“The higher the abdominal pressure, the less the secretion of urine”: Another target disease for renal ultrasonography?

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D r. Barozzi and colleagues have written an excellent manuscript describing the use of bedside ultrasound to assess and direct the early treatment of the critically ill with acute renal failure (ARF) (1). These techniques should be welcomed by the critical care community, because the current treatments for these patients leave much to be desired. Despite much attention to the prevention and management of postinjury or surgical ARF, mortality continues to exceed 50%, even with the institution of continuous renal replacement therapies (2–4). Isolated acute renal failure is rare in the intensive care unit (ICU), with most renal failure occurring in the setting of sepsis and multi-organ failure, or severe medical conditions, and after surgical procedures (5). One related condition that has not been mentioned in this manuscript, however, is intraabdominal hypertension (IAH) and its manifestation as the abdominal compartment syndrome (ACS). These conditions are important to consider given our current practices in the ICU.

Despite controlled evaluations of many potential renal-protective strategies, the only proven management strategies for the prevention of postoperative renal failure are adequate volume expansion and avoidance of hypovolemia (3, 4). Current management of both sepsis and traumatic injury also emphasizes early aggressive fluid resuscitation (6, 7). It has long been understood that prompt and adequate resuscitation of hypovolemia and shock is critical to avoid prerenal causes and to minimize the ischemia of renal causes such as acute tubular necrosis (ATN). The recognition that the shock state necessitated an obligatory loss of fluid to the intracellular, or “third,” space represented a seminal advance in trauma care that has saved numerous lives, and has all but eliminated renal failure as a complication of burn injury (8, 9). It is too simple, however, to simply administer excess quantities of fluids. Overly vigorous crystalloid resuscitation may complicate the ischemia/reperfusion consequences of critical illness and shock, culminating in excessive tissue edema, and often ascites, to produce pathologic raised IAH that itself induces renal dysfunction and failure (10–14). Renal dysfunction in this setting constitutes a diagnosis of ACS. The World Society of the Abdominal Compartment Syndrome has defined ACS as the presence of both an intraabdominal pressure (IAP) ≥20 mm Hg, with or without an abdominal perfusion pressure (APP) <50 mm Hg, recorded by a minimum of three standardized measurements conducted 1 hrs to 6 hrs apart and single- or multiple-organ system failure that was not previously present (15).

It has become apparent that IAH is both very common in the critically ill and also an independent cause of renal dysfunction (16). Over 100 yrs ago, Wendt reported, “The higher the abdominal pressure, the less the secretion of urine” (17). Despite this, the adverse influence of IAH on renal function is often unappreciated or discounted by clinicians. The subject is not mentioned in several of the recent review articles referenced on acute renal failure (5, 18). Nonetheless, the kidneys are particularly susceptible to IAH (19), presumably because of the serial nature of the renal vasculature (20). Thus, raised IAP may begin to effect renal function at only 10 mm Hg, and markedly impairs renal function at pressures as low as 20 mm Hg (21, 22). This low pressure threshold suggests that the kidney might be a potential marker to indicate adverse physiologic effects, thus prompting intervention at an earlier spectrum of disease.

Rates of pressure >18 mm Hg in post-surgical patients have ranged from 32% to 40% when looked for (14, 19, 23–25). A 1995 study reported that IAP measurements exceeded 20 mm Hg in 33% of postoperative patients when routinely studied. This level of IAP carried a 12- and 11-fold increased association of renal dysfunction and death, respectively (19). Biancofiore reported that 31% of postliver transplant patients who developed renal failure had significantly higher mean IAP (28 vs. 19 mm Hg) than those who did not. Another prospective study of 263 postoperative patients found that an IAP >18 mm Hg occurred in 40% of patients, and that renal impairment occurred in 32% of the group, being independently associated with increased IAP, as well as hypotension, sepsis, and older age (16). Further, the incidence of renal impairment appeared to be dose-related, with the incidence of renal impairment doubling at 25 mm Hg compared with 18 mm Hg (16). Intervening after renal dysfunction occurs may also be too late, and does not ameliorate the course of renal dysfunction. When critically ill patients (mean Acute Physiology and Chronic Health Evaluation II score of 27) with IAH were decompressed, both the IAP (mean IAP decrease from 24 to 14 mm Hg) and lung compliance (mean increase, 24–28 mL/cm H₂O) significantly improved (24). However, there was no overall improvement in hourly urine output, and the serum creatinine continued to worsen (24).
Dr. Barozzi and colleagues (1) have described the utility of a number of indices generated by studying vascular flow in the kidneys, the most studied evaluation of arterial spectral analysis being the resistive index (RI). There have been varied clinical and laboratory experiences suggesting that this index is either very useful (26–28) or quite unreliable (29, 30). It has emerged from further analysis of these seemingly paradoxical results that it is the intrarenal pressure per se that most affects the conductance and arterial distensibility of the renal vascular bed resulting in elevated resistive indexes, rather than the overall mean renal vascular resistance (31, 32).

The frequency of the association of IAH with ARF, coupled with the pressure sensitivity of the kidneys, suggests another potential role for renal Doppler sonography, that being the early bedside detection of vascular changes associated with ARF before established ARF. Using a porcine model of IAH, we have demonstrated that a linear relation between increasing IAP and RI exists, which may be described by a regression equation (33). This equation predicts an RI of 0.81 for an IAP of 25 mm Hg, an RI of major clinical significance as noted by Dr. Barozzi and colleagues (1). Congruous with the theme of their article, we feel that this is another example of an indication that holds promise, but that requires more extensive study before being considered for routine use. The typical critically ill patient is much more difficult to image than the anesthetized pig, but as mentioned, improvements in technique, technology, and information processing will continue to assist the clinician in assessment at the point of care.

REFERENCES