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A CLINICAL CASE OF REFRACTORY VENTRICULAR TACHYCARDIA TREATMENT: FROM SIMPLE TO COMPLEX AND BACK AGAIN

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The article presents a clinical case of a patient with recurrent refractory ventricular tachycardia (VT) on the background of postinfarction cardiosclerosis. Despite optimal drug therapy and several catheter ablation procedures using modern navigation technologies, episodes of VT persisted. The key factor of ineffectiveness of the endocardial approach turned out to be the intramural location of arrhythmogenic zones. Therefore, a decision was made to perform surgical intervention with cryoablation of scar tissues. After the surgery, a stable remission was achieved, which was confirmed by the data of regular checks of the implantable cardioverter-defibrillator. The presented case emphasizes the importance of individualized and multidisciplinary approach in the choice of treatment tactics for patients with refractory VT.

Key words: ventricular tachycardia; catheter ablation; cryoablation; implantable cardioverter-defibrillator; ischemic heart disease

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Cardiovascular diseases remain the leading cause of mortality worldwide, with sudden cardiac death (SCD) accounting for 40-45% of such cases. More than 70% of SCD episodes are associated with ventricular tachycardia (VT), and survival after its occurrence remains extremely low, at only 3-5% [1, 2]. Despite advances in the treatment of patients with chronic heart failure (CHF) and reduced left ventricular ejection fraction (LVEF), the residual risk of SCD remains high. The main method of SCD prevention in patients with reduced LVEF, in addition to optimal medical therapy, is the use of implantable cardioverter-defibrillators (ICDs) - devices capable of recognising and terminating life-threatening arrhythmias. However, even with a comprehensive treatment approach, many patients experience appropriate ICD therapies, which negatively affect patients' psycho-emotional state and prognosis [3].

Advances in catheter treatment of VT in patients with structural heart disease make it possible to significantly reduce arrhythmia burden and improve prognosis and represent the treatment of choice for recurrent arrhythmias resistant to medical therapy [4, 5]. Nevertheless, in some patients endocardial ablation does not produce the desired effect, which is associated with an intramural and/or epicardial localisation of the arrhythmogenic substrate. In such cases, consideration may be given to cardiac surgical treatment, including resection of scar tissue. We present a demonstration of cardiac surgical treatment in a patient with VT recurrent despite prior endocardial radiofrequency ablation (RFA).

A 64-year-old man presented to the centre's clinic with complaints of recurrent episodes of dizziness and loss of consciousness occurring during attacks of rapid heartbeat lasting up to several minutes and terminating with ICD activation. According to the medical history, at the age of 49 years (2009) the patient sustained a myocardial infarction in the territory of the left anterior descending artery (LAD), followed by stent implantation. In 2016, 2019, and 2023, repeat endovascular revascularisation of the LAD territory was performed due to restenosis.

In October 2023 (at the age of 63 years), following a long-haul flight, the first episode of VT accompanied by dizziness was documented. During the patient's stay in the intensive care unit, the episodes recurred, requiring electrical cardioversion. Amiodarone was initiated as antiarrhythmic therapy, followed by implantation of a dual-chamber ICD. Device interrogations revealed episodes of VT with ineffective antitachycardia pacing (ATP) requiring shock therapy. During hospitalisations for these events, no reversible causes were identified. In October 2023, an endocardial electrophysiological study was performed, during which sustained VT was not inducible and ablation was not performed.

Subsequently, ICD therapies continued to occur, and in November 2023 RFA of arrhythmogenic areas along the anterior wall of the right ventricle was performed. During the control endocardial electrophysiological study, VT with a QRS morphology different from the clinical VT was induced. At that time, a decision was made to refrain

from further continuation of the procedure. During the same hospitalisation, coronary angiography was also performed, which revealed a haemodynamically significant LAD stenosis; percutaneous coronary intervention with implantation of a drug-eluting stent was carried out.

The patient continued to experience episodes of palpitations accompanied by device therapies. ICD interrogation documented episodes of fast VT with a cycle length (CL) of 280-330 ms, occasionally terminated by shock delivery due to ineffective ATP (Fig. 1).

According to transthoracic echocardiography, the LVEF was 31%, with a left ventricular apical aneurysm measuring 52 × 38 mm, an end-diastolic volume of 150 mL, and an end-systolic volume of 93 mL. These findings were also confirmed by cardiac magnetic resonance imaging. Laboratory test results were within normal limits. A decision was made to proceed with repeat catheter-based treatment.

The procedure was performed under combined anaesthesia with invasive haemodynamic monitoring. Left ventricular mapping was carried out via antegrade and retrograde approaches using the Abbott EnSite X system (Abbott Laboratories, USA) and the Advisor™ HD Grid diagnostic catheter (Abbott Laboratories, USA). Vascular access and transseptal puncture were performed under ultrasound guidance. Intracardiac echocardiography (ICE) revealed pronounced spontaneous echo contrast localised within the apical aneurysm, characterised by markedly thinned walls.

Using programmed ventricular stimulation according to a standard protocol, the clinical VT with a CL of 315 ms was repeatedly induced. Attempts to terminate VT using ATP resulted in a change in arrhythmia morphology followed by rhythm acceleration (CL 307 ms) and subsequent transformation into ventricular fibrillation during repeated attempts at termination with burst pacing, which necessitated electrical cardioversion (Fig. 2).

Given the haemodynamic instability, ventricular substrate mapping was performed during the patient's intrinsic rhythm using both antegrade and retrograde pacing. According to the activation map, a zone of late myocardial activation was identified within the post-infarction aneurysm, closer to the interventricular septum (Fig. 3a). At the first stage, a strategy aimed at eliminating excitation entry channels into the late activation zones was selected. Analysis of the activation pattern revealed the presence of two channels (Fig. 3a). However, frequency analysis of the recorded electrograms demonstrated that only one channel had a true endocardial localisation.

Pacing from the zone of late activation resulted in a match between the paced QRS morphology and that of the clinical VT. However, entrainment mapping could not be performed due to the development of haemodynamically unstable VT. Elimination of the endocardial channel was therefore performed as the first stage (Fig. 3b, c). At the second stage, homogenisation of the scar tissue within the apical aneurysm was carried out. The total ablation time was 20 minutes at a power of 40 W, irrigation flow of 25 mL/min, and contact force exceeding 8 g at each point.

It should be noted that RFA was performed under intracardiac echocardiography guidance, which provided the necessary visualisation of anatomical structures during the intervention. During the procedure, it was

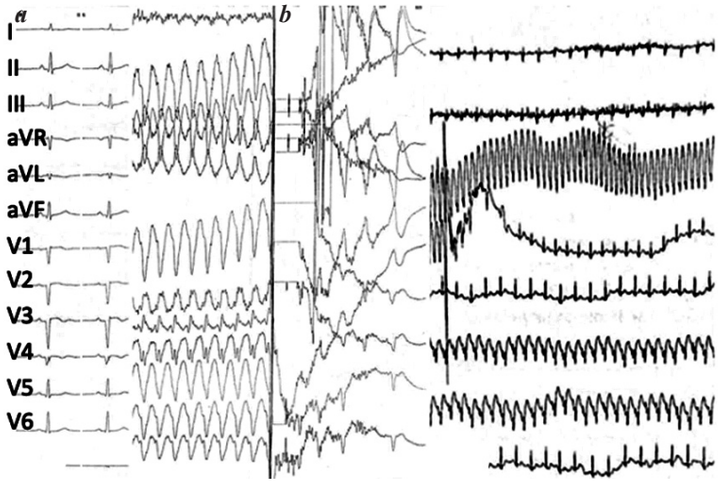

Fig. 1. Data from 24-hour ECG monitoring: (a) an episode of fast monomorphic VT with a CL of 250 ms, terminated by shock therapy delivery (analysis of QRS morphology during VT suggests localisation of the arrhythmogenic zone within the apical aneurysm with exit to the inferior wall of the left ventricle); (b) multiple episodes of VT with ineffective antitachycardia pacing (ATP) and termination by shock therapy.

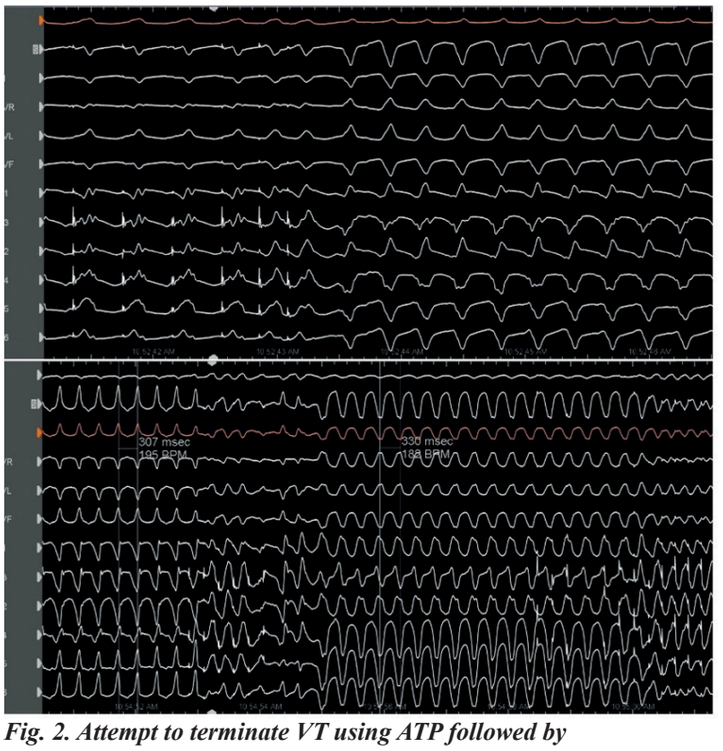

Fig. 2. Attempt to terminate VT using ATP followed by transformation into ventricular fibrillation.

found that areas of interest with significant myocardial thickness were localised in the region of the anterior interventricular groove, where ablation lesions had previously been delivered by colleagues from the right-sided, septal aspect. Epicardial access in this region was limited by the close proximity of a coronary artery and a pronounced epicardial fat layer, which restricted the feasibility of this approach.

After RFA in the region of late potential recording, myocardial capture during pacing from the scar tissue was absent. During repeat induction testing, VT was not induced, which led to termination of the procedure. On the second postoperative day, a recurrence of VT with a CL of 315 ms was recorded and terminated by shock therapy. Subsequently, VT episodes again acquired a recurrent pattern (Fig. 4).

Due to the ineffectiveness of repeated endocardial ablation procedures, a decision was made to perform aneurysm resection with left ventricular reconstruction and cryoablation of the border zones. Upon opening the left ventricular cavity, a pronounced layer of epicardial fat located along the perimeter of the left ventricular aneurysm was noted, which had also been previously identified by intracardiac echocardiography. Along the scar tissue of the anterior wall, a fresh mural thrombus measuring 2 × 3 cm was visualised. In the demarcation zone, circumferential cryoablation of the endocardium and epicardium was performed: applications were delivered along the perimeter of the resection area, forming a continuous ring of necrosis. The objective of cryoablation was to isolate potential arrhythmogenic foci within the transition zone between viable myocardium and scar tissue, as well as to prevent recurrent ventricular arrhythmias. Subsequently, the left ventricle was reconstructed using a double-layer continuous suture with Teflon pledgets (Fig. 5). During one year of follow-up after cardiac surgery, no recurrence of cardiac rhythm disturbances was recorded, and the patient continued to receive optimal medical therapy.

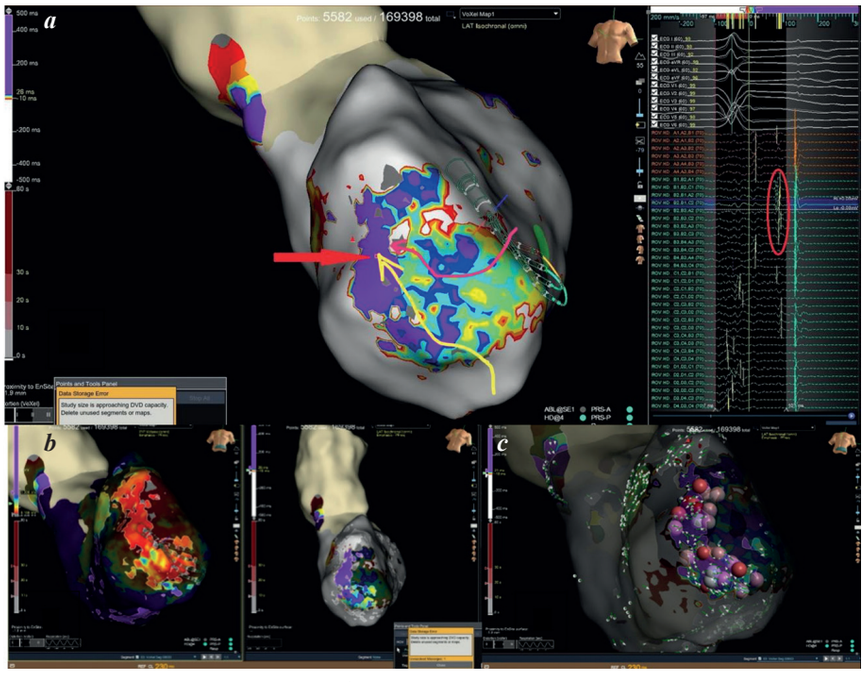

Fig. 3. Substrate mapping data: (a) area of late left ventricular myocardial activation within the post-infarction aneurysm; (b) area of late myocardial activation in the apical region displayed in frequency-domain signal analysis mode (window 250-1000 Hz); notably, only part of the scar zone demonstrates far-field signals, indicating an intramural/epicardial localisation of a proportion of the conduction channels; (c) radiofrequency ablation of the channel within the scar region.

DISCUSSION

One of the main methods of SCD prevention is implantation of an implantable cardioverter-defibrillator (ICD); however, these devices do not address the problem of recurrent cardiac arrhythmias, and each delivery of shock therapy is associated with a worsening of prognosis [6]. Endocardial ablation is the gold standard for the treatment of therapy-refractory sustained VT in patients with structural myocardial disease leading to ICD therapies [7]. One of the pioneers in the study of VT in patients with structural heart disease was Mark Josephson, who confirmed the presence of critical zones within the border area between scar tissue and intact myocardium, which became the starting point for subsequent research and led to the development of electrophysiological techniques and navigation systems [8, 9]. Since then, catheter ablation of VT has been increasingly performed worldwide and has demonstrated favourable outcomes.

Thus, the randomised controlled trial PARTITA demonstrated a significant reduction in the risk of death or hospitalisation due to decompensated CHF from 42% in the control group to 4% in the ablation group (HR 0.11; 95% CI 0.01-0.85; p = 0.034) in patients after the first appropriate ICD therapy [10]. The most pronounced benefit of interventional therapy has been observed in patients with post-infarction cardiosclerosis, which is attributable to the extensive clinical experience in treating this population and the presence of scar-related substrates responsible for VT maintenance.

According to a meta-analysis of five randomised controlled trials including 635 patients with post-infarction cardiosclerosis, catheter ablation was associated with a 51% reduction in the likelihood of ICD shock therapy (HR 0.49; 95% CI 0.28-0.87). In addition, the risks of electrical storm and hospitalisation were reduced by 36% (HR 0.64; 95% CI 0.43-0.95) and 33% (HR 0.67; 95% CI 0.46-0.97), respectively [11]. In the VANISH2 randomised controlled trial, which included 416 patients with ischaemic cardiomyopathy and clinically significant VT, catheter

ablation was shown to be superior to antiarrhythmic drug therapy as a first-line strategy: over a median follow-up of 4.3 years, the composite primary endpoint (all-cause mortality or clinically significant ventricular tachyarrhythmia) occurred in 50.7% of patients in the ablation group and in 60.6% of those receiving medical therapy (HR 0.75; 95% CI 0.58-0.97; p = 0.03) [4]. In addition, the catheter ablation group demonstrated a lower incidence of treatment-related adverse events (12.3% vs 22.1%) as well as a lower rate of therapy-related mortality.

The study findings once again confirm the appropriateness of catheter ablation as the preferred first-line therapy in patients with VT of ischaemic origin, aimed at improving quality of life and reducing the risk of adverse outcomes. However, the efficacy of endocardial ablation does not reach 100%, which is attributable to multiple factors, including the presence of numerous arrhythmogenic zones, their de novo formation, and the "non-subendocardial" localisation of conduction channels, which limits the effective delivery of radiofrequency energy into deeper myocardial layers. At the same time, aggressive catheter ablation is associated with an increased risk of myocardial perforation in areas of post-infarction aneurysms with thinned walls.

In cases of ineffective repeated endocardial ablation, epicardial and/or cardiac surgical interventions with excision of scar tissue and ablation of border zones represent the treatment of choice. These techniques have a long historical background, beginning with Charles Bailey's report of successful elimination of recurrent VT in a patient with post-infarction cardiosclerosis through aneurysm resection, which marked a turning point in the development of surgical treatment strategies for this patient population and led to the introduction of a new technique-subendocardial resection of arrhythmogenic zones, known as the "Pennsylvania peel" method [12].

Several important factors merit attention in the present clinical case. First, the large size of the apical left ventricular scar with markedly thinned walls limited the feasibility of aggressive ablation. Second, the presence of an intramural component responsible for VT maintenance was identified. As demonstrated in a number of studies, analysis of the frequency characteristics of electrograms correlates with near-field signal components [13]. The use of algorithms designed to identify regions with target frequency characteristics allows prediction of the effectiveness of endocardial ablation [14]. Returning to the mapping results (Fig. 4), only a portion of the signals within the scar region demonstrated a true subendocardial origin. Despite homogenisation of the scar tissue, VT recurrence was observed. In such cases, the only viable option is a combined epi-endocardial approach. In the present case, taking into account all myocardial characteristics, a decision was made to proceed with additional cardiac surgical intervention; however, given the small left ventricular volumes, complete resection of the aneurysmal tissue was abandoned in favour of circumferential cryoablation along the perimeter of the aneurysm using both epicardial and endocardial approaches.

CONCLUSION

Modern arrhythmology and cardiac surgery have demonstrated substantial progress in the development of minimally invasive methods for the treatment of VT since the early decades of the twentieth century. These advances have made it possible to significantly reduce the recurrence rate of arrhythmias, minimise intraoperative complications, improve patients' quality of life, and increase survival. However, as clinical practice shows, including the case presented herein, in certain situations there remains a need for individualised arrhythmological strategies, including those involving the use of radical treatment approaches. Such cases require particularly thorough analysis of clinical data and a personalised approach to selecting the optimal management strategy, which was implemented in the present clinical case.

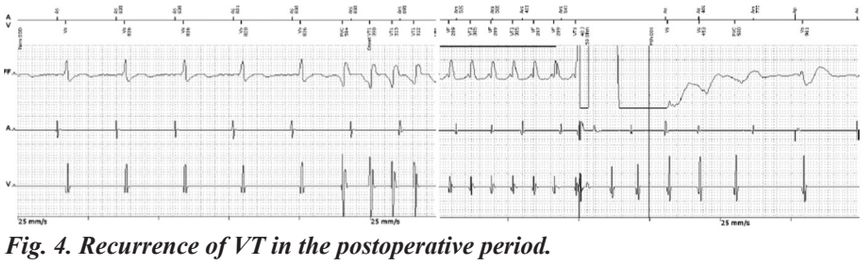

Fig. 4. Recurrence of VT in the postoperative period.

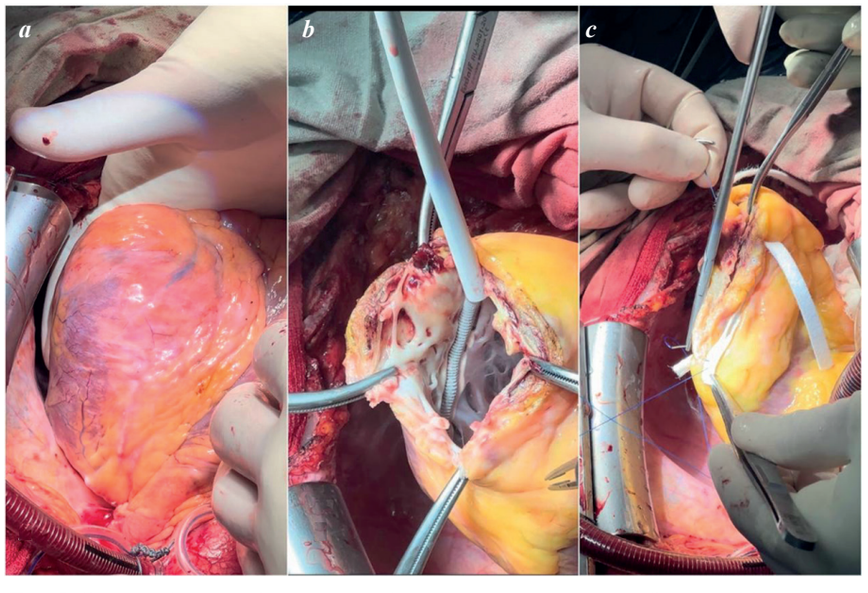

Fig. 5. Stages of surgical treatment: (a) prior to intervention; (b) cryoablation of the border zones; (c) left ventricular reconstruction.

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