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Letter to the Editor

An underestimated tool for both cooling and circulatory support in cardiac arrest survivors developing severe hyperthermia

Dear Editor,

The onset of severe hyperthermia in the early phase following cardiac arrest (CA) increases dramatically the risk for adverse neurological outcomes and mortality [1]. However, the management of this catastrophic complication still remains challenging. Herein we wish to communicate our clinical experience of continuous venovenous hemodiafiltration (CVVHDF) as an efficient and safe cooling method in treating CA patients with severely compromised hemodynamics manifesting uncontrolled fever. We would also like to highlight the "pleiotropic" beneficial effects that CVVHDF may exhibit on several cerebral, circulatory, pulmonary, renal and inflammatory pathways in the acute phase post-CA.

Four men who survived from CA due to ventricular fibrillation (age = 63.25 ± 2.63; in-hospital/out-of-hospital CA = 2/2; STEMI/NSTEMI = 3/1) underwent percutaneous revascularization of the infarct-related artery (IRA) and the non-IRA significant stenoses in multivessel disease. The common characteristic of our cases was that all of them developed intractable fever (38.9–40.2 °C) and received CVVHDF renal replacement therapy (RRT) as a cooling method early (8–19 h) post-resuscitation. The rationale was to take advantage of CVVHDF-induced hypothermia, a well-known side effect of the technique [2]. Notably, literature data regarding the possible beneficial effect of CVVHDF in CA patients without renal failure (like our ones) is extremely sparse [3]. We also aimed at counteracting pulmonary congestion, problematic oxygenation and severe acidosis ensued post-CA. Additionally, this method was considered ideal for our hemodynamically unstable patients, as CVVHDF is characterized by gradual fluid and solute removal instead of large osmotic shifts [4].

Clinical and echocardiographic data before and after CVVHDF application are demonstrated in Table 1. First, temperature control (≤37.2 °C) was efficient and tight throughout CVVHDF sessions, while side-effects were not recorded. Second, CVVHDF was associated with (i) cardiac output increase [as assessed by ultrasound-derived velocity time integral (VTI) in left ventricular outflow track] despite intensive fluid removal (≥100 mL/h) [5], (ii) blood pressure increase and vasopressor-dose de-escalation, (iii) central venous pressure (CVP) decrease, (iv) venous decongestion (reduction in inferior vena cava diameter and increase in its respiratory fluctuation in echocardiography) and (v) pulmonary edema decrease (marked reduction in B-lines in lung ultrasonography) [6]. We hypothesized a "type of positive inotropic effect", as CVVHDF seems to transpose Frank-Starling's cardiac performance curve upward and leftward (towards lower CVP and higher VTI values). Third, CVVHDF was associated with significant improvement in diuresis, acidosis and lacticemia. Both increased cardiac output/renal perfusion and decreased CVP/renal vein congestion may have preserved renal function [2,7]. Foremost, CVVHDF was associated with favorable neurological outcomes [(Glasgow Coma Scale of 15 in 4/4 patients (100%)] and increased survival overall (3/4;75%); possible mediating mechanisms include (i) successful thermoregulation, (ii) maintenance of the stability of hemodynamic/cerebral coupling and cerebral autoregulation [4,8], (iii) reduction of cerebral congestion/edema through CVP decrease [9], and (iv) attenuation of inflammatory mediators [10].

Our report indicates that CVVHDF may represent an underrated weapon in our armamentarium not only for cooling but also for hemodynamic support and beyond-cooling neuroprotection in febrile CA survivors in circulatory collapse. The pleiotropic benefits of CVVHDF suggested herein remain to be elucidated in larger-scale studies in the future.

Competing Interests.

Nothing to disclose.

Acknowledgments

Nothing to disclose.

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Ethics approval

This is an observational study. The Research Ethics Committee has confirmed that no ethical approval is required in view of the retrospective nature of the study and all the procedures being performed were part of the routine care.

Table 1 – Patients' clinical characteristics and echocardiographic measurements before and after CVVHDF.

	Before	After	Difference between before and after CVVHDF (95% CI) [a]	*P* value
Clinical parameters				
Temperature (°C)	39.45 ± 0.56	36.77 ± 0.43	2.68 (1.54 to 3.81)	**0.005**
Mean Blood Pressure, mmHg	59 ± 7.9	86.3 ± 8.5	−27.25 (−43.55 to −10.95)	**0.013**
Noradrenaline (μg/kg/min)	0.843 ± 0.427	0.026 ± 0.023	0.816 (0.16 to 1.48)	**0.029**
Heart Rate (bpm)	118 ± 9.93	81.75 ± 5.91	36.2 (13.14 to 59.36)	**0.015**
Urine Output, mL/hour	27.5 ± 10.4	143.7 ± 14.9	−116.2 (−150.2 to −82.3)	**0.002**
Lactate, mmol/L	9.07 ± 2.29	1.02 ± 0.26	8.05 (4.1 to 11.95)	**0.007**
Serum creatinine, mg/dL	1.525 ± 0.15	1.4 ± 0.346	0.125 (−0.251 to 0.5)	0.166
pH	7.132 ± 0.105	7.455 ± 0.046	−0.32 (−0.54 to −0.11)	**0.018**
CVP, mmHg	17.75 ± 3.3	6.75 ± 3.5	11 (2.09 to 19.9)	**0.029**
Echocardiographic parameters				
LVEF, %	33 ± 6.27	46.25 ± 15.48	−13.25 (−29.6 to 3.1)	0.107
VTI, cm	9.25 ± 1.71	16.5 ± 2.65	−7.25 (−10.26 to −4.24)	**0.005**
B-lines on lung ultrasound	37.5 ± 11.5	13 ± 4.4	24.5 (1.24 to 47.76)	**0.044**
IVCd, cm	3.17 ± 0.22	2.5 ± 0.34	0.67 (0.47 to 0.87)	**0.002**
IVCrf, %	7.5 ± 2.5	45.75 ± 9	−38.25 (−54.08 to −22.42)	**0.005**

Numerical data are expressed as mean ± standard deviation. Differences between pre- and post-CVVHDF values are examined by paired t-test (a P-value less than 0.05 is considered statistically significant).

CVVHDF = continuous venovenous hemodiafiltration; CI = confidence interval; CVP = central venous pressure; LVEF = left ventricular ejection fraction; VTI velocity time integral; IVCd = diameter of inferior vena cava; IVCrf = respiratory fluctuation of inferior vena cava.

[a] Data are expressed as mean difference [95% confidence interval (CI)].

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Abbreviations: CVVHDF, continuous venovenous hemodiafiltration

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