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► **To cite this version:**

Mikael Laredo, Ludivine Oliveira da Silva, Fabrice Extramiana, Nicolas Lellouche, Emilie Varlet, et al.. Catheter ablation of electrical storm in patients with arrhythmogenic right ventricular cardiomyopathy. Heart Rhythm, 2020, 17 (1), pp.41-48. 10.1016/j.hrthm.2019.06.022 . hal-02446179

HAL Id: hal-02446179

<https://hal.sorbonne-universite.fr/hal-02446179v1>

Submitted on 20 Jan 2020

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1 **Catheter Ablation of Electrical Storm in Patients with Arrhythmogenic Right**
2 **Ventricular Cardiomyopathy**

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18 **Brief Title:**

19 Electrical Storm Ablation in ARVC

20 **Word count:** 4973

21 **Source of funding:**

22 None

23 **Disclosures:**

24 The authors report no conflict of interest relevant to the present study to disclose.

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30

31 **Structured Abstract**

32

33 **Background**

34 Therapeutic strategies for electrical storm (ES) in patients with arrhythmogenic right
35 ventricular cardiomyopathy (ARVC) are not well defined.

36 **Objective**

37 To report the acute and long-term results of ventricular tachycardia (VT) radiofrequency
38 catheter ablation (RFCA) as treatment for ES in patients with ARVC.

39 **Methods**

40 This multicenter study retrospectively enrolled 23 consecutive patients with ARVC (mean age
41 43.6 ± 16.7 years; all male) who underwent 24 RFCA procedures for ES between 2003 and
42 2015.

43 **Results**

44 Thirteen (57%) patients had a previous VT RFCA; 14 (61%) had right ventricular dysfunction
45 and 7 (30%) left ventricular ejection fraction $\leq 50\%$. The clinical VT was inducible in 19
46 (79%) procedures. Epicardial ablation was performed in 4 (17%) procedures. The median
47 number of targeted VTs was 1 [1–6]. Complete acute success (no VT inducible) was achieved
48 in 11 (46%) procedures and partial acute success (clinical VT nor inducible) in 11 (46%).

49 After a median follow-up of 3.9 years [1 month–10 years], ES recurred in 2 patients and end-
50 stage heart failure developed in 4 (17%), leading to 1 death and 3 heart transplantations. At 1-
51 year follow-up, the probability of freedom from VT recurrence was 75% and did not
52 significantly predict long-term survival. At last evaluation, 8 (35%) patients were free of non-
53 beta-blocker anti-arrhythmic drugs as compared with 1 (4%) at baseline ($p = 0.02$).

54 **Conclusion**

55 Catheter ablation was efficient to prevent ES recurrence in patients with ARVC. However,

56 these patients were at high risk of evolution toward ARVC-related heart failure that was not
57 associated with VT recurrence.

58 **Introduction**

59

60 Arrhythmogenic right ventricular cardiomyopathy (ARVC), a type of arrhythmogenic
61 cardiomyopathy is a rare inherited disease associated with progressive fibrofatty myocardial
62 remodeling affecting mostly the right ventricle (RV)^{1,2}. Structural alterations associated with
63 ARVC predispose to reentrant ventricular tachycardia (VT), and patients may experience a
64 high burden, especially those at advanced disease stage³. Implantable cardioverter-
65 defibrillators (ICDs) can efficiently prevent sudden cardiac death; however, VT events among
66 ICD carriers with ARVC are frequent and are associated with ICD firing and increased
67 cardiovascular morbidity³.

68 Electrical storm (ES) is a life-threatening complication of structural heart diseases
69 associated with advanced disease stage, with poor short- and long-term prognosis, that
70 requires specific therapeutic interventions^{4,5}. Patients with ARVC may experience ES, but the
71 clinical implications and prognostic significance in this setting are less well understood.

72 Multiple studies have demonstrated a benefit of radiofrequency catheter ablation
73 (RFCA) for treating ARVC-associated VT⁶⁻⁸, which is therefore recommended in addition to
74 ICD^{9,2}. Several reports also suggest that RFCA is effective in reducing the ventricular
75 arrhythmia burden in the context of ES, when evaluated in various heart diseases^{4,10}.
76 However, the benefits of RFCA as a treatment for ES in ARVC has not been specifically
77 assessed. In this multicenter observational study, we analyzed the clinical and
78 electrophysiological characteristics of ARVC-related ES and report the outcomes of RFCA.

79

80

81 **Methods**

82

83 *Patients and definitions*

84 Patients who underwent catheter ablation for ES from 2003 to 2015 in 7 tertiary care French
85 centers were retrospectively included (n=23) if they 1) had a definite diagnosis of ARVC
86 according to the 2010 revised Task Force Criteria (TFC)¹¹; 2) presented ES defined by at least
87 3 separate sustained VT episodes or ventricular fibrillation episodes within 24 hr, documented
88 by 12-lead electrocardiography (ECG), Holter monitoring or ICD interrogation; 3) and were
89 referred for radiofrequency ablation as an urgent treatment for ES. Any ICD therapy
90 (antitachycardia pacing or shock) was considered a sustained VT episode. In cases of
91 unsuccessful antitachycardia pacing or shock, further therapies related to the same episode
92 were not counted as an individual VT episode.

93 Patient screening involved reviewing all consecutive VT RFCA procedures in each
94 center during the inclusion period, identifying those performed for ARVC-related VT (n=121
95 across the 7 centers), and selecting procedures that were specifically performed as urgent
96 treatment for ES in the days or weeks after ES.

97 ARVC was prospectively diagnosed according to the current consensus documents
98 and was retrospectively assessed for final diagnosis according to the 2010 revised TFC¹¹ at
99 the time of data collection. The TFC criteria regarding the arrhythmia refer to an evaluation
100 before the ES event.

101 Severe RV systolic dysfunction was defined by RV ejection fraction < 40% on MRI or
102 RV angiography and/or RV fractional area change < 33% on transthoracic echocardiography
103 in the apical 4-chamber view (major TFC). Diffuse RV involvement was arbitrarily defined
104 by the presence of ≥ 3 akinetic or dyskinetic RV regions including RV apex, outflow tract,

105 free wall and peritricuspid region and diagnosed by echocardiography, MRI or RV
106 angiography.

107 Patients with available samples underwent mutation screening for desmosomal genes
108 *PKP2*, *DSG2*, *DSP*, *JUP* and *DSC2* by Sanger or next-generation sequencing after informed
109 written consent. All patient data were anonymized prior to data collection. No authorization
110 approval from our Institutional Committee on Human Research was required for this
111 retrospective study, in accordance with French law.

112

113 ***Electrophysiological study and catheter ablation***

114 Electrophysiological studies (EPS) and catheter ablation procedures (n=24) were performed
115 under continuous invasive pressure monitoring. Programmed ventricular stimulation was
116 performed in patients in sinus rhythm at the beginning of the procedure, according to standard
117 protocols by using intravenous isoproterenol infusion if necessary. Ablation strategies
118 included VT activation mapping with characterization of the VT critical isthmus and ablation
119 targeting mid-diastolic potentials, pace-mapping based on the clinical VT 12-lead ECG
120 morphology and substrate mapping based on delineation of scar areas identified by bipolar
121 voltage mapping and maximal elimination of late potentials or Local Abnormal Ventricular
122 Activities in sinus rhythm or ventricular pacing. Ablation strategies were chosen at the
123 discretion of the electrophysiologist according to the patient's history, number of previous
124 ablation procedures, VT characteristics and clinical tolerance. For the purpose of the study,
125 ablation strategies were retrospectively classified as VT mapping (activation, entrainment and
126 pace-mapping), substrate mapping and mixed VT + substrate mapping.

127 Electro-anatomical mapping systems (CARTO, Biosense Webster Inc., Diamond Bar,
128 CA, or NavX, St. Jude Medical Inc., St. Paul, MN) were used in 19/24 procedures and a
129 multipolar electrode diagnostic catheter was used in 3/24 procedures. When indicated,

130 epicardial access was obtained by a percutaneous subxiphoid puncture. Procedures were
131 performed with 4-mm conventional or irrigated-tip radiofrequency catheters. Induced VTs
132 were considered clinical when the 12-lead ECG morphology was identical to the clinical VT
133 with the same rate \pm 20 bpm. Complete acute success was defined as no sustained VT induced
134 at final EPS, partial acute success as no clinical VT induced, acute procedural failure as the
135 ability to induce a sustained clinical VT. Procedural success was considered undetermined
136 when no VT was induced at the beginning of the procedure or when no final EPS was
137 performed.

138

139 ***Follow-up and endpoints***

140 Patients were followed routinely by their treating electrophysiologist or cardiologist. VT
141 recurrence was defined as the recurrence of any documented sustained VT lasting \geq 30 sec or
142 any appropriate ICD therapy including anti-tachycardia pacing. Recurrence of ES followed
143 the same definition as for the initial presentation. Medical records and stored electrograms
144 from ICD interrogations were reviewed at each center to identify VT recurrence and assess
145 ICD therapies.

146 ***Data collection***

147 Data regarding demographics, medical history, clinical evaluation, 12-lead ECG, genetic
148 analyses, echocardiography and computed tomography imaging, ICD interrogations,
149 electrophysiology studies and catheter ablation procedures were retrieved from medical
150 records at each center.

151 ***Statistical analyses***

152 Continuous data are reported as mean \pm SD or median [range] for normally or non-normally
153 distributed data. Categorical variables are presented as number (%). Comparative statistics
154 involved the exact Fisher test. Survival curves were created with the Kaplan-Meier method,

155 with comparisons involving the Log-Rank test. Univariate regression analyses were
156 performed with the Cox proportional-hazards model, estimating hazard ratios (HRs) and 95%
157 confidence intervals (CIs). All tests were two-sided, with $p < 0.05$ denoting statistical
158 significance. All statistical analyses involved using IBM SPSS v23 (IBM Corp., Armonk,
159 NY, USA).

160

161 **Results**

162

163 *Patient characteristics*

164 Overall, 23 consecutive patients with ARVC (mean age at diagnosis 43.6 ± 16.7 years)
165 underwent 24 RFCA procedures for ES were retrospectively enrolled (Table 1). All patients
166 had a definite ARVC diagnosis according to the 2010 revised TFC¹¹. Individual TFC criteria
167 are available in Supplementary Table 1 and ES episode descriptions are in Supplementary
168 Table 2. Patients presented ES at a mean of 7.3 ± 9.6 years after ARVC diagnosis. Three
169 (13%) patients had a history of resuscitated sudden cardiac death. Overall, 17 (74%) patients
170 had previously experienced at least one episode of sustained VT, 18 (83%) had previous ICD
171 placement, and 13 (57%) had previous VT catheter ablation (median 1, range [0–7]). Results
172 from genetic analysis were available for 15 (65%) patients; 10/15 (67%) carried an ARVC-
173 related pathogenic mutation (Table 1). Twelve (52%) patients presented incessant not-well
174 tolerated VT despite repeated intravenous amiodarone boluses, electrical cardioversion or
175 general anesthesia (Supplementary Table 2). The median number of ICD shocks related to ES
176 was 13 (range 1–30). Most patients showed typical depolarization and repolarization
177 abnormalities (Supplementary Table 1). Overall, 14 (61%) patients had severely impaired RV
178 systolic function, 8 (35%) had at least mild LV systolic function impairment with LV ejection
179 fraction (LVEF) $\leq 50\%$, and one had severe LV dysfunction (LVEF = 20%).

180

181 ***Procedural characteristics and acute results***

182 In most cases, a single monomorphic VT was responsible for the ES [20 (83%) procedures],
183 whereas multiple sustained monomorphic VTs could be induced by ventricular programmed
184 stimulation in 15 (63%) procedures (Table 2 and Supplementary Table 2 for individual
185 electrophysiological data). Median clinical VT rate was 171 bpm (range 130–230).
186 Endocardial+epicardial mapping and ablation was performed in 4 (17%) procedures. In one
187 patient with severely depressed LVEF, the RFCA procedure was performed under
188 extracorporeal membrane oxygenation support for incessant hemodynamically unstable VT.
189 Irrigated radiofrequency was used in 21 (88%) procedures and non-irrigated radiofrequency
190 in 3 (12%). Ablation strategies consisted of VT mapping [7 (29%)], substrate mapping [8
191 (33%)] and mixed VT and substrate mapping [9 (38%)]. The median number of targeted VT
192 was 1 [range 1–6] and targeted RV areas 1 [1–5]. More than one RV area was targeted in 9
193 (38%) patients and 10 (42%) procedures. Targeted RV areas included RV free wall [12
194 (50%)], RV outflow tract [7 (29%)], RV sub-tricuspid region [9 (38%)], RV septum [2 (8%)]
195 and left ventricle [2 (8%)] (Supplementary Table 2). Complete acute success was achieved in
196 11 (46%) procedures and partial acute success in 11 (46%). An undetermined result was
197 reported in 2 (8%) procedures. There was no acute procedural failure. Acute procedural
198 complications included one minor groin hematoma and one femoral arteriovenous fistulae
199 requiring percutaneous treatment.

200

201 ***Long-term outcomes***

202 During a median follow-up of 3.9 years (range 1 month–10 years), ES recurred in 2 patients
203 (see Supplementary Table 3 for detailed individual outcomes). The first patient underwent
204 redo RFCA for ES at 2.5 years after the initial procedure but died 2.8 years later in the post-

205 operative course of heart transplantation (HT) that was performed for end-stage heart failure.
206 The other had ES recurrence 1.5 years after the initial ES; he underwent redo RFCA and
207 remained free of VA recurrence in a subsequent follow-up period of 1 year. The cumulative
208 probability of freedom from ES recurrence at 1 and 5 years after the initial ES RFCA
209 procedure was 100% and 85% (Figure 1A & Supplementary Table 4 for 95% CIs of survival
210 rates). Six patients had VT recurrence during follow-up. All VT recurrences were
211 monomorphic and triggered appropriate ICD therapies (details in Supplementary Table 3).
212 Five patients underwent redo VT ablation, including 4 who remained free of VT recurrence at
213 the end of follow-up. The cumulative probability of freedom from VT recurrence at 1 and 5
214 years after the initial ES RFCA procedure was 77% and 66% (Figure 1B). Complete acute
215 procedural success was not significantly associated with freedom from VT recurrence after
216 the initial ES ablation procedure (HR = 0.27, 95% CI [0.05–1.43], p = 0.13). Baseline LVEF
217 and presence of RV systolic dysfunction were not associated with VT recurrence. After the
218 initial ES catheter ablation procedure, 4 patients had ICD placement, so all patients had an
219 ICD during follow-up. At the end of follow-up, 8 (35%) patients were not taking non-beta-
220 blocker anti-arrhythmic agents as compared with 1 (4%) at baseline (p = 0.02).

221 Three patients died during follow-up (2 immediately after HT and 1 from hemorrhagic
222 stroke while awaiting HT) and 1 patient survived HT. These 4 patients had end-stage heart
223 failure related to ARVC with severe biventricular systolic dysfunction. The cumulative
224 probability of survival without death or HT at 1 and 5 years after initial ES catheter ablation
225 was 90% and 84% (Figure 2A). Neither VT recurrence during follow-up (HR 2.32, 95% CI
226 [0.32–16.74], p = 0.40) (Figure 2B) nor complete acute procedural success (HR 0.96, 95% CI
227 [0.13–6.86], p = 0.97) was significantly associated with death or heart transplantation during
228 follow-up. Among the 4 patients who died or underwent HT, 2 did not show VA recurrence
229 after the initial RFCA. Baseline LVEF was significantly associated with risk of death or HT

230 (HR for a 1% LVEF increase: 0.90, 95% CI [0.81–60.99], $p = 0.045$) (Figure 2C). The
231 detailed course of adverse events during follow-up is in Supplementary Table 3.

232

233 **Discussion**

234

235 This is the first study to report outcomes after RFCA performed for ES in patients with
236 ARVC. RFCA was safe and effective to prevent ES recurrence, with no periprocedural
237 adverse outcomes and only 2 ES recurrences. In addition, 1) patients presenting ES frequently
238 had ARVC with overt RV structural alterations and a significant history of ventricular
239 arrhythmias with previous VT ablations, 2) usually one monomorphic VT was responsible for
240 the ES event, 3) complete procedural success was achieved in 11 (46%) procedures and
241 elimination of the clinical VT could be achieved in 22 (92%), 4) the 5-year estimated VT
242 recurrence rate was 34%, and 5) we found a 24% 5-year estimated rate of mortality or HT
243 after an ES event — mortality consecutive to heart failure related to ARVC structural
244 dysfunction and that was not associated with the long-term arrhythmic outcome after RFCA.

245

246 *Clinical features associated with ES in ARVC*

247 In our series of ARVC patients presenting ES and referred for RFCA, we found a high
248 prevalence of electrical and structural abnormalities as compared with large cohorts of ARVC
249 patients^{12,13}. Most patients had severe RV systolic dysfunction, 35% had LV dysfunction,
250 43% showing epsilon-wave and 53% previous VT RFCA. All were male, and the mean age at
251 ES was 43.6 years, when most ARVC patients are symptomatic¹⁴. Overall, 66% of screened
252 individuals had a pathologic mutation in desmosomal genes, which is higher than what is
253 usually found in ARVC cohorts and associated with worse prognosis¹². These high-risk
254 features were also more frequent than in a recent large cohort of ICD carriers¹⁵. However,

255 patient characteristics were overall comparable to studies enrolling ARVC patients
256 undergoing VT ablation^{8,16}, except in terms of the frequency of ECG depolarization
257 abnormalities and previous VT ablation, which were more frequent in our patients and
258 suggests that ARVC patients experiencing ES have a more extensive electrical substrate than
259 those with isolated VT.

260 The study of Carbuciccio *et al.* reported outcomes of ES ablation among 95 patients
261 (76% with ischemic cardiomyopathy and 13 [14%] with ARVC)¹⁰. Mean age was higher than
262 in our series (64±13 years), 38% of patients had several clinical VTs and 11% required
263 periprocedural hemodynamical support. In our ARVC series, a single VT was responsible for
264 most of the ES; we did not observe any patient with repetitive polymorphic VT; and only 1
265 patient had > 5 inducible VTs. Hemodynamic instability was rare, which is probably related
266 to the conserved or mildly impaired LV function in most ARVC patients.

267

268 ***Procedural outcomes***

269 We report a relatively low rate of complete acute procedural success, 46%, as compared with
270 the 72% in the largest series of ES ablation¹⁰ and the 71% to 77% in a recent series of VT
271 ablation in ARVC⁸. The low rate may have several explanations: first, our series included rare
272 patients across a 12-year period that saw major progresses in RFCA techniques. Particularly,
273 a combined endocardial+epicardial approach, associated in several studies with improved
274 acute procedural success and long-term freedom from VT recurrences^{7,8,17,18}, was performed
275 in only 18% of our cases because of the inclusion period. Epicardial ablation likely would
276 have enhanced our acute and late success rates. Nonetheless, recent evidence has shown a
277 negative correlation between the extension of RV endocardial scarring and the presence of
278 arrhythmogenic epicardial scar because of disease progression from the epicardium to the
279 endocardium¹⁹. Considering the severity of RV involvement in our series, a first-line

280 epicardial approach would probably have been less profitable than in a regular case of VT
281 RFCA in ARVC. Also, epicardial access may be deleterious in patients with ES by
282 compromising the chance of inducing the clinical VT and by increasing the risk of
283 hemodynamic instability during VT mapping. That being said, there is sufficient evidence to
284 favor an initial endocardial+epicardial approach in stable patients presenting ES, and it should
285 be particularly considered in patients with early-stage ARVC or when evidence for extensive
286 epicardial substrate or clinical VT originating from the epicardium is known from a previous
287 VT ablation. Second, 3D electroanatomic mapping systems, which significantly improve VT
288 RFCA⁷ in ARVC, were unavailable at the time of RFCA in 25% of our cases. Finally, our
289 patients had marked RV structural alterations, which are correlated with scar extension and
290 diffuse arrhythmogenic substrate²⁰.

291 All our patients were amenable to RFCA, including those with several previous
292 ablations and biventricular dysfunction, and no severe complications were observed,
293 including no hemodynamic deterioration. Although we obtained a relatively low rate of
294 complete acute success, clinical VT could not be induced in most patients (92%) and only 2
295 patients experienced ES recurrence after RFCA. This finding may suggest that inability to
296 induce clinical VT is a reasonable endpoint to prevent ES recurrence. Catheter ablation is a
297 Class I indication as an urgent first-line treatment for ES²¹ regardless of the heart disease, and
298 should also be considered as such in ARVC.

299

300 *Long-term survival after ES in ARVC*

301 ES is an independent predictor of death among various heart diseases⁵. However, the
302 mechanisms by which ES leads to increased mortality remain poorly understood, and its
303 clinical significance in ARVC as compared with other heart diseases is poorly established. In
304 a recent meta-analysis of 471 ES patients from 39 publications, only 22 had ARVC, which

305 precluded generalizing findings to patients with ARVC⁴. A key question is whether RFCA
306 improves long-term survival after an ES event in ARVC, or if RFCA only permits a palliative
307 benefit of alleviating the VT burden. In the study of Carbuciccio *et al.*, long-term survival
308 was worse for patients for whom RFCA resulted in partial success or procedural failure, with
309 a significant occurrence of sudden cardiac death related to untractable ES recurrence¹⁰. In the
310 above-mentioned meta-analysis, arrhythmic sudden death accounted for 23% of long-term
311 mortality⁴. These findings suggest that VA recurrence after RFCA for ES contributes to
312 survival to some extent. In our study specifically addressing ARVC, we found no association
313 between long-term survival and procedural success or VT/VF recurrence during follow-up.
314 Fatal outcomes were all related to ARVC-related structural dysfunction, and we found no
315 temporal association between VT recurrence and mortality or HT. These results are in line
316 with a recent multicenter observational study finding no difference in survival in RFCA-
317 versus AAD-treated patients despite a significantly reduced VT burden in the former group²².
318 Also, a prospective study including patients with ES occurring in various forms of
319 cardiomyopathies showed that patients with ES shared many similarities with those
320 presenting severe decompensated heart failure and a comparable prognosis²³. ES clinical
321 presentation and accessibility to RFCA seems more favorable in ARVC than in the overall ES
322 landscape, but eliminating the arrhythmic substrate responsible for ES may have little impact
323 on the progressive structural deterioration. For ARVC patients with advanced disease, ES
324 might be considered an epiphenomenon of disease progression toward end-stage heart failure.

325

326 ***Limitations***

327 The main limitations of our study are its retrospective nature and the long inclusion period.
328 The low number of included patients over a 12-year inclusion period in a specialized center
329 certainly does not reflect the ES incidence in ARVC, data that would have been interesting to

330 report but given the nature of our study, cannot be provided. The patient screening
331 methodology that we used identified patients who underwent VT ablation specifically for ES
332 treatment and not those who were managed medically. A recent retrospective study of 31 ICD
333 carriers reported an annual incidence of ES of 31%²⁴, so the definition of ES itself
334 encompasses diverse and arrhythmia profiles that range from 3 separate episodes of well-
335 tolerated VT to the critical situation of incessant VT, which was the most frequent scenario in
336 our study. The RFCA strategies and techniques were heterogenous, considering that they
337 were performed in 7 different centers over 12 years, beginning in 2003. Much progress has
338 been made in the field of VT ablation over the last decade, including refinement of
339 electroanatomic mapping, substrate identification and epicardial ablation; therefore, our acute
340 and late results might have been improved if RFCA procedures were performed in a
341 contemporary setting. Also, we lack quantitative data regarding ICD data at baseline and
342 during follow-up, which would have been helpful to assess the reduction in VT burden
343 provided by RFCA. Finally, the small study population warrants caution in interpreting
344 descriptive and survival statistics.

345

346 **Conclusions**

347

348 Patients with ARVC experiencing ES exhibited features of structurally advanced disease and
349 frequently had a long history of ventricular arrhythmias, but ES was characterized by
350 relatively good hemodynamic tolerance and single monomorphic VT in most cases. Despite a
351 relatively low complete procedural success rate, RFCA was effective in preventing ES
352 recurrence. Long-term mortality was significant and was associated with the ARVC-related
353 structural dysfunction rather than the arrhythmic outcome. Further studies are needed to
354 define the optimal first-line strategy for ES ablation in the setting of ARVC.

355

356 **Acknowledgments**

357

358 This work was supported by grants from the “Fédération Française de Cardiologie”, “Société
359 Française de Cardiologie” and “Ligue contre la Cardiomyopathie.”

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Figure title and legends

Figure 1. Long-term arrhythmic outcomes in patients with arrhythmogenic right ventricular cardiomyopathy/dysplasia (ARVC) after catheter ablation for electrical storm (ES). A) Kaplan-Meier representation of cumulative probability of freedom from ES recurrence after the initial ES ablation procedure. B) Kaplan-Meier representation of cumulative probability of freedom from ventricular tachycardia (VT) recurrence after the initial ES ablation procedure.

Figure 2. Long-term survival in patients with ARVC after catheter ablation. A, B, C) Kaplan-Meier representation of cumulative probability of survival without death and heart transplantation after the initial ES ablation procedure with and without VT recurrence and LVEF > 50% and \leq 50%. Vertical bars represent the 95% confidence interval of the estimated probability.

Table 1: Characteristics of patients with arrhythmogenic right ventricular cardiomyopathy (ARVC) undergoing catheter ablation as urgent treatment for electrical storm (ES) (n=23)

Age at procedure, years	43.6 ± 16.7
Time from diagnosis to ES, years	3.4 (-4.4–39.2)
Male sex	23 (100)
Definite ARVC diagnosis (TFC)	23 (100)
Pathogenic mutation	
<i>PKP2</i>	6 (26)
<i>DSG2</i>	1 (4)
<i>DSP</i>	1 (4)
<i>PKP2 + DSP VUS</i>	2 (9)
None	5 (12)
Unknown	8 (35)
ECG abnormalities	
Inverted T-waves beyond V2	17 (74)
Epsilon wave	10 (43)
QRS width in V1, ms	108 ± 20
Structural abnormalities	
LVEF	56 (20–70)
LVEF ≤ 50%	7 (30)
RVEF	45 (29–63)
RV systolic dysfunction*	14 (61)
Diffuse RV involvement	16 (70)
ICD before ablation	19 (83)
Previous VT ablation	12 (52)

Anti-arrhythmic drugs

Beta-blocker alone	6 (26)
Flecainide	10 (43)
Amiodarone	4 (17)
Flecainide + sotalol	1 (4)
Sotalol	1 (4)
None	1 (4)

ES clinical tolerance

Syncope	12 (52)
Hemodynamic failure	4 (17)

Data are n (%), median (range) or mean \pm SD.

*: defined by RVEF < 40% with RV angiography or fractional area change < 33% with transthoracic echocardiography.

Table 2: Arrhythmia characteristics and results of electrophysiological study for each ES catheter ablation procedure (n=24)

Clinical VT	
Number of documented VTs	1 (1–2)
>1 documented VT	4 (17)
Clinical VT morphology*	
LBBB, inferior axis	12
LBBB, superior axis	10
RBBB	2
Electrophysiological study and catheter ablation	
Clinical VT cycle length	348 ± 51
>1 induced VT	15 (63)
Use of a 3D electroanatomic mapping system	18 (75)
Number of induced VTs	2 ± 1.36
Number of targeted VTs	1 (1–6)
Number of targeted sites	1 (1–5)
Radiofrequency time, s	790 (180–6150)
Procedure duration, min	180 (120–360)

Data are n (%), median (min-max) or mean ± SD.

