The role of hypotension in the development of acute renal failure

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Introduction

Acute renal failure in critically ill patients is common and is associated with high morbidity and mortality rates [1]. When defined as a serum creatinine of 300 µmol/l (3.5 mg/dl) or more and/or a urine output of <500 ml/day, acute renal failure was shown to affect almost 25% of patients in the ICU [2]. Using the RIFLE criteria, studies have reported that between 10 and 67% of ICU patients have acute kidney injury [3]. Moreover, by increasing the hospital length of stay [2,4,5] and by inducing the need for renal replacement therapy, acute renal failure has substantial economic consequences [5,6].

Acute renal failure is typically multifactorial. The classical classification of renal diseases seen in any nephrology textbook does not apply to the vast majority of critically ill patients, who usually develop what is called 'acute tubular necrosis', even though the term is a misnomer as few of the tubular cells are actually necrotic [7]. The typical course of acute renal failure in a severely ill patient involves several factors including sepsis with hypovolaemia, (possibly transient) hypotension and administration of nephrotoxic therapeutic agents and/or contrast material, in a patient with co-morbidities such as diabetes and arteriosclerosis [2,8].

In this issue of *Nephrology Dialysis and Transplantation*, Liu and co-workers report that development of acute renal failure is usually preceded by an episode of relative hypotension [9]. The authors retrospectively selected two cohorts of patients, one with acute kidney injury as defined by the RIFLE criteria and one without. Blood pressure readings were obtained for 3 days prior to the development of acute kidney injury and for a similar 3-day period in patients without renal failure, and compared to baseline values or population norms where baseline values were unavailable. Interestingly, the hypotensive episode preceding the development of acute renal failure did not need to be severe, indeed patients with systolic pressures of <90 mmHg were excluded from the study. Importantly, in multivariable analysis, a decrease in systolic blood pressure relative to pre-morbid values was independently associated with the occurrence of acute renal failure.

Even with the limitations of this study (especially its retrospective, single-centre nature) acknowledged by the authors, these data are credible and, in fact, not so surprising. The authors are correct in stating that ‘normotensive renal failure’ is in fact quite a rare phenomenon if one monitors arterial pressure closely, as was the case in this study. It would have been interesting to also collect data on cardiac output, as these measurements may be more sensitive than blood pressure measurements for the detection of transient ischaemic challenges to the kidneys. To quantify the severity of acute renal failure, the authors used the RIFLE criteria, which include relative increases in creatinine concentration. We believe that it would have been interesting to report the time course of the sequential organ failure assessment (SOFA) score [10] as a means of following the changes in renal function (as of other organs) over time.

A key question is whether the apparent link between relative hypotension and acute renal failure is a simple association or a cause-and-effect relationship. Although a cause-and-effect relationship is possible, other mechanisms may be responsible for the apparent link. For example, sepsis is a major contributor to the development of acute renal failure in critically ill patients [2,8,11] and is also often associated with hypotension. In the present study by Liu et al. [9], the number of patients with sepsis is not provided, only that about one half of the patients had a ‘vasodilatory state’.

If we decide to accept that there is a cause-and-effect relationship, does it actually matter? If hypotension, even relative, does indeed cause, or increase the risk of, acute renal failure, then maintaining systolic blood pressure at pre-morbid values would become a key target in the prevention of acute renal failure. But is therapy tailored to pre-morbid blood pressure values a valid and feasible approach to managing patients at risk? How could such an approach be instituted? First, correction of hypovolaemia...
would obviously be of paramount importance. In the study by Liu et al. [9], hypovolaemia was identified in only 20% of patients, but identifying hypovolaemia retrospectively is difficult so this number may not be accurate. In the clinical situation, assessing ongoing fluid needs and responsiveness is complex as physicians balance the risks of fluid overload with those of hypovolaemia. Second, should vasopressors be used to restore baseline blood pressure or limit the risks of hypotension? This is a controversial area but there is no good evidence that liberal use of vasopressors decreases the incidence of acute renal failure. Finally, what level of arterial pressure should be chosen as the target? Finding one value that could be applicable to all patients would be difficult, perhaps such a value should be related to the patient’s own baseline blood pressure value which may be unknown.

Clearly, even if relative hypotension does participate in the development of acute renal failure, focusing primarily on arterial blood pressure as a target may be rather limited. Rather than providing a new goal, these observations by Liu et al. [9] are more a message supporting the importance of early, rapid and complete resuscitation in all patients.

Conflict of interest statement. None declared.

(See related article by Y. L. Liu et al. Changes in blood pressure before the development of nosocomial acute kidney injury. Nephrol Dial Transplant 2009; 24: 504–511.)

References


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Which hypoglycaemic agents to use in type 2 diabetic subjects with CKD and how?*

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DCCT [1], UKPDS [2,3] and Kumamoto studies [4] clearly indicate that good glycaemic control can reduce the risk of nephropathy in subjects with both type 1 and type 2 diabetes. Furthermore, the recent ADVANCE trial confirmed that much tighter glycaemic control (mean HbA1c = 6.5%) is beneficial for nephropathy in subjects with type 2 diabetes [5]. In order to achieve tight glycaemic control, we would like to try to describe which hypoglycaemic agents to use in type 2 diabetic subjects with CKD and how, in this comment.

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