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Newphrologist: The Intensive Care Kidney Specialist

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EDITORIAL

Newphrologist: The Intensive Care Kidney Specialist

The tsunami of CKD has now possibly generated an even greater wave of acute kidney injury (AKI) that is sweeping over the world. The causes and frequencies of AKI differ markedly between developed and developing nations, but the wave of AKI continues to rise.[1] The pre-eminent risk factor for the development of AKI is CKD, and older patients are at risk for multiple reasons.[2] The elderly have less kidney regenerative capacity and often have chronic medical conditions that render them more greatly susceptible to kidney insults, such as ischemic heart disease. Notably, an episode of AKI may follow several courses: (1) complete recovery, (2) progressive CKD, (3) worsening of the rate of progression of pre-existent CKD, and (4) evolution to ESRD.[2] In fact, the number one reason why a patient is initiated onto dialytic therapy today in the United States is AKI, and AKI is common in intensive care units, where nephrologists now spend an ever-increasing amount of time as consultants.

This issue of *Advances in Chronic Kidney Disease* underscores a call to action for nephrologists who ply their tools of trade in intensive care units. The average nephrologist devotes nearly half of the working day to hospitalized patients, and a disproportionate amount of this time is spent in intensive care units. The most common reasons for the appearance of kidney specialists in intensive care units include AKI, particularly in septic patients, hypertension, electrolyte and acid-base disturbances, and patients with ESRD and a host of distressing conditions, including hemorrhage, cardiac failure, myocardial ischemia, pulmonary edema, postsurgical recovery, and infection-related problems. As such, nephrologists must become facile with the tools of intensivists, whose numbers now include experts in infectious disease, neurology, surgery, anesthesiology, and emergency medicine. Moreover, nephrologists must collaborate with intensivists to provide cohesive and coherent care plans because critically ill patients cannot be treated in isolation, as a multitude of other factors impinge on their health.

Sepsis is the most common cause of AKI and AKI is a harbinger of sepsis.[3] Therefore, recognition of sepsis is paramount followed by its treatment with antibiotics. An accrual of knowledge about newer antibiotics and their respective dosing regimens, especially those reserved for more exotic infections and those that are intrinsically nephrotoxic, must be achieved firsthand. In addition, systematic avoidance of nephrotoxic antibiotics must be practiced, since such nephrotoxins may prove injurious when combined with other injurious renal exposures, such as radiocontrast media. Protocolized avoidance of nephrotoxins, especially iodinated radiocontrast media, requires collaborative leadership and input from nephrologists at pharmacy therapeutics committees. This knowledge must be judiciously distributed among other involved health care personnel, pharmacists, and the electronic health record, with fault-free dispersal through computerized order entry systems, given the emerging specter of stage 2 meaningful use criteria that begin in 2013.[4] These criteria encourage information technology in health care as a driver of continuous quality improvement at the point of care and information exchange in a highly structured format.

AKI runs rampant in intensive care units and has multiple and often overlapping causes. AKI is frequently complicated by a reduction of uremic solute clearance and/or urine output to the extent that renal replacement therapy is necessary. The intensivist-nephrologist must now be conversant in continuous renal replacement therapies because intermittent hemodialysis may not be feasible in hypotensive individuals, especially those who become vasopressor-dependent for several days or more. Whether using continuous venovenous therapy as purely hemofiltration or as hemodiafiltration, the

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critical care kidney specialist must appreciate not only the positive aspects of continuous therapy but also the negative ones, including net negative potassium and phosphorus balance,[5] as well as reductions in vasopressor concentrations and antibiotic concentrations. With regard to the latter, continuous venovenous therapy may offer the intensive care pharmacy an easier pathway to effective antibiotic dosing,[6] compared with intermittent dialysis or sustained low-efficiency dialysis, unless the latter modality is on a continuous basis as is possible with regional citrate anticoagulation.[7] Recently, sustained low-efficiency dialysis/regional citrate anticoagulation has been proved effective and safe in individuals with severely compromised hepatic function. Furthermore, patients with portal-systemic encephalopathy or cerebral edema from other causes who are at risk for impending cerebral herniation may benefit from continuous therapies.

The effective circulatory volume can be manipulated, as can the prevailing tonicity in which the swollen brain is bathed. The manipulation of serum sodium with renal replacement therapy[8] may now supplant or augment conventional osmotherapy[9] and is now within the purview of the nephrologist-intensivist who may now be called upon to enact treatments for brain shrinkage. Likely, consultations requesting these treatments will increase. Even if armed with more advanced techniques of renal replacement therapy, the nephrologist-intensivist must now acknowledge that the determination of "volume status" is ever more difficult in desperately ill individuals. Protocolized volume resuscitation algorithms have proved worthy but sometimes at a cost.[10]

Volume or fluid overload has become the rule rather than the exception. The nephrologist must exercise judgment jealously on behalf of the patient because fluid overload is associated with worse outcomes, including increased intubation and ventilator times, suboptimal recovery from AKI, and mortality.[11] However, this judgment must be annealed to more modern and sophisticated techniques of volume assessment, such as inferior vena cava ultrasonography that demonstrates luminal obliteration, central venous pressure monitoring in selected cases, and dynamic measurements of stroke volume changes during mechanical ventilation that use Doppler ultrasonography, pulse contour analysis, and bioreactance measurements.[12] These data, in combination with traditional measures of perfusion (vital signs, arterial blood gas and lactate measurements) may prove more informative than clinical "guesstimation." Finally, a simple maneuver such as the assessment of fluid responsiveness to passive leg raising may prove rapidly and clinically useful.[13] Unlike the aforementioned methods, this subtle and sublime technique is also exceedingly inexpensive.

Recognition of when fluid overload has occurred is a minimal requirement, but preventing it is better. Prevention requires not only an assessment of fluid inputs and outputs but also much more careful analysis: the calculus of mass balance and electrolyte–free water determination, with appropriate forecasting.[14,15] Notably, no large scale, commercially available electronic health care record carries out these calculations in a truly meaningful fashion that amplifies care of the patient. All account for fluid inputs and outputs, colloids, and blood products as equivalents, and 10 L of 5% dextrose in water is ranked equally with 10 L of 0.9 saline solution. Ionic compositions are systematically ignored, especially with regard to enteral and parenteral nutrition solutions, with brutal consequences for patients. Patients may be overly ultrafiltered by nephrologists who fail to perform such mass balance. Conversely, intensivists who ignore mass balance may induce dysnatremias with or without fluid overload. Lastly, simple clinical acuity cannot be forsaken. Otherwise, differentiation of AKI from an abdominal compartment syndrome[16] with elevated bladder pressure, right-sided heart failure, or hepatic sinusoidal hypertension will not occur.

Certainly, the nephrologist's scope of business has been forcefully expanded in conjunction with the enlarging critical care space. This has provoked the evolution of a new brand of nephrology and nephrologist. This discipline, "newphrology," welcomes and actively engages the nephrologist as an intensive care kidney specialist. These individuals will increase in number and take their place among those nephrologists who became disciples of other former "new" disciplines of nephrology, including kidney transplantation and interventional nephrologists. These "newphrologists" are favorably positioned to participate in and impact patient care and research in medical, surgical, neurosurgical, and pediatric intensive care units. These individuals will be dedicated to breaking the wave of AKI through the earlier recognition, mitigation of aggravating factors, and treatment of AKI—the selfsame mantra of nephrologists who treat CKD. In fact, they are already doing so as delineated by the series of articles in this issue of *Advances in Chronic Kidney Disease*, as collated by our Guest Editor, Kathleen Liu, herself a nephrologist-intensivist.

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References

1. Cerda J, Lameire N, Eggers P, et al. Epidemiology of acute kidney injury. *Clin J Am Soc Nephrol*. 2008;3:881-886.
2. Anderson S, Eldadah B, Halter JB, et al. Acute kidney injury in older adults. *J Am Soc Nephrol*. 2011;22:28-38.
3. Uchino S, Kellum JA, Bellomo R, et al. Acute renal failure in critically ill patients: a multinational, multicenter study. *JAMA*. 2005; 294:813-818.
4. Centers for Medicare & Medicaid Services. Medicare and Medicaid programs: electronic health record incentive program—stage 2;

proposed rule. Washington, DC: US Department of Health and Human Services. *Fed Regist.* 2012;77:1-133. Available at: http://www.gpo.gov/fdsys/pkg/FR-2012-03-07/pdf/2012-4443.pdf. Accessed October 24, 2012.
5. Palevsky PM, Zhang JH, O'Connor TZ, et al. Intensity of renal support in critically ill patients with acute kidney injury. *N Engl J Med.* 2008;359:7-20.
6. Mueller BA, Scoville BA. Adding to the armamentarium: antibiotic dosing in extended dialysis. *Clin J Am Soc Nephrol.* 2012;7:373-375.
7. Szamosfalvi B, Frinak S, Yee J. Automated regional citrate anticoagulation: technological barriers and possible solutions. *Blood Purif.* 2010;29:204-209.
8. Bender F. Successful treatment of severe hyponatremia in a patient with renal failure using continuous venovenous hemodialysis. *Am J Kidney Dis.* 1998;32:829-831.
9. Berger S, Schurer L, Hartl R, et al. Reduction of posttraumatic intracranial hypertension by hypertonic/hyperoncotic saline/dextran and hypertonic mannitol. *Neurosurgery.* 1995;37:98-107.
10. Rivers E, Bryant Nguyen B, Havstad S, et al. Early goal-directed therapy in the treatment of severe sepsis and septic shock. *N Engl J Med.* 2001;345:1368-1377.
11. Heung M, Wolfgram DF, Kommareddi M, et al. Fluid overload at initiation of renal replacement therapy is associated with lack of renal recovery in patients with acute kidney injury. *Nephrol Dial Transplant.* 2012;27:956-961.
12. Marik PE, Monnet X, Teboul J-L. Hemodynamic parameters to guide fluid therapy. *Ann Intensive Care.* 2011;1:1.
13. Benomar B, Ouattara A, Estagnasie P, et al. Fluid responsiveness predicted by noninvasive bioreactance-based passive leg raise test. *Intensive Care Med.* 2010;36:1875-1881.
14. Davids MR, Edoute Y, Mallie JP, et al. Body compartment volumes and composition after giving a vasopressin antagonist: changes are revealed by a tonicity balance. *Nephrol Dial Transpl.* 2002;17:300-303.
15. Shafiee MAS, Bohn D, Hoorn EJ, et al. How to select optimal maintenance intravenous fluid therapy. *QJM.* 2003;96:601-610.
16. Cheatham ML. Intraabdominal pressure monitoring during fluid resuscitation. *Curr Opin Crit Care.* 2008;14:327-333.