

In conclusion, we believe that the most important consideration in acute myocarditis is support measures in the initial phases. Some patients required ventricular assistance, and this was not associated with poor prognosis in the long term. With regard to diagnosis, magnetic resonance imaging is a very useful technique. The main challenge is to obtain high-quality images in younger patients (neonates and infants). Endomyocardial biopsy should only be used in patients with deterioration. With regard to prognosis, right ventricular involvement may be more common in neonates, as they have a marked pulmonary vascular reactivity. In these patients, right-ventricular dysfunction may be secondary to pulmonary hypertension and does not necessarily indicate poor prognosis.

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Ventricular Support With Extracorporeal Membrane Oxygenation: A New Rescue Alternative for Refractory Cardiogenic Shock

Asistencia ventricular con membrana de oxigenación extracorpórea: una nueva alternativa al rescate del shock cardiogénico refractario

To the Editor,

In recent years, ventricular support devices have emerged as a rescue option for patients with cardiac arrest or refractory cardiogenic shock.¹ Ventricular support with venoarterial extracorporeal membrane oxygenation (VA-ECMO) enables a less invasive percutaneous approach, biventricular support, and better

Table

Baseline Characteristics, Hemodynamic Status, Indications for Ventricular Support With Venous Arterial Extracorporeal Membrane Oxygenation, and Clinical Evolution

| Sex | Age | Diagnosis | Duration ECMO | LVEF | Lactate | Baseline creatinine | INTERMACS value | Initial plan | Evolution |
|--------|-----|--|---------------|------|---------|---------------------|-----------------|--------------|-----------|
| Female | 56 | Valvular cardiomyopathy | 6 | 13 | 4.3 | 256 | 1 | CT | Death |
| Male | 54 | Valvular cardiomyopathy | 2 | 30 | | 159 | 2 | CT | CT |
| Male | 59 | Anterior AMI Killip IV | 6 | 15 | 1.7 | 255 | 1 | CT | CT |
| Female | 55 | Pheochromocytoma | 8 | 33 | 3.9 | 380 | 1 | Recovery | Weaned |
| Male | 64 | Anterior AMI, VSD | 10 | 30 | 2.9 | 193 | 1 | CT | Death |
| Male | 66 | NSTEACS Killip IV | 8 | 25 | 10.6 | 215 | 1 | Decision | Death |
| Female | 67 | Valvular cardiomyopathy | 10 | 55 | 2.1 | 208 | 1 | CT | Death |
| Male | 34 | Fulminant myocarditis | 1 | 15 | 8.5 | 137 | 1 | Decision | Death |
| Female | 32 | Dilated cardiomyopathy, CRA | 1 | 30 | 10.4 | 140 | 1 | Decision | Weaned |
| Male | 52 | Ischemic cardiomyopathy, CRA | 9 | 21 | 15.9 | 191 | 1 | CT | CT |
| Male | 52 | Pheochromocytoma, CRA | 1 | 25 | 18.9 | 224 | 1 | Decision | Death |
| Male | 53 | Ischemic cardiomyopathy, AMI Killip IV, SI | 8 | 10 | 2.5 | 195 | 1 | Decision | Weaned |
| Male | 68 | Ischemic cardiomyopathy | 9 | 20 | 12.8 | 240 | 1 | CT | CT |
| Male | 46 | AMI Killip IV, arrhythmic storm | 4 | 10 | 7.8 | 125 | 1 | Recovery | Weaned |
| Female | 50 | AMI Killip IV | 2 | 20 | 4.2 | 149 | 1 | Recovery | Death |
| Male | 65 | AMI no Q Killip IV | 11 | 30 | 2.9 | 162 | 2 | CT | Death |

AMI, acute myocardial infarction; CRA, cardiorespiratory arrest; CT, cardiac transplant; ECMO, venoarterial extracorporeal membrane oxygenation; LVEF, left ventricular ejection fraction; NSTEACS, non-ST elevation acute coronary syndrome; SI, surgical intervention; VSD, ventricular septal defect.

tissue oxygenation; hence, it can be used as a bridge to decision for patients with severe multiorgan failure in whom a complete assessment cannot be carried out.² The published experience with adults in Spain is scant^{3,4} and most of the reported cases are patients who have undergone cardiectomy.⁵ The lack of randomized studies, small sample sizes, and heterogeneity of the published series (most with elevated mortality²) make it difficult to precisely define the profile of the ideal patient for VA-ECMO.

Our objective was to analyze the baseline characteristics, indications, and duration of VA-ECMO in patients undergoing this procedure in our coronary unit, as well as the management and evolution of these patients during hospitalization and follow-up.

Between December 2009 and October 2012, 16 patients underwent VA-ECMO in our center. A Quadrox-D oxygenator system (Maquet; Wayne, New Jersey, United States) and a Jostra RotaFlow centrifugal pump (Maquet) were used in all patients, except for one case in which a CentriMag centrifugal pump (Thoratec; Pleasanton, California, United States) was used.

The characteristics of the patients are shown in the Table. Mean age was 54.6 years and 11 of the 16 patients (68.7%) were men. The main indication was cardiogenic shock, secondary to dilated cardiomyopathy (7/16, 43.7%), followed by myocardial infarction (6/16, 37.5%) and myocarditis (3/16, 18.7%). Four patients had experienced prolonged cardiorespiratory arrest previously.

Most of the patients (14/16, 87.5%) were in catastrophic hemodynamic collapse when admitted (INTERMACS⁶ category 1), despite maximum inotropic and vasoconstrictor treatment.

Half the cases were referred from third-level centers that did not have a cardiac transplantation unit. All patients required invasive mechanical ventilation. A Swan-Ganz catheter was used in 68.8% of the patients, intra-aortic counterpulsation in 75%, and renal replacement therapy in 31%.

In 50% of cases, a bridge to cardiac transplantation was initially contemplated, in 18.7%, a bridge to possible recovery, and in the remaining 31.3% a bridge to decision, pending the clinical evolution.

Nine of the 16 patients presented clinically relevant hemorrhage (hemodynamic deterioration, intervention required, or life-threatening) and 9 patients developed infections that required intravenous antibiotic therapy.

Successful weaning from VA-ECMO was achieved in 4 (25%) cases, and heart transplantation was performed in 4 others (25%). The remaining 8 patients died during VA-ECMO support. In 2 patients who achieved a degree of stability, VA-ECMO was changed to a CentriMag central biventricular assist device of medium-term duration ("bridge-to-bridge" strategy). One transplant recipient died of infection, and one patient moved to medium-term assistance died while receiving this support. In-hospital mortality was 62.5%. The progression of mortality is shown in the Figure. The main cause of death was sepsis (5/10, 50%), followed by hemorrhage (4/10, 40%) and refractory lactic acidosis, secondary to prolonged arrest in the remaining case. The 6 patients surviving hospitalization were alive at the last follow-up (mean, 441 days).

Our results show elevated mortality, which is in keeping with the rates reported in most published series.² In light of the extreme severity of the patients' condition and the fact that this was our initial experience, the results can be considered reasonable. The documented requirement for invasive procedures and complication rates make proper case selection especially important. It is recommended to reserve this technique for potential heart transplant candidates or cases of potentially reversible acute heart disease in patients younger than 70 years.

The main limitation of this study is that it is a single-center registry with a small number of patients. We would like to

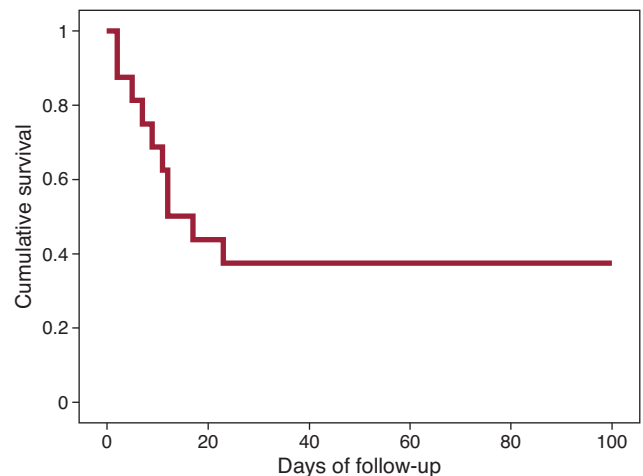


Figure. Progression of mortality in the cohort.

highlight the importance of creating a systematic multicenter registry of cases in the future, which would provide a larger sample size and more effective use of the data.

In conclusion, the use of VA-ECMO led to rescue of 38% of patients in catastrophic hemodynamic collapse without other therapeutic options. We believe that the introduction of VA-ECMO in coronary units with a sufficient volume of highly complex patients would be a notable step ahead in the management of critically ill patients with advanced heart failure.

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Efficacy of Tolvaptan in Patients Hospitalized for Heart Failure With Refractory Hyponatremia. Clinical Experience in Daily Practice

Eficacia de tolvaptán en pacientes ingresados por insuficiencia cardíaca e hiponatremia refractaria. Experiencia en la práctica clínica diaria

To the Editor,

Hyponatremia (plasma sodium ≤ 135 mEq/L) is one of the most common electrolyte abnormalities in patients with acute heart failure (HF) and is considered a marker of poor prognosis.¹

The latest European Society of Cardiology, Guidelines for HF included tolvaptan, a vasopressin V_2 -receptor blocker that inhibits free water reabsorption,² as a valid treatment for patients with refractory hyponatremia.³

We present our experience with tolvaptan for the treatment of refractory hyponatremia in patients admitted by HF.

A retrospective study was conducted in patients treated with tolvaptan who were admitted for HF between February 2011 and August 2012 with refractory hyponatremia (sodium < 135 mEq/L despite "classic" treatment, mainly fluid intake restrictions and/or administration of hypertonic saline solution) and persistent symptoms of HF.

Plasma sodium, potassium, and creatinine concentrations, glomerular filtration rate (calculated by the Modification of Diet in Renal Disease equation), weight, and excretion rhythm at the start of tolvaptan therapy were assessed 24 h and 48 h after tolvaptan was given.

The ESTATA/SE 11.1 software package was used for the statistical analysis.

A total of 30 patients (57% women; mean age, 72 ± 14 years) were included. The most common cause of HF (33% of all patients) was ischemic heart disease; 54% of patients presented ventricular dysfunction (mean ejection fraction, $48\% \pm 16\%$). All were following optimal treatment for HF and all were receiving diuretic therapy at home.

Treatment was started at a daily dose of 15 mg of tolvaptan in 90% of patients and 30 mg in all others. At the start of treatment, sodium was 129 ± 3 mEq/L. Natremia was significantly increased at 24 h, an effect that persisted at 48 h (129 ± 3 mEq/L at baseline; 134 ± 3 mEq/L at 24 h; 135 ± 3 mEq/L at 48 h; $P < .001$) (Fig. 1). No significant changes were observed in potassium concentrations after the drug was administered (4 ± 0.5 mEq/L at baseline; 4 ± 0.4 mEq/L at 24 h; 4.2 ± 0.4 mEq/L at 48 h; $P > .05$).

After treatment, diuresis was significantly increased at 24 h, and the effect was maintained at 48 h (80 ± 45 mL/h, 138 ± 60 mL/h, and 136 ± 64 mL/h, respectively; $P < .001$) (Fig. 2). Likewise, a significant decrease in patient weight was observed at 48 h (67.1 ± 17.8 kg vs 64.1 ± 15.1 kg; $P = .01$).

No statistically significant differences were observed in creatinine (1.3 ± 0.5 mg/dL at baseline; 1.4 ± 0.4 mg/dL at 24 h; 1.6 ± 1.1 mg/dL at 48 h; $P > .05$) or Modification of Diet in Renal Disease (54 ± 20 , 50 ± 16 , and 50 ± 18 mL/min/1.73 m², respectively; $P > .05$).

Patients who presented moderate-to-severe hyponatremia (sodium < 130 mEq/L) showed a larger post-treatment increase in natremia and excretion rhythm than those who presented mild

hyponatremia. These results were similar in patients with ventricular dysfunction or HF with preserved systolic function.

The only adverse event occurred in a patient with no known renal impairment who experienced acute renal failure while receiving treatment and it resolved without dialysis.

In patients with HF, a significant relationship between natremia and in-hospital mortality was observed, along with an increase in readmissions and long-term morbidity and mortality.^{1,2}

Various mechanisms promote this hyponatremia: increased vasopressin due to low cardiac output and decreased renal blood flow, increased perception of thirst, and the use of diuretics.

In patients admitted to our hospital for HF and refractory hyponatremia, we observed a significant increase in the natremia after tolvaptan therapy. Additionally, patients with lower sodium were those who most benefited from treatment, as shown in other studies.⁴⁻⁶ Our study also observed a significant increase in the excretion rhythm and a decrease in weight without significant decline in kidney function or potassium levels,

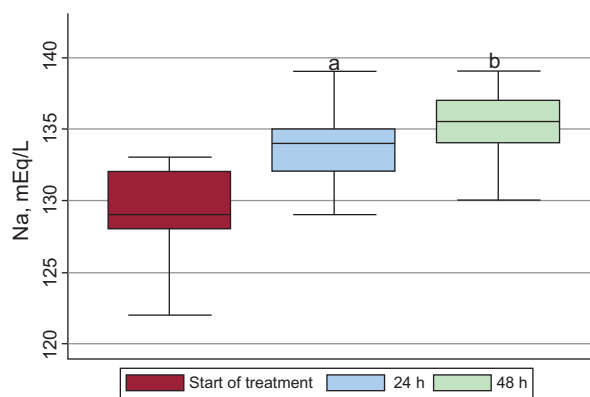


Figure 1. Course of natremia after tolvaptan therapy in patients with acute heart failure and refractory hyponatremia. ^a $P < .05$ (natremia at the start of treatment vs natremia at 24 h). ^b $P < .05$ (natremia at 24 h vs natremia at 48 h).

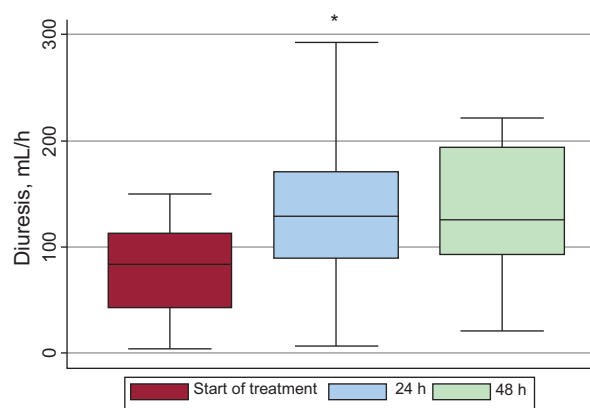


Figure 2. Course of diuresis after tolvaptan therapy in patients with acute heart failure and refractory hyponatremia. ^{*} $P < .05$ (diuresis at the start of treatment vs diuresis at 24 h).