CHAPTER 38

Acute Kidney Injury in Burns and Trauma

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OBJECTIVES

This chapter will:

1. Provide an overview of acute kidney injury (AKI) occurring in the two major forms of external injury causing critical illness trauma and burns.
2. Review the incidence and clinical implications of AKI complicating major trauma and burns.
3. Examine the cause and pathogenesis of AKI in burns and trauma.
4. Discuss aspects of AKI management specific to burns and trauma.

TRAUMATIC ACUTE KIDNEY INJURY

Worldwide more than 5 million people die every year as a result of an injury. Road traffic accidents alone are the ninth leading cause of death projected to rise to the seventh by 2030.[1] Globally, young people between the ages of 15 and 44 years account for almost 50% of the world's injury-related deaths, lending trauma an even greater economic significance because it often affects healthy wage earners with dependents.[1] Although many trauma patients die immediately of their injuries, increasing expertise in prehospital care and centralized major trauma centers led to increasing numbers of the sickest patients surviving to reach the hospital, be admitted to the intensive care unit (ICU), and require multiorgan support. Currently major trauma accounts for 15% of intensive care unit admissions annually in the United States.[2] The management of traumatic multiorgan failure, including acute kidney injury (AKI), is likely to be a growing challenge for intensivists and nephrologists over the next 20 years as better prehospital care increases immediate survival.

The study of traumatic AKI has a central role in the history of critical care nephrology. The first recognition of acute renal injury as a clinical entity with descriptions of the clinical, biochemical, and histopathologic findings were made in victims of traumatic injury during World War II; in particular, the seminal paper by Baywaters and Beall described acute renal failure as a cause of death after crush injuries during the 1940 to 1941 London blitz.[3] Indeed, before the development of the specialties in nephrology and intensive care medicine, pioneering research on acute renal failure with a major focus on trauma and transfusion was led by surgeons and pathologists.[4,5]

EPIDEMIOLOGY OF ACUTE KIDNEY INJURY IN MAJOR TRAUMA

Over the last 10 years since the development of consensus diagnostic criteria, the incidence of AKI as a complication of major trauma in the ICU has been examined in a number of publications (Table 38.1). Overall, rates of AKI differ from 6% to 50%, reflecting a diversity in trauma ICU admissions between centers and countries. In almost all studies AKI is associated with increased mortality and length of hospitalization, data that agree with the authors' local data in an ICU serving a level 1 trauma center in London (see Table 38.1, Fig. 38.1). This association persists after adjustment for trauma severity and other major confounders. However, although most cases of AKI complicating trauma are stage 1, complications are more severe in stages 2 and 3 AKI, including those requiring renal replacement therapy (RRT), which account for around one third of all cases of traumatic AKI. These are associated most consistently with the highest risk of death.

As in other AKI settings (such as septic or post-operative AKI), these findings raise the argument as to what extent AKI is serving as a "better measure" of illness severity and physiologic reserve, and to what extent it is an avoidable risk factor that plays a causative role in clinical deterioration. Although stage 1 AKI may be more a marker of severe injury, acid-base and electrolyte abnormalities, fluid overload, and need for invasive vascular access and anticoagulation for RRT accompanying severe AKI are likely to contribute to risk of death and progression of multiorgan failure in major trauma patients, together with more subtle effects of AKI on systemic inflammation and organ cross-talk. Thus the prevention, recognition, and appropriate management of severe AKI is a key process in the care of critically ill trauma patients and a strong indicator of adverse outcomes.

CAUSE OF ACUTE KIDNEY INJURY AFTER MAJOR TRAUMA

Several studies have examined the risk factors for AKI defined by consensus criteria in trauma patients.[2,6–13] Interestingly, injury severity as measured by Injury Severity Score (ISS) is associated poorly with risk of AKI[7] except possibly in the most severe injuries (ISS > 40); conversely comorbid disease and physiologic instability at presentation are better predictors of AKI.[7] Specific factors that have been associated with the development of AKI include older age,[6,10,12] prior comorbid disease[10] (particularly diabetes mellitus[6,10,13]), black race,[13] hypothermia,[7] acidosis[12] or hyperlactatemia[7] at presentation, massive blood transfusion,[7,13] site of injury other than the brain,[6,9,13] and use of hydroxyethyl starch.[10] There is conflicting evidence on the effect of gender with reports of greater risk in women[6] or men[10] in or no effect of sex in various studies. However, the true effect of gender on AKI risk is something that is difficult to judge in the major trauma population, who is predominantly male and in whom mechanism of injury tends to vary greatly between male and female patients. Interestingly there is little evidence on the influence of intravenous (IV) contrast exposure[10,12] on the development

TABLE 38.1

Contemporary Studies Examining the Incidence and Associated Mortality of Acute Kidney Injury in Major Trauma Admitted to the Intensive Care Unit

AUTHOR	COUNTRY	PROPORTION WITH AKI	CRUDE HOSPITAL MORTALITY		ADJUSTED OR FOR DEATH WITH AKI	NUMBER RECEIVING RRT
			NO AKI	AKI		
Bagshaw 2008[6]	Australia, NZ	1711/9449 (18%)	7.8%	16.7%	1.8	ns
Bihorac 2010[7]	USA	255/982 (26%)	5%	32%	3.05	28/255 (11%)
Brandt 2007[8a]	USA	246/1033 (24%)	2.3%	24.4%	7.19	25/246 (10%)
de Abreu 2010[9]	Brazil	52/129 (40%)	98%	93%	ns	19/52 (36.5%)
Eriksson 2015[10]	Sweden	103/413 (25%)	5.8%	17.5%	ns	27/103 (26%)
Gomes 2010[11]	Portugal	217/436 (50%)	37%	22%	ns	0/217 (0%)
Podoll 2013[2]	USA	54/901 (6%)	9.2%	29.6%	3.41	10/54 (19%)
Prowle 2016[b]	UK	169/858 (20%)	18.1%	34.9%	2.43	43/169 (25%)
Shashaty 2012[12]	USA	147/400 (36%)	3.8%	14.9%	1.97	9/147 (6%)
Skinner 2013[13]	South Africa	102/666 (15%)	ns	57%	8.5	39/102 (38%)
Yuan 2009[84c]	China	423/3945 (11%)	7.1%	54%	ns	59/423 (13.9%)

[a]AKI definition non-standard.
[b]Author's local data.
[c]Road accidents only.
AKI, Acute kidney injury; *ns,* not stated; *RRT,* renal replacement therapy.

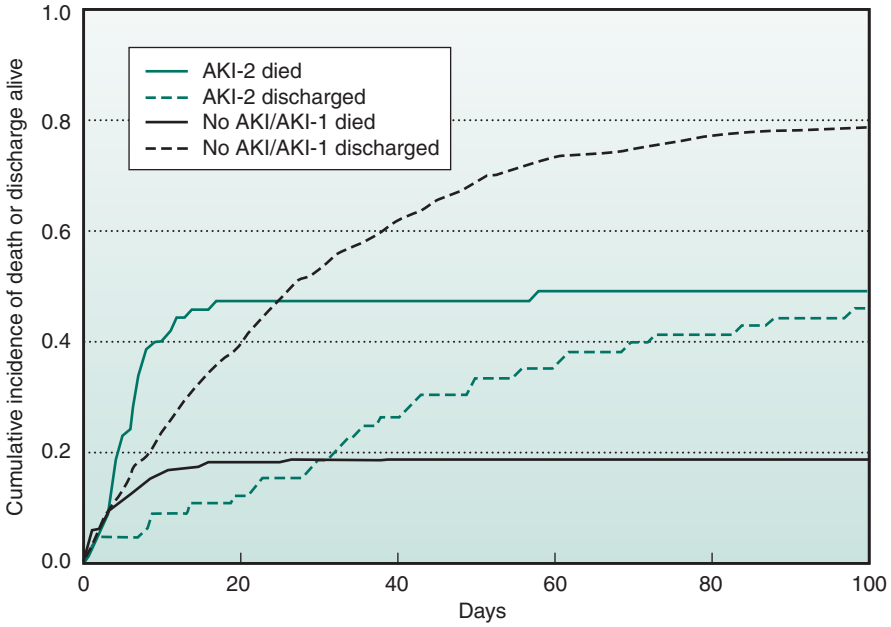

FIGURE 38.1 More severe acute kidney injury (AKI) is associated with significant increased early mortality and significantly delayed hospital discharge after major trauma. Data is provided from 8585 trauma intensive care unit (ICU) admissions during 2013 and 2014 in the author's institution.

of AKI, even though this is a near universal exposure as part of initial evaluation in major trauma centers in the developed world. Certainly there is no evidence to justify limiting contrast procedures (which are essential to the identification and management of occult hemorrhage) related to any fear of AKI in this population.

Overall acute kidney injury in trauma is usually multifactorial with specific risks for immediate, early, or late AKI occurring in combination (Table 38.2). Indeed the role of multiple causative factors in the causation of AKI in trauma has been well appreciated since the 1940s.[5] These risk factors may interact in complex fashion; for instance, severity of trauma and blood transfusion may result in secondary immunosuppression, predisposing to later infectious complications and risk of late AKI.[14] Given this interplay of causative factors, management of AKI risk in the trauma patient should focus on control of the primary source of injury and avoidance of secondary injury, particularly through minimizing avoidable nephrotoxin exposure and prevention of important complications such as sepsis.

RHABDOMYOLYSIS

Any consideration of trauma as a cause of AKI is incomplete without consideration of rhabdomyolysis and renal toxicity from myoglobin as a specific cause of AKI. Although typically regarded as an outcome of crush injury and/or the development of compartment syndromes, prolonged circulatory shock or ischemia reperfusion resulting from vascular injuries is also an important cause of muscle injury and rhabdomyolysis in trauma. Increasingly maneuvers to acutely mange bleeding, including the employment of temporary aortic occlusion devices and internal iliac artery

TABLE 38.2

Multifactorial Cause and Pathogenesis of Acute Kidney Injury After Major Trauma

FINDING	ETIOLOGY	PATHOPHYSIOLOGY	TIMING
Hypovolemic shock	Hemorrhage		
Extravasation of plasma water	Low cardiac output		
Tissue hypoperfusion			
Secondary reperfusion injury after resuscitation	Immediate		
Direct renal injury	Abdominopelvic trauma	May be directly to the kidney, to the lower urinary tract, or to the vascular supply to the kidneys	Immediate
Rhabdomyolysis	Crush injury		
Therapeutic embolization or vascular occlusion devices for bleeding	Hypovolemia		
Heme-pigment nephropathy			
Calcium phosphate			
Uric acid	Early		
Massive transfusion	Massive hemorrhage and consumptive coagulopathy	Heme-pigment nephropathy	
Systemic inflammation			
Immunosuppression	Early		
Coagulopathy	Consumptive coagulopathy	Systemic inflammation	
Microvascular thrombosis	Early		
Intraabdominal hypertension	Primary abdominal injuries requiring major surgery	Renal venous congestion, compression, and hypoperfusion	Early
Systemic inflammation	Direct and reperfusion tissue injury with release of damage-associated molecular patterns	Vasoplegia and systemic hypotension	
Endothelial activation and microvascular injury	Early-late		
Nephrotoxins	Need for emergent radiocontrast investigations and procedures		
Medications including antibiotics and nonsteroidal antiinflammatories	Direct nephrotoxic injury particular in combination with shock and systemic inflammation	Early-late	
Secondary sepsis	Trauma and transfusion induced immunosuppression	Sepsis-associated AKI	Late
Preexisting comorbidities	CKD, diabetes, chronic liver disease, heart failure	Preexisting AKI risk factors	Any

AKI, Acute kidney injury; *CKD,* chronic kidney disease.

embolization for pelvic bleeding, also may be intragenic causes of ischemic tissue injury and rhabdomyolysis in the major trauma patient. Thus rhabdomyolysis usually coexists with other AKI causes, including shock and renal ischemia, systemic inflammation, contrast exposure, and massive transfusion (which is also a source of free circulating heme). Thus, although a modestly raised creatinine kinase (CK) of more than 5000 IU/L (the most widely used clinical definition of rhabdomyolysis[15]), or an elevated serum or urinary myoglobin may correlate with the occurrence of AKI, they also correlate with the severity of trauma and a range of other renal insults so that direct contribution of rhabdomyolysis to AKI is often uncertain. In more profound rhabdomyolysis with admission CK levels more than 15 to 20,000 IU/L (that are in isolation more consistently associated with AKI[16]), a causative role for myoglobin toxicity in AKI is more certain; however, rhabdomyolysis of this level is a relatively rare occurrence among general trauma admissions who develop AKI (5% to 10% in the Royal London Hospital). The role of rhabdomyolysis is much more prominent in situations in which crush injuries predominate, such as earthquakes and blast injuries.[17–19] These mass-casualty situations involve complexities of search and rescue, advanced prehospital care, triage, and management of large patient numbers in the setting of damaged infrastructure[17,18] that are beyond the scope of this chapter.

Rhabdomyolysis[16] involves the dissolution of striped (skeletal) muscle either by direct sarcolemmic injury resulting from trauma or ischemia reperfusion and inflammation. Unregulated intracellular Ca^{2+} increase causes production of reactive oxygen species destruction of myofibrillar, cytoskeletal, and membrane protein and release of muscle cell contents, including myoglobin, phosphate, and potassium. Conversely fluid is lost into newly permeable muscle compartments via osmotic forces, causing systemic hypovolemia and secondary compartment syndromes within the swollen muscle compartment, further compounding injury. Pigment nephropathy occurs from the intravascular release and subsequent filtration of the heme-containing proteins hemoglobin and myoglobin.[20–25] These pigments are filtered freely and concentrated in the renal tubules. Although tubular obstruction with casts is observed, this is likely to be a consequence of low urine flow in AKI rather than the proximal cause of renal dysfunction, which is most related to direct toxic effects on the tubular epithelium.[24] Heme contains redox-active iron, which can stimulate lipid peroxidation and catalyze the formation of hydroxyl radicals, with heme-induced oxidant injury likely to be a consequence of lipid peroxidation via the formation of potent renal vasoconstrictors, F2-isoprostanes, and direct cellular injury.[24–26] Direct oxidative activity of free labile iron[27] and/or redox cycling between IV (ferryl) and III (ferric)

oxidation states within the heme-moiety[24] are potential chemical mechanisms for the formation of free radicals.

Risk prediction for the development of AKI in rhabdomyolysis has focused on the interpretation of serum creatine kinase, in which admission and peak levels do correlate with the development of AKI on a population basis, but with wide overlap making diagnostic prediction in individual patients difficult[28] and only very high levels consistently associated with AKI.[15,16] Plasma myoglobin levels may be more valuable in stratifying with patients with a rhabdomyolysis[29] but are not widely available and may still overlap significantly between the AKI and non–AKI populations.[30] In a large study[31] of more than 2000 patients with elevated CK above 5000 IU/L, only 25% of rhabdomyolysis patients had trauma as a cause. The authors examined a composite end point of death or need for RRT; independent risk factors for this composite outcome were older age, female sex, cause of rhabdomyolysis (not seizures, syncope, exercise, statins, or myositis), initial CK > 40,000 IU/L, higher initial serum phosphate or creatinine, and lower initial serum calcium or bicarbonate.

The primary treatment of rhabdomyolysis is the recognition and management of the underlying source of muscle injury. In the trauma patient this most often is the treatment of underlying compartment syndromes or vascular compromise but also may involve the control of patient temperature, treatment of underlying infection, and withdrawal of any offending medication.[15] This is combined with the supportive management of immediate life-threatening complications, including hypovolemic shock, profound hyperkalemia, and hypocalcemia, which may be immediately life threatening. Electrolyte abnormalities may require emergency medical management and/or rapid institution of renal replacement therapy (RRT), particularly in situations in which reperfusion injury may cause further acute releases particularly of potassium.[16] One challenge is the treatment of coexisting hypocalcia, hyperkalemia, and acidosis when bicarbonate administration or RRT may cause further falls in ionized calcium. Conversely, rapid correction of serum calcium in the context of hyperphosphatemia may cause deposition of calcium phosphate in tissues, including injured muscle and the kidney.[16] Continuous RRT may lack the intensity to rapidly control serum potassium at conventional doses and higher intensity CRRT or hemodialysis may be required in refractory hyperkalemia; however, when used intermittently, significant rebound in potassium may occur after dialysis. Thus there is no easy recipe to the ICU management of profound rhabdomyolysis with electrolyte disturbance and severe AKI, which requires close monitoring and attention to detail. Removal of myoglobin by extracorporeal methods has been advocated as a treatment for severe rhabdomyolysis, only anecdotal evidence of clinical benefit.[32] As a 17.8 kDa middle molecule conventional RRT results in relatively poor myoglobin clearance; however, high-dose techniques using highly permeable membrane have been successful in rapidly lowering serum concentrations.[33] However, at present these techniques cannot be recommended outside of clinical trials, and if RRT is required after trauma with rhabdomyolysis it should be in conventional dose to standard indications.[32]

Conversely, in less severely sick patients, management of rhabdomyolysis is focused on the prevention of severe AKI and for this vigorous fluid resuscitation (>6 L/day) remains a cornerstone of conventional management.[15–17,19,34] In theory fluid resuscitation corrects hypovolemia and improves glomerular filtration rate (GFR) and dilutes tubular fluid enhancing elimination of toxins while minimizing toxicity in the tubules.[16] However, the evidence base for this practice relies largely on uncontrolled case series performed outside the ICU in the setting of crush injuries, where overt hypovolemia is much more likely.[15,34] Similarly, although urinary alkalinization to solubilize and reduce the redox activity of myoglobin has a theoretic rationale, there is no evidence to support any specific formulation of fluid (buffered, unbuffered, or alkaline) nor any consensus on the use of diuretics.[15,34] The deleterious effects of fluid overload in critical illness, and in AKI in particular, are now well recognized[35] and the potential harm from a vigorous fluid regimen targeting only a modest evaluation in CK should not be underestimated. The author's own non–evidenced-based practice[36] in adequately resuscitated patients admitted to the Royal London Hospital ICU with rhabdomyolysis not needing RRT is to provide fluid therapy as a one-off bolus followed by 100% replacement of hourly urine output using a buffered solution, employing diuretics if necessary, to achieve a target urine output of more than 2 mL/kg/hr, and with the addition of sodium bicarbonate if an acidic urine (pH < 6) develops. However, if oliguria occurs despite diuretic challenge, focus should shift to prevention of the global harm from fluid overload, with the discontinuation of continuous IV fluid infusion and use of RRT if necessary.

BURN INJURIES AND ACUTE KIDNEY INJURY

An estimated 265,000 deaths per year worldwide are attributed to burn and inhalation injuries, mainly in low-income countries in southeast Asia.[37] Risk of death in this population is associated strongly with the occurrence of AKI. When defined by the risk, injury, failure, loss of kidney function, and end-stage kidney failure (RIFLE) classification, AKI occurs in one quarter of patients with severe burn injury and is associated with mortality of 35%. However in the small proportion of burn patients (~3%) who require RRT mortality can be as high as 80%.[38] Thus in severe burn patients the occurrence of AKI is associated with a sixfold greater risk of death,[38] far stronger than, for instance, that association between AKI and risk of death in the septic population.[39] Furthermore, the low prevalence of RRT may indicate reluctance to initiate treatment because of potential treatment-related adverse events (such as bleeding or catheter-related bloodstream infection) or perception of therapeutic futility. However, bleeding is now much less a problem because regional citrate anticoagulation (RCA) does not carry any more the risk of increased bleeding,[40] Although in burns patients, the occurrence of catheter related bloodstream infection (CRBSI) was not associated with an increased need of RRT,[41] suggesting that burn severity is an overriding factor in the development of AKI in this population[41] and also that the fear of CRBSI with vascular access for RRT may be overvalued. Thus it is likely that RRT is, in fact, underused in the burns population and should be considered early in the clinical course because AKI-related complications, including fluid overload, acidosis, and electrolyte abnormalities may be particularly harmful in the major burns patient.

PATHOPHYSIOLOGY OF ACUTE KIDNEY INJURY IN BURNS

The pathophysiology of AKI in burn patients is multifactorial but can be divided into three phases.[42] In the "toxic" phase,

burn shock is accompanied by severe hypovolemia, leading to renal and medullary hypoperfusion. Ample volume-resuscitation aims to rapidly correct organ, including kidney, perfusion. However, excess fluid may increase the risk for abdominal compartment syndrome or elicit renal vein congestion, which increases kidney "afterload."[43–45] Both conditions paradoxically may aggravate AKI.[43] The second "inflammatory" phase[43] is apparent approximately 48 hours later after the insult and is characterized by cytokine-induced changes in intrarenal microcirculation, release of caspase components attracting inflammatory cells into the renal parenchyma,[46] and plasminogen activator inhibitor type 1–mediated intraglomerular thrombosis with disseminated intravascular coagulopathy.[47] Shedding of apoptotic cells may induce obstruction of tubules.[47] Importantly the effects of AKI on distant organ function is increasingly well recognized (so-called "organ cross-talk"), with an increasing recognition of the contribution of AKI on susceptibility to other complications of critical illness.[48,49] For example, the presence of AKI, through effects on the innate and acquired immunity, compounds the existing predisposition to nosocomial infection in the burns patients; this may in turn exacerbate the severity of AKI and other organ dysfunction.[50] Finally, a third or "late" phase is identified when AKI occurs in isolation rather than as a part of multiorgan dysfunction[49] associated with a late increase of calcium. Hypercalcemia therefore must be excluded when AKI becomes manifest at this late stage of the burn process.[51] Finally, a number of other injuries may coexist with burns and play a causative role in AKI; electrocution burns may cause deep muscle injury and induce rhabdomyolysis;[52] similarly carbon monoxide and inhalational hydrogen cyanide exposure may contribute to multiorgan dysfunction in the burned patient.

MANAGEMENT OF THE BURNS PATIENT WITH ACUTE KIDNEY INJURY

Fluid resuscitation remains a cornerstone of the hemodynamic challenge during the initial phase of burn shock. Most units use buffered crystalloids following the Parkland resuscitation formula first described in 1968 (4 mL/kg body weight per 1% total burned surface area in the first 24 hours). However, most patients are receiving more fluids than this, with volumes up to 6 mL/kg/total being reported.[53] The inevitable consequence of this high-volume resuscitation to preserve cardiac output in the face of fluid losses is that most burns patients become grossly fluid overloaded, which may have adverse effects on multiple organ systems, including the kidney.[35,54] Consequently, other types of fluids, such as hypertonic saline or the use of albumin, may decrease volume loading and thus decrease the incidence of intra-abdominal hypertension, venous pressure, and risk of AKI[55] via "increasing kidney afterload."[45,50] However, prospective evidence for fluid resuscitation strategies in burns is limited, and treatment inevitably will be individualized. It is as yet unclear whether trends toward lower volume resuscitation in general critical care translate to the burn population.

Historically, burns patients who developed AKI or severe metabolic abnormalities were treated with intermittent hemodialysis (IHD). Experience with continuous RRT (CRRT) techniques in general medicosurgical ICUs has been transmitted progressively to specialist burns ICUs, however, with some delay.[56–58] Importantly, use of CRRT has been shown improve mortality in burn AKI when compared with historical controls treated with IHD,[56] a benefit that has been difficult to demonstrate in general ICU populations, possibly because higher baseline risk of death in the burns patient with severe AKI makes it easier to demonstrate survival benefits of interventions.[59]

Inserting a jugular or subclavian venous catheter (CVC) for RRT is hazardous in patients with severe burn injuries located at the upper body half. Jugular access is preferred and should be preserved for initiating CRRT.[60] To limit infection risk, it has been suggested that catheters be changed to another site every 5 days.[61,62] RCA is the preferred method of anticoagulation in CRRT in general.[58] Unlike unfractionated heparin, RCA can be used at low blood flow and higher filtration fraction,[63–65] which may be beneficial in burns patients who often have less efficient or lower CRRT blood flow because of precarious vascular access. Because regional citrate anticoagulation (RCA) diminishes the bleeding risk, surgical procedures and dressing changes that frequently are required in burns patients are performed more safely, and less blood transfusion may be required. Burns patients are often hypercongealable, and citrate better counteracts hypercoagulability inside the CRRT circuit.[66] In contrast with heparin, it never is associated with thrombopenia. Good circuit patency and minimal down-time because of circuit clotting is of great importance in the burns patient and regular patient bathing, wound dressing, often required daily, may result in frequent enforced breaks from CRRT.[56,67]

The healing process (i.e., wounds, autografts, and/or concomitant organ failure) in severely burned patients is highly dependent on providing adequate nutrition. An energy delivery below 30 kcal/kg has been associated with significant weight loss and lower pre-albumin levels.[68] Daily calculation or measurement of energy needs is recommended.[68] CRRT with ultrafiltration enables increased nutrition volume without excessive volume loading. CRRT may be associated with increased losses of amino acids and other nutrients. Two small studies assessed the effect of high-dose vitamin C (66 mg/kg/hr) given within the first 24 hours after thermal injury. One prospective study randomized patients to receive fluid resuscitation with or without adjuvant vitamin C. Vitamin C treatment reduced resuscitation volume and resulted in better gas exchange and less days on mechanical ventilation.[69] A more recent retrospective study confirmed reduced fluid requirements and also reported a better diuresis in vitamin C recipients[70]; however, mortality benefit was not observed. Vitamin C is cleared substantially by dialysis,[71] and at least 50% of ascorbate may be lost during CRRT.[40] Current guidelines recommend a high-normal vitamin C intake during CRRT[72] that may not cover the needs in critically ill patients on CRRT; a temporary increase of up to 12 g per day has been suggested.[61,70,72] Other substances such as glutamine, zinc, copper, and selenium often are supplemented during CRRT; however, evidence in favor of these interventions critically ill patients generally and AKI patients in particular has not been sustained.[73,74] Given their special requirements, more studies on dosing of nutrients are required in CRRT and burns patients to guide clinicians.[75]

OUTCOMES AND FOLLOW-UP AFTER ACUTE KIDNEY INJURY IN TRAUMA OR BURNS

Overall survival from the acute traumatic episode is associated with recovery of renal function and, in the absence of destructive injury such as bilateral renal artery avulsion, survivors of trauma or burns have a very low incidence of

immediate end-stage renal disease. This should not be taken as implying these patients are not at increased long-term risk of renal disease. Because trauma and burn patients are significantly younger with a low incidence of chronic kidney disease (CKD), significant nephron loss may occur without the development of overt CKD as assessed by estimated GFR or even measured GFR (because of loss of functional renal reserve). Most importantly, serum creatinine is likely to be an invalid measure of renal recovery in these patients because of the extensive muscle wasting that accompanies prolonged critical illness[76] and trauma[77,78] and burns[79] in particular so that apparently normal or baseline serum creatinine may be associated with substantial persistent renal dysfunction.[80] Thus trauma and burns survivors could be at significant risk of progressive CKD and related cardiovascular mortality over time, something of great relevance when considering a relatively younger population with many years of life ahead.

These concerns are supported in a recent study of US army troops who required RRT after traumatic injury during conflicts in Iraq or Afghanistan, where, although no survivor had an eGFR below 60 mL/min per 1.73 m^2, 25% had developed proteinuria.[81] Proteinuria is a strong risk factor for progressive CKD and cardiovascular disease,[82] suggesting a significant burden of covert renal disease may be present in these young trauma survivors. Notably more than half of these patients experienced a major amputation, further confounding the interpretation of their serum creatinine because of the attendant loss of muscle mass. The same considerations apply to burns patients, in whom persistent proteinuria is a common finding strongly linked to occurrence of AKI and adverse ICU outcomes[83] but has been less studied as a prognostic marker in survivors. Currently we would recommend that all trauma or burns patients who have experienced AKI should have their renal function assessed at least 3 months after hospital discharge including urinalysis. Given the difficulty in assessing renal function accurately, long-term follow-up would be advised in trauma or burns AKI patients who have experienced more severe kidney injury or have persistently reduced muscle mass (such as in amputees or spinal injuries) and in any patients with evidence of persistent renal disease including proteinuria at discharge or follow-up.

Key Points

1. Acute kidney injury (AKI) is a relatively frequent complication of trauma and burns injury that is associated independently with morbidity and mortality.
2. Multiple coexisting risk factors predispose to the development of AKI in these groups, some of which are specific to the setting and some more general AKI risk factors.
3. Although management of rhabdomyolysis is often a focus, outside of disaster situations it is usually not the sole cause of AKI in major trauma.
4. Prompt and adequate fluid therapy is a vital aspect of the management of major burns, trauma, and rhabdomyolysis; however, an awareness of the deleterious effects of fluid overload should be maintained especially after the initial resuscitation phase.
5. Long-term outcomes of AKI often are neglected in these populations.

Key References

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