Intensive Care Med (2001) 27: 1194–1199
DOI 10.1007/s001340101002

ORIGINAL

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Acute renal failure after successful cardiopulmonary resuscitation

Received: 21 September 2000
Final revision received: 28 March 2001
Accepted: 8 May 2001
Published online: 15 June 2001

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Abstract *Objective*: To assess the frequency and independent predictors of severe acute renal failure in patients resuscitated from out-of-hospital ventricular fibrillation cardiac arrest.
Design: A cohort study with a minimum follow-up of 6 months.
Setting: Emergency department of a tertiary care 2200-bed university hospital.
Patients and participants: Consecutive adult (> 18 years) patients admitted from 1 July 1991 to 31 October 1997 after witnessed ventricular fibrillation out-of-hospital cardiac arrest and successful resuscitation.
Measurements and results: Acute renal failure was defined as a 25 % decrease of creatinine clearance within 24 h after admission. Out of 187 eligible patients (median age 57 years, 146 male), acute renal failure occurred in 22 patients (12 %); in 4 patients (18 %) renal replacement therapy was performed. Congestive heart failure (OR 6.0, 95 % CI 1.6–21.7; $p = 0.007$), history of hypertension (OR 4.4, 95 % CI 1.3–14.7; $p = 0.02$) and total dose of epinephrine administered (OR 1.1, 95 % CI 1.0–1.2; $p = 0.009$) were independent predictors of acute renal failure. Duration of cardiac arrest, pre-existing impaired renal function and blood pressure at admission were not independently associated with renal outcome.
Conclusions: Severe progressive acute renal failure after cardiopulmonary resuscitation (CPR) is rare. Pre-existing haemodynamics seem to be more important for the occurrence of acute renal failure than actual hypoperfusion during resuscitation.

Keywords Acute renal failure · Cardiac arrest · Ventricular fibrillation

Introduction

Temporary impairment of renal function is common in patients resuscitated from cardiac arrest [1, 2]. However, severe ongoing acute renal failure (ARF) occurs infrequently after resuscitation and persistent renal failure in patients with primarily normal renal function is rare [2]. Although a moderate rise of serum creatinine levels after resuscitation is reversible in most patients, impaired renal function after cardiac arrest is associated with poor outcome [1, 2, 3, 4, 5, 6].

So far ARF after resuscitation has been defined by elevated serum creatinine levels at a single time point [1, 2]. A dynamic parameter like the course of serum creatinine or creatinine clearance is more accurate for estimating the impairment of renal function, but has

not been investigated yet. The frequency and determinants of ARF after cardiac arrest have been evaluated only in small case series [1, 2]. Congestive heart failure (CHF), cumulative epinephrine administered, coronary artery disease, pre-existing renal insufficiency and low flow time were described as possible risk factors of ARF after cardiac arrest. To the best of our knowledge, a multivariate analysis to determine independent predictors of ARF has not been performed in a representative cohort.

The aim of the study was to assess the frequency and predictors of severe ARF in a larger cohort of patients resuscitated from witnessed out-of-hospital ventricular fibrillation cardiac arrest.

Materials and methods

Patients

The study was designed as a retrospective cohort study. However, patients' data were collected in a prospective manner according to a standardised protocol of a cardiac arrest registry according to the Utstein Style. We included all consecutive patients who were treated at the emergency department of the Vienna General Hospital, a 2200-bed tertiary care university hospital, following cardiac arrest from 1 July 1991 to 31 October 1997. The minimum follow-up period was 6 months. The data of approximately 40 eligible patients had previously been analysed in a pilot study and are therefore partly overlapping [2].

Inclusion criteria

Patients with witnessed out-of-hospital cardiac arrest and ventricular fibrillation as the first presenting rhythm, more than 18 years of age and who were successfully resuscitated from cardiac arrest and survived at least for 24 h were included.

Exclusion criteria

Patients whose cardiopulmonary arrest was of non-cardiac origin (primary respiratory arrest, trauma, hypothermia, near drowning, drug overdose or haemorrhage) were not included in the analysis. Further, we excluded patients with contrast agent-induced renal failure and patients with urinary outflow tract obstruction.

Definitions

Cardiopulmonary arrest was defined as the absence of both spontaneous respiration and palpable pulse. Return of spontaneous circulation (ROSC) was defined as electrical activity on the electrocardiogram and palpable pulses. Treatment was in accordance with the guidelines of the American Heart Association and the European Resuscitation Council for basic and advanced cardiac life support and for post resuscitation care [7, 8]. Acute renal failure (ARF) following resuscitation from cardiac arrest was defined as a 25% decrease of renal function within 24 h estimated by Cockcroft and Gaults's formula for creatinine clearance [9], which is equivalent to an increase of one-third of serum creatinine. Cockcroft creatinine clearance is calculated: $C_{crea} = [(140\text{-age}) \times (\text{weight in kg})]/[72 \times \text{serum creatinine in mg/dl}]$. Data on renal function before cardiac arrest in the setting of an emergency department are frequently missing. We considered medical history alone not accurate enough and, therefore, arbitrarily defined pre-existing impairment of renal function by the presence of the combination of admission baseline creatinine more than 1.4 mg/dl and a creatinine clearance less than 70 ml/min. Normotension was defined as systolic blood pressure from 90 to 160 mmHg, values below were classified as hypotensive and values above were classified as hypertensive. Oliguria was defined as an urine volume less than 30 ml/h. History of hypertension, diabetes mellitus and congestive heart failure were assumed if previous history and corresponding medication could be recorded.

Study design and data collection

Acquisition of data concerning cardiopulmonary resuscitation (CPR) was performed systematically on the arrival of each patient according to the "Utstein Style" – the recommended guidelines for uniform reporting of data on out-of-hospital cardiac arrest [10]. Evaluated data included duration of the interval from time of collapse until ROSC, first electrocardiogram-rhythm and history of the individual patient, in particular concerning cause of cardiac arrest. The time of recognition of collapse until time of calling the emergency medical system was evaluated by one of the investigators personally interviewing one or more witnesses. The time of cardiac arrest was estimated from time of calling the emergency medical system and the time of recognition of collapse. The "Utstein Style" protocol described above was extended at our institution and also included questions for the following medical pre-arrest diagnoses: coronary artery disease (includes angina pectoris, aorto-coronary bypass grafting, history of myocardial infarction), CHF, hypertension, cerebrovascular disease, diabetes mellitus and chronic pulmonary disease. Anuric patients were evaluated by ultrasound to exclude urinary outflow tract obstruction.

A complete series of routine laboratory investigations including serum creatinine and blood urea nitrogen (BUN) were performed on arrival and 24 h after cardiac arrest. Measurements were carried out with a Hitachi 717 autoanalyser (Boehringer Mannheim, Mannheim, Germany).

One or a combination of the following conditions were regarded indicative for renal replacement therapy: metabolic acidosis and hyperkaliaemia unresponsive to other interventions, BUN more than 100 mg%, intractable fluid and volume problem.

Statistical analysis

Continuous data are presented as the median and interquartile range (IQR, range from the 25^{th} to 75^{th} percentile). Percentages were calculated for dichotomous variables. The Wilcoxon test for paired samples and McNemar tests were used to compare within subject variation. The chi-square test or Fisher's exact test, if appropriate, were performed to compare proportions. The Mann-Whitney-U test was used for univariate comparison of continuous data. A multivariate logistic regression model was applied to assess the independent effect of baseline variables on renal failure. Before conducting the statistical analysis we defined the parameters from the registry data set which should be entered in the model to avoid the negative effects of multiple testing. Variables which had a p value less than 0.2 in univariate comparison as well as biologically reasonable variables were entered as predictor variables in the model. A p value of less than 0.05 was considered as statistical-

Table 1 Baseline data of 187 patients after successful resuscitation

Median age (years) (IQR)	57 (51–68)
Male sex	146 (78 %)
Diabetes mellitus	15 (8 %)
Nicotine abuse	67 (36 %)
Hypertension	42 (22 %)
History of congestive heart failure	27 (14 %)
Coronary artery disease	67 (37 %)
History of myocardial infarction	52 (28 %)
Cerebrovascular disease	8 (4 %)
COPD	8 (4 %)
Pre-existing renal impairment	66 (35 %)
Median no flow time (min) (IQR)	1 (0–2)
Median time to ROSC (min) (IQR)	20 (12–30)
Median number of countershocks (IQR)	3 (2–6)
Median milligrams of epinephrine administered (IQR)	2 (0–6)

ly significant. The goodness-of-fit for the model was assessed by the Hosmer Lemeshow test [11]. All calculations were performed with MS Excel for Windows 97 and SPSS for Windows (Version 10.0).

Results

Patients

During the observation period 1032 patients following cardiac arrest were admitted to the emergency department. Seven hundred seventeen patients (69 %) had out-of-hospital cardiac arrest. Cardiac arrest of cardiac origin was diagnosed in 511 patients (50 %), of these 471 patients (46 %) had a witnessed arrest. In this group ventricular fibrillation was found in 314 patients (30 %), ROSC was achieved in 264 patients (26 %). Of these, 24 patients (2 %) died within the first 24 h and were excluded. We had to exclude another 53 patients (5 %), as their data were incomplete, and one patient under the age of 18, and included 187 patients (18 %) in the final analysis. Baseline data of the 187 patients included in the final analysis are presented in Table 1.

Renal function, blood pressure and creatinine kinase (CK) values of the 187 patients at baseline and 24 h after admission to the emergency department are presented in Table 2. Overall there was a significant decrease of serum creatinine during the first 24 h after presentation. Renal function estimated by Cockcroft creatinine clearance also improved. Median serum CK rose to 985 mg/dl (IQR 396–2003, range 15–7900). Ten patients had a serum CK of more than 4000 mg/dl; ARF occurred in two of these patients (24 h CK 6820 mg/dl; 24 h CK 5050 mg/dl).

Overall ARF occurred in 22 patients (12 %), who were all male. In four patients renal replacement therapy was performed. In one of these patients renal function recovered completely after 16 days of haemofiltration, the remaining three patients did not survive until hospital discharge. Seventy-two patients out of the 187 died within 30 days after the index event. Patients with ARF died more often within the first 30 days after admission than patients without ARF (19/22 versus 53/165, $p < 0.0001$). Causes of death of patients with and without renal failure are presented in Table 3.

An univariate comparison of baseline variables between patients with and without renal failure is presented in Table 4. Patients with ARF, compared to patients without ARF, were older and all patients were male. High cumulative doses of epinephrine, hypotension at presentation and longer time to ROSC were more frequently found in patients with ARF. Furthermore preexisting renal function, history of hypertension, coronary artery disease and CHF were significantly different. The number of countershocks, oliguria at admission, history of diabetes mellitus and smoking did not differ between patients with and without ARF. As only 16 (10 %) and 12 (7 %) patients have been treated after successful resuscitation with dopamine and dobutamine, respectively, for more than 12 h we did not look for differences between the groups of patients with and without ARF concerning this parameter.

A multivariate model was applied to determine independent predictors of ARF and to control for confounding. Results of the logistic regression model are shown in Table 5. In this multivariate model CHF, history of hypertension and total dose of epinephrine administered were independent predictors of ARF. Coronary artery disease, time to ROSC, history of diabetes mellitus and blood pressure at admission showed no significance. The sex variable was not entered in the model because all 22 patients with ARF were male. The log-likelihood ratio of the model was 96.1, $r^2 = 0.37$. The model had an acceptable fit (Hosmer Lemeshow $\chi^2 = 11.0$, degrees of freedom = 8, $p = 0.2$).

Table 2 Renal function, blood pressure and CK within 24 h after cardiac arrest ($n = 187$) (*oliguria* urine volume < 30 ml/h, *hypotension* systolic blood pressure < 90 mmHg, *hypertension* systolic blood pressure > 160 mmHg)

	Baseline	24 h	*p*
Serum creatinine (mg/dl) median (IQR)	1.4 (1.2–1.6)	1.1 (0.9–1.5)	< 0.0001
Serum BUN (mg/dl) median (IQR)	19.4 (15.2–24.3)	21.1 (14.1–29.0)	0.002
Creatinine clearance (ml/min) median (IQR)	67 (54–81)	81 (54–107)	< 0.0001
Oliguria	63 (34 %)	9 (5 %)	< 0.0001
Hypertension	17 (9 %)	7 (4 %)	0.02
Hypotension	99 (53 %)	72 (38 %)	0.001
CK (U/l) median (IQR)	79 (46–124)	985 (396–2003)	< 0.0001

Table 3 Comparison of the cause of death of patients with *(ARF)* and without *(no ARF)* acute renal failure

Cause of death	No ARF ($n = 53$)	ARF ($n = 19$)
Congestive heart failure	23 (43%)	14 (74%)
Arrhythmia	3 (6%)	0
Cerebrovascular event	19 (36%)	4 (21%)
Sepsis	5 (9%)	0
Pulmonary embolism	1 (2%)	0
ARDS	1 (2%)	0
Multi-organ failure	1 (2%)	1 (5%)

Discussion

Acute renal failure related to cardiac arrest occurred in 12% of the patients. A history of CHF, hypertension and the cumulative dose of epinephrine administered during CPR were independent predictors of ARF.

Specific nephro-protective strategies for prevention of ARF are not available yet. The general approach to the management of this disorder is preventing complications while awaiting recovery of renal function. Early identification of patients at risk for progressive ARF after successful resuscitation should result in close monitoring of volume changes and metabolic parameters in these patients. Preventive measures include appropriate dosing of all medications for glomerular filtration rate and careful avoidance of unnecessary nephrotoxins including radio-contrast agents.

In contrast to the pilot study [2], the present analysis investigates renal function after resuscitation in a large and homogenous cohort of patients surviving 24 h after resuscitation from out-of-hospital ventricular fibrillation cardiac arrest. In comparison to previously published data [1, 2] on small and selected groups of patients, increased strength and power of these results can be expected. The former studies used a cut-off value of baseline creatinine (1.4 mg%) for the definition of renal failure. This definition certainly has several limitations, in particular, the progress of renal function is not considered for categorisation of ARF [12]. In the present analysis renal function quantified by serum creatinine, BUN and Cockcroft's formula for creatinine clearance was assessed over a period of at least 24 h after CPR giving a more adequate estimate of the course of renal function. In patients without any impairment of excretory renal function within this period, ARF caused by CPR seems very unlikely. Moreover, the 24 h serum creatinine levels showed the best association with outcome compared to 3- and 7-day measurements [2].

Similar to former findings, we observed a significant overall decrease of the median serum creatinine level within 24 h after resuscitation [2], which suggests that resuscitation induces a transient mild rise of serum creatinine. Interestingly, serum creatinine values varied around 1.4 mg/dl, which was formerly taken as the cut-off for categorisation of ARF. The rise of median serum BUN after 24 h (Table 1) might be interpreted as being due to the transient catabolism following cardiac arrest and pre-renal azotaemia. This contrasts to the decrease of serum creatinine level.

Pre-existing impairment of renal function is one of the strongest predictors for the development of ARF in patients undergoing cardiac surgery [13, 14, 15]. This effect has not been investigated in patients after CPR, possibly because exact data on pre-arrest renal function are often not available. Mattana et al. [1] reported a his-

Table 4 Comparison of patients with *(ARF)* and without *(no ARF)* acute renal failure ($n = 187$)

	No ARF ($n = 165$, 88%)	ARF ($n = 22$, 12%)	p
Age median (years) (IQR)	57 (50–68)	62 (56–70)	0.09
Male sex *(n)*	124(85%)	22 (12%)	0.005
Median number of countershocks(IQR)	3 (2–5)	4 (1–7)	0.4
Median epinephrine (mg) (IQR)	2 (0–5)	5 (2–12)	0.002
Median time to ROSC (min) (IQR)	18 (11–30)	27 (16–35)	0.015
Blood pressure at admission			0.04
Hypotension	82 (50%)	17 (77%)	
Normotension	68 (41%)	3 (14%)	
Hypertension	15 (9%)	2 (9%)	
Impaired renal function at presentation *(n)*	51 (31%)	15 (68%)	0.001
Oliguria at presentation *(n)*	53 (32%)	10 (46%)	0.2
History of hypertension *(n)*	32 (19%)	10 (46%)	0.01
Diabetes mellitus *(n)*	12 (7%)	3 (14%)	0.4
Smoking *(n)*	61 (37%)	6 (27%)	0.5
History of congestive heart failure *(n)*	18 (11%)	9 (41%)	0.001
Coronary artery disease *(n)*	56 (34%)	13 (59%)	0.03

Table 5 Logistic regression model for independent predictors of acute renal failure

	Odds ratio	95 % Confidence interval	*p*
Congestive heart failure	6.0	1.6–21.7	0.007
History of hypertension	4.4	1.3–14.7	0.02
Cumulative dose of epinephrine (mg)	1.1	1.0–1.2	0.009
Pre-existing impaired renal function	2.1	0.7–6.8	0.2
History of diabetes mellitus	1.9	0.4–9.8	0.4
Blood pressure at admission			0.2
Hypotension	1.0		
Normotension	1.2	0.2–8.2	
Hypertension	0.3	0.03–3.3	
Age (years)	1.0	1.0–1.1	0.4
Time to ROSC (min)	1.0	1.0–1.0	0.5
Coronary artery disease	0.9	0.3–2.7	0.8

tory of pre-existing compromised renal function in 23 % of 56 patients. These data are based only on clinical history and probably underestimate the frequency of renal impairment on presentation. We found pre-existing impaired renal function on admission in 35 % of our patients. The higher incidence of pre-existing renal impairment in our analysis seems more accurate, because the definition is based on objective measurement and calculations of baseline serum creatinine and creatinine clearance.

We investigated the entity of severe ongoing renal failure related to cardiac arrest, which was found in 12 % of the victims. In this group of patients the extent of renal dysfunction per se reached clinical relevance, which is underlined by the frequency of renal replacement therapy required in this group ($n = 4$, 18 %). In former studies mild impairment of renal function was also registered, because of the single time point cut-off value of serum creatinine 1.4 mg/dl.

In a multivariate model three parameters were independent predictors of ARF: history of CHF, history of hypertension and cumulative dose of epinephrine administered. For CHF and epinephrine, this is in line with former findings [1, 2]. To the best of our knowledge history of hypertension is identified as a relevant predictor for the first time. The small number of patients included in former publications [1, 2] might explain the fact that history of hypertension did not reach statistical significance in these reports.

In patients with CHF, chronic renal hypoperfusion and disturbed counterregulatory reflexes during reperfusion are known mechanisms in augmenting renal dysfunction after acute ischemia [16, 17, 18, 19]. The pathophysiology by which chronic hypertension triggers ARF in patients after CPR can only be suspected. Chronic hypertensive micro-angiopathy and damage to the glomerula and the juxtaglomerular apparatus might restrict renal functional capacity. High serum levels of epinephrine have been reported to induce ARF [20, 21]. Moreover, the cumulative dose of epinephrine administered during CPR is known to be inversely associated with outcome [22, 23]. Epinephrine increases the severity of post resuscitation myocardial dysfunction and aggravates renal ischemia.

Pre-existing impaired renal function and blood pressure on admission showed no significant difference between patients with and without ARF in the regression model. Mattana et al. [1] found baseline renal insufficiency univariate a risk factor for the development of ARF, which is similar to our findings. However, in the multivariate regression model co-factors like CHF, hypertension and diabetes mellitus reduce the valence of pre-existing renal impairment as significant predictor because these comorbidities are confounding factors. Moreover, the exclusion of patients with chronic renal replacement therapy might partially explain the fact that pre-existing renal failure did not reach statistical significance in this model, although we found a higher incidence of pre-existing renal impairment.

Blood pressure on admission was no independent predictor of ARF in this patient series, although univariate analysis revealed that patients with ARF were significantly more frequently hypotensive at admission. When adding the variable CHF to the model, the predictive value of the admission blood pressure significantly decreased, indicating statistical interaction between these two terms.

In contrast to previously published data, time to ROSC [1] and diabetes mellitus [2] did not show significant differences between patients with and without ARF. Time to ROSC in the univariate analysis differed significantly between the two groups. In the multivariate model, however, time to ROSC turned out not be an independent predictor of ARF. ARF seems rather to be explained by cumulative dose of epinephrine, which is also determined by the duration of time to ROSC. Mattana et al. [1] also reported diabetes mellitus as not significant.

There are some limitations to this study. Because of the lack of pre-arrest serum creatinine values as a baseline, the influence of CPR on the course of post resuscitation serum creatinine and BUN levels is based on admission values and implies some kind of uncertainty. This, however, seems to be negligible because of the short time delay in time to ROSC and acquisition of first serum samples immediately after admission. Furthermore, no direct measurements of serum creatinine clearance were performed, however, calculation by Cockcroft's formula is a well accepted surrogate for quantification of renal function [9]. Although the data were analysed retrospectively, acquisition of data in our cardiac arrest registry is, according to Utstein style, in a prospective fashion.

In conclusion, severe progressive ARF after CPR is rare. Congestive heart failure, history of hypertension and cumulative epinephrine administered independently predict the occurrence. Pre-existing haemodynamics seem to be more important than actual hypoperfusion during resuscitation.

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