Fluid Management in Major Burns

19

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Contents

IFA Commentary (MLNGM)

Burn care has greatly improved over the past few decades, thanks to better understanding of burn shock pathophysiology and the development of targeted burns resuscitation. While inadequate fluid resuscitation shock is now rare in clinical practice due to early aggressive intervention, attention has shifted to the morbidity and mortality related to post-resuscitation oedema and fluid creep in burns care. Severe burns cause systemic inflammation and fluid extravasation due to transendothelial hyperfiltration, with patients with more than 20% total burn surface area at the greatest risk. The disruption of large areas of normal skin results in both sustained heat

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and fluid loss, complicating temperature regulation. In the absence of adequate fluid resuscitation immediately following the burn, a patient would rapidly develop intravascular hypovolaemia due to elevated capillary transendothelial pressure and hyperfiltration across the injured endothelium, resulting in low-output shock. While adequate fluid resuscitation is essential in preventing critically low cardiac output, the presence of elevated transendothelial pressure means there is significant extravasation of resuscitation fluid and oedema formation. This oedema can develop in both burned and unburned tissue, occurring within minutes after the injury and peaking at 12–24 h after injury. Endothelial dysfunction and capillary leak are present within 2 h post-burn. There is no consensus on the ideal resuscitation fluid and strategy, nor on how to achieve adequate resuscitation while avoiding the adverse effects of excessive resuscitation. Significant variability exists in fluid strategies and haemodynamic monitoring during burn care by clinicians. The latest evidence regarding the choice of fluids, adjunctive treatments, the role of abdominal hypertension, and the end points used to guide fluid resuscitation in burn patients are summarised in Table 19.1. The IFA suggests a novel, holistic, and dynamic resuscitation protocol with targets and end points for the more challenging burn cases that includes an active de-resuscitation phase according to the ROSE concept and based on newly available physiologic parameters from transpulmonary thermodilution [1, 2].

Suggested Reading

1. Peeters Y, Lebeer M, Wise R, Malbrain M. An overview on fluid resuscitation and resuscitation endpoints in burns: past, present and future. Part 2—avoiding complications by using the right endpoints with a new personalized protocolized approach. Anaesthesiol Intensive Ther. 2015; 47:S15–26.
2. Peeters Y, Vandervelden S, Wise R, Malbrain M. An overview on fluid resuscitation and resuscitation endpoints in burns: past, present and future. Part 1—historical background, resuscitation fluid and adjunctive treatment. Anaesthesiol Intensive Ther. 2015; 47:S6–S14.

Table 19.1 Recommendations regarding fluid resuscitation and resuscitation end points in severe burn patients

Fluids	
1. Normal saline	Given the fact that fluid resuscitation in burn management requires large volumes, the use of saline cannot be recommended in a burn resuscitation protocol
2. Balanced crystalloid	Based on the available evidence, balanced crystalloid solutions are a pragmatic initial resuscitation fluid in the majority of acutely ill (and burn) patients

Table 19.1 (continued)

Fluids	
3. Semi-synthetic colloids	Given the recent data concerning the use of semi-synthetic colloids (and starches in particular), their use in critically ill patients including burn patients cannot be recommended
4. Albumin	Based on the available evidence, the use of albumin 20% can be recommended in severe burns, especially in the de-resuscitation phase guided by indices of capillary leak, body weight, (cumulative) fluid balance, fluid overload, extravascular lung water, and intra-abdominal pressure
5. Hypertonic solutions	To this day, there is insufficient evidence to reach consensus regarding the safety of hypertonic saline in burn resuscitation. Whenever using hypertonic saline in clinical practice however, close monitoring of sodium levels is highly advised
Adjunctive therapy	
6. Vitamin C	Vitamin C prevents intra-abdominal hypertension in burns patients. However the current level of evidence for Vitamin C means that it cannot be recommended routinely
7. Plasmapheresis	The benefit of plasmapheresis on outcome in burn patients still needs to be validated in large prospective, randomised trials. As such, its use cannot be recommended
8. Other therapy Hydrocortisone Oxygen Hydroxocobalamin Sedation	In case of use of etomidate for intubation, the secretion of cortisone could be suppressed for up to 18 h as for patients regularly taking corticoids High levels of oxygen (100%) for up to 6–18 h are required for CO intoxication and smoke inhalation trauma Severely burn casualties can suffer a very early refractory shock – Most of the time outright on scene – During house fire with smoke inhalation injury and cyanide intoxication. The antidote consists of intravenous hydroxocobalamin 70 mg/kg of body weight Avoid hypotensive and cardiodepressive sedation
Abdominal hypertension	
9. Intra-abdominal pressure (IAP)	During the resuscitation phase as well as the recovery phase, intra-abdominal pressure (IAP) needs to be measured in burn patients at least four to six times per day
10. Medical treatment	Medical management (improvement of abdominal compliance, evacuation of intra-abdominal contents, evacuation of intra-luminal contents, limitation of fluid intake, optimisation of organ perfusion) comes first and should be initiated whenever IAP increases above 12 mmHg
11. Surgical treatment	Escharotomies should be performed in case of circular thoracic or abdominal eschars, while surgical decompressive laparotomy is only a last resort in case medical management fails

(continued)

Table 19.1 (continued)

Fluids	
Resuscitation end points	
12. Monitoring	Every severely burned patient (>20% TBSA in adults or > 15% TBSA in children) should be adequately monitored with regard to fluid status, fluid responsiveness, and organ perfusion
13. Urine output	Diuresis is a poor end point in the complex cases (many recent articles still recommend UO as the criteria with the other classical haemodynamic parameters) that may lead to over- or underestimation of fluid resuscitation and as such can no longer be recommended; however, in situations with limited monitoring techniques, it can still be used to guide fluid resuscitation (see further under urine output algorithm)
14. Barometric preload	Barometric preload indicators like central venous pressure (CVP) or pulmonary artery occlusion pressure (PAOP) should not be used to guide fluid resuscitation in burn patients. It can still be used or least the trend of the CVP in situations without modern monitoring
15. Volumetric preload	Volumetric preload indicators (like right ventricular or global end-diastolic volume) are superior compared to barometric ones and are recommended to guide fluid resuscitation, especially in burn patients with increased IAP. (see further under GEDVI algorithm.)
16. Lung water	The use of extravascular lung water is recommended to guide de-resuscitation in burn patients not transgressing spontaneously from ebb to flow phase
17. Fluid responsiveness	Fluid resuscitation in burn patients should be guided by physiological parameters or tests that are able to predict fluid responsiveness. (see further under PPV algorithm.)
18. Perfusion	Fluid resuscitation should only be given/increased in case of evidence of tissue hypoperfusion (base deficit, lactate, etc.)
Stepwise approach	
19. PPV algorithm	If a patient is sedated and mechanically ventilated, an algorithm based on pulse pressure variation (PPV) can be used in severe burns, under the condition that PPV measurements are reliable with an experienced staff (Fig. 19.1)
20. GEDVI algorithm	If PPV is unreliable, volumetric parameters obtained with transpulmonary thermodilution can be used to guide fluid resuscitation in severe burns. Here, the GEDVI is interpreted as a measure of preload and EVLWI as a safety parameter warning for pending pulmonary oedema (Fig. 19.2). If the GEDVI is high, the measurement needs to be corrected with the global ejection fraction as this leads to a more accurate estimation of preload
21. Urine output algorithm	If PPV or volumetric parameters are unreliable, or when monitoring possibilities are limited, urine output can be used to guide fluid resuscitation in severe burns (Fig. 19.3)

CVP central venous pressure, *EVLWI* extravascular lung water index, *GEDVI* global end-diastolic volume index, *IAP* intra-abdominal pressure, IVIG: intravenous immunoglobulins, PAOP: pulmonary artery occlusion pressure, TBSA: total burned surface area

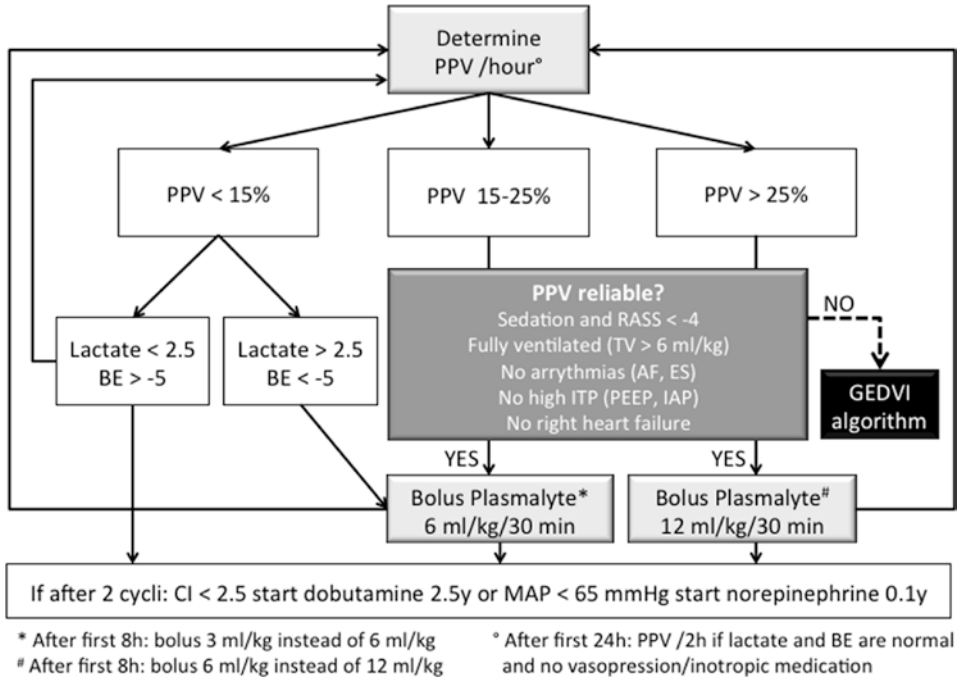

Fig. 19.1 Pulse pressure variation algorithm to guide resuscitation in severely burned patients. If the patient is mechanically ventilated and PPV is reliable, fluid resuscitation is guided by the PPV algorithm [14, 15]. *AF* atrial fibrillation, *BE* base excess, *CI* cardiac index, *ES* extrasystole, *GEDVI* global end-diastolic volume index, *IAP* intra-abdominal pressure, *ITP* intrathoracic pressure, *MAP* mean arterial pressure, *PEEP* positive end-expiratory pressure, *PPV* pulse pressure variation, *TV* tidal volume

Learning Objectives

After reading this chapter, you will:

1. Understand the pathophysiology of burn shock and the development of targeted burn resuscitation.
2. Identify the risk factors for post-resuscitation oedema in burn patients.
3. Evaluate the evidence regarding the choice of fluids and adjunctive treatments for burn patients.
4. Describe the role of abdominal hypertension in burn patients and its implications for fluid resuscitation.
5. Analyse the end points used to guide fluid resuscitation in burn patients and their appropriateness in different clinical scenarios.
6. Discuss the principles of a holistic resuscitation protocol for burn patients, including targets and end points for the more challenging cases.

7. Explain the concept of transendothelial hyperfiltration and its role in the generation of tissue oedema in burn patients.
8. Assess the importance of avoiding capillary hypertension in preventing transendothelial hyperfiltration and associated complications in burn patients.
9. Identify the biomarkers and techniques that can be used to track the development of burn injury and guide resuscitation.
10. Recognise the adverse effects of excessive fluid resuscitation, including intra-abdominal hypertension and compartment syndromes, and strategies to mitigate these risks.

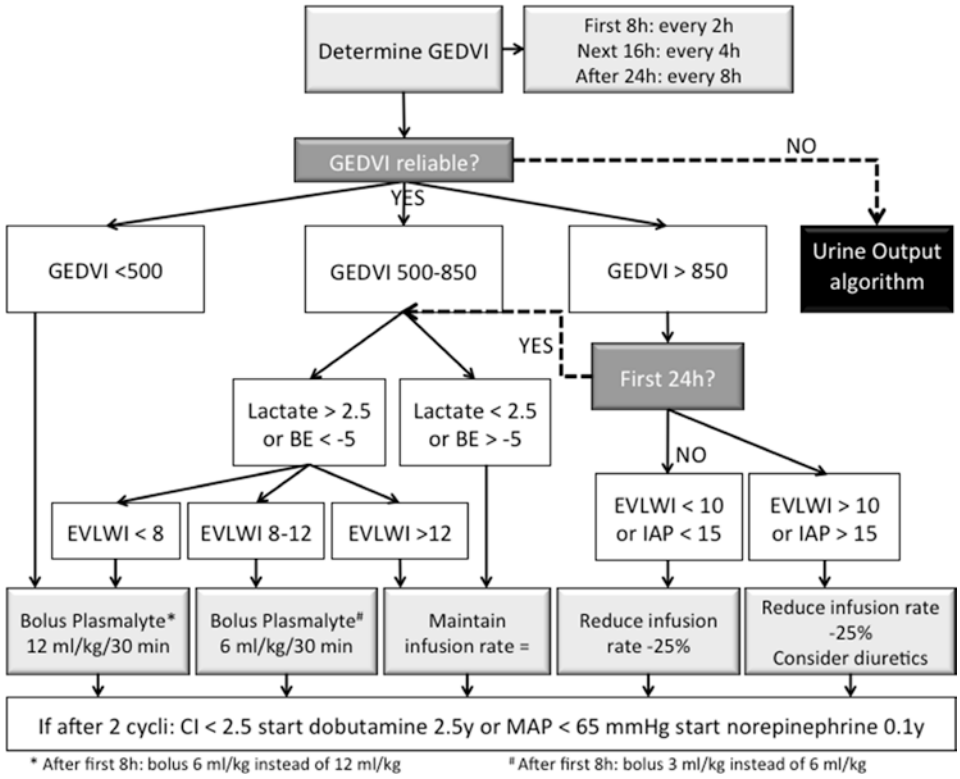

Fig. 19.2 Global end-diastolic volume index algorithm to guide resuscitation in severely burned patients. If PPV is unreliable and the patient has a PiCCO catheter and GEDVI is reliable, fluid resuscitation is guided by the GEDVI algorithm [14, 15]. *BE* base excess, *CI* cardiac index, *EVLWI* extravascular lung water index, *GEDVI* global end-diastolic volume index, *IAP* intra-abdominal pressure, *MAP* mean arterial pressure

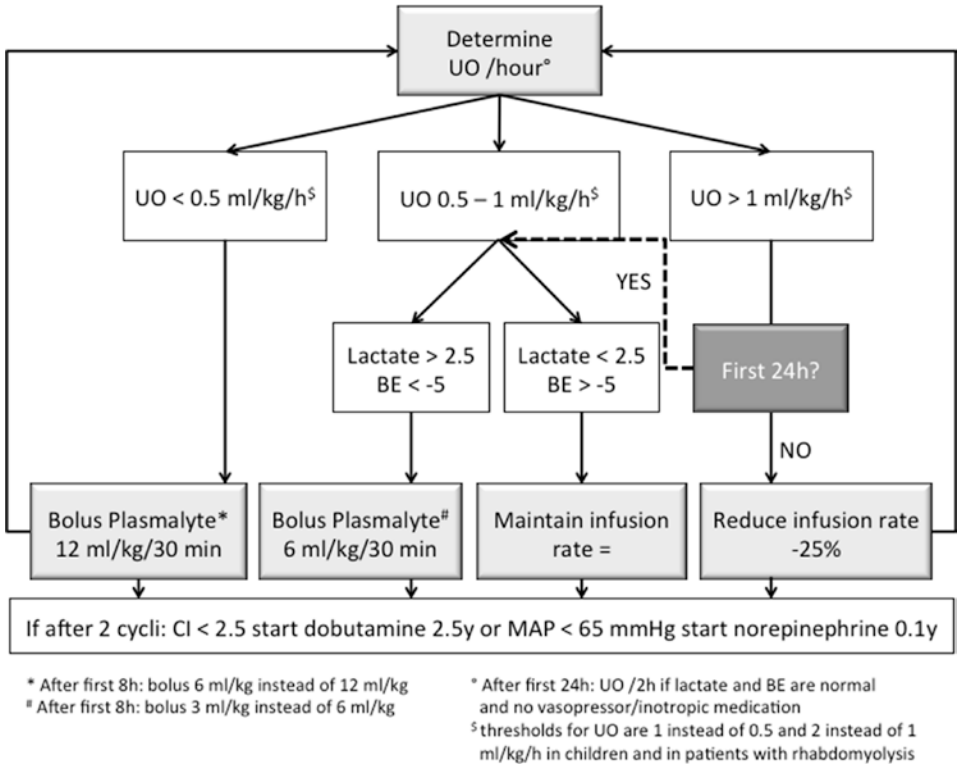

Fig. 19.3 Urine output algorithm to guide resuscitation in severely burned patients. If the patient has no PiCCO catheter (or GEDVI is not reliable) and PPV is not reliable, fluid resuscitation is guided by the UO algorithm [14, 15]. *BE* base excess, *CI* cardiac index, *IAP* intra-abdominal pressure, *MAP* mean arterial pressure, *UO* urine output

Case Vignette:

Mr. S, a 42-year-old male, was brought to the emergency department with second-degree burns covering 25% of his total body surface area. He was in pain and hypotensive with a blood pressure of 90/60 mmHg, heart rate of 120 beats per minute, and a urine output of 20 mL/h. He had no significant medical history, and his initial labs showed elevated serum lactate levels. The medical team initiated fluid resuscitation using intravenous fluids.

Questions

Q1. Why is intravenous fluid resuscitation essential for burn patients?

Q2. What is the goal of fluid resuscitation in burn patients?

Introduction

Burn management has undergone a lot of changes with advancements in the medical field over the past few decades. As our understanding of the pathophysiological consequences of burns has evolved, so has our approach in the daily management of the burn patients. Early resuscitation is essential as hypovolaemia sets in rapidly, with compromised cardiac function leading to what has been called as burn shock. If left untreated, burn shock can cause 58% of deaths within 72 h of major thermal injury [1]. Appropriate and timely resuscitation is essential to offset the development of burn shock and has been clearly shown to reduce mortality. However, fluid management in this subset of patients is based on formulae and concepts elucidated decades ago, with no clear consensus on the ideal resuscitation fluid. Colloid use in burns remains mired in controversy; resuscitation targets and ideal monitoring tools are still poorly defined.

The ideal fluid for burns resuscitation is debatable, though there appears to have been a shift back towards usage of colloids along with crystalloids.

Colloids were used as early as 1942 when Cope and Moore, following the Cocoanut Grove disaster [2]. Over time, with the knowledge that capillary leakage led to accumulation of sodium-rich fluid in the burned tissues and that the intravascular volume could be corrected with balanced salt solutions, crystalloids became favoured over colloid use during the first 24 h. Resuscitation formulae utilising crystalloids include the Baxter and Shires Parkland formula and modified Brooke formula by Pruitt, both using Ringer's lactate for resuscitation during the first 24 h [3]. There was also increased recognition that over-resuscitation is as dangerous as under-resuscitation and leads to a vast array of complication ranging from kidney injury, worsening of oedema, ARDS, airway oedema, and loss of skin grafts secondary to tissue oedema, a concept known as "fluid creep" as proposed by Pruitt [4].

Early and rapid resuscitation of the burn patient is a priority, as hypovolaemia and burn shock sets in rapidly especially in patients with major burns, generally defined as burns involving more than 20% total body surface area. Shock in burns has features of distributive, hypovolaemic, and cardiogenic shock; fluid administration needs to be tailored to the characteristics of the patient, as the rapid sequestration of the intravascular volume into the second and third space needs to be counterbalanced to maintain adequate tissue perfusion and prevent organ damage. This chapter will focus on adult patients, and more information on fluid therapy in children can be found in Chap. 20. Some other chapters will discuss fluids in specific populations: sepsis (Chap. 14), heart failure (Chap. 15), trauma (Chap. 16), neurocritical care (Chap. 17), perioperative setting (Chap. 18), liver failure (Chap. 21), abdominal hypertension (Chap. 22), and COVID-19 (Chap. 26).

Pathophysiology

Burns to a large surface area cause extensive damage, not only directly but also by causing a cascade of changes which ultimately increases the morbidity and mortality. Burns to more than 15–20% of total body surface area are a major cause of mortality unless they receive prompt and adequate resuscitation.

Injury to the tissue in and around the burns area causes disruption of sodium ATPase, leading to intracellular accumulation of sodium and fluid shift resulting in cellular oedema and intravascular depletion.

There is disruption of the collagen and hyaluronic acid scaffolding which normally maintains the integrity of the interstitial space, leading to loss of fluid in the extravascular spaces. Furthermore, the loss of intravascular proteins to the interstitial spaces reduces plasma oncotic pressure; these changes are profound in the injured area but also occur in the non-injured areas. This reduced plasma oncotic pressure leads to intense oedema formation especially once fluid resuscitation starts [5].

In addition, thermal injury results in the release of inflammatory mediators and vasoactive products like histamines, prostaglandins, and leukotrienes along with activation of complement system; these have a twofold effect. Firstly, they contribute to increased vascular permeability leading to extensive fluid shifts from the intravascular compartment resulting in oedema, hypovolaemia, and haemoconcentration. Secondly, these mediators appear to cause cardiac dysfunction – the intense local vasoconstriction coupled with systemic vasodilatation result in increased afterload and reduced preload. Tumour necrosis factor alpha and IL-1 have been found to be elevated in burn patients with apoptotic cells detected in left ventricular tissue; these changes are hypothesised to be contributors to the cardiac dysfunction. Cardiac output is not fully restored with fluid resuscitation as the preload improves and the complete resolution may take 48–72 h [5].

Burn shock is a combination of hypovolaemic, cardiogenic, and distributive shock. These changes become evident as early as within first five to 6 h post injury, more so in the injured area; therefore, prompt adequate fluid resuscitation and maintaining the intravascular volume in the face of ongoing third spacing are extremely important while managing a patient with burns especially during the first 24 h. Over-resuscitation leads to a whole set of different complications and should be avoided. Capillary integrity in non-injured tissue gradually recovers in 12–24 h, beyond which intravascular losses decrease and aggressive fluid management can be tailored down to maintain adequate tissue and organ perfusion (Fig. 19.4).

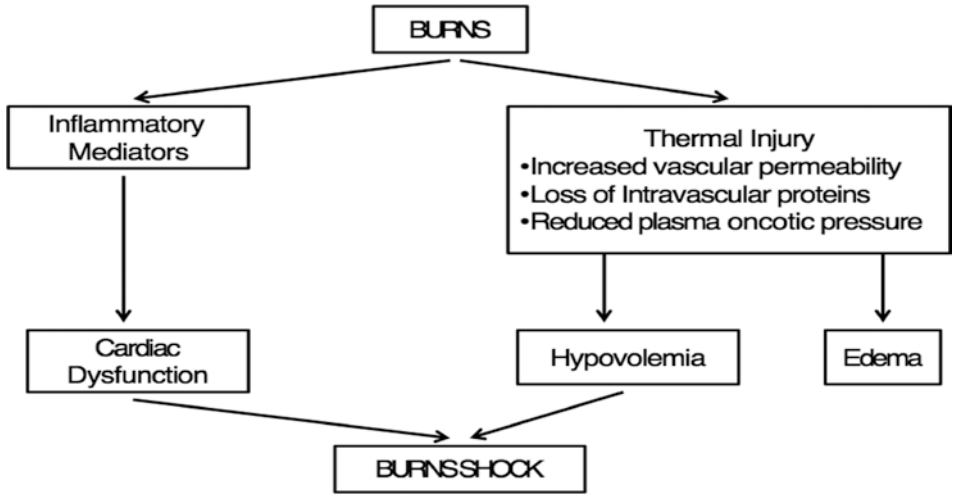

Fig. 19.4 Pathophysiology of burn shock

Fluid Estimation and Administration

Estimation of percentage of body surface area (BSA) burnt is the first step when managing a patient. The Wallace rule of nines, the Lund and Browder chart (in paediatric patients), and the rule of palm (using the patient's palm without fingers and wrist as 1%) can all be used to estimate the BSA. During BSA estimation, only deep partial-thickness burns (previously called second-degree burns) and full-thickness burns (previously called third- and fourth-degree burns) are calculated for fluid administration; superficial partial-thickness burns (previously called first-degree burns) are not included.

Numerous formulae for fluid resuscitation have been proposed; however, the formula given by Baxter and Shires at the Parkland Hospital (referred to as the Parkland formula) is the most commonly followed universally, using Ringer's lactate up to 4 mL/kg/% TBSA (total burned surface area) in the first 24 h, half during the first 8 h and the rest over the next 16 h. It is important to remember that time is estimated from the time of injury, and in cases of delayed presentation a more rapid fluid administration may be required.

The intravenous route of fluid resuscitation is preferred, although oral or enteral routes can also be used in cases of limited resources. Vomiting and gastric ileus may however make it difficult to deliver large amounts of fluids orally. Intraosseous lines can be used in emergencies or if intravenous access is difficult.

IV cannulae need to be properly secured as the developing oedema may dislodge the cannulae or make further access difficult; a central venous catheter may be prudent in cases of significant torso and limbs burns.

Arterial catheterisation can be helpful especially for blood pressure monitoring and repeated sampling and also in cases where the burns make it difficult to place the cuffs or electrodes.

Early and adequate resuscitation is important to prevent renal failure, organ dysfunction, and death. Hypovolaemia and shock rapidly develop in the absence of timely fluid administration, and inadequate fluid resuscitation leads to worsening of burns shock. On the contrary, overzealous and unchecked fluid administration results in "fluid creep" [4]—this tends to occur when the total volume of resuscitation exceeds 6 mL/kg/% TBSA, or the Ivy index of 250 mL/kg. Fluid creep causes worsening of oedema in the injured and uninjured area, intra-abdominal hypertension, ARDS, intraocular hypertension, conversion of superficial to deep burns due to impaired vascularity, and organ failure. Fluid administration needs to be initiated timely especially during the initial hours, as ongoing third spacing of fluid causes intense intravascular losses and maintenance of adequate intravascular volume is paramount to ensure adequate tissue perfusion. Care should be taken while assessing patients, as patients presenting with shock probably have some other underlying injury causing hypotension since burn shock generally sets in gradually.

Choice of Fluid and Monitoring

Colloids were initially the fluid of choice for burn resuscitation, earlier formulae advocated plasma as the replacement fluid, e.g. Harkins, or body weight burn budget, Evans formulae. Gradually, it was observed that the fluid lost in burn tissue was rich in sodium and proteins, and the volume could be replaced with balanced solutions only, whereas (ab) normal saline (NaCl 0.9%) given in large amounts more rapidly results in hyperchloraemic metabolic acidosis.

Currently, most patients receive Ringer's lactate or Plasma-Lyte during initial resuscitation, as per the Parkland formula and modified Brooke formula. Ringer's lactate while hypotonic tends to correct the volume status and electrolyte imbalance and also avoids hyperchloraemic acidosis that occurs with the use of normal saline. The problem with lactate containing buffered solutions is that when they are given in vast amounts as is the case in severe burns, plasma lactate levels may increase due to exogenous lactate accumulation in combination with diminished metabolisation especially in shock and liver failure.

The problem with crystalloid resuscitation is the large volume of fluids involved, which is recognised to result in worsening oedema, renal dysfunction, airway complications including ARDS, abdominal compartment syndrome, and intraocular hypertension. This has led to renewed interest in colloids, especially human albumin. Colloid use reduces the volume required for resuscitation especially in cases of anticipated larger volume losses including delayed presentation, large burn area, and inhalational injuries [6]. Post major burns, there is generalised increase in capillary permeability even in the unburned tissues

with loss of albumin and smaller proteins molecules; this is however transient lasting for up to 12 h. Oedema and further third spacing are nonetheless persistent due to hypoproteinaemia and reduction in plasma oncotic pressures; increased lung water also occurs as a result. Colloids maintain the intravascular volume by maintaining the plasma oncotic pressure and decreasing third spacing [7]. Various formulae utilise colloids early or late as rescue therapy; Slater and Haifa formulae utilise FFP and plasma immediately post-injury. Goodwin et al. used albumin early in resuscitation and found reduced volume requirement and improved cardiac function [8]. Others have used albumin 8–12 h later as rescue fluid in cases of high projected volume losses and have shown a trend towards reduced volume of resuscitation and mortality reduction[6, 9] . However, data regarding timing and dose are still lacking – a meta-analysis by Navickis et al. [10] found no significant effect on mortality when albumin was used during the first 24 h; but when they excluded two studies with high risk of bias, there was in fact a reduction in mortality with albumin as well as marked reduction in the development of a compartment syndrome.

Hydroxyethyl starch (HES) is associated with increased risk of renal dysfunction and their use in burn resuscitation remains controversial and should be abandoned.

Hypertonic saline has been used for resuscitation keeping in mind that large-volume crystalloids will result in more severe oedema and complications including acute kidney injury [11].

Fresh frozen plasma (FFP) is not without its own problems, namely, infection risk and TRALI; hence, it is not the first choice of colloid for burns resuscitation.

Urine output monitoring guides fluid resuscitation, with a target urine output of 0.5 mL/kg to 1 mL/kg, although this parameter is unreliable in renal dysfunction. There is a danger of increasing the volume of infusion to offset low urine output without appropriate subsequent de-escalation. Goal-directed fluid therapy has been studied in burns including transpulmonary thermodilution [12]. However, there is insufficient data to support its use; these devices are also unavailable in many centres.

Lactate and base deficit as monitoring tools have been studied but their role in the face of ongoing third spacing is not well established. B-type natriuretic peptide (BNP) and proteinuria have also been studied as potential monitoring tools during resuscitation; a high BNP level with low proteinuria was associated with better outcomes [13]. We refer to the IFA commentary and some recent papers looking more in detail at the different monitoring targets and goals [14, 15].

Case Vignette

- Why is intravenous fluid resuscitation essential for burn patients?

  Answer: Intravenous fluid resuscitation is essential for burn patients to prevent hypovolaemic shock and acute kidney injury. Burn injuries cause the loss of intravascular volume, leading to hypovolaemia, hypotension, and inadequate tissue perfusion. IV fluids are given to restore intravascular volume, correct electrolyte imbalances, and improve organ perfusion.

- What is the goal of fluid resuscitation in burn patients?

  Answer: The goal of fluid resuscitation in burn patients is to maintain adequate organ perfusion and treat shock. The Parkland formula can be used to determine the initial volume of fluid needed for resuscitation, which involves giving of lactated Ringer's solution/Plasma-Lyte 4 mL/kg/body weight/percentage of the total body surface area (TBSA) burned. Half of the calculated volume is given in the first 8 h post-burn, with the remaining half given in the next 16 h. The goal is to maintain a urine output of 0.5–1 mL/kg/h, which indicates adequate organ perfusion.

  Fluid management should be tailored to the patient's condition, urine output, etc., and strict adherence to resuscitation formula is not recommended.

Conclusion

Burn resuscitation poses a unique challenge, as progressive intravascular depletion leads to burn shock; early and appropriate therapy aims to minimise or prevent burns shock, tissue hypoperfusion, and organ dysfunction. Under-resuscitation increases morbidity and mortality, whereas overzealous fluid administration is equally harmful and causes fluid creep. Fluid administration needs to be tailored to the patient's condition and strict adherence to resuscitation formulae may not be prudent; physicians need to tailor their resuscitation strategy to the evolving targets. Colloids have a place later on in the resuscitation process particularly in cases of anticipated larger fluid volume requirement including deeper burns, inhalational injuries, and late presentation.

Take Home Messages

- Estimation of the percentage of body surface area (BSA) burnt is the first step when managing a patient, and there are several methods to estimate BSA.
- During BSA estimation, only deep partial-thickness and full-thickness burns are calculated for fluid administration; superficial partial-thickness burns are not included.
- The most commonly followed formula for fluid resuscitation is the Parkland formula, which uses Ringer's lactate up to 4 mL/kg/%TBSA in the first 24 h, half during the first 8 h and the rest over the next 16 h.
- Time is estimated from the time of injury, and in cases of delayed presentation a more rapid fluid administration may be required.
- Early and adequate resuscitation is important to prevent renal failure, organ dysfunction, and death.

- Care should be taken while assessing patients, as patients presenting with shock probably have some other underlying injury causing hypotension since burn shock generally sets in gradually.
- Most patients receive Ringer's lactate or Plasma-Lyte during initial resuscitation, as per the Parkland formula and modified Brooke formula; however, fluids should be tailored on the patient's individual needs following the ROSE concept.
- Crystalloid resuscitation results in worsening oedema, renal dysfunction, airway complications including ARDS, abdominal compartment syndrome, and intra-ocular hypertension.
- Colloid use reduces the volume required for resuscitation, especially in cases of anticipated larger volume losses including delayed presentation, large burn area, and inhalational injuries.
- Clear data on the type timing and dose of colloids early or late as rescue therapy are still lacking.

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