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Arrhythmia-induced cardiomyopathy: a potentially reversible cause of refractory cardiogenic shock requiring veno-arterial Extracorporeal Membrane Oxygenation

Short title: Arrhythmia-induced cardiogenic shock on ECMO

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AC has received lecture fees from GETINGE, Baxter and XENIOS. EG has received consulting fees from MICROPORT, MEDTRONIC, ABBOTT and educational fees from BOSTON. GL has received educational fees from MEDTRONIC, JARVIK and SYNCARDIA and lecture fees from ABBOTT and MEDTRONIC. PL has received a research grant from GETINGE. CEL reports consulting fees from CARMAT, THERMOFISCHER BRAHMS, FARON, BAYER HEALTHCARE and lecture fees from MERCK and BIOMERIEUX. MS has received lecture fees from GETINGE, XENIOS, and DRAGER. The other authors have reported that they have no relationships relative to the contents of this paper to disclose.

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ABSTRACT 250/250:

BACKGROUND: The most severe form of arrhythmia-induced cardiomyopathy in adults—refractory cardiogenic shock requiring mechanical circulatory support—has rarely been reported.

OBJECTIVE: To describe the management of critically ill patients admitted for acute, nonischemic or worsening of previously known cardiac dysfunction and recent-onset supraventricular arrhythmia who developed refractory cardiogenic shock requiring venoarterial-ECMO (VA-ECMO).

METHODS: This study is a retrospective analysis of prospectively collected data.

RESULTS: Between 2004 and 2018, 35 patients received VA-ECMO for acute, nonischemic cardiogenic shock and recent supraventricular arrhythmia (77% atrial fibrillation). Cardiogenic shock was the first disease manifestation in 21 (60%) patients. Characteristics at ECMO implantation (median [IQR]) were: SOFA score: 10 [7–13], inotrope score: 29 [11–80], left ventricular ejection (LVEF) fraction: 10% [10–15] and lactate level: 8

[4–11] mmol/l. For 12 patients, amiodarone and/or electric cardioversion successfully reduced arrhythmia, improved LVEF and enabled weaning-off VA-ECMO; 11 had long-term survival without transplantation or long-term assist device. Eight patients experiencing arrhythmia-reduction failure underwent ablation procedures (7 atrioventricular node with pacing and 1 atrial tachycardia) were weaned-off VA-ECMO; 7 survived. Among the remaining 15 patients without arrhythmia reduction or ablation, only the 6 bridged to heart transplantation or left ventricular assist device survived.

CONCLUSION: Arrhythmia-induced cardiomyopathy, mainly atrial fibrillation-related, is an underrecognized cause of refractory cardiogenic shock, and should be considered in patients with nonischemic cardiogenic shock and recent-onset supraventricular arrhythmia. VA-ECMO support allowed safe arrhythmia reduction or rate control by atrioventricular-node ablation while awaiting recovery, even among those with severe left ventricular dilation.

Keywords: ECMO; Cardiogenic shock; Arrhythmia; Ablation; Atrial fibrillation

INTRODUCTION

Arrhythmias are frequent in patients with heart failure and left ventricular dysfunction, and have long been considered consequences of the cardiomyopathy¹. However, it has been known for several decades that tachyarrhythmia alone can result in reversible nonischemic cardiomyopathy, called arrhythmia-induced cardiomyopathy (AiCM). The risk of developing AiCM depends on the type of arrhythmia, but also its duration and rate. Children are more prone to AiCM, resulting mainly from atrial tachycardia and permanent reentrant tachycardia, while atrial fibrillation (AF) is the most common cause in adults¹. Premature ventricular contraction (PVC) is another recognized cause of AiCM and PVC burden has been shown to impact LVEF². AiCM has been reported in 4% of patients with AF referred for pulmonary vein isolation³, 10–37% of patients with atrial tachycardia referred for radiofrequency ablation⁴, 20–50% of patients with permanent junctional reciprocating tachycardia⁵, and 25%–50% of patients with AF and left ventricular dysfunction undergoing atrioventricular node (AVN) ablation^{6,7}. Furthermore, AiCM occurrence could also worsen any underlying ischemic or idiopathic cardiomyopathy. AiCM should be suspected in any patient with tachyarrhythmia and dilated cardiomyopathy of no obvious etiology; however, cardiomyopathy reversal after arrhythmia reduction is the only way to confirm its diagnosis.

Few data on arrhythmia-induced cardiogenic shock (AiCS) have been published and most focused on children⁵. Furthermore, the potential of reversibility of the most advanced forms, with severely impaired left ventricular function and left ventricular dilation, is questionable. For patients with severe cardiomyopathy requiring circulatory venoarterial-extracorporeal membrane oxygenation (VA-ECMO) support, transplantation is often considered the unique solution for survival⁸. However, some patients with recent AF and cardiogenic shock complicating previously unknown dilated cardiomyopathy or normal hearts may have refractory AiCS and might theoretically recover, at least partially.

Management of adults with suspected AiCS on VA-ECMO has not been described previously. Hence, this retrospective study was undertaken to report our experience managing patients with potential refractory AiCS receiving VA-ECMO support.

METHODS

We retrospectively reviewed the prospectively constituted ECMO database of our 26-bed intensive care unit (ICU), 2004 to 2018, to identify patients given VA-ECMO support for cardiogenic shock who had supraventricular tachycardia at shock onset. Patients with refractory cardiogenic shock complicating ischemic cardiomyopathy, previously known cardiomyopathy with left ventricular ejection fraction (LVEF) <20%, previously known permanent AF/flutter or with paroxysmal/persistent atrial arrhythmia following ECMO implantation were excluded.

DATA COLLECTION. The following information was recorded prospectively: age, sex, severity of underlying condition according to the McCabe & Jackson criteria, medical history, clinical and biological parameters at ICU admission and during the stay. The Sequential Organ Failure Assessment (SOFA) score before ECMO implantation was calculated.

VA-ECMO MANAGEMENT is described in the online supplementary file.

ARRHYTHMIA MANAGEMENT. Electrical arrhythmia cardioversion was attempted with biphasic electrical anterior–posterior shock at maximum energy (first attempt at 150 J, further were performed at 200 J). Right-side AVN catheter ablation was performed through femoral vein access with a 4-mm (Celsius™, Biosense Webster®, Mariner™ MC, Medtronic) or 8-mm nonirrigated radiofrequency deflectable ablation catheter (Blazer II EPT 4500 TK2™, Boston Scientific®). A temporary external ventricular pacemaker was always implanted before AVN ablation during the same procedure through either subclavian or jugular venous access with a screw-in pacemaker lead (CapSureFix Novus 5076™, Medtronic® or Solia™, Biotronik®)

positioned on the right ventricular septum and connected to a temporary external generator. The correct functioning of the external pacemaker was checked before AVN ablation. No other back-up ventricular lead was used during the procedure. Atrial tachycardia ablation was achieved with 3D-mapping (Carto 3, Biosense Webster®) and a 4-mm irrigated-tip contact-force catheter (Smart Touch SF, Biosense Webster®).

OUTCOME VARIABLES. The main outcome variable was in-hospital mortality. Secondary outcomes included LVEF evolution, 90-day mortality, heart transplant, LVAD and long-term survival. Survivors to hospital discharge were contacted by phone 1-year post-refractory AiCS to evaluate their vital status (alive or dead) and time to death, if relevant.

STATISTICAL ANALYSES. Data are expressed as number (percent) for categorical variables or median [interquartile range (IQR)] for continuous variables. Percentages were compared using chi-square or Fisher's exact tests; continuous variables were compared with Student's t- or Wilcoxon's signed-rank tests. Patients' demographic, clinical and biological characteristics were entered into univariable analyses to determine their association with hospital mortality. A p value < 0.05 was considered statistically significant. Analyses were computed with StatView v5.0 (SAS Institute, Cary, NC) and SPSS v22.0 software (SPSS, Chicago, IL).

ETHICS. This study was conducted in accordance with the declaration of Helsinki and the ethical standards of our hospital's institutional review board (Committee for the Protection of Human Subjects). Because this observational study did not modify existing diagnostic or therapeutic strategies, informed consent was not obtained for demographic, physiological and hospital-outcome data analyses. However, patients and/or their relatives were informed about the anonymous data collection and that they could decline inclusion. This database was registered at the Commission Nationale l'Informatique et des Libertés (CNIL, registration no. 1950673).

RESULTS

Between 2004 and 2018, 448 patients were admitted in our ICU for refractory cardiogenic shock with supraventricular arrhythmia requiring VA-ECMO. After exclusion of patients with ischemic cardiomyopathy, previously known cardiomyopathy with LVEF <20%, previously known permanent AF/flutter or with paroxysmal atrial arrhythmia following ECMO implantation, the 35 patients who had received VA-ECMO support for acute nonischemic refractory cardiogenic shock and recent-onset supraventricular arrhythmia were included in our study (Figure 1). Their baseline characteristics are reported in Table 1. Briefly, they were mostly men (77%) and young (median [IQR] age, 48 [39–60] years). Fourteen (40%) patients had known nonischemic dilated cardiomyopathy (DCM) and 10 (29%) known paroxysmal AF. Refractory cardiogenic shock was the first manifestation of AiCM in 21 patients.

ARRHYTHMIA MANAGEMENT. Characteristics of the arrhythmia and its initial management are detailed in Table 2. AF was the main cause of arrhythmia (77% of the cases). Median [IQR] heart rate before ECMO implantation was 150 [140–168] beats/minute. Half the patients were given amiodarone as first-line therapy and only 5 patients received electric shock before ECMO implantation.

DETAILS OF MECHANICAL SUPPORT. Characteristics at ECMO implantation and details regarding ECMO support are given in Table 1. Briefly, median [IQR] SOFA score was 10 [7–13], lactate concentration 8 [4–11] mmol/l, LVEF 10% [10–15%], Ao VTI 6.5 [6–7] cm and end-diastolic left ventricular diameter 60 [57–65] mm. ECMO complications are listed in the online table 1.

OUTCOMES. Patient outcomes are reported in Figure 1 and in the online table 2. Twelve patients had sustained successful reduction after amiodarone and/or electric shock; median

LVEF improved from 12% [10–17%] to 28% [25–40%]; all were weaned-off ECMO and 11 survived without transplantation or long-term LVAD. One patient recovered (LVEF: 30%; Ao VTI: 18) but died of multiorgan failure 40 days after ECMO removal. Median [IQR] times between ECMO implantation and successful sustained reduction, and between successful reduction and ECMO weaning for these 12 patients were 3 [1–5] and 5 [3–8] days, respectively.

Among the 21 patients with amiodarone/electric shock failure to reduce arrhythmia, 7 underwent AVN ablation (6 with temporary or 1 with definitive pacing) while on ECMO, with no complication. One patient with drug-refractory atrial tachycardia underwent 3D activation mapping which identified ectopic focal atrial tachycardia as coming from the right appendage, and underwent successful ablation, with no further recurrence. LVEF in all 8 patients improved from 10% [10–15%] to 28% [17–35%] (Figure 2), all were weaned-off ECMO and 7 survived; 1 patient's LVEF improved to 40% and Ao VTI to 17 cm but he subsequently died of septic shock 54 days after ECMO removal. Median [IQR] time between ECMO implantation and the ablation procedure was 8 [4–11] days.

When considering the 20 patients with successful arrhythmia reduction or AVN/atrial tachycardia ablation procedure, the median [IQR] time between reduction or ablation and ECMO-weaning was 6 [3.5–4.5] days; their median left ventricular end diastolic diameter was 61 [56–65] mm.

Among the remaining 13 patients with failure of arrhythmia control, 7 had previously known heart failure with LVEF at 30% [25–50%] and 3 had known paroxysmal atrial fibrillation. Five had at least 1 failed electrical cardioversion and thrombosis of the left atrium contraindicated electrical cardioversion in 2. Among these 13 patients without reduction or ablation procedure, only the 6 patients bridged—4 to heart transplantation and 2 to LVAD—survived. The 7 deaths of patients who could not be weaned-off ECMO were attributed to

multiorgan failure for 6 and hemorrhage for 1.

COMPARISON BETWEEN SURVIVORS AND NONSURVIVORS. The 24 survivors and 11 nonsurvivors were comparable for age, sex, body-mass index, and Simplified Acute Physiology Score (SAPS) II- and SOFA score-assessed critical illness severity (Table 1). Nonsurvivors were more likely to have comorbidities, especially previously known cardiomyopathy or paroxysmal AF. According to univariate analysis, previously known heart disease, heart rate, renal replacement therapy, prothrombin time, N-terminal prohormone of brain natriuretic peptide level and failed rhythm or rate control were significantly associated with death.

Ninety-day mortality was similar to hospital mortality (24 survivors and 11 nonsurvivors). Median [IQR] long-term follow-up, available for 22 of the 24 survivors, was 300 [158–1417] days. These 22 patients were alive, with and NYHA class 1 or 2 heart failure. Among the 18 patients who recovered and survived without transplantation or LVAD (11 with successful arrhythmia reduction and 7 with successful ablation), median [IQR] LVEF increased from 10% [10–15%] before ECMO implantation to 50% [45–55%] at long-term follow-up (Figure 2).

DISCUSSION

Herein, we described a large series of adults with refractory AiCS benefiting from VA-ECMO support. Among 35 patients with potential AiCS, it was confirmed in 18 by recovery from left ventricular dysfunction after electrical cardioversion restored sinus rhythm or an ablation procedure obtained rate control (with temporary external right ventricular pacing for those undergoing AVN ablation). According to our results, rhythm or rate control obtained with either amiodarone infusion, DC cardioversion or AV node ablation during VA-ECMO support was the most effective treatment.

AiCS has been described almost exclusively in children^{5,9,10}. In those pediatric series, 79%–100% arrhythmias were reentrant supraventricular tachycardia or ectopic atrial tachycardia, while 77% of our adult cases had AF. Thirty-nine children on mechanical circulatory support for AiCS were described¹⁰ in the largest multicentric international series. Their median age was 5.5 months and 61% of them had preexisting cardiac disease (half with congenital heart disease). Their median heart rate was 230 beats/minute, which is much higher than for our population. Nine children underwent successful radiofrequency ablation and 23 survived without transplantation. We found only 2 case reports of successful ECMO management of reentrant tachycardia-induced cardiogenic shock in adults^{11,12} and another on ECMO-rescued refractory cardiac arrest following AF¹³. We show here that AiCS can also be observed in the adult population and is not confined in the paediatric population.

Management of patients with cardiogenic shock and supraventricular arrhythmias is particularly difficult. Inotropic treatment, such as dobutamine, is recommended for cardiogenic shock but may worsen supraventricular tachycardia or accelerate heart rate in these patients. In our series, only half the patients received amiodarone as first-line therapy and only 5 patients underwent electrical cardioversion before ECMO implantation. Those therapeutic choices might be explained for some patients by AF not being considered responsible for cardiogenic shock but only a consequence of terminal DCM. Indeed, study results suggest that AiCM appears to be an underestimated and undertreated cause of left ventricular systolic dysfunction and underline AiCM as a relevant component of idiopathic DCM in most patients with AF¹⁴. Another reason might be that, due to the risk of stroke in patients with undated AF without long-term anticoagulation, clinicians were reluctant to try to restore sinus rhythm. A third explanation might be the risk of worsening circulatory failure or cardiac arrest during electrical cardioversion in these patients with cardiogenic shock.

After ECMO implantation, 13 patients were considered to have terminal

cardiomyopathies without potential recovery and were bridged to transplantation without considering sinus-rhythm restoration. However, it is possible that some of them had AiCM and may have recovered with arrhythmia control. Interestingly, in our study, we observed recovery even in patients with severe left ventricle dilation. All patients with successful reduction experienced significant and sustained LVEF improvement allowing ECMO removal.

In patients with suspected AiCS after failed amiodarone and/or electrical shock reduction, urgent AF ablation might be an option, although it has never reported to our knowledge in patients on VA-ECMO. Indeed, AF ablation might be superior to antiarrhythmic drugs in heart failure patients¹⁵, with an 8%–18% absolute LVEF increase in 60%–70% of patients randomized to ablation^{3,14–17} and even better survival in two studies^{17,18}. We did not use that procedure for the following reasons: first, it was considered dangerous because our patients suffered from severe circulatory failure; second, it can be technically difficult because of the position of the ECMO cannula in the right atrium; third, immediate return to sinus rhythm is more difficult to achieve in patients with heart failure and persistent AF, with a single procedure success-rate range of 29%–61% and a 30% recurrence rate at 24 months of follow-up, as recently reported¹⁸. Therefore, we decided to use a simple strategy, i.e., AVN ablation and temporary external pacing, which allows immediate and definitive rate control and is known to be effective against drug-refractory atrial tachyarrhythmia^{6,7,19,20}.

Our results showed that, for patients with refractory atrial arrhythmia and suspected AiCM, urgent AVN ablation with temporary external right ventricle pacing was safe and always led to hemodynamic improvement allowing ECMO weaning. Because septal right ventricle stimulation has been suggested to be less deleterious on LVEF than apical stimulation²¹, the right ventricle lead was implanted in the septal position in all patients.

However, since the results of the PROTECT-PACE did not show any short term clinical benefit²², the superiority of septal vs. apical pacing remains questionable. For all survivors, temporary external ventricular pacing was successfully converted to definitive cardiac resynchronization therapy in all cases. Recent study results showed the benefit of cardiac resynchronization combined with AVN ablation for patients with permanent AF, narrow QRS and heart failure, compared to medical therapy²³. For patients with less complex arrhythmia, e.g., right atrial flutter or focal atrial tachycardia, first-line catheter ablation may be proposed because the acute success rate of these procedures is high (>90%)²⁴ and may avoid the need for permanent pacing. One of our case-series patients underwent successful focal atrial tachycardia ablation allowing hemodynamic improvement and ECMO weaning.

LIMITATIONS. Our study has several limitations that should be mentioned. First, this was a monocenter study, meaning that extrapolation of our results to other patients in other ICUs could be difficult. The small sample size decreases the power of this study to detect differences between groups and which factors are associated with outcome. However, an adult population with suspected AiCS on VA-ECMO has not been described previously. Second, due to the retrospective design, the management of patients was not formalized. So, despite precise inclusion criteria and exclusion of many patients in order to describe a homogeneous population, it is possible that some factors influencing the management were not taken into account and that patients who underwent ablation or achieved reduction were different from the patients bridged to transplantation. Therefore, comparison between groups should be very cautious. It remains challenging to recognize whether the arrhythmia is the cause or the consequence of the cardiomyopathy. However, we think that some of these patients have true AiCS and that achieving rhythm or rate control is essential. Third, our analysis focused on ECMO-treated patients—not all AiCS patients who were potentially ECMO candidates or not. Some patients may have had AiCS but for several reason(s) (too sick, not sick enough or

ECMO contraindication) might not have been offered ECMO. Those patients were not systematically referred to our ICU and the treatment plan was discussed in other primary-care hospitals. Thus, it is not possible at present to identify which patients, among those with suspected AiCS, could benefit from ECMO. Only a large multicenter registry including all suspected AiCS patients could answer that important question.

In conclusion, AiCM is a possible and probably underrecognized cause of heart failure that may be complicated by refractory cardiogenic shock. AiCM should be suspected in any patient with nonischemic cardiogenic shock and recent-onset supraventricular arrhythmia, mainly AF in adults. VA-ECMO may provide time to wait for recovery after sinus-rhythm restoration, even in patients with severe left ventricular dilation. Aggressive rate control by AVN ablation with ventricular pacing appears to be warranted when reduction fails, and may enable recovery and a favorable outcome.

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REFERENCES

1. Huizar JF, Ellenbogen KA, Tan AY, Kaszala K: Arrhythmia-Induced Cardiomyopathy: JACC State-of-the-Art Review. *J Am Coll Cardiol* 2019; 73:2328–2344.
2. Baman TS, Lange DC, Ilg KJ, et al.: Relationship between burden of premature ventricular complexes and left ventricular function. *Heart Rhythm* 2010; 7:865–869.
3. Gentlesk PJ, Sauer WH, Gerstenfeld EP, et al.: Reversal of left ventricular dysfunction following ablation of atrial fibrillation. *J Cardiovasc Electrophysiol* 2007; 18:9–14.

4. Medi C, Kalman JM, Haqqani H, et al.: Tachycardia-mediated cardiomyopathy secondary to focal atrial tachycardia: long-term outcome after catheter ablation. *J Am Coll Cardiol* 2009; 53:1791–1797.
5. Moore JP, Patel PA, Shannon KM, et al.: Predictors of myocardial recovery in pediatric tachycardia-induced cardiomyopathy. *Heart Rhythm* 2014; 11:1163–1169.
6. Redfield MM, Kay GN, Jenkins LS, Mianulli M, Jensen DN, Ellenbogen KA: Tachycardia-related cardiomyopathy: a common cause of ventricular dysfunction in patients with atrial fibrillation referred for atrioventricular ablation. *Mayo Clin Proc* 2000; 75:790–795.
7. Heinz G, Siostrzonek P, Kreiner G, Gössinger H: Improvement in left ventricular systolic function after successful radiofrequency His bundle ablation for drug refractory, chronic atrial fibrillation and recurrent atrial flutter. *Am J Cardiol* 1992; 69:489–492.
8. Dangers L, Bréchet N, Schmidt M, et al.: Extracorporeal Membrane Oxygenation for Acute Decompensated Heart Failure. *Crit Care Med* 2017; 45:1359–1366.
9. Salerno JC, Seslar SP, Chun TUH, et al.: Predictors of ECMO support in infants with tachycardia-induced cardiomyopathy. *Pediatr Cardiol* 2011; 32:754–758.
10. Silva JNA, Erickson CC, Carter CD, et al.: Management of pediatric tachyarrhythmias on mechanical support. *Circ Arrhythm Electrophysiol* 2014; 7:658–663.
11. Cheruvu C, Walker B, Kuchar D, Subbiah RN: Successful ablation of incessant AV reentrant tachycardia in a patient on extracorporeal membrane oxygenation. *Heart Lung Circ* 2014; 23:e12-15.
12. Magro P, Calquinha J, Adragão P, Neves J: Extracorporeal membrane oxygenator assistance as bridge to recovery in a case of tachycardiomyopathy. *Rev Port Cir Cardio-Torac E Vasc Orgao Of Soc Port Cir Cardio-Torac E Vasc* 2015; 22:211–213.
13. Lee JW, Ahn HJ, Yoo YH, Lee JW, Kim SW, Choi SW: Extracorporeal CPR and intra-aortic balloon pumping in tachycardia-induced cardiomyopathy complicating cardiac arrest. *Am J Emerg Med* 2017; 35:1208.e5-1208.e7.
14. Prabhu S, Taylor AJ, Costello BT, et al.: Catheter Ablation Versus Medical Rate Control in Atrial Fibrillation and Systolic Dysfunction: The CAMERA-MRI Study. *J Am Coll Cardiol* 2017; 70:1949–1961.
15. Trulock KM, Narayan SM, Piccini JP: Rhythm control in heart failure patients with atrial fibrillation: contemporary challenges including the role of ablation. *J Am Coll Cardiol* 2014; 64:710–721.
16. Chen MS, Marrouche NF, Khaykin Y, et al.: Pulmonary vein isolation for the treatment of atrial fibrillation in patients with impaired systolic function. *J Am Coll Cardiol* 2004; 43:1004–1009.
17. Marrouche NF, Brachmann J, Andresen D, et al.: Catheter Ablation for Atrial Fibrillation with Heart Failure. *N Engl J Med* 2018; 378:417–427.

18. Di Biase L, Mohanty P, Mohanty S, et al.: Ablation Versus Amiodarone for Treatment of Persistent Atrial Fibrillation in Patients With Congestive Heart Failure and an Implanted Device: Results From the AATAC Multicenter Randomized Trial. *Circulation* 2016; 133:1637–1644.
19. Manolis AG, Katsivas AG, Lazaris EE, Vassilopoulos CV, Louvros NE: Ventricular performance and quality of life in patients who underwent radiofrequency AV junction ablation and permanent pacemaker implantation due to medically refractory atrial tachyarrhythmias. *J Interv Card Electrophysiol Int J Arrhythm Pacing* 1998; 2:71–76.
20. Brignole M, Gianfranchi L, Menozzi C, et al.: Influence of atrioventricular junction radiofrequency ablation in patients with chronic atrial fibrillation and flutter on quality of life and cardiac performance. *Am J Cardiol* 1994; 74:242–246.
21. Shimony A, Eisenberg MJ, Filion KB, Amit G: Beneficial effects of right ventricular non-apical vs. apical pacing: a systematic review and meta-analysis of randomized-controlled trials. *Eur Eur Pacing Arrhythm Card Electrophysiol J Work Groups Card Pacing Arrhythm Card Cell Electrophysiol Eur Soc Cardiol* 2012; 14:81–91.
22. Kaye GC, Linker NJ, Marwick TH, et al.: Effect of right ventricular pacing lead site on left ventricular function in patients with high-grade atrioventricular block: results of the Protect-Pace study. *Eur Heart J* 2015; 36:856–862.
23. Brignole M, Pokushalov E, Pentimalli F, et al.: A randomized controlled trial of atrioventricular junction ablation and cardiac resynchronization therapy in patients with permanent atrial fibrillation and narrow QRS. *Eur Heart J* 2018; 39:3999–4008.
24. Brugada J, Katritsis DG, Arbelo E, et al.: 2019 ESC Guidelines for the management of patients with supraventricular tachycardia. The Task Force for the management of patients with supraventricular tachycardia of the European Society of Cardiology (ESC). *Eur Heart J* 2019; .

Figure legends

FIGURE 1. Study Flow Chart

AVN = atrioventricular node; ECMO = extracorporeal membrane oxygenation; ICU = intensive care unit; LVAD = left ventricular assist device. *Survivors without heart transplantation or LVAD. †Survivor with heart transplantation.

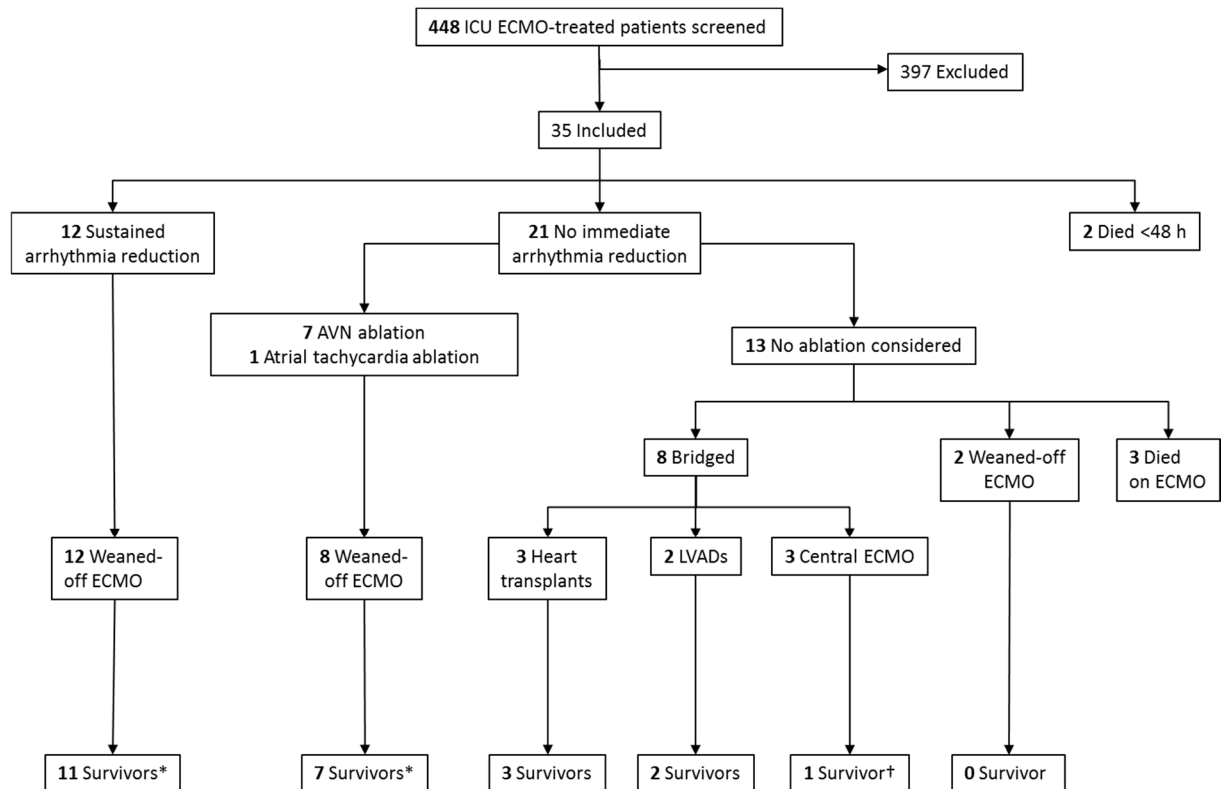


FIGURE 2. Left Ventricular Ejection Fraction (LVEF) of Survivors without Transplantation or Left Ventricular Assist Device

Blue: patients with succesful arrhythmia reduction, red: patients who underwent an ablation procedure.

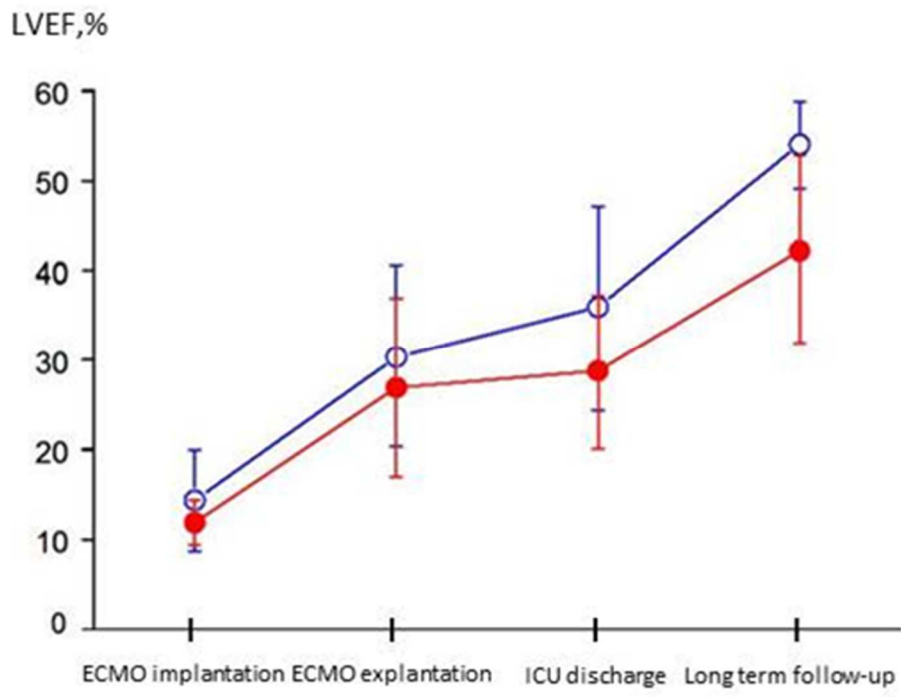


TABLE 1 Patient Characteristics at Baseline, before and at ECMO Start as a Function of Outcome.

Characteristic	All Patients n = 35	Survivors n = 24	Nonsurvivors n = 11	p Value
At baseline				
Age, yrs	48 [39–60]	47 [39–53]	59 [41–65]	0.21
Male sex	27 (77)	20 (83)	7 (64)	0.36
Body-mass index	26 [23–29]	27 [23–29]	26 [25–31]	0.56
Known nonischemic cardiomyopathy	14 (40)	5 (21)	9 (82)	0.002
LVEF*	30 [30–49]	45 [35–60]	30 [25–0]	0.11
Known paroxysmal atrial fibrillation	10 (29)	4 (17)	6 (55)	0.04
Ongoing treatment				
Beta-blocker	14 (40)	6 (25)	8 (73)	0.01
ACE inhibitor or angiotensin II receptor antagonist	12 (34)	3 (13)	9 (82)	0.0001
Antiarrhythmia drugs	9 (26)	3 (13)	6 (55)	0.01
ICU admission SOFA score	14 [9–16]	14 [12–16]	11 [7–17]	0.97
ICU admission SAPS II	60 [34–73]	60 [35–72]	58 [33–74]	0.93
McCabe & Jackson score	1 [0–2]	0 [0–1.5]	2 [1–2]	0.01
3	3 (9)	1 (4)	2 (18)	0.03
1 or 2	18 (51)	10 (42)	8 (73)	
0	14 (40)	13 (54)	1 (9)	

Before ECMO implantation

SOFA score	10 [7–13]	10 [7–12]	12 [8–14]	0.29
Inotrope score	29 [11–80]	20 [12–63]	67 [11–173]	0.29
Heart rate, beats/min	150 [140–168]	158 [140–175]	140 [120–150]	0.01
Lactate, mmol/l	7.9 [4.3–11.3]	8 [4.3–11.4]	7.2 [4.1–10.5]	0.61
pH	7.28 [7.20–7.37]	7.31 [7.21–7.35]	7.26 [7.15–7.38]	0.79
PaO ₂ /FiO ₂ , mm Hg	360 [250–400]	371 [184–418]	350 [296–400]	0.96
Bilirubin μmol/l	46 [18–64]	41 [18–54]	46 [20–86]	0.28
Alanine aminotransferase, IU/l	637 [98–1505]	655 [100–1455]	565 [115–1869]	0.97
Aspartate aminotransferase, IU/l	449 [120–1799]	458 [126–1675]	412 [120–1773]	0.83
Prothrombin time, %	35 [25–59]	45 [29–61]	20 [18–24]	0.008
Serum creatinine, mg/dl	1.61 [1.30–2.18]	1.63 [1.31–2.17]	1.57 [1.24–2.26]	0.99
NT Pro BNP, pg/ml	8503 [3283–16899]	4741 [2521–8503]	20019 [14250–34534]	0.0005
Troponin I, pg/mL	67 [23–115]	43 [16–79]	97 [74–215]	0.24
Pre-ECMO echocardiography				
LVEF, %	10 [10–15]	10 [10–15]	10 [10–15]	0.83

Ao VTI, cm	6.5 [6– 7]	6.5 [6–7]	6.5 [6–7]	0.99
LVEDD, mm	60 [57–65]	59 [56–64]	62 [60–68]	0.08
At ECMO implantation				
Days from shock onset to ECMO	1 [0–2]	0.5 [0–1.5]	1 [0–2]	0.8
ECMO under CPR	1 (3)	1 (4)	0	0.99
Cardiac arrest before ECMO	3 (9)	3 (13)	0	0.54
ECMO implanted by mobile team	17 (49)	13 (54)	4 (36)	0.47
Intra-aortic balloon pump	18 (51)	15 (63)	3 (27)	0.07
Percutaneous/surgical ECMO	14/21	11/13	3/8	0.46

Results are expressed as n (%) or median [IQR], unless stated otherwise. Ao VTI = aortic velocity time integral; CPR = cardiopulmonary resuscitation; ECMO = extracorporeal membrane oxygenation; ICU = intensive care unit; LVEDD = left ventricular end diastolic diameter; LVEF = left ventricular ejection fraction; NT Pro BNP = N-terminal prohormone of brain natriuretic peptide; PaO₂/FiO₂ = partial oxygen pressure in arterial blood/fraction of inspired oxygen; SAPS II = Simplified Acute Physiology Score; SOFA = Sequential Organ Failure Assessment.

*For the 14 patients for whom it was available.

Table 2 Characteristics and Arrhythmia Treatment

	All Patients	Survivors	Nonsurvivors	
Characteristics	n = 35	n = 24	n = 11	p Value
Type of supraventricular arrhythmia				
Atrial fibrillation	27 (77)	19 (79)	8 (73)	0.69
Atrial flutter	3 (9)	1 (4)	2 (18)	0.23
Atrial tachycardia	2 (6)	2 (8)	0	0.99
Junctional tachycardia	3 (9)	2 (8)	1 (9)	0.99
Preexisting symptoms				
Palpitation	16 (46)	14 (58)	2 (8)	0.04
Duration of palpitations before admission	8 [4–15]	7 [4–26]	9 [2–13]	0.64
Dyspnea	35 (100)	24 (100)	11 (100)	
Treatment before ECMO implantation				
Electrical cardioversion	5 (14)	5 (21)	0	0.16
Beta-blockers	11 (31)	8 (33)	3 (27)	0.99
Digoxin	11 (31)	9 (38)	2 (18)	0.44

Amiodarone	18 (51)	13 (54)	5 (46)	0.72
Atrial thrombosis*	5/17	3/13	2/4	
Treatment after ECMO implantation				
Electrical cardioversion	20 (57)	15 (63)	5 (45)	0.47
Number of electrical cardioversions	3 [2–4]	3 [2–4]	3 [3–8]	0.3
Amiodarone load	30 (86)	22 (92)	8 (73)	0.09
Total amiodarone dose, mg	4100 [825–7550]	4650 [1400–7800]	1200 [300–7725]	0.007
Results of arrhythmia management				
Sustained sinus rhythm after amiodarone				
load and/or electric shock	12 (34)	11 (46)	1 (9)	
Successful ablation procedure	8 (23)	7 (29)	1 (9)	
Failure of arrhythmia or rate control	15 (43)	6 (25)	9 (82)	

Results are expressed as n (%) or median [IQR]. ECMO = extracorporeal membrane oxygenation.

*Number of thromboses seen on transesophageal echocardiography (TEE) over the number of TEE performed. TEE was not done in 15 patients: 8 survivors and 7 nonsurvivors