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Early hemodynamic improvement is a prognostic marker in patients treated with continuous CVVHDF for acute renal failure

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Key words

CRRT. Hemodynamics. Outcome.

Abstract

We examined whether hemodynamic improvement after high flow hemofiltration predicts survival in patients treated with standard continuous renal replacement therapy (CRRT).

This was a prospective observational cohorts study of 169 patients, measuring the mean arterial pressure (MAP) and norepinephrine (NE) dosage before and 24 hours after CRRT. Responders (Rs) were defined as having a 20% reduction in NE dosage or a 20% rise in MAP with no increase in NE, and compared with non-responders (NRs). Patients were considered to be unstable if they were receiving NE or their MAP was lower than 60 mmHg prior to CRRT.

169 patients, 68% male, mean age: 53.8 (52.7-54.9) with a mean APACHE II at admission of 21.8 (21.2-22.3), of whom 114 were unstable at the start of CRRT. Overall mortality 15 days after the end of CRRT was 54.3% (57.7% in stable vs. 52.9% in unstable patients, P=NS). There were 99 Rs and 70 NRs, the only differences being NE dosage (higher in Rs, $p<0.01$) and mortality (Rs 30% vs. NRs 74.7%, $p<0.001$). In unstable patients, mortality was 30% in Rs vs 87% in NRs ($p<0.001$) (72% sensitivity and 86% specificity for predicting mortality). Logistic regression analysis showed the only variables associated with mortality were APACHE II at admission (OR: 1.06, 95% CI 1.0-1.12), percent creatinine decrease (OR: 0.98, CI 0.96-1.0) and lack of hemodynamic response to CRRT (OR: 7.04, CI 3.3-15.02).

Hemodynamic improvement after 24 hours CRRT is a strong predictor of survival.

Introduction

Continuous renal replacement therapy (CRRT) has gained popularity in the ICU as the preferred method for managing acute renal failure (ARF) when renal replacement therapy (RRT) is needed.[1-3] CRRT is preferable to intermittent hemodialysis because of the hemodynamic tolerance shown by even the most critically ill patients when treated with continuous techniques.[4]

Because CRRT is mostly used in unstable ICU patients, the mortality rate in this group is high and some concern has been raised over its cost.[5] Although many attempts have been made to determine prognostic factors that could aid in the decision regarding whether to start CRRT, the results have been poor, and widely used scores for critically ill patients and for acute renal failure patients have not proven not as valid in this population.[6-10] A possible explanation for the lack of success at predicting outcome with CRRT could be the low doses prescribed in earlier years and the varying patterns of use by the different centers.

Hemodynamics and respiratory parameters improve with higher doses of hemofiltration, and these higher doses may also have an impact on prognosis.[11] Recent studies on the use of high-flow hemofiltration for the management of septic patients have shown that early hemodynamic improvement after initiation of this therapy predicts a high rate of survival,[12] but earlier reports using standard hemofiltration had shown this effect as well.[13]

In a group of patients treated with a unified protocol and a dosage based on recent standards,[11] we attempted to determine whether patients treated with CRRT who have hemodynamic improvement within 24 hours of starting treatment have a better prognosis than those who do not respond within this time.

Methods

The study was undertaken in a 42-bed polyvalent ICU in a third level teaching hospital in southern Spain from January 2001 to December 2004. The study was based on a prospective

registry applied to all patients treated with CRRT in our Unit. This registry has been in use for ten years. We designed, based in this registry (adapted specifically for this purpose), a prospective observational cohorts study. We recorded age, gender, diagnosis, date of hospital and ICU admission, date of ARF diagnosis, APACHE II on admission and CRRT initiation, indication for treatment, modality and fluid used, anticoagulation regime, mean hourly dosage of convective + diffusive treatment (taking into consideration losses), vascular access, complications, duration of therapies, indication for ending treatment, and outcome. Analytical data (creatinine, blood urea, coagulation status and platelets) were recorded daily. In 2001 we added to the registry data concerning mean arterial pressure (MAP) and norepinephrine (NE) use at the start of CRRT and 24 hours later. The registry remained unchanged during the study period and all the data were introduced prospectively by the authors. Our CRRT protocol, which also remained unchanged during the study period, consisted of a Prisma® monitor primed in CVVHDF mode and a femoral venous 12G access with a double lumen catheter. The initial dosage was 35 mL/Kg/hour and convective treatment (ultrafiltrate) was administered in a dose as high as the vascular access permitted, aiming for a filtration fraction lower than 20%. The rest of the dosage was administered as diffusive therapy. The overall dose remained unchanged for at least the first 24 hours and was then increased if metabolic control (serum creatinine below 2 mg/dL and pH normalization) was not acceptable. The same bicarbonate buffered solution was used for hemofiltration and dialysis and was supplemented with sodium up to 148 meq/L in all patients. The anticoagulation regime depended on the patient characteristics.

Patients were considered to be unstable if they were receiving NE or had a MAP lower than 60 mmHg at the start of CRRT. Two groups of patients were defined after 24 hours treatment: responders (Rs), i.e., those with a 20% decrease in NE dosage or a 20% increase in MAP with no increase in NE dosage; and non-responders NRs. All patients were followed-up for

15 days after withdrawal of CRRT to determine the relationship between the study variables and mortality.

Statistical Analysis

The results are expressed as the mean (95% CI for mean) for continuous variables and n (%) for categorical variables. Variables indicating time are expressed as the median (25-75 percentiles). The statistical analysis was done with the Student t test for continuous variables and the chi-square test for categorical variables. An alpha error of 5% was used in all tests. Kaplan-Meier and Log-Rank were used to plot survival graphs. To detect variables associated with mortality we used backward stepwise logistic regression analysis, introducing in the model all variables related to mortality with a significance level of 0.15 in the previous tests. For the regression analysis itself a significance of 0.05 was used. Results are presented as OR (95% confidence interval). All calculations were made with SPSS for Windows®.

Results

We studied 169 patients, 115 (68%) male, mean age: 53.8 (52.7-54.9) years. The mean APACHE II at admission was 21.8 (21.2-22.3) and the reason for admission was sepsis in 65 patients (38.5%), liver transplant or liver failure in 31 (18.3%), cardiac surgery in 28 (16.5%), trauma in 15 (8.9%), abdominal surgery in 11 (6.6 %), and other reasons in 19 (11.2%).

Of these 169 patients, 99 were classified as responders (Rs) and 70 as non-responders (NRs). The main characteristics of the patients in both groups and the analysis of the differences are shown in **Table 1**. The only significant difference between Rs and NRs was in the NE dosage ($p<0.01$), higher in the Rs. Median survival was statistically different in both groups: 21(15-30) days for Rs vs 12 (3-22) days for NRs ($p < 0.001$) as shown in **Figure 1**.

At the start of CRRT, 114 patients (67.5%) were unstable, but their profile did not differ significantly from the stable patients, even though the unstable patients, as expected, had a lower

MAP and required NE. Mortality was similar in both groups (65 of 114 unstable patients -57%- and 30 of 55 stable patients -54.5%; p=NS) **(Table 2)**.

Then, we performed a specific analysis in the subgroup of patients unstable when initiating CRRT. In this case, similarity between both groups was maintained and the differences are presented in **Table 3**.

We detected significant differences in mortality between Rs and NRs in all patients (30% mortality in Rs vs 74.7% in NRs) and in the subgroup of unstable patients (30% vs 87% mortality). In the group of patients who were stable at the start of CRRT, mortality was also higher in NRs (64.4%) than Rs (30%) but no clear relationship could be demonstrated (p 0.08) **(Table 4)**. When selecting only the 65 patients admitted because of sepsis, the results were similar; mortality in Rs was 11 out of 30 (29.7%) patients vs 26 out of 35 (70.3%) in NRs ($p<0.005$).

Univariate analysis showed that only age, APACHE II at admission, APACHE II at start of CRRT, and oliguria were related to mortality in all patients. In unstable patients, the effluent was also related to mortality **(Table 5)**.

In the overall group of patients and the unstable patients, lack of hemodynamic improvement was also the main factor related to mortality. In unstable patients, a negative response predicted mortality with a sensitivity of 72% and a specificity of 86% (positive predictive value of 87% and negative predictive value of 70%).

The logistic regression analysis to detect possible confounding variables and to evaluate the real weight of NRs in predicting mortality showed that the only variables related to mortality in our patients were APACHE II at admission (not at CRRT), percentage decrease of creatinine, and lack of hemodynamic response to CRRT **(Table 6)**.

Discussion

The practice of CRRT for the management of ARF in the ICU setting is increasing steadily, and it is currently the preferred method for RRT by intensive care specialists in several different countries (1-3).[1-3] Increasing knowledge is being gained about the results of this type of therapy and the different safety factors involved. However, the variety of techniques available, e.g., continuous hemofiltration (CHF), continuous hemodiafiltration (CHDF), continuous hemodialysis (CHD), high flow therapies (HFHF), slow dialysis (SLEDD), and their different methods of implementation, such as anticoagulation regimes, dosage, and criteria for initiation or withdrawal, hinder harmonization of the data so far published.

Even though the main issue concerning RRT remains unsolved (which therapy is best in terms of outcome),[14,15] some key points regarding CRRT are accepted. One of these points is that hemodynamics remain unchanged even in the most unstable patients[4] or, more frequently, improve after initiation of these therapies (as our present results clearly show). This improvement has been detected in animal studies[16,17] and, later, in different clinical studies[18-20] and has been partly explained by a positive effect in the elimination of inflammatory mediators.[21,22] Another explanation could be an immunomodulatory effect of CRRT, as shown by Yekebas *et al*.,[23] which would also help explain the possible benefit obtained when instituting these therapies early in the course of the inflammatory process.[24,25] Other possible factors affecting improvement have also been suggested; for example, vascular resistance and venous tone, as well as arterial blood pressure, are significantly higher during cold hemofiltration,[26] and a decreased temperature could explain the improvement in some patients.[27] Use of bicarbonate buffer, even with inconclusive evidence, has been shown in some reports to have a better hemodynamic profile than lactate based solutions.[28] In our study, both temperature maintained and percentage of patients treated with bicarbonate were similar in both groups and so, even

though we are unable to draw conclusions about the possible effect of these variables in the hemodynamic response, we can assume that did not interfere with our results. On the other side, an elevation of the concentration of sodium in the dialysate has been unequivocally related to the hemodynamic stability of intermittent hemodialysis and, as we raise sodium concentration in our fluids, this procedure could play a part in our results. Against this possibility, we must mention that, in the first place, this effect has not been proved in CRRT and, in second place, as the sodium concentration is a standard procedure in our center, it has been applied to all our patients (Rs and nonRs).

As mentioned before, an early start of therapy is another factor that can interfere with the results[24,25,29] (perhaps as an expression of the above-mentioned immunomodulation),[30] but we were unable to demonstrate this effect. In fact, the mean delay was somewhat shorter in the group of patients who died, possibly because our protocol involved the early initiation of therapy in the course of ARF.

Another important aspect associated with clinical improvement and outcome is the volume of the ultrafiltrate. In a recent controlled study, Ronco *et al*.[11] demonstrated that a starting ultrafiltrate of 35 mL/Kg/h is significantly better in terms of outcome compared with 25 mL. This figure of 35 mL/Kg/h can now be considered the adequate starting dose for patients in ARF under hemofiltration. Further study of their data showed that a higher dosage is even better for septic patients. Thus, even though the higher the fluid exchange the better the prognosis,[31] further studies are needed to detect which patients would benefit from this increased dosage.

In view of these data, we opted in our protocol for 35 mL/Kg/h as the initial dose in all patients, but increased this figure after 24 hours treatment if adequate metabolic control was not achieved. Our data show that mean ultrafiltrate exchange (real ultrafiltrate, not taking into account the hours of treatment losses) was associated with outcome, and differences in this

figure can only be explained by the mentioned treatment losses or by a mismatch between patient needs and dose delivered. This last concept is intriguing: is it possible that the lack of hemodynamic response could be a marker of a mismatch between patient needs and dose administered?

Another interesting point raised in our results is the fact that responders did receive higher doses of NE which could mean that we detected a group of patients under-resuscitated. Against this possibility is the fact that NE dosage was similar in survivors and non-survivors.

Considering these observations (a possible immunomodulatory effect and a proven effect of ultrafiltrate removal) high-flow hemofiltration presents itself as an attractive alternative. Indeed, in 1999 Oudemann-van Straaten *et al*. showed that patients treated with a mean ultrafiltrate rate of 63 ml/min had an ICU mortality of 33%, in comparison with a predicted mortality of 67%.[32] More recently, Honore *et al*. treated 20 patients with intractable cardio-circulatory failure complicating septic shock, who had failed to respond to conventional therapy, by removing 35 L of ultrafiltrate in four hours and continuing conventional hemofiltration for at least four days. They defined a group of responders (improvement in cardiac index, mixed venous saturation, increase in arterial pH, and reduction in epinephrine dose) and compared mortality with non-responders, and showed how survival to 28 days was improved for the responders (81% vs. 0%).[12] This same association between improvement and prognosis had already been mentioned by Gotloib *et al*. in a study using mixed hemodialysis and hemofiltration.[13] The effect of high-flow hemofiltration on mortality has recently been challenged and remains to be demonstrated.[33] Based on these studies, we designed our protocol to evaluate hemodynamic response as a marker of mortality with a more conventional treatment (35 mL/Kg/h), and showed that this association is maintained (OR for death in the non-responders of 7 versus the responders).

Although different studies have shown that CRRT has a possible benefit in terms of outcome, mortality remains high and many attempts have been made to define the characteristics of those patients with a poorer prognosis at the start of the procedure. Different factors associated with a worse outcome include age, need for mechanical ventilation, vasopressors, urine volume, serum bilirubin, arterial base deficit, serum creatinine,[34] septicemia,[35] less fluid removal, rising blood urea nitrogen and serum creatinine levels after ultrafiltrate,[36] hepatic failure, or coagulopathy.[37] Our data are coincident with most of these results, but it is important to point out that, even though NA use was higher in responders than non-responders, the vasopressor dosage was not related to mortality in our patients, and this variable had no effect on our results.

An additional problem is that widely used prognostic indexes do not perform well in these patients: APACHE II, III or SAPS[9,10] and specific indexes for ARF overstate the actual mortality.[6,7] There is more agreement when referring to the number of failing organs and outcome.[9,29] An interesting work recently published shows that the number of failing organs and APACHE III at day 3 of initiation of therapy were much more powerful predictors of outcome in such patients.[38] Thus, factors associated with the technique that may affect our results include ultrafiltrate volume, temperature, buffer used and precocity in the initiation of CRRT, and patient-related factors include age, severity of the process and severity of renal dysfunction. Because all these factors have been taken into account in our analysis, we do not consider there to be any confounding variables that could explain part of the association between the hemodynamic response and survival.

Nevertheless, even though our study is a prospective cohort, it comprises different groups of patients with different etiologies, and this can diminish the validity of our results. Another important aspect to consider is that we used mixed dialysis and convection to reach the final desired dosage of 35 mL/Kg/h, and so our results cannot be explained only by the effect of the

convective therapy. On the other hand, the fact that it is a protocol based on our clinical practice and complies with the standards in use can make our results widely reproducible.

In designing this protocol, we did not seek to define the hemodynamic response to CRRT but to evaluate it usefulness as an aid in determining patients' outcomes. We selected a somewhat long period of delay. It can be argued against our results that, by selecting this 24-hour delay (and not a shorter one), we can be detecting more of the natural course of the disease than the effect of the CRRT per se. Because we did not find differences between responders and not responders in therapy related aspects, we can conclude that a possible confusing effect of differences in the therapy is not affecting the results and, on the other side, because both groups are well balanced regarding epidemiological variables and severity scores, the situation at the start of the treatment can be assumed to be similar as well. In this context, even though we cannot answer unequivocally whether the effect is due to the evolution of the disease or the effect of the treatment, this question does not invalidate our conclusion that the hemodynamic response to the treatment can be of aid in predicting outcome in ICU patients under CRRT

Finally, we should point out that our intention was not to perform a complete outcome study, but rather to validate the hemodynamic response as an isolated parameter. Accordingly, we conducted the regression analysis to discard possible confounding variables and included in the model only those variables (already discussed) that could possibly affect the main result. Because of the relatively small number of patients and the fact that this was a single-center study, we did not attempt to calculate an outcome-predicting formula.

Conclusion

Hemodynamic improvement after 24 hours CRRT is closely related to survival in ICU patients, and this association is even stronger for patients who are unstable at the start of CRRT. Our results warrant larger multicenter studies addressing outcome and considering hemodynamic

response as a main factor, in order to generate a specific outcome index for patients undergoing CRRT.

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Table I. Difference between responders and non-responders at initiation of CRRT

	All patients n=169	**Responders n=70**	**Non-responders n=99**	***P***
Age	53.8 (52.7-54.9)	52.7 (51.2-54.3)	54.6 (53.1-56.0)	Ns
Gender (% male)	115 (68%)	50 (71.4%)	65 (65.7%)	Ns
APACHE II admission	21.8 (21.2-22.3)	21.8 (21.1-22.6)	21.8 (21.0-22.5)	Ns
Sepsis	65 (38.5%)	30 (40%)	35 (37.2%)	Ns
Liver transplant	31 (18.3%)	15 (20%)	16 (17.1%)	
Cardiac surgery	28 (11.9%)	6(8%)	15 (16%)	
Trauma	15 (8.9%)	10 (13.3%)	5 (5.3%)	
Abdominal surgery	11 (6.6%)	1 (1.3%)	5 (5.3%)	
Other	19 (11.2%)	13 (17.3%)	18 (19.2%)	
APACHE II at CRRT	22.7 (22.1-23.2)	21.7 (21.0-22.4)	23.3 (22.6-24.1)	Ns
MAP at CRRT	77.4 (76.3-78.5)	75.6 (74.1-77.1)	78.6 (77.1-80.1)	Ns
ARF at CRRT	164 (97%)	72 (96%)	92 (97.9%)	Ns
Temperature decrease	0.67 (0.56-0.78)	0.87 (0.70-0.98)	0.53 (0.39-0.67)	Ns
Serum creatinine at CRRT	354 (340-367)	374 (358-397)	338 (321-355)	Ns
% oliguria at CRRT	111 (65.7%)	48 (68.6%)	63 (63.6%)	Ns
Norepinephrine at CRRT	29.3 (27.2-31.5)	37.2 (33.1-41.2)	18.7(16.1-21.1)	< 0.001
Delay in CRRT after ARF	3.0 (2.7-3.4)	3.2 (2.7-3.7)	2.9 (2.5-3.3)	Ns
% decrease creatinine	24.4 (23.0-25.8)	26.7 (25.3-27.4)	22.7 (21.6-24.3)	Ns

Data are presented as the mean (confidence interval for mean) or n (%). Age in years. MAP (mean arterial pressure) in mmHg. Vasopressor in μgr/min. Creatinine in mmol/L. Delay in CRRT in median days (25-75 percentiles). ARF (acute renal failure). % decrease creatinine over first 24 hours CRRT. Temperature in ºC

Table II. Differences between stable and unstable patients at the start of CRRT

	All patients n=169	**Stable n=55**	**Unstable n=114**	***P***
Age	53.8 (52.7-54.9)	51.9 (50.1-53.7)	54.7 (53.4-56.0)	ns
Gender (% male)	115 (68%)	35 (63.3%)	80 (70.2%)	ns
APACHE II admission	21.8 (21.2-22.3)	21.3 (20.2-22.4)	22.0 (21.4-22.6)	ns
APACHE II at CRRT	22.7 (22.1-23.2)	22.1 (21.0-23.2)	22.9 (22.3-23.5)	ns
MAP at CRRT	77.4 (76.3-78.5)	84.1 (82.3-85.9)	74.1 (72.9-75.3)	< 0.001
Serum creatinine at CRRT	354 (340-367)	390 (363-416)	335 (320-350)	ns
% oliguria at CRRT	111 (65.7%)	30 (54.5%)	65 (57%)	ns
Norepinephrine at CRRT	---	*None*	29.3 (27.2-31.5)	---
Delay in CRRT after ARF	3.0 (2.7-3.4)	4.2 (3.5-4.9)	2.5 (2.2-2.8)	< 0.05
% decrease creatinine	24.4 (23.0-25.8)	26.4 (24.1-28.7)	23.5 (22.7-25.3)	Ns
Died	95 (56.2%)	30 (54.5%)	65 (57%)	Ns

Data are presented as the mean (confidence interval for mean) or n (%). Age in years. MAP (mean arterial pressure) in mmHg. Vasopressor in μgr/min. Creatinine in mmol/L. Delay in CRRT in days. ARF (acute renal failure). % decrease creatinine over first 24 hours CRRT.

Table III. Difference between responders and non-responders at start of CRRT in the group of unstable patients (n=114)

	Responders n=60	**Non-responders n=54**	***P***
Age	53.2 (51.5-54.9)	56.4 (54.3-58.5)	Ns
Gender (%male)	44 (73%)	36 (66.7%)	Ns
APACHE II admission	21.9 (21.1-22.7)	22.2 (21.2-23.2)	Ns
APACHE II at CRRT	21.9 (21.1-22.7)	24.1 (23.2-25.0)	Ns
MAP at CRRT	74.8 (73.1-76.5)	73.4 (71.6-75.2)	Ns
Serum creatinine at CRRT	369 (346-392)	294 (276-313)	< 0.05
% oliguria at CRRT	43 (71.7%)	37 (68.5%)	Ns
Norepinephrine at CRRT	32.5 (29.3-35.7)	25.7 (23.2-28.3)	Ns
Delay in CRRT after ARF	2.52 (2.14-2.90)	2.47 (2.00-2.94)	Ns
% decrease creatinine	26.6 (25.5-27.7)	19.4 (16.3-22.5)	0.05

Data are presented as the mean (confidence interval for mean) or n (%). Age in years. MAP (mean arterial pressure) in mmHg. Vasopressor in µgr/hour. Creatinine in mmol/L. Delay in CRRT in days. ARF (acute renal failure). % decrease creatinine over first 24 hours CRRT.

Table IV. Percent mortality for all patients and for responders and non-responders

	Responders	**Non-responders**	**OR (CI)**
All patients (n=169)	30% (21 out of 70)	74.7% (74 out of 99)	6.9 (3.5-13.7)
Stable (n=55)	30% (3 out of 10)	60% (27 out of 45)	3.5 (0.79-15.3)
Unstable (n=114)	30% (18 out of 60)	87% (47 out of 54)	15.7 (5.9-41.2)

P<0.001 for all patients and unstable patients, P=0.08 for stable patients

Table V. Variables associated with mortality

All patients	**Survivors n=74**	**Died n=95**	***P***
Age	51.7 (50.2-53.2)	55.4 (53.9-56.9)	0.09
Gender (%male)	51 (68.9%)	64 (67.4%)	ns
APACHE II admission	20.4 (19.6-21.2)	22.9 (22.2-23.6)	< 0.05
APACHE II at CRRT	20.8 (20.1-21.5)	24.1 (23.3-24.9)	< 0.005
Temperature decrement	0.63 (0.45-0.81)	0.7 (0.56-0.84)	ns
% oliguria at CRRT	43 (58.1%)	68 (71.6%)	0.07
Norepinephrine at CRRT	20.7 (17.7-23.6)	19.1 (16.8-21.3)	ns
Delay in CRRT after ARF	3.9 (3.3-4.5)	2.4 (2.1-2.7)	< 0.05
% decrease creatinine	27.7 (25.9-29.5)	21.6 (19.5-23.7)	< 0.05
Volume of effluent	2.22 (2.16-2.28)	2.16 (2.10-2.22)	ns
Unstable	**Survivors n=49**	**Died n=65**	***P***
Age	51.9 (50.0-53.8)	56.8 (55.0-58.6)	0.07
Gender (%male)	37 (75.5%)	43 (66.2%)	ns
APACHE II admission	20.4 (19.521.3)	23.2 (22.4-24.0)	< 0.05
APACHE II at CRRT	21.1 (20.2-22.0)	24.3 (23.4-25.2)	< 0.05
Temperature decrease	0.58 (0.34-0.82)	0.66 (0.51-0.81)	ns
% oliguria at CRRT	32 (65.3%)	48 (73.8%)	ns
Norepinephrine at CRRT	31.2 (27.7-34.7)	27.9 (25.3-30.4)	ns
Delay in CRRT after ARF	2.7 (2.2-3.2)	2.4 (2.0-2.8)	ns
% decrease creatinine	28.8 (26.5-31.1)	18.9 (16.3-21.5)	< 0.005
Volume of effluent	2.32 (2.24-2.40)	2.09 (2.04-2.14)	< 0.05

Data are presented as the mean (confidence interval for mean) or n (%). Age in years. MAP (mean arterial pressure) in mmHg. Vasopressor in µgr/hour. Creatinine in mmol/L. Delay in CRRT in days. ARF (acute renal failure). % decrease creatinine over first 24 hours CRRT. Mean effluent in L/hour, accounting for treatment losses. Temperature in ºC.

Table VI. Logistic regression analysis for mortality 15 days after withdrawal from CRRT in unstable patients

	Wald	OR (CI)	P
Non-responders	25.49	7.04 (3.30-15.02)	< 0.001
% decrease creatinine	3.98	0.98 (0.96-1.00)	< 0.05
APACHE II at admission	3.87	1.06 (1.00-1.12)	< 0.05
Oliguria	3.25	2.06 (0.94-4.53)	= 0.07

Only statistically significant variables are shown

Figure 1. Differences in survival between responders (Rs) and non-responders (NRs) from the start of CRRT to 15 days after withdrawal from CRRT *($p<0.001$)*

Figure

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