanical injury to the leaflets by the striking lead (16 patients) and lead adhesion to the leaflet (14 patients). In the first case "the spinning electrode" resembled a cone with the TV as its basis and a tip of the electrode as its apex. In 7 patients leaflet perforation was found, whereas in 4 patients the leaflet was wrapped around the lead [10]. The present study, apart from the above mentioned mechanisms, documented also a significant relationship between the development of LDTD and the existence of lead loops crossing TV, probably strongly irritating the tricuspid leaflets, which leads to malcoaptation. This complication is becoming visible especially in patients undergoing PM implantation at a younger age and with a longer lead created for future growth [11]. Another cause for the lead loops development is failure of the lead affixing. The visualization of the lead loops is relatively easy — a circle is visible in RTG and echocardiography. In the present material in 10 LDTD cases lead loops were found with advantage of atrial lead (7 cases). In these patients the problem of LDTD development was evidently connected with irritating TV by the lead loops.

Another important clinical finding in LDTD patients was severe cardiac decompensation, found in 75% of them, coexisting with a relatively high mean LVEF (44.8%) and a relatively low PASP (42.1%). This fact appears to confirm lead-dependent cause of tricuspid dysfunction resulting in an increase in central venous pressure through increasing RV preload. Higher RV end-diastolic volume causes an increase in right atrial pressure which in turn decreases venous return and cardiac output [12]. Prior to TLE all patients underwent venography which revealed contrast-enhanced blood back flow from the innominate vein and superior vena cava to the jugular veins to the rhythm of the heart. As the present study was based on echocardiography the phenomenon was not explored any further.

An extremely important aspect is the best choice of treatment in patients with LDTD. There is a considerable amount of controversy surrounding the therapy. The basic approach appears to be the lead removal as the factor that causes TR. Our current state of knowledge allows to repair valves only during open-heart surgery. Currently available guidelines do not recommend lead extraction in patients with LDTD, probably for fear of potential aggravation of valve dysfunction in the case of lead adherence to the leaflet. Paradoxically, one of the complications of TLE is TR due to rupture of tendinous cords. TR as a complication after lead extraction is very rare — as shows available evidence — despite a larger number of technical problems among patients with LDTD (33.3% vs. 17.1% in

the controls; p = 0.04). The radiological success and the number of major and minor complications related to TLE were comparable in both groups. The present study shows that lead extraction with its reimplantation in the RV is sufficient to produce long-term clinical improvement in 75% of patients, with a dramatic improvement in objective cardiovascular parameters in some of those patients. This is the result of changing the site of leads crossing the TV, frequently unblocking the leaflets and improving coaptation. There are single reports in the literature on the efficacy of such an approach [13]. An alternative option is lead reimplantation, omitting the TV, i.e. LV pacing. In the present study this treatment option was used in 1 patient achieving a positive outcome. LV pacing via the cardiac vein was attempted in patients after prosthetic TV implantation, which appeared effective; similar results were obtained in patients with defibrillator leads [14, 15]. Positive effects of more synchronous ventricular pacing without crossing the TV may also be achieved through atrioventricular pacing as animal studies promisingly show [16].

In case there is no improvement after TLE, TV repair is recommended. The commonest and the most sparing surgical option is tricuspid annuloplasty. Annuloplasty may be combined with implantation of epicardial leads instead of those crossing the TV. Only epicardial ICD leads fail to produce expected results. They are left in the repaired TV. Because of technical problems during TLE in the present study this therapeutic option was used in 2 patients. In case of TS percutaneous annuloplasty is possible; this approach was used in 1 patient about a year after TLE.

If native valves cannot be saved and it is necessary to correct a dysfunction, a prosthetic TV is implanted. An optimal option is simultaneous explantation of the lead and its replacement for the epicardial one, but repositioning of the lead in the cleft between septal and inferior leaflet is also performed. In one report this approach appeared to be effective as there were no symptoms of RV decompensation at 1 year follow-up [17]. However, implantation of a prosthetic TV is associated with high risk of periprocedural death (7–40%) and high (30–50%) 10-year mortality. Leaving the lead *in situ* predisposes to LDTD recurrence, and positioning the lead in the cleft between prosthetic sutures makes it impossible to perform any percutaneous intervention in case of future problems with the lead or development of infection [18–20]. In the present study 1 patient necessitated implantation of a prosthetic TV; during the operation the pacing system was explanted and replaced for the epicardial one.

Limitations of the study

The main limitation of the present study is its retrospective character. A more accurate analysis would be necessary to compare the degree of TV dysfunction in both groups. Currently, only qualitative evaluation of the degree of TR in group I and similar data regarding mitral valve are available. Undoubtedly, appropriate documentation of LDTD requires further prospective studies using new echocardiographic techniques.

Conclusions

1. LDTD is a diagnostic and therapeutic challenge.
2. Probably, TLE procedure with lead reimplantation is a safe and effective option in LDTD management.
3. Change of the lead route through the TV causes improvement of the leaflets' coaptation with significant clinical status improvement.
4. An alternative option is TLE with omitted TV reimplantation/repair.
5. Cardiac surgery with epicardial lead placement should be reserved for patients with ineffective previous procedures.

Conflict of interest: none declared

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