

Institutional report - Cardiac general

Early dialysis in acute kidney injury after cardiac surgery

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Abstract

Acute kidney injury following cardiac surgery (AKICS) remains a frequent cause of major morbidity and mortality. The aim of this study was to examine the influence of timing of dialysis. A retrospective analysis of 3528 patients undergoing cardiac surgery between April 1995 and July 2006 was performed. In group 1 (April 1995–January 2000) intermittent haemodialysis was resorted to when other supportive measures failed. In group 2 (January 2000–July 2006) intermittent haemodialysis was commenced immediately when oliguria did not respond to fluid replacement or single-dose diuretics. In group 1, 49/1511 (3.2%) patients developed AKICS. Thirty-four patients did not receive dialysis and six patients died (18%). Of the remaining 15 patients who underwent dialysis, 13 died (87%). The overall mortality for group 1 AKICS patients was 19/49 (39%). In group 2, 87/2017 (4.3%) patients developed AKICS. Thirty-one patients did not require dialysis and none died. Of the 56 patients who were dialysed, 14 died (25%). During January 2005–July 2006, mortality following dialysis fell further to 17% (4/24). The overall mortality for group 2 patients developing AKICS was 14/87 (16%). Although the incidence of AKICS increased from 3.2% to 4.2%, earlier dialysis resulted in significantly improved survival ($P=0.00001$).

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1. Introduction

Acute kidney injury is a serious complication after cardiac surgery. It is well recognised that patients who develop acute kidney injury following cardiac surgery (AKICS) have a high mortality rate, both perioperatively and in the medium term [1, 2]. The term acute renal injury includes a broad range of definitions. Hence, the varying incidences reported after cardiac surgery, from as low as 1% [3] to as high as 7% [4]. Arbitrary values for acute renal injury have been used in various studies, such as a serum creatinine exceeding 200 mmol/l or a rise of 50 mmol/l over a baseline of 175 mmol/l [5]. Severe acute renal injury has been defined as a serum creatinine exceeding 500 mmol/l or a clinical situation requiring renal replacement therapy [6]. This last definition is of course open to the widest interpretation but is, perhaps, the most relevant when it comes to clinical practice. The definition of AKICS in this study was oliguria and/or the need for renal replacement therapy.

In those patients undergoing renal replacement therapy the mortality has been variously reported in the range of 22–67% [2, 4]. Interestingly, a high mortality has been reported both in studies prior to 2000, as in the study from the Brigham and Women's Hospital, Boston, Massachusetts, quoting a mortality of 63.7% [1] as well as in recent studies, such as the one from San Raffaele Hospital, Milan published in 2006, quoting a mortality of 66.7% [7]. However, a study

from Leicester [8] reported in 2004 showed that early renal replacement therapy during the first day post surgery, this time with continuous veno-venous haemofiltration, achieved a significantly lower mortality of 22% when compared with the same treatment started two and a half days after surgery when the mortality was 43%. Other studies, using early veno-venous haemofiltration [9] or early veno-venous haemodiafiltration [10] have corroborated these findings. Although these different studies cannot be compared because they used different criteria for defining acute renal injury and for instituting renal replacement therapy, the studies comparing early with late renal replacement therapy within the same population draw a very valid conclusion.

2. Materials and methods

We retrospectively analysed 71 patients who were dialysed after cardiac surgery, from a single surgeon's practice, spanning an 11-year period from April 1995 to July 2006. Data were obtained from the patients' files as well as from our cardiac surgery database. Data regarding the timing and number of dialysis sessions were cross-referenced with our renal unit.

We divided the population under study into two groups: group 1, from April 1995 to January 2000, comprised 15 patients and group 2, from January 2000 up to July 2006, comprised 56 patients. The two groups reflect our policy change with regard to the treatment of acute renal injury after cardiac surgery.

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We obtained the following data: the patient's age, history of diabetes, hypertension, prostatism, and use of ACE inhibitors, the preoperative baseline creatinine and urea, the percentage of patients with raised creatinine and urea as well as the peak values before dialysis was started; the potassium and pH values immediately before dialysis; the time lapse from surgery to dialysis and from the onset of oliguria (<0.5 ml/kg/h) to dialysis, and the number of sessions of dialysis administered. These values are recorded in Table 1. Statistical significance was accepted at a level of $P < 0.05$.

Our policy with regard to treatment of renal injury changed with our understanding of this complex clinical problem. Initially, renal support measures were instituted and utilised to the full, only reserving renal replacement therapy as a last resort when it became impossible to correct hyperkalaemia. This policy was based on our untoward early experience with dialysis and the high mortality related to this treatment modality in our unit. It later became apparent that our poor results may have resulted from the late institution of dialysis, and we therefore embarked on a programme of early dialysis to test this hypothesis.

The renal support measures were employed when oliguria, defined as <0.5 ml/kg/min urine output, set in. These included fluid replacement when the central venous pressure permitted, intravenous loop diuretics, in single doses followed by infusion, osmotic diuretics, low dose dopamine as a diuretic, other inotropes as indicated by the clinical condition, dextrose-insulin infusion and enteral calcium resonium, in varying combinations. In group 2, dialysis was commenced immediately when oliguria occurred and did not respond to fluid replacement and single dose intravenous diuretics (limited renal support measures). In two cases renal replacement therapy was required for correction of hyperkalaemia in the absence of oliguria.

3. Results

During the study period from April 1995–July 2006, 71 out of 3528 (2.01%) cardiac surgery patients were treated with dialysis postoperatively: 15/1511 (0.99%) patients in group 1 from April 1995 to January 2000 and 56/2017 (2.78%) patients in group 2 from January 2000 to July 2006. Full renal support measures were instituted in 34/1511 (2.25%) in group 1 and limited renal support measures in 31/2017 (1.54%) in group 2. Thus, the use of renal support measures declined in favour of an increase in the use of dialysis (Fig. 1).

3.1. Demographics

Groups 1 and 2 did not differ significantly in age distribution, incidence of diabetes, hypertension or prostatism. The preoperative use of ACE inhibitors was not statistically different in the two groups, with a trend away from enalapril in favour of perindopril.

3.2. Biochemical parameters

The mean preoperative creatinine and urea as well as the percentages of patients with raised preoperative values were not significantly different.

The pre-dialysis creatinine was significantly lower in group 2 (403.7 ± 183.5 $\mu\text{mol/l}$ vs. 233.9 ± 73.7 $\mu\text{mol/l}$, $P = 0.003$) as was the pre-dialysis urea (35.2 ± 17.7 vs. 14.4 ± 3.1 mmol/l, $P = 0.004$) as a result of the institution of early dialysis. The pre-dialysis potassium was similar in the two groups. Early dialysis prevented the development of metabolic acidosis.

3.3. Timing and number of sessions of dialysis

The time lapse from surgery to dialysis was significantly reduced in group 2 (from 139.1 ± 137.1 h to 41.6 ± 36.2 h,

Table 1
Demographics, biochemical parameters, timing and number of sessions of dialysis, and mortality in groups 1 and 2

	Group 1	Group 2	P-value	
			t-test	χ^2 -test
Age (years)	63.7 ± 10.9	66 ± 9.1	0.49	–
Diabetes	47%	47%	–	0.92
Hypertension	60%	63%	–	0.76
Prostatism	13%	9%	–	0.61
ACE inhibitors	27%	31%	–	0.78
Enalapril	20%	11%	–	0.34
Perindopril	7%	20%	–	0.23
Pre-op creatinine ($\mu\text{mol/l}$)	111.6 ± 57.0	118.1 ± 42.2	0.69	–
Pre-op urea (mmol/l)	8.5 ± 3.8	10.0 ± 3.6	0.20	–
Creatinine > 100 $\mu\text{mol/l}$	40%	55%	–	0.20
Urea > 10 mmol/l	33%	40%	–	1.0
Predialysis creatinine ($\mu\text{mol/l}$)	403.7 ± 183.5	233.9 ± 73.7	0.003*	–
Predialysis urea (mmol/l)	35.2 ± 17.7	14.4 ± 3.1	0.004*	–
Predialysis K ⁺ (mmol/l)	5.3 ± 0.8	4.9 ± 0.5	0.13	–
Predialysis pH	7.256 ± 0.08	7.353 ± 0.06	0.0004*	–
Surgery to dialysis (h)	139.1 ± 137.1	41.6 ± 36.2	0.015*	–
Oliguria to dialysis (h)	41.2 ± 22.8	8.6 ± 8.2	0.000064*	–
Number of dialysis sessions in survivors	6.5	1.8 ± 0.9	0.684	–
Survival	13%	75%	–	0.0000125*

*Denotes statistical significance.

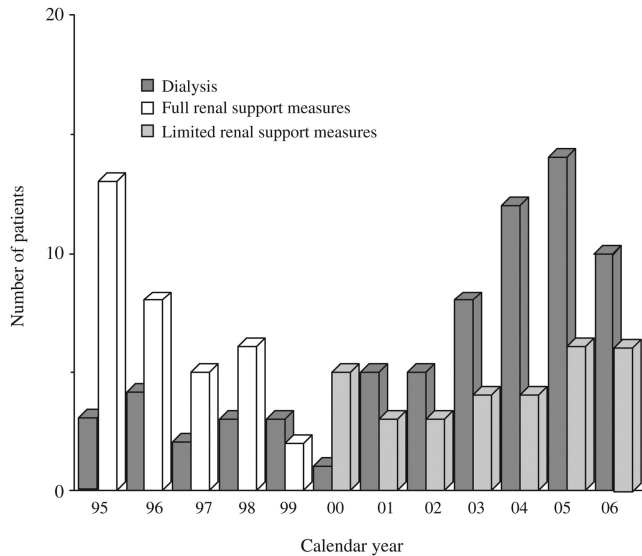


Fig. 1. Management of acute kidney injury, showing number of patients treated per year in this study with differing renal support.

$P=0.015$) as was the period from the onset of oliguria to dialysis (from 41.2 ± 22.8 h to 8.6 ± 8.2 h, $P=0.000064$). In group 1 the two dialysis survivors required 7 and 6 sessions, respectively, before recovery. In group 2 the 42 dialysis survivors required a mean of 1.8 ± 0.9 dialysis sessions before recovery.

3.4. Mortality after dialysis

In group 1, prior to January 2000, 13 of the 15 patients who received dialysis after cardiac surgery died peri-operatively, giving a mortality of 87%. In group 2, from January 2000 until July 2006, 14 out of 56 patients died after receiving dialysis, giving a mortality of 25%. If we look at our results from the last two years of the study, only 4 out of 24 patients who received dialysis died peri-operatively, and so the mortality has recently dropped further to 17%. There has been a significant and steady reduction in mortality as we have pursued our policy of early dialysis.

4. Discussion

Our management of acute renal injury after cardiac surgery has evolved over the study period. A policy decision was taken in January 2000 to perform early dialysis in an attempt to decrease the high mortality that was prevalent up to that time. This has resulted in an almost three-fold rise (0.99–2.78%) in the use of dialysis. During this same period our combined mortality (following renal supportive measures and dialysis) fell from 39% to 16%, a 2.4-fold reduction. In the latter part of the study, from January 2005 to July 2006 ($n=445$), the combined mortality following limited renal support measures only (administered in 12 patients) and early dialysis (administered in 24 patients) was 11% (4/36), representing a 3.5-fold reduction. This policy has encouraged us to accept patients for surgery who would otherwise have been refused in the past because of impaired renal function. Dialysis is now started as soon

as oliguria ceases to respond to limited renal support measures. A double lumen catheter is inserted in the femoral vein using local anaesthetic and a 4 h dialysis session performed via this route. The commonest time for this to happen is during the first and second postoperative days (median 32 h). Additional support measures at this stage such as increasing inotropic support, further diuretic treatment or a dextrose insulin infusion are unlikely to result in renal recovery. Our results demonstrate that although full renal support measures may correct the serum potassium levels to a degree (5.3 ± 0.8 vs. 4.9 ± 0.5 mmol/l, $P=0.13$), patients in group 1 were significantly acidotic, a situation avoided in group 2 (pH 7.256 ± 0.08 vs. 7.353 ± 0.06 , $P=0.0004$). The consequent delay in dialysis which resulted from this policy as practised in group 1 resulted in a high mortality.

When dialysis is administered in the first 24 h post-operatively it is our policy to avoid a net removal of fluid. After this time a 2 l reduction is normally well tolerated. We do not fully anti-coagulate the patient with heparin but infuse a dose of 5000 U into the dialysis circuit. We invariably dialyse against a potassium concentration of 1 mmol/l. The occurrence of atrial fibrillation is not uncommon and we have also observed this event to herald the early resumption of a diuresis. Renal recovery occurs early following this strategy, with a mean of 1.8 ± 0.9 dialysis sessions in group 2 survivors. One may argue that we are perhaps instituting dialysis in patients who may not require it and may go on to recover spontaneously. In patients who are still passing urine in adequate quantity, a rising creatinine is not, in itself, an indication for renal replacement therapy. However, when the patient becomes oliguric, anuric or hyperkalaemic in spite of a normal diuresis there is no advantage in delaying renal replacement therapy. Our experience from group 1 patients prior to January 2000 clearly demonstrates the dangers of delay. Our policy of early dialysis has also widened our scope for revascularisation in patients with preoperative renal impairment in whom surgery would, in many cases, have been denied prior to January 2000.

When acute kidney injury presented as part of multi-organ failure, our experience with dialysis was uniformly unsuccessful. This clinical picture always included cardiogenic or septic shock, singly or in combination. Multi-organ failure was the cause of death of 9 of the 13 deaths in group 1 and in 9 of the 14 deaths in group 2. The incidence of this condition and the poor outcome has remained unchanged over the study period and early dialysis in this situation has not improved our results. Furthermore, our results do not support the notion that early dialysis may prevent the progression from isolated kidney injury to multi-organ failure. Although the use of early dialysis has increased three-fold, the mortality from dialysis in the setting of multi-organ failure has remained unchanged. In a recent paper by Tillyard et al. [5] the authors concluded that acute kidney injury in intensive care is a mongrel and not a pedigree diagnosis and serves only to identify when another organ has failed. Acute kidney injury as a single system failure is a distinct entity and carries a vastly different prognosis from that of acute kidney injury as part of a syndrome of multi-organ failure.

The jury is still out on which renal replacement therapy is the most appropriate [11, 12]. We have gained experience with haemodialysis having encountered early coagulation problems with the use of continuous veno-venous dialysis. Clearly each unit will utilise the method which they have gained more experience with and which best suits their needs. Our results are comparable with those obtained with continuous veno-venous dialysis.

Whereas ten years ago the onset of postoperative acute kidney ushered a sense of doom, our experience with early dialysis has changed this outlook and renal replacement therapy has become simply another modality of postoperative care in the cardiac patient.

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