Management of Severely Burned Adult Patients: From Sedation to Organ Dysfunction

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Received: 17 July 2020; Accepted: 23 July 2020
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ABSTRACT

Burns are devastating and debilitating injuries leading to high morbidity and mortality, emotional stress and they add to the financial burden. Severely burned patients should preferably be managed in burn centre with dedicated intensive care unit (ICU). Severity of burns are classified according to the degree of burns and total body surface area (TBSA) with burns. Rule of nine is used for calculation of TBSA with burn. Severe burns will cause cellular leak, hypovolemia, storm of proinflammatory markers and cardiovascular impairment leading to the burn shock and multiple organ dysfunction.

Intensive care management of severe burns includes resuscitation and organ supportive care. There are number of formulae for fluid resuscitation in these patients, these formulas give initial guidance for fluid resuscitation and further fluid therapy should be guided by dynamic preload parameters. The opioids and benzodiazepines are frequently used for analgesia and sedation respectively whereas ketamine and dexmedetomidine have added advantage of opioid and benzodiazepine sparing effects. In these patient’s hyperglycaemia and hypercatabolism should be controlled. ARDS (acute respiratory distress syndrome) occurs in up to 50% of the ventilated burns patient. High frequency percussive ventilation and use of ARDS adjuvant therapies will have better outcome. Acute kidney injury occurs in 30% of severe burns, renal replacement therapy should be started early. Early enteral feeds, gastric ulcer prophylaxis and adequate fluid resuscitation will prevent the GI dysfunction. Burns shock will improve with adequate fluid resuscitation and supportive care. The common neurological dysfunction in burns is delirium. Delirium should be managed with pharmacological and non-pharmacological therapies. There is a decreasing trend in the mortality of severely burns patients and it is around 10%.

Keywords: Airway, Burn Shock, Multiorgan dysfunction, Sedation, Sepsis

Introduction

Although majority of burns are preventable, burns remained 4th major trauma related injuries. The burn injuries are difficult and devastating injuries. They are more common in low socioeconomic group and in lesser developed countries. These injuries cause high morbidity and mortality, emotional imbalance, additional socioeconomic burden and can be stressful (Smolle, 2017). Severe burns are the
burns involving more than 20% of the body surface area or inhalational injury, chemical, electric burns, lightening injury and burns associated with other traumatic injuries.

In October 2012 a fire broke out in a marriage party in which 25 people were killed and 30 people sustained injuries (Kattan et al., 2016). In Kuwait in 2009 electric short circuit and fire led to death of 57 people and caused injuries to 90 more people (Shih et al., 2017). Both these major fire accidents were preventable and use of fire alarms and exist doors decreased the incidence of burn injuries (Sheridan, 2016). Coconut Grove pub fire raised the initial alarm to have a better preventive measures and the recent advances in the medical science namely in resuscitation, wound care, infection control and management of inhalation injuries improved the care of severely burned patients. The complex metabolic and glycaemic changes, multiple organ dysfunction, repeated long term interventions remained worrisome factors (Herndon and Thompkins, 2004).

**Epidemiology**

World Health Organization (WHO), reports that more than 300,000 people per year die from burn injuries all over the world, the highest deaths are from Southeast Asia and the Middle East. Majority of burn (90%) related fatalities occur in moderate and low income countries due to a lack of medical and resuscitative facilities (Seyed-Forootan et al., 2016). Smolle C, et al. reviewed 46 burn publications, majority of studies come from highly developed (HD) countries, few from medium developed (MD) and no reports from low developed (LD) countries. Australia, Bulgaria, China, the Czech Republic, Finland, Iran, Israel, the Netherlands, the Oman and the United Kingdom are reporting an increasing trend in burn incidence and hospital admissions. Austria, Canada, Chile, China, Germany, Hong Kong, India, Japan, Mexico, Netherlands, Singapore, South Africa, South Korea, Sweden and the United States are showing decreasing trend in burn incidences (Smolle et al., 2017). Studies from Australia, Austria, Bulgaria, Chile, China, Germany, Israel, Netherlands, Taiwan and the United States are showing a decreasing trend in severity of burns (Smolle et al., 2017).

Burn injuries are frequent in younger age group, the average age of patients with burn injuries was 30.38 years. The hip is the most frequently burned area (45.9%), followed by thigh (22%) and wrist and hand (19%) (Sarbazi et al., 2019).

The common traumatic injuries associated in burns patients are multiple fractures (64%), complex soft tissue injuries (52%), head injuries (26%) and thoraco-abdominal injuries in 24% (Table 1) (Santaniello et al., 2004).
**Types of Burn**

The following are the common types of burns (Yasti *et al.*, 2015).

**Flame burn:** Common type of burn, caused by burning flames.

**Electric burn:** This burn injury occurs due to the electric current causing arc burns. These patients will have entry and exit points. This type of burns is known to cause high cardiac and muscular injuries.

**Chemical burn:** It occurs when skin or eyes comes in contact with chemical irritants acid or base.

**Thermal burn:** It is the burns caused by heated objects or liquids.

**Inhalation injuries:** These are the burns causing injury to the upper and lower airway (Yasti *et al.*, 2015).

**Severity of Burn**

Severity of burns is classified as follows

The 1st degree burns injuries epidermis is intact, there is erythema. In 2nd degree burns, the epidermal integrity is lost. When all the layers of dermis are involved, it is 3rd degree burns. In 4th degree burns all layers of skin, subcutaneous tissues and muscles are injured and there is carbonised appearance (Santaniello *et al.*, 2004; Yasti *et al.*, 2015).

Minor burns are the 2nd degree burns involving less than 10% of total body surface area (TBSA) in adults. Moderate burns are the 2nd degree burns involving 15 to 20% TBSA. Major or severe burns are when burn injury occurs to more than 20% of the TBSA (Sarbazi *et al.*, 2019; Santaniello *et al.*, 2004; Yasti *et al.*, 2015).

<table>
<thead>
<tr>
<th>Type of Injuries</th>
<th>%</th>
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<tbody>
<tr>
<td>Multiple skeletal fractures</td>
<td>64%</td>
</tr>
<tr>
<td>Soft tissue injuries</td>
<td>52%</td>
</tr>
<tr>
<td>Traumatic brain injuries</td>
<td>26%</td>
</tr>
<tr>
<td>Thoraco-abdominal injuries</td>
<td>24%</td>
</tr>
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**Table1:** The traumatic injuries associated with burns.
Calculation of Percentage of Burns

The extent of burns is described by the percentage of burns involving total body surface area (TBSA). TBSA is important to calculate TBSA for prescribing the required amount of fluids and to decide the need to transfer the patient to specialized burn centres (Kagan and Smith, 2000). There are three methods to calculate the TBSA with burns 1. Wallace rule of nine 2. Palm method 3. Lund and Browder chart.

Wallace rule of Nine: In this formula various body sections represent 9% each of the TBSA. It is the most commonly used formula for calculation of burn percentage in adult, it is not accurate in pediatric population. In Palm method, each palm including fingers represents 1% of TBSA. Lund and Browder chart is used in pediatric population as the surface area of the neck and head is much larger compared to the limbs in children (Kagan and Smith, 2000).

Pathophysiology of Shock in Severe Burns

When the burns are severe or involve more than 1/3rd of the TBSA leading to typical derangements of cardiovascular functions and shock, it is commonly called as burn shock (Fig. 1). The etiopathology of burn shock is combination of direct tissue injury, hypovolemia and the storm of pro-inflammatory mediators generated by burn injuries. The strong negative interstitial fluid pressure, capillary leak, loss of glycocalyx and endothelial activation lead to rapid edema formation in burn patients. Although the colloids are blamed for the increased edema formation in burns, the recent study showed that the use of crystalloid fluids in resuscitation increases the glycocalyx damage and shock thus increasing the cellular leak and more edema formation (Wurzer et al., 2018).

Figure 1: Pathophysiology of edema and shock in burns.
Indications for Intensive Care Unit (ICU) Admission of Burn Patients

Following burn patients should be admitted to intensive care unit (ICU) for monitoring and/or management (Spiro and Lambert, 2015),

1. Patients with chemical and electric burns
2. Inhalation injury patients
3. Burns involving more than 20% of the TBSA
4. Patients with burns shock and organ dysfunction
5. Burns involving extremities, genitalia and major joints.

Burn patient management in ICU

Moderate to severe burns and above-mentioned patients are better managed in a specialized burn centre with dedicated burns ICU as multidisciplinary teamwork and skilful management in the intensive care unit is essential for outcome of severely injured patients (Schiefer et al., 2016).

Airway management: Inspite of recent developments in airway management and ventilation, the inhalation injuries remained a leading cause of morbidity and mortality in burn patients. The risk of inhalation injury increases with percentage of burns and approximately 2/3rd of patients with 70% of TBSA burns will have inhalation injuries (Lpaktchi and Arabi, 2006). Oxygen supplementation should be started as early as possible to neutralize the effects of toxic gases in these patients. The involvement of upper airway in inhalation injury can progress rapidly and lead to airway catastrophes, concomitant fluid resuscitation can exacerbate the laryngeal edema making endotracheal intubation difficult or even impossible. Hence the ATLS (advance trauma life support) course advice that patient with inhalation injury should be intubated early. Other indications in inhalation and major burn injury patients are before transferring them to the specialised burn centre or anticipated to develop ARDS (acute respiratory distress syndrome) (Lpaktchi and Arabi, 2006).These patients should be intubated with a larger possible size ETT (endotracheal tube) to facilitate bronchoscopy and the ETT should be secured well to avoid any dislodgments. Initial mode of ventilation should be the one which the treating physicians are familiar with, using100% oxygen. After obtaining the ABG (Atrial blood gas) and the levels of carbon monoxide we can titrate the Ventilatory setting accordingly. Lpaktchi K, et al. found that the lung protective ventilation reduces the ventilator associated events in burn patients (Oribabor et al., 2018). Chung KK, et al. suggested that the high frequency percussion and high frequency oscillatory ventilation are useful in ventilating major burn patients (Lpaktchi and Arabi, 2006). Aerosol heparin and N-acetylcysteine nebulisations are found to reduce ventilator days in the inhalation injury patients (McIntire et al., 2017).
Fluid therapy: In patients with severe burns and burn shock due to capillary and cellular leakage, there will be depletion of the intravascular volume, hence aggressive fluid resuscitation is required for initial 48 hours to replenish the intravascular volume status and to maintain the end organ perfusion. Any delay or inadequate resuscitation in severely burn patients will increase the morbidity and mortality (Spiro and Lambert, 2015). At the same time one must be careful as over resuscitation can complicate into compartment syndromes, pneumonia and ARDS. A survey of burn resuscitation revealed that up to 58% patients receive more than recommended fluids (Oribabor et al., 2018).

There are number of formulas available to estimate the initial fluid requirement in severely burned patients. Unfortunately, any of these formulae will not provide precise method to determine the fluid requirement, but they describe the starting points and initial guidelines for fluid management (Schiefer et al., 2016). We should tailor the fluid therapy in severely burn patients according to the age, associated injuries and comorbidities by using dynamic parameters of preload namely ITBVI (Intrathoracic blood volume index) and GEDVI (Global end diastolic volume index) to avoid the complications of under or over resuscitation.

European burn association found a wide variation in the formulas for initial fluid resuscitation in burn patients. Parkland formula was preferred by 69.3% and others used Brook 6 to 9%, Galveston 8.9%, Warden 5.9% and colloids by 11.9% (Greenhalgh, 2010). The difference between the Parkland and modified Brook formula is the amount of crystalloid prescription, in former it is 4ml/kg, in latter formula it is 2ml/kg (Table 2a & b). Commonly used crystalloid fluid is ringer lactate and out of the total calculated fluid requirement, half amount should be given in the initial 8 hours and remaining half in the next 16 hours. (Table 2a & b) Additional daily maintenance fluid should run separately.

US army institute of surgical research (USAISR) proposed rule of 10 (ISR rule of 10) for the resuscitation of severe burn patients (Table 2c). Most of the care givers also use 5% albumin as colloid resuscitation decreases the requirement of the crystalloids. The use of albumin in initial 12 hours is discouraged due to the cellular leak and creep out of the albumin, decreasing the intravascular volume (Lundy et al., 2016). However a recent meta-analysis showed decrease in mortality in burn patients with use of albumin (Navickis et al., 2014). USAISR and modified Brook formula advice to use 5% albumin as shown in Table 2.

Adjuvant for fluid therapy: The use of vitamin C in high dosage reduces the endothelium dysfunction and reduces the fluid requirement and increases the urine output. The risk of acute kidney injury and formation of renal stones limit routine use of vitamin C (Buehner et al., 2016).
Table 2: Formulas for fluid resuscitation in severe burns.

<table>
<thead>
<tr>
<th>Formulae</th>
<th>Total Volume and Plan of administration</th>
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<tbody>
<tr>
<td>Parkland Formula</td>
<td>Volume: 4ml/kg Lactated Ringer/% TBSA</td>
</tr>
<tr>
<td></td>
<td>Plan administration: 50% to be given in 8 hours &amp; 50% to be given in next 16 hours</td>
</tr>
<tr>
<td>MODIFIED BROOKE’S Formula</td>
<td>Volume: 2ml/kg Lactated Ringer/% TBSA</td>
</tr>
<tr>
<td></td>
<td>Plan administration: 50% to be given in 8 hours &amp; 50% to be given in next 16 hours</td>
</tr>
<tr>
<td>Rule of 10</td>
<td>Estimates Burn Area to nearest of 10%</td>
</tr>
<tr>
<td></td>
<td>Initial rate (ML/ Hour) equal to TBSA×10 (Body weight 40-80 kgs)</td>
</tr>
<tr>
<td>USAISR MODIFIED BROOKE’S Formula</td>
<td>TBSA burn 0-29%: No albumin</td>
</tr>
<tr>
<td></td>
<td>TBSA burn 30-49%: 0.3ml/kg/% of burn</td>
</tr>
<tr>
<td></td>
<td>TBSA burn 50-69%: 0.4ml/kg/% of burn</td>
</tr>
<tr>
<td></td>
<td>TBSA burn 70-100%: 0.5ml/kg/% of burn</td>
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**Analgesia and Sedation**

A recent survey about analgesia and sedation in critically ill burn patients showed that opioids are commonly used analgesic and 45% of the care givers use morphine analgesia (Depetris et al., 2018). The Society of Critical Care Medicine (SCCM) guidelines for management of analgesia in ICU patients heavily rely on narcotic / opioids, but there is awareness about opioid induced hyperalgesia where opioids are increasingly blamed for hyperalgesia (Holtman and Jellish, 2012). The opioid sparing effects of dexmedetomidine, ketamine, NSAID (non-steroidal anti-inflammatory drugs) and paracetamol are well described in the literature (Asmussen et al., 2013). Hence we should maximise the use of opioid sparing agents to reduce the requirement of opioids.

The practice pattern of sedation in burn ICU patients varies widely all over the globe. The choice of sedative agents in burns patients depends on severity, duration of mechanical ventilation, hemodynamic status and the resources available. In a recent survey about analgesia and sedation in critically ill burn patients. Depetris N, et al. (2018) found that the commonly used sedative agent was midazolam (72% of the burn patients) (Depetris et al., 2018). Inspite of strategies to use non-benzodiazepine sedatives (dexmedetomidine, ketamine and propofol) as they reduce opioid requirements and delirium. The benzodiazepine remained an important sedative agent due to deep sedation, amnesia, anxiolytic effects and sedation sparing effect. European intensivists use benzodiazepines initially and later on during weaning from the ventilator shift to other non-benzodiazepines (Barr et al., 2013). Ketamine is often used for sedation and analgesia in severely
burned patients due to its favourable effects on hemodynamics, opioid sparing effects and potential to decrease the long term neurocognitive impairments (McGhee et al., 2011). The use of continuous dexmedetomidine infusion provides bridging effect during weaning from the mechanical ventilation, reduces the requirement of higher doses of opiates and benzodiazepines, decreases incidence of delirium and provides modification in hypermetabolic response similar to the β-blockers with improved nutrition support (Asmussen et al., 2013). Asmussen S, et al. in their meta-analysis found that dexmedetomidine as an adjuvant in burn patients improves sedation compared to the midazolam and ketamine alone (Asmussen et al., 2013).

**Wound Management**

In burn patients' eschars are formed due to deep and full thickness burns and become stiff and unyielding causing circumferential chest movement restrictions, resulting in ventilation difficulties, abdominal and extremity compartment syndromes affecting perfusion to vital organs and the extremities. Hence these burn patients should have escharotomy earlier before it complicates into the compartment syndromes (Lundy et al., 2016).

After cleaning the burn wounds, a fine non-adherent mesh should be applied over the burned area, deeper burn wounds will require multiple debridements and at a later stage skin grafting and/or flap coverage. Various local antimicrobial creams are used in burn patients, commonly used are silver sulphadiazine, Nano crystalline silver, fucidin, bismuth impregnated petroleum gauze which improved the wound care and decreased the incidence of infection in burn patients (Norman et al., 2017).

**Sepsis in Burn Patients**

The loss of skin barrier, immune dysfunction and multiple invasive devices predispose these patients for higher risk for infection and sepsis and as high as 61% of the burn patients die of sepsis (Lundy et al., 2016; Navickis et al., 2014; Buehner et al., 2016; Depetris et al., 2018; Holtman and Jellish, 2012; Asmussen et al., 2013; Barr et al., 2013; McGhee et al., 2011; Norman et al., 2017; Gómez et al., 2009). Inappropriate use of antibiotics in burn patients will lead to development of multidrug resistant bacteria, antibiotic associated diarrhoea or clostridium difficile colitis whereas perioperative use of antibiotics will not decrease the burn wound infections (Lundy et al., 2016; Navickis et al., 2014; Buehner et al., 2016; Depetris et al., 2018; Holtman and Jellish, 2012; Asmussen et al., 2013; Barr et al., 2013; McGhee et al., 2011; Norman et al., 2017; Gómez et al., 2009; Barajas-Nava et al., 2013). Antibiotic should only be prescribed if patients who had raised sepsis markers associated with clinical signs and symptoms of sepsis.
Pneumonia is the common infection in patients with burns, reported to occur in 38% of the patients. Four percent of critically ill burn patients will have bacteraemia and the common bacteria is staphylococcus. Before the practice of applying the local antibiotic cream to the burns area, the invasive gram -ve burn wound infection was the common cause of death (Navickis et al., 2014; Buehner et al., 2016; Depetris et al., 2018; Holtman and Jellish, 2012; Asmussen et al., 2013; Barr et al., 2013; McGhee et al., 2011; Norman et al., 2017; Gómez et al., 2009; Barajas-Nava et al., 2013, Brown et al., 2004). The routine change of catheters are not recommended, the antimicrobial coated or impregnated catheters are of benefit in these patients (Ramos et al., 2006).

**Nutrition, Hyperglycaemia and Hypermetabolism**

Major burn injury causes hypermetabolic state resulting in protein utilization, loss of lean body mass and hyperglycaemia. This hypermetabolic state helps in providing glucose to the glucose dependent tissues but ultimately causes immune impairment, sepsis and multiorgan dysfunction leading to adverse outcome in post-burn patients (Holm et al., 2013). This post burn hyperglycaemia and hypermetabolism are associated with increased catabolism, bacteraemia, fungemia, skin graft loss and mortality. Use of intensive insulin therapy is associated with increased survival in burn patients (Swell et al., 2013). Insulin has various beneficiary effects such as immunomodulation, trophic effects on mucosa and skin barrier thus reducing the bacterial invasion as well as translocations (Swell et al., 2013). Hence the random blood sugar should be controlled between 8-10 mmols by using insulin in all these critically ill burn patients.

Hart, et al. found that the high carbohydrate diet and early enteral feeding in these critically ill burn patients has a beneficial effect, whereas use of selenium, zinc and other trace elements has not found to have definitive beneficial effects in burn patients (Lundy et al., 2016; David et al., 2001). In burn patients if hypermetabolic response is not controlled, it can lead to increased morbidity and mortality. Early burn wound debridement and closure, maintaining environmental temperature 30°C, use of anabolic steroids and b-blockers are associated with reduction in hypermetabolism and inflammation in severely burned patients (Herndon et al., 2001).

**Acute Respiratory Distress Syndrome (ARDS)**

Acute respiratory distress syndrome (ARDS) occurs in up to 17% of all burn patients, it can increase up to 40 to 50 % in severely burned patients requiring mechanical ventilation. Ventilation options for these ARDS patients are low tidal volume ventilation (LTV), high frequency ventilation and open the lung and keep it open techniques. In severely ill burn patients with increased metabolic demand, loss of chest compliance due to edema and eschar makes LTV less practical. Chung, et al. in
their randomized controlled trial found that in 1/3rd of patients LTV had failed to ventilate and high frequency percussion was better option for ventilating burn patients with ARDS (Chung et al., 2010). Other non-Ventilatory therapies used in burn patients are inhalation of nitric oxide, prostacyclin, fluid restriction, diuresis, prone position, ECMO (extracorporeal membrane oxygenation) (Schuster et al., 2008; Hale et al., 2012).

**Acute Kidney Injury (AKI)**

Acute kidney injury (AKI) occurs in up to 30% of the severe burn patients increasing the mortality from 80 to 100% if AKI requires haemodialysis (Brusselaers et al., 2010). There are various risk factors of AKI in severely burn patients namely hypovolemia, sepsis, medications and abdominal compartment syndrome (ACS). The therapeutic approaches to prevent AKI in these patients are adequate fluid resuscitation, avoiding nephrotoxic medications, early detection and treatment of sepsis and earlier escharotomy in patients with ACS. In patients with AKI haemodialysis should be initiated early without waiting for traditional indications for haemodialysis. In burn victims if renal failure is effectively predicted and promptly managed, there will be a significant reduction in morbidity and overall mortality, especially in severe burn injury (Emara and Alzalia, 2013).

**Gastrointestinal Dysfunction**

Various factors cause gastrointestinal dysfunction in severely burn patients leading to mucosal hypoperfusion, ileus and feed intolerance and Curling’s ulcers with increase in morbidity and mortality (LeVoyer et al., 1992). The combination of early optimal fluid resuscitation, earlier starting of enteral feeds (within 24 to 48 hours of injury), gastric ulcer prophylaxis, use of prokinetics and avoidance of NSAID and steroid will prevent gastrointestinal dysfunctions in severely burn patients (Lundy et al., 2016).

**Cardiovascular Dysfunction**

Burn shock, as described initially in pathophysiology, needs adequate resuscitation otherwise the combination of hypovolemia, decreased myocardial contractility and increased blood viscosity will lead to persistent shock state. Refractory shock in burns patients can be due to infections, full thickness burns, adrenal insufficiencies and vasoplegic shock. The septic shock should be managed as per the Surviving Sepsis Guidelines and in patients with vasoplegic shock the use of methylene blue may be of benefit (Shaefi et al., 2018).
Neurologic Dysfunction

Inhalational injury may cause hypoxic or anoxic brain injury which may lead to brain death in burn patients. The electric burn may cause life threatening arrhythmias and cardiac arrest may complicate into severe brain injuries (Jonathan et al., 2013). The common neurological problem in burn patient is delirium due to benzodiazepines sedation, disturbance of normal rhythm of circadian, frequent sepsis comorbidities particularly CVA (cerebro vascular accidents). Fortunately, 70% of the burn care givers assess these patients for delirium and 62.5% give combined pharmacological and non-pharmacological treatment in burn patients with delirium (Depetris et al., 2018).

Mortality in Burns

According to the estimation of WHO (World Health Organization) around 265 000 fatal burns occur annually (Lee et al., 2014). Peck, et al. reported decline in burn mortality from various regions in the world with decrease of burn incidence and burn severity as well as an overall reduction of mortality rates and LOS (Peck, 2011). Severe burn mortality is 10% (Pavoni et al., 2010).

Reports from Iran, shows that 9% of burn victims die with annual mortality of 4.6 to 5.6 per 100,000 people (Sarbazi et al., 2019). Adolescence, illiteracy, unemployment, residing in rural areas, low socioeconomic status and percentage of TBSA burned were found to be significant factors in increasing the mortality rate (Sarbazi et al., 2019). The improvement of health care facilities and preventive measures will decrease the burns mortality. The nature of burn, burned body surface over 40% and timing of first debridement are associated with mortality (Tabiee and Nakhaee, 2004). Dastgiri, et al. found that the TBSA of more than 75% has a higher mortality risk. Burns on the head and neck, trunk and upper extremity increase the mortality risk (Dastgiri et al., 2006). The improved awareness of burns pathophysiology with the recent advances in intensive care, renal replacement therapy, and nutritional support had contributed for the reduction of burns patients’ mortality (Stylianou et al., 2015; Dokter et al., 2014). Simultaneously the improvements in infection control had added to the decline in mortality of these patients (Al-Shaqsi et al., 2013). The reduction of length of hospital stay was the main improvement in burn treatment particularly due to early surgical therapy, newer wound dressings which allow less frequent burn dressing changes, reduced infection rates in combination with above mentioned patient care developments (Harats et al., 2015; Dokter et al., 2014; Jonsson et al., 2015).

Conclusion

The overall incidence and severity of burns are showing descending trend, still burn injuries remain a major trauma related injury. Major /severe burn injuries are the injuries involving more than
20% of total body surface area (TBSA) and these severe burn injuries patients should be managed in burn centre with a dedicated ICU, where the teamwork and skillful critical management will have a better impact. Burn shock occurs due to tissue injury, hypovolemia and storm of proinflammatory markers.

The intensive care management of severely burned patients includes securing the airway and optimal ventilation, fluid therapy initially guided by various formulas and meticulously tailored by using the dynamic preload parameters, using opioid sparing sedatives to minimize use of opioids and benzodiazepines to avoid opioid hyperalgesia and delirium. Early escharotomy will prevent the occurrence of various body compartment syndromes in severely burned patients. These patients are at a higher risk for infection and sepsis due to loss of dermatological protective barrier. Early detection and appropriate management of sepsis is necessary for better outcome. Hyperglycaemia and hypermetabolism should be controlled by insulin therapy, use of dexmedetomidine and b-blockers. Severely burned patients frequently complicate into multiorgan dysfunction namely ARDS, AKI, burn shock, gastrointestinal and CNS dysfunction and should be managed as described above.

The recent advances in intensive care therapy, burn wound care, organ and nutritional support and infection control had improved the outcome in severe burn patients.

Burns injuries are preventable and impact can be minimized by using smoke alarms, exit doors and other preventive measures developed over the last few years.

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Review Article


