The Role of Nephrologist in the Intensive Care Unit

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Acute kidney injury · Chronic kidney disease · Dialysis · Progression · Complex metabolic and electrolyte disorders

Abstract
Participation by nephrologists is needed in most intensive care units, even when such units are ‘closed’. This participation should assist with diagnosis and management of intrinsic and complex renal diseases such as vasculitis, complex metabolic and electrolyte disorders including hyponatremia, and acute kidney injury (AKI) with and without underlying chronic kidney disease (CKD). Early nephrologist involvement will also facilitate transition to continuing care and follow-up after an episode of AKI, but may also assist in avoiding dialysis where treatment is futile. Management of AKI by intensivists should be in partnership with nephrologists to oversee and hopefully to minimize progression to CKD.

Introduction

The role of a nephrologist in the intensive care unit (ICU) has previously been discussed in the context of ICU format [1, 2]. Except in the United States, the closed model of intensive care is the most widely prevalent format, see summary by Vincent [3]. The general benefits and limitations of open and closed ICUs have been widely debated, and surprisingly remains controversial [1, 3–6]. However, they will not be elaborated here, except for highlighting that the role of nephrologist in the ICU will obviously depend on the ICU model. A closed ICU model has been the norm in large Australian and New Zealand Hospitals for several decades, so my comments are largely directed at the closed ICU model.

An expansion of the role of nephrologists in the closed ICU model appears desirable, but this may also be necessary for some other physician groups, for example, infectious disease physicians to assist with antimicrobial stewardship [7]. This should not be construed as seeking to open ICUs, but rather to recognize that the supervising intensivists, who regionally come from training pathways as either physicians or anaesthetists, may require nephrology support in areas in which they do not have relevant training experience, such as complex metabolic or electrolyte disorders. With a few exceptions, intensivists in our ICUs are responsible for using continuous modes of dialysis, while machines for intermittent dialysis and trained dialysis nurses are provided and supervised by nephrologists. Also, local practice and geography (rural versus urban) play a role. The nephrologists in US ICUs provide renal replacement therapy and assist in the diagnosis and treatment of complex acid-base disorders [8], however, in Australia and New Zealand, most ICUs independently commence dialysis with continuous venous hemodiafiltration, and nephrologists are consulted when intermittent dialysis is required in the ICU or after
discharge from the ICU. The timing of dialysis is thus important. However, the decision to dialyse is one in which nephrologists should participate, since they may ultimately need to continue dialysis after discharge from ICU, and neither short- nor long-term dialysis should be undertaken where this exercise may be considered futile [9].

After being invited to write this article, I informally asked colleagues in my own hospital and also nephrologists from 7 other local hospitals for their views regarding the role of nephrologists in the ICU. The view shared by nephrologists from 7 hospitals was that the intensivists provided a high standard of care but the quality and timing of communication with nephrologists was suboptimal. Such communication is highly dependent on individuals concerned rather than protocol driven, and often limited to the time of transfer of care of patients needing ongoing intermittent dialysis. Many nephrologists expressed concern that dialysis was regularly initiated in ICU for reasons that were either controversial, such as acidosis, or not consistent with the current teaching or guidelines. These nephrologists were aware of, but not participants in, the current debate regarding early initiation of dialysis [10–12].

Most nephrologists expressed the view that they would like to be more involved, and involved early in the care of patients who were likely to come eventually under their care. Where existing nephrology patients with chronic kidney disease (CKD) or a kidney transplant were admitted to ICU, more timely communication is usually the norm. My comments focus on patients developing acute kidney injury (AKI) in the ICU. With over 30 years of clinical experience and research interest in AKI, I am keenly appreciative of the concerns raised.

<table>
<thead>
<tr>
<th>Table 1. Potential roles for the nephrologist in ICU</th>
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<tr>
<td><strong>AKI</strong></td>
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<td>Differential diagnosis</td>
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<td>Oversight investigation of aetiology</td>
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<td>Oversight some management, especially</td>
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<td>Immune-mediated disorders (vasculitis, glomerulonephritis etc.)</td>
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<td>Complex metabolic and electrolyte disorders</td>
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<td>Identify where dialysis is futile</td>
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<td>Manage transition to discharge including dialysis</td>
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<td>Identify recovery of renal function</td>
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<td>Follow-up after discharge</td>
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<td>Promote research</td>
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<td><strong>CKD</strong></td>
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<td>Preserve function</td>
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<td>Oversight management – identify nephrotoxins</td>
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<td>Follow-up after discharge</td>
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**Nephrologist Involvement in AKI (Table 1)**

Overall, most AKIs occur outside the ICU and dialysis is also frequently initiated for AKI outside the ICU. In this location, the National Confidential Enquiry into Patient Outcome and Death study in the UK reported that 43% of the patients had an improper delay in diagnosis of post-hospitalisation AKI [13]. Dialysis-dependent AKI in the presence of other organ failure usually begins or ends in the ICU [13]. Most nephrologists expressed a desire to be involved as early as possible in the care of ICU patients where dialysis was contemplated, regardless of whether such patients needed dialysis and the dialysis modality. On the contrary, in the 1980s and 1990s most nephrologists in this region were comfortable with intensivists looking after AKI associated with multiorgan failure, especially given the absence of any successful therapy for intervention. However, this has undergone a change.

There has been a slowly accelerating interest in AKI among nephrologists following, first, the harmonization of AKI definitions by the Acute Dialysis Quality Initiative and Acute Kidney Injury Network groups leading to the current Kidney Disease: Improving Global Outcomes (KDIGO) definitions [14]. The clarity of definition has increased the awareness of the global burden of AKI [15, 16]. Second, the realization that biomarkers of kidney damage usually preceded renal functional decline and could potentially facilitate early intervention in AKI has sparked nephrologist and industry interest [17–19]. The suggested incorporation of damage biomarkers into standard definitions of AKI [20] awaits agreement regarding the context-specific thresholds for such biomarkers and
the need for validation in relevant clinical scenarios [21]. Nevertheless, US Food Drug Administration approval of a device to measure the combination of tissue inhibitor of metalloproteinases-2 (TIMP2) and insulin-like growth factor-binding protein 7 (IGFBP7) to predict the progression of AKI is a much needed first step to incorporation of such damage biomarkers into routine clinical practice [22] and L-type fatty acid-binding protein, neutrophil gelatinase-associated lipocalin are approved for use in some jurisdictions [21].

Increases in renal injury biomarkers provide evidence that renal damage is occurring and highlight the need to investigate and intervene earlier in AKI than permitted by serum creatinine-based changes. The short- and long-term mortality associations of even stage 1 AKI have been extensively corroborated [16, 23]. These mortality associations and remote organ injury to heart, lung and brain are real and probably represent direct injury with active participation of innate immunity, mediated through some combination of the damage/danger-associated molecular pattern molecules and pathogen-associated molecular pattern molecules (e.g. [24, 25]). There is an urgent need to recognise AKI as a disease with systemic manifestations [25], and not just accept the old view of AKI as a syndrome of multiple causation associated with logical and temporary reduction of the glomerular filtration rate (GFR). This mimics and probably contributes to the clearly bidirectional relationship between cardiac disease and CKD. While ischaemia-reperfusion injury and sepsis are the most common causes of AKI in both ICU and other hospitalized patients, other common causes of AKI need to be identified, including drug toxicity and vasculitis. While notable groups of intensivists have driven research in some of these domains, the role of nephrologists should be to oversee and drive differential diagnosis, to help define critical investigation and further promote research (table 1).

There is now an increasing awareness that AKI is a significant precursor of CKD and that CKD is the most critical risk factor for AKI. Not only are AKI and CKD interconnected by the same general risk factors, precipitants and prognosis [26], they may really be an integrated syndrome characterized by reduced GFR with acute and chronic stages. Subjects are at risk of CKD and death, even those with apparently normal prior renal function and with apparently complete recovery after an episode of AKI [27]. There is, therefore, a critical need for early and long-term nephrology follow-up after any episode of AKI (table 1). AKI is inadequately documented and follow-up rates are poor. For example, a study examining chart documentation after a major surgery showed that AKI may be appropriately documented in as few as 16% of patients based on serum creatinine [28]. This is consistent with anecdotal evidence from subjects discharged from ICU who remain unaware that an episode of AKI has occurred. Formal documentation of AKI during admission is associated with increased rates of nephrology consultation (31 vs. 6%), and with reduced mortality 30 days after discharge (OR 0.81, 95% CI 0.68–0.96, p = 0.02) [29]. These data suggest that nephrology consultation should be encouraged for AKI occurring both within and outside the ICU.

In reality, the majority of nephrology consultations within the hospital and outside the ICU are for AKI. It is appropriate that we focus on harmonising the notification, investigations and first response to detection of ‘renal angina’, the name currently given to the category of patients at high risk of AKI [30], across the population of hospitalized patients including those in the ICU. The way forward is suggested by the response in the UK, where eAlerts, guidelines for investigation and intervention have been mandated [13, 16]. Initial treatment is suggested in the KDIGO guidelines [31]. Additional investigations that predict progression to more severe stages of AKI need to be validated in large cohorts. Investigations should include urinary biomarkers, such as but not limited to IGFBP7 and TIMP2 [22], the furosemide stress test in patients with an indwelling catheter [32] and probably kinetic GFR [33, 34]. Given the lack of insight into the pathophysiology of the early phase of injury in human AKI, we need to encourage critical investigations before a modest increase in creatinine becomes so convincing as to be too late for useful intervention. The role of nephrologist within and outside the ICU should be to argue for such investigations. Together with intensivists, we must drive future research on AKI.

**Disclosure Statement**

The author declares no conflicts of interest.

**References**


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