World Journal of Critical Care Medicine

Online Submissions: http://www.wjgnet.com/esps/
bpgoffice@wjgnet.com
doi:10.5492/wjccm.v3.i1.1

World J Crit Care Med 2014 February 4; 3(1): 1-7
ISSN 2220-3141 (online)

EDITORIAL

Rhabdomyolysis, compartment syndrome and thermal injury

Yusuf Kenan Coban

Yusuf Kenan Coban, Burn Unit and Department of Plastic Surgery, Inonu University Turgut Ozal Medical Centre, 44280 Malatya, Turkey
Author contributions: Coban YK solely contributed to this paper.
Correspondence to: Yusuf Kenan Coban, Professor, Burn Unit and Department of Plastic Surgery, Inonu University Turgut Ozal Medical Centre, 44280 Malatya, Turkey. ykenanc@yahoo.com
Telephone: +90-422-2374780 Fax: +90-422-2374780
Received: May 7, 2013 Revised: September 4, 2013
Accepted: October 16, 2013
Published online: February 4, 2014

Abstract

Rhabdomyolysis (RML) after electrical burns and crush injuries is a well-known clinical entity, but its occurrence following thermal injury has not gained so much attention. Capillary leak syndrome and following polycompartmental syndrome are devastating end results of major thermal injuries. In the current review, polycompartment syndrome within the clinical picture of systemic oedema and its relationship to RML is discussed along with its management and prevention.

Key words: Capillary leak syndrome; Rhabdomyolysis; Thermal injury

Core tip: In the current review, polycompartment syndrome within the clinical picture of systemic oedema and its relationship to rhabdomyolysis is discussed along with its management and prevention.

Coban YK. Rhabdomyolysis, compartment syndrome and thermal injury. *World J Crit Care Med* 2014; 3(1): 1-7 Available from: URL: http://www.wjgnet.com/2220-3141/full/v3/i1/1.htm DOI: http://dx.doi.org/10.5492/wjccm.v3.i1.1

DEFINITION

Rhabdomyolysis (RML) is defined as muscle damage with dissolution of the skeletal muscle fibre and results in release of potentially toxic intracellular components into the systemic circulation. A classical triad of symptoms includes muscle pain, prostration and dark pigmented urine. Urine pigmentation only manifests itself when the renal threshold for myoglobulin (MB) is exceeded. In 1940-1941, RML syndrome was first described in patients with crush injury secondary to building destruction during wartime[1]. In later decades, it was understood that RML is not confined to crush injuries, but is also associated with thermal injuries and other medical causes. Although direct muscle injury remains the most common cause of RML, additional causes include toxins, endocrinopathies, malignant hyperthermia, neuroleptic malignant syndrome, electrolyte alterations, diabetic ketoacidosis, non-ketotic hyperosmolar coma, severe hypo- or hyperthyroidism and bacterial or viral infections. Generally speaking, RMLs can be divided into two categories as shown in Figure 1.

PATHOPHYSIOLOGY

The scenario is seen after severe crush and thermal or electric injuries in addition to direct muscle injury. In other words, there may be a combination of both direct muscle injury and compartment syndrome in the same clinical picture. On the other hand, the primary mechanism for position-related RML is reperfusion of damaged tissue after a period of ischaemia and the release of necrotic muscle material into the circulation after pressure is relieved. In severe burns, capillary leak syndrome leading to polycompartmental syndrome is also responsible for the development of RML *via* several mecha-

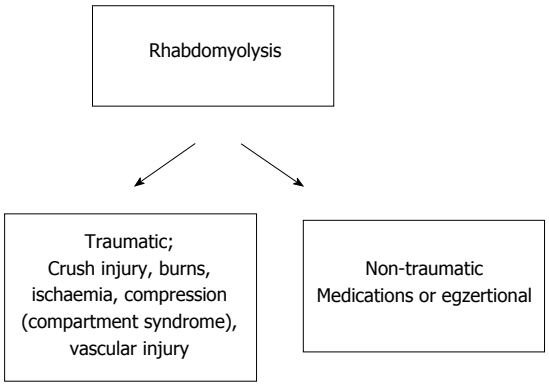

Figure 1 General classification of rhabdomyolysis.

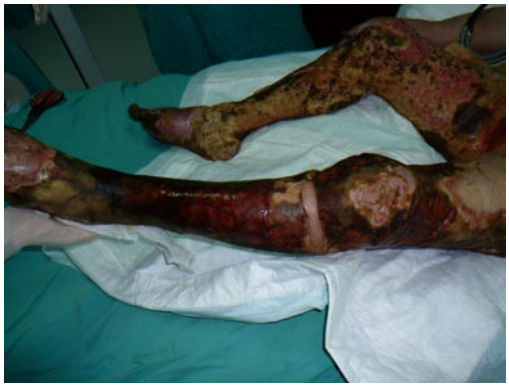

Figure 2 An example for full-thickness burns of both lower extremities.

nisms including loss of vasomotor tone of arterioles, collapse of thinner-walled veins, and the overall loss of the pressure gradient between the arterial and venous system. As the blood flow decreases, the tissues become progressively more ischaemic, thereby leading to further necrosis and oedema.

Muscle necrosis is triggered by derangements in oxidative or glycolytic energy production with resulting ATP depletion. In the presence of concomitant ATP depletion, free Ca^{2+} content in the myocyte increases due to failure of Ca^{2+} efflux mediated by the ATPase-driven Ca^{2+} pump. Influx from the extracellular compartment into muscle cells includes water, Na^{+}, Cl^{-} and Ca^{2+}. Muscle cells swell due to the accumulation of intracellular solutes and a reduction in active ion extrusion. Within minutes of trauma, intramuscular pressure may exceed arterial blood pressure within the intracompartmental space. Hypovolaemia develops, followed by haemodynamic shock, hypocalcaemia, and hyponatraemia in the context of trauma and burns. Progressive hypovolaemia is thought to contribute to the formation of casts that obstruct renal tubules and to renal vasoconstriction involving afferent glomerular arterioles and glomerular capillaries[2].

As the skeletal muscle (SM) is a principal actor that plays a major role in the pathophysiology of RML, its distribution within the body is worthy of mention. Subcutaneous muscle accounts for approximately 40% of total body mass for adult males and 29% for adult females. Depending on gender, age and health status, one-third and one-half of body protein resides within SM[3]. There is growing awareness of the importance of SM in many physiological and disease processes[4]. Janssen *et al*[5] studied skeletal muscle mass measurement in whole body distribution by magnetic resonance imaging. They found that the lower body had more SM mass than upper body regions in both sexes. As the lower extremity bears much of SM mass of total body SM mass, full-thickness burns of the lower extremity pose a higher risk for development of fatal RML (Figure 2). One might speculate that immobilization is more commonly seen after lower extremity full-thickness burns when compared to the upper body parts, and this may mean increased risk of RML for lower extremity burns.

LABRATORY DIAGNOSIS AND MORTALITY

The diagnosis of RML is based on the measurement of creatinine kinase (CK) in serum or plasma. Plasma and urine myoglobin CK measurement might be useful in the early stage of the syndrome[6]. Patient monitoring is pivotal (the mortality rate is as high as 8%) and should be aimed at preventing the detrimental consequences. Mortality for patients with RML secondary to flame burns seems to be high[7]. Stewart *et al*[8] studied percentage of full-thickness burns, percentage of total body surface area (TBSA) burned, injury severity score, peak CK and acute kidney injury (AKI) in burn patients. They found that the log peak CK correlated with stage of AKI[8]. Another study revealed that 28% of severely burned patients developed AKI during acute resuscitation[9]. In a retrospective study of 714 patients, eight were reported to have RML and these cases had poor survival[10].

THERMAL INJURY AND RHABDOMYOLYSIS

Several issues related to the subject are present. These are the role of albumin and fluid therapy, capillary leak syndrome and comorbid situations.

Albumin in severe burns

Lower albumin concentrations are commonly observed in older persons and are associated with worse health outcomes and mortality[11]. Albumin is a negative acute-phase protein that decreases with ongoing inflammation, and many of the reported associations with albumin may reflect this[12]. Serum albumin concentration $\leqslant$ 30 g/L was reported to be associated with a twofold increase in organ dysfunction[13]. The optimal resuscitation algorithm including albumin supplementation remains elusive for patients with large burn injuries. Park *et al*[14] compared the use of 5% albumin in the first 24 h with other protocols not using albumin solution. They found

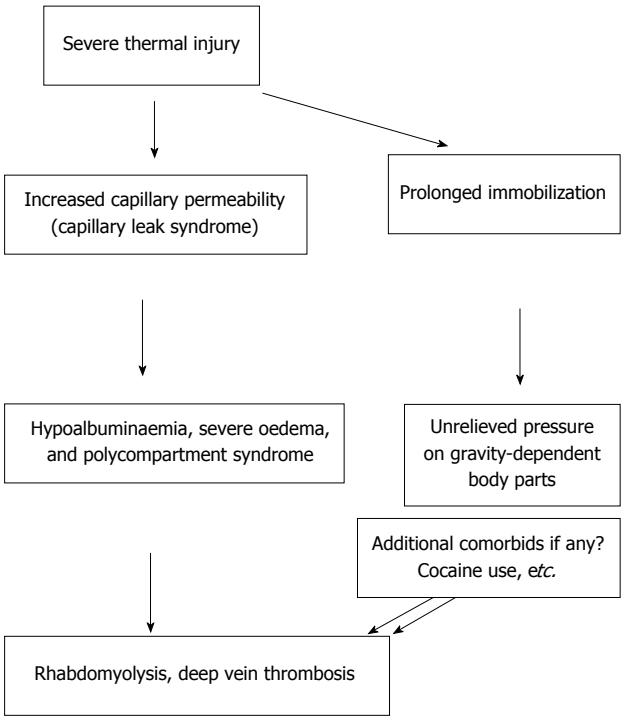

Figure 3 Pathogenic mechanisms for development of rhabdomyolysis in severe thermal injury.

that their new protocol decreased ventilator days and mortality, whereas another study revealed an opposite outcome. Currently, there is no consensus on using albumin replacement therapy for acute burn resuscitation[15]. The albumin molecule, being smaller (69 kDa) than the globulin molecule (90-156 kDa), will leak at a relatively earlier stage of the disease (with a moderate increase in capillary pore size) than globulin. This leads to albumin/globulin reversal. In cases with severe permeability changes related with rapid progression to larger pore size with simultaneous leak of both albumin and globulin, albumin/globulin reversal will not occur. Kumar's[16] study showed that patients with albumin protein values less than 5.0 g/dL showed higher mortality (95%) compared to those in other groups with more than 5.0 g/dL.

Capillary leak syndrome

Major burns are characterised by an initial capillary leak that requires fluid resuscitation for haemodynamic stabilization. The extensive capillary damage that follows thermal injury is responsible for massive plasma extravasation into burned tissues, with consequent hypovolaemia, abdominal hypertension and extremity compartmental syndrome necessitating fasciotomy, prolonged mechanical ventilation and hospital stay. Increased compartmental pressure poses a risk for RML. In local compartmental syndrome, the pathology is restricted to a unique body region and does not cause a life-threatening systemic condition. However, capillary leak syndrome, whether the cause is idiopathic or due to severe burns, causes hypovolaemic shock due to marked plasma shifts from the intravascular to the extravascular space. This presents as the characteristic triad of hypotension, haemoconcentration and hypoalbuminaemia. The reason is due to leakage of fluids and macromolecules (up to 900 kDa) into tissues. Systemic capillary leak syndrome is a transient event, and less than 150 cases have been reported. Systemic oedema causes multiple compartment syndrome, which needs emergency fasciotomies[17]. Hypoalbuminaemia always associates with the picture of systemic oedema and polycompartmental syndrome.

Direct damage to the striated muscle by high electrical voltage is well understood[18]. In thermal burns without direct muscle injury, the actual trigger for RML often remains unexplained. The majority of the publications on the subject are single case reports[19,20]. Prolonged immobilization following unconsciousness and repeated surgical procedures has been suggested to trigger RML in thermally injured patients. However, there may be several factors contributing to the development of RML in the context of severe thermal burns (Figure 3).

Ideal formulation of fluid therapy in burn shock

Less than 20% of burn injuries are associated with minimal fluid shifts and can generally be resuscitated with oral hydration. Current recommendations are to initiate formal intravascular fluid resuscitation when the surface area burned is greater than 20%. The ideal burn resuscitation is the one that effectively restores plasma volume, with no adverse effects. Isotonic crystalloids, hypertonic solutions and colloids have been used for this purpose, but every solution has its advantages and disadvantages. None of them is ideal, and none is superior to any of the others. Too vigorous resuscitative efforts may lead to severe protein depletion and further oedema accumulation into both burned and unburned tissues.

A condition of fluid unresponsiveness is present throughout the first 12 h of the post-burn period. The administration of supranormal volumes fails in the first 24 h to achieve normal preload volumes. The fluid creep started in the 1990s with an increasing proportion of the first 24 hours' fluid delivery above the 4 mL/kg TBSA% (Parkland formula). The first alerts were published under the form of case reports of increased mortality due to abdominal compartment syndrome and respiratory failure. While under-resuscitation was the major cause of mortality among burned patients until the 1980s, over-resuscitation has become an important source of complications[21]. Several studies have supported that patients who receive larger volumes of resuscitation fluid are at higher risk of injury complications such as pneumonia and extremity compartment syndrome. Hypertonic saline should be reserved for providers experienced in this approach. Plasma sodium concentrations should be closely monitored to avoid excessive hypernatraemia. Administration of high-dose ascorbic acid may decrease the overall fluid requirements, and is worthy of further study[22].

Resuscitation fluids influence the inflammatory response to burns in different ways and it may be possible

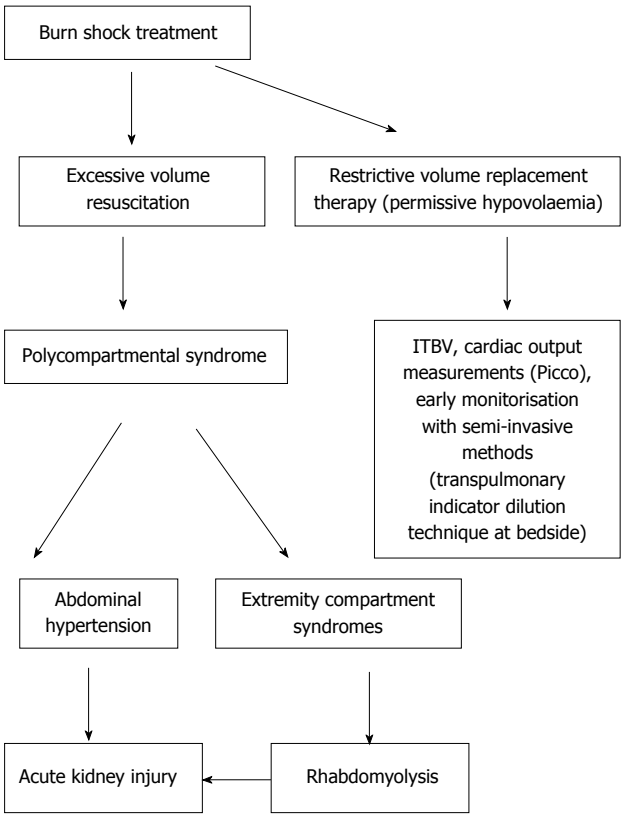

Figure 4 Possible scenarios during burn shock resuscitation. ITBV: Intrathoracic blood volume.

Table 1 Therapeutic goals of fluid resuscitation and therapeutic measures in thermal injury

Goal	Measure
Preventing hypovolaemia and shock	Fluid overloading (with rationing prehospital fluid delivery)
Improving organ perfusion	Mannitol, high dose vitamin C administration (avoiding early colloids)
Reducing capillary leak and oedema	Not known
Reducing inflammatory storm	Haemodialysis, plasmapheresis, *etc.*
Avoiding polycompartment syndrome	Permissive hypovolaemia

therefore to affect this response using appropriate fluid at the appropriate time[23]. Table 1 shows the goals of early fluid resuscitation in major burns.

Starches are effective volume expanders and early use of newer formulations may limit resuscitation requirements and burn oedema[24]. It has been shown that post-burn oedema is detrimental to organ function and that the deleterious effect is proportional to the amount of extravasated fluids[25]. Arlati *et al*[26] compared permissive hypovolaemia administered by a hemodynamic-oriented approach with Parkland formula resuscitation throughout the first 24 h period. They found permissive hypovolaemia allowed for less volume infusion, a reduced positive fluid balance and significantly lesser multiple organ dysfunction syndrome (MODS) score values than the Parkland formula.

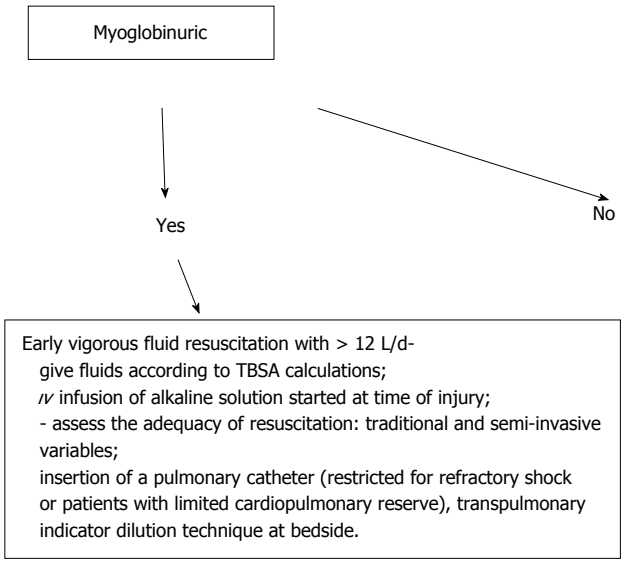

Figure 5 Algorithm for established clinical picture of rhabdomyolysis in burn victims. TBSA: Total body surface area.

Permissive hypovolaemia seems safe and well tolerated by burn patients. It seems effective in reducing multiple organ dysfunction due to induced oedema fluid accumulation and inadequate O_2 tissue utilization. It has been speculated that the insensitivity of cardiac preload to increase by even the most aggressive regimen might derive from the combination of both increased capillary permeability and higher hydrostatic pressure than most forms of hypovolaemic shock. A supranormal resuscitation volume might exacerbate post-burn oedema accumulation by unnecessarily increasing both the amount and length of fluid extravasation. The reduction of volume given was obtained throughout the period of maximum capillary damage. This would be impossible without the use of invasive haemodynamic monitoring as both intrathoracic blood volume (ITBV, as a cardiac preload indicator) and cardiac output measurements generated[26]. The PICCO system allows both ITBV and cardiac output measurements. ITBV and cardiac output measurement are earlier and more sensitive indicators of critical hypovolaemia than vital signs, hourly urine output and central venous pressure. A haemodynamic-oriented approach to burn shock resuscitation is gaining more acceptance for direct cardiac preload estimation nowadays (Figure 4).

Compartment syndrome

Burns and toxic causes may lead to polycompartment syndrome during fluid resuscitation if given at too high a dose. Compartment syndrome can be classified as primary (pathology within the compartment) and secondary (no primary pathology or injury within the compartment)[27]. SM compartments are especially susceptible to this type of injury. Thermal injuries, in particular full-thickness burns, cause secondary tissue constriction eschars and oedema. Fluid shifts associated with major burns adding to extravolume pressure may contribute to

Table 2 Risk factors for rhabdomyolysis

Risk factors for position-related rhabdomyolysis
Long-lasting surgery (more than 5-6 h) or prolonged immobilization (coma, unconsciousness)
Body weight more than 30% of ideal body weight
Pre-existing azotaemia
Diabetes
Hypertension
Uncontrolled extracellular volume depletion
Associated drug abuse (cocaine, *etc.*)

the development of compartment syndrome. The obtruded patient with prolonged limb compression, either during surgery or postoperative sedation, is at particular risk for development of an extremity compartment syndrome that may go unnoticed. In terms of the role of increasing CK levels and myoglobinuria, these are nonspecific signs of muscle necrosis and late signs of untreated irreversible compartment syndromes.

In most compartments, pressures > 30 mmHg critically compromise organ perfusion. According to the most recognised explanation of the syndrome, A-V gradient pressure theory, the perfusion of the intracompartmental tissues is hindered by the elevation of the interstitial fluid pressure above the level of the capillaries. The capillary pressure on the arterial end and the venous ends are 30 and 10 mmHg, respectively. Excessive volume resuscitation to prevent and treat burn shock may lead to intra-abdominal compartment syndrome, which is probably an underestimated contributor to the development of acute kidney injury after burn shock. The earliest effect of raising the intra-abdominal pressure (IAP) is to reduce visceral perfusion to organs. Subtle interactions with other noxious organ-damaging effects may be indistinguishable in a multifactorial setting. With higher IAP, more overt symptoms may be seen such as hypercarbic and hypoxaemic respiratory failure. Mortality rates of this clinical picture are between 70% and 100%.

Comorbid situations

The only cause of raised CK in thermal burns patients remains the involvement of muscles in deep burns. However, RML may occur in patients with superficial burns who had cocaine abuse[28]. Table 2 shows risk factors for development of RML. If any of these is present in the burn victim, RML may occur without the effect of presence of burn injury. Prolonged immobilization (*e.g.,* anaesthesia, coma, drug- or alcohol-induced unconsciousness) has been reported to induce RML due to unrelieved pressure on gravity-dependent body parts. Lateral decubitus, lithotomy, sitting, knee-to-chest and prone positions are reported to be the most common positions leading to RML. One of the risk factors for position-related RMLs was identified as having a bodyweight more than 30% greater than the ideal body weight .

RHABDOMYOLYSIS-INDUCED RENAL FAILURE

Leakage of intracellular contents such as myoglobulin (MB), CK, K, aldolase phosphate, lactate dehydrogenase, aspartate transaminase and urate into the extracellular space occurs in RML[29]. After complete sarcolemmal destruction, MB is released into the systemic circulation, leading to renal tubular obstruction. The extent of renal damage is dependent on the amount of volume deficit and renal ischaemia. When MB levels reach 100 mg/dL, dark, tea-coloured urine is seen. The principal goal is to prevent renal failure in cases of RML by aggressive fluid replacement and forced diuresis. If acute renal failure cannot be prevented by these measures, renal replacement therapy becomes mandatory. CK and MB levels must be routinely measured in all patients on admission.

In human patients, increased serum and perfusate levels of MB and CK during isolated limb perfusion have been shown for melanoma and sarcoma treatments[30]. Exertional muscle damage produced by eccentric exercise in healthy individuals has been shown to cause profound CK and MB elevations without renal impairment[31,32].

RML-induced renal failure is caused by the precipitation of myoglobin in the renal tubules. Early aggressive resuscitation with either normal saline or ringer lactate to maintain an adequate urine output is the most employed intervention in preventing the development of renal failure[33]. Therapeutic options include the correction of the hypovolaemia with sufficient fluid supply, the prevention of oliguria using loop diuretics, alkalinization of urine, normalization of serum electrolytes and decomposition of compartment syndromes. RML can be complicated by ARF occurring in 4%-33% of the patients[34]. Emerging data overwhelmingly suggest that fluid overload in critically ill patients may be associated with adverse outcomes. Over and under fluid resuscitation may endanger renal function in several ways. So, management of such patients should include a strategy of early resuscitation followed by a careful assessment of fluid status and early initiation of renal replacement therapy[35].

With increased pulmonary intestinal fluid during fluid administration, hypoxia is a frequent sequela in the context of systemic oedema. The septic patient with capillary leak syndrome is then diagnosed as having acute respiratory distress syndrome (ARDS) and placed on mechanical ventilation. Hyroxyethyl starch (HES) has negative effects on coagulation and causes an osmotic nephrosis that can lead to renal impairment. So, HES usage during fluid management of severe burn injuries may increase the risk of acute kidney injury (AKI)[36]. On the other hand, persistent fluid overload in AKI patients may lead to development of ARDS. Abdominal compartment syndrome (ACS) has been described with extensive abdominal fluid and impaired renal function[37]. ACS is associated with resuscitation volumes of 300

mL/kg per 24 h. Figure 5 shows possible negative consequences of over-resuscitation. Burn physicians must evolve their practices to avoid over-resuscitation or they should use more sensitive markers of organ perfusion than urine output.

Figure 5 proposes that myoglobinuric burn patients must be protected from AKI by *iv* infusion of alkaline solution. This regimen stabilizes the circulation and mobilizes oedema fluids sequestered in the injured muscles into the circulation, corrects hyperkalaemia and acidosis and protects against the nephrotoxic effects of myoglobulinuria and uricosuria[38].

Muscle injury as a consequence of burn injury may have led to elevation in serum creatinine concentrations by increased release of creatinine in circulation while glomerular filtration rate is unaffected. On the other hand, serum creatinine may already be increased on admission leading to a false low prevalence of AKI when defined as a relative increase of serum creatinine. Burn injury patients with AKI have a worse prognosis that is almost linearly correlated with severity of AKI. Although AKI with renal replacement therapy remains prevalent in populations with severe burn injury, the outcome improved[26,39].

CONCLUSION

RML is still one of the leading causes of fatality in major burns. Massive destruction of muscular tissue leads to RML, defined as CK elevation combined with organ damage, which requires immediate diagnostic and therapeutic intervention. It appears that RML following extensive full-thickness burns may be more common than previously suggested. So health professionals dealing with burn therapy must pay close attention against possible development of RML. Especially prolonged immobilization and surgeries in certain positions can be preventable, while health professionals are taking measures against these risk factors[40-42]. Judicious fluid resuscitation remains a basic, but potentially life-saving duty of all involved in the care of the severely burned patient. However, it is not known whether serious complications like secondary abdominal hypertension are iatrogenic or truly unavoidable in the most seriously burned patients. Our uncertainty regarding the basic pathophysiology of thermal injury and resuscitation may be the explanation. A number of alternative strategies have been explored in relatively small trials. Further advances may potentially arise from modulation of the inflammatory response through improved therapies and fluid or from new insights into the basic mechanism of cellular injury and its treatment.

REFERENCES

1 **Better OS**, Stein JH. Early management of shock and prophylaxis of acute renal failure in traumatic rhabdomyolysis. *N Engl J Med* 1990; **322**: 825-829 [PMID: 2407958 DOI: 10.1056/NEJM199003223221207]

2 **Wiedemann HP**, Wheeler AP, Bernard GR, Thompson BT, Hayden D, deBoisblanc B, Connors AF, Hite RD, Harabin AL. Comparison of two fluid-management strategies in acute lung injury. *N Engl J Med* 2006; **354**: 2564-2575 [PMID: 16714767]

3 **Heymsfield SB**, McManus C, Stevens V, Smith J. Muscle mass: reliable indicator of protein-energy malnutrition severity and outcome. *Am J Clin Nutr* 1982; **35**: 1192-1199 [PMID: 6805298]

4 **Gallagher D**, Heymsfield SB. Muscle distribution: variations with body weight, gender, and age. *Appl Radiat Isot* 1998; **49**: 733-734 [PMID: 9569594 DOI: 10.1016/S0969-8043(97)00096-1]

5 **Janssen I**, Heymsfield SB, Wang ZM, Ross R. Skeletal muscle mass and distribution in 468 men and women aged 18-88 yr. *J Appl Physiol* (1985) 2000; **89**: 81-88 [PMID: 10904038]

6 **Visweswaran P**, Guntupalli J. Rhabdomyolysis. *Crit Care Clin* 1999; **15**: 415-428, ix-x [PMID: 10331135 DOI: 10.1016/S0749-0704(05)70061-0]

7 **Lazarus D**, Hudson DA. Fatal rhabdomyolysis in a flame burn patient. *Burns* 1997; **23**: 446-450 [PMID: 9426917 DOI: 10.1016/S0305-4179(97)89767-7]

8 **Stewart IJ**, Cotant CL, Tilley MA, Huzar TF, Aden JK, Snow BD, Gisler C, Kramer KW, Sherratt JR, Murray CK, Blackbourne LH, Renz EM, Chung KK. Association of rhabdomyolysis with renal outcomes and mortality in burn patients. *J Burn Care Res* 2013; **34**: 318-325 [PMID: 22955163]

9 **Mosier MJ**, Pham TN, Klein MB, Gibran NS, Arnoldo BD, Gamelli RL, Tompkins RG, Herndon DN. Early acute kidney injury predicts progressive renal dysfunction and higher mortality in severely burned adults. *J Burn Care Res* 2010; **31**: 83-92 [PMID: 20061841 DOI: 10.1097/BCR.0b013e3181cb8c87]

10 **Stollwerck PL**, Namdar T, Stang FH, Lange T, Mailänder P, Siemers F. Rhabdomyolysis and acute renal failure in severely burned patients. *Burns* 2011; **37**: 240-248 [PMID: 20965664 DOI: 10.1016/j.burns.2010.09.009]

11 **Naber TH**, de Bree A, Schermer TR, Bakkeren J, Bär B, de Wild G, Katan MB. Specificity of indexes of malnutrition when applied to apparently healthy people: the effect of age. *Am J Clin Nutr* 1997; **65**: 1721-1725 [PMID: 9174466]

12 **Huang Y**, Shinzawa H, Togashi H, Takahashi T, Kuzumaki T, Otsu K, Ishikawa K. Interleukin-6 down-regulates expressions of the aldolase B and albumin genes through a pathway involving the activation of tyrosine kinase. *Arch Biochem Biophys* 1995; **320**: 203-209 [PMID: 7625825 DOI: 10.1016/0003-9861(95)90001-2]

13 **Eljaiek R**, Dubois MJ. Hypoalbuminemia in the first 24h of admission is associated with organ dysfunction in burned patients. *Burns* 2013; **39**: 113-118 [PMID: 22683139]

14 **Park SH**, Hemmila MR, Wahl WL. Early albumin use improves mortality in difficult to resuscitate burn patients. *J Trauma Acute Care Surg* 2012; **73**: 1294-1297 [PMID: 23117385 DOI: 10.1097/TA.0b013e31827019b1]

15 **Melinyshyn A**, Callum J, Jeschke MC, Cartotto R. Albumin supplementation for hypoalbuminemia following burns: unnecessary and costly! *J Burn Care Res* 2013; **34**: 8-17 [PMID: 23128130]

16 **Kumar P**. Grading of severity of the condition in burn patients by serum protein and albumin/globulin studies. *Ann Plast Surg* 2010; **65**: 74-79 [PMID: 20548219 DOI: 10.1097/SAP.0b013e3181c47d71]

17 **Pitt RM**, Parker JC, Jurkovich GJ, Taylor AE, Curreri PW. Analysis of altered capillary pressure and permeability after thermal injury. *J Surg Res* 1987; **42**: 693-702 [PMID: 3586633 DOI: 10.1016/0022-4804(87)90013-8]

18 **Fish RM**. Electric injury, Part II: Specific injuries. *J Emerg Med* 2000; **18**: 27-34 [PMID: 10645833 DOI: 10.1016/S0736-4679(99)00158-4]

19 **Guéchot J**, Cynober L, Lioret N, Bétourné C, Saizy R, Giboudeau J. Rhabdomyolysis and acute renal failure in a patient with thermal injury. *Intensive Care Med* 1986; **12**:

159-160 [PMID: 3734248]
20 **Lee MT**, Lee XL, Hsieh CS. Survival of near fatal rhabdomyolysis following flame burn in a 25-year-old patient. *Burns* 2006; **32**: 634-639 [PMID: 16764995 DOI: 10.1016/j.burns.2005.12.007]
21 **Dulhunty JM**, Boots RJ, Rudd MJ, Muller MJ, Lipman J. Increased fluid resuscitation can lead to adverse outcomes in major-burn injured patients, but low mortality is achievable. *Burns* 2008; **34**: 1090-1097 [PMID: 18468802 DOI: 10.1016/j.burns.2008.01.011]
22 **Baxter CR**. Problems and complications of burn shock resuscitation. *Surg Clin North Am* 1978; **58**: 1313-1322 [PMID: 734611]
23 **Pham TN**, Cancio LC, Gibran NS. American Burn Association practice guidelines burn shock resuscitation. *J Burn Care Res* 2008; **29**: 257-266 [PMID: 18182930]
24 **Tricklebank S**. Modern trends in fluid therapy for burns. *Burns* 2009; **35**: 757-767 [PMID: 19482429 DOI: 10.1016/j.burns.2008.09.007]
25 **Dries DJ**, Waxman K. Adequate resuscitation of burn patients may not be measured by urine output and vital signs. *Crit Care Med* 1991; **19**: 327-329 [PMID: 1999092 DOI: 10.1097/00003246-199103000-00007]
26 **Arlati S**, Storti E, Pradella V, Bucci L, Vitolo A, Pulici M. Decreased fluid volume to reduce organ damage: a new approach to burn shock resuscitation? A preliminary study. *Resuscitation* 2007; **72**: 371-378 [PMID: 17137702 DOI: 10.1016/j.resuscitation.2006.07.010]
27 **Sanghavi R**, Aneman A, Parr M, Dunlop L, Champion D. Systemic capillary leak syndrome associated with compartment syndrome and rhabdomyolysis. *Anaesth Intensive Care* 2006; **34**: 388-391 [PMID: 16802499]
28 **Welch RD**, Todd K, Krause GS. Incidence of cocaine-associated rhabdomyolysis. *Ann Emerg Med* 1991; **20**: 154-157 [PMID: 1996798]
29 **Khan FY**. Rhabdomyolysis: a review of the literature. *Neth J Med* 2009; **67**: 272-283 [PMID: 19841484]
30 **Hohenberger P**, Haier J, Schlag PM. Rhabdomyolysis and renal function impairment after isolated limb perfusion--comparison between the effects of perfusion with rhTNF alpha and a 'triple-drug' regimen. *Eur J Cancer* 1997; **33**: 596-601 [PMID: 9274441 DOI: 10.1016/S0959-8049(97)00013-0]
31 **Clarkson PM**, Kearns AK, Rouzier P, Rubin R, Thompson PD. Serum creatine kinase levels and renal function measures in exertional muscle damage. *Med Sci Sports Exerc* 2006; **38**: 623-627 [PMID: 16679975 DOI: 10.1249/01.mss.0000210192.49210.fc]
32 **Oh JY**, Laidler M, Fiala SC, Hedberg K. Acute exertional rhabdomyolysis and triceps compartment syndrome during a high school football cAMP. *Sports Health* 2012; **4**: 57-62 [PMID: 23016070]
33 **Shapiro ML**, Baldea A, Luchette FA. Rhabdomyolysis in the intensive care unit. *J Intensive Care Med* 2012; **27**: 335-342 [PMID: 21436168 DOI: 10.1177/0885066611402150]
34 **Bagley WH**, Yang H, Shah KH. Rhabdomyolysis. *Intern Emerg Med* 2007; **2**: 210-218 [PMID: 17909702]
35 **Yerram P**, Karuparthi PR, Misra M. Fluid overload and acute kidney injury. *Hemodial Int* 2010; **14**: 348-354 [PMID: 20955269 DOI: 10.1111/j.1542-4758.2010.00498.x]
36 **Vlachou E**, Gosling P, Moiemen NS. Hydroxyethylstarch supplementation in burn resuscitation--a prospective randomised controlled trial. *Burns* 2010; **36**: 984-991 [PMID: 20558004 DOI: 10.1016/j.burns.2010.04.001]
37 **Shibagaki Y**, Tai C, Nayak A, Wahba I. Intra-abdominal hypertension is an under-appreciated cause of acute renal failure. *Nephrol Dial Transplant* 2006; **21**: 3567-3570 [PMID: 16935902 DOI: 10.1093/ndt/gfl496]
38 **Better OS**, Abassi ZA. Early fluid resuscitation in patients with rhabdomyolysis. *Nat Rev Nephrol* 2011; **7**: 416-422 [PMID: 21587227 DOI: 10.1038/nrneph.2011.56]
39 **Holm C**, Melcer B, Hörbrand F, Wörl H, von Donnersmarck GH, Mühlbauer W. Intrathoracic blood volume as an end point in resuscitation of the severely burned: an observational study of 24 patients. *J Trauma* 2000; **48**: 728-734 [PMID: 10780609 DOI: 10.1097/00005373-200004000-00023]
40 **Penn AS**, Rowland LP, Fraser DW. Drugs, coma, and myoglobinuria. *Arch Neurol* 1972; **26**: 336-343 [PMID: 5015592 DOI: 10.1001/archneur.1972.00490100066006]
41 **Szewczyk D**, Ovadia P, Abdullah F, Rabinovici R. Pressure-induced rhabdomyolysis and acute renal failure. *J Trauma* 1998; **44**: 384-388 [PMID: 9498517 DOI: 10.1097/00005373-199802000-00028]
42 **Biswas S**, Gnanasekaran I, Ivatury RR, Simon R, Patel AN. Exaggerated lithotomy position-related rhabdomyolysis. *Am Surg* 1997; **63**: 361-364 [PMID: 9124760]

P- Reviewers: Al-Benna S, Jeschke M
S- Editor: Zhai HH **L- Editor**: Wang TQ **E- Editor**: Liu XM

Published by **Baishideng Publishing Group Co., Limited**
Flat C, 23/F., Lucky Plaza,
315-321 Lockhart Road, Wan Chai, Hong Kong, China
Fax: +852-65557188
Telephone: +852-31779906
E-mail: bpgoffice@wjgnet.com
http://www.wjgnet.com