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JSM Burns and Trauma

Editorial

Burns Fluid Resuscitation with Considerations for Changing Target Goals

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Submitted: 09 May 2018

Accepted: 09 May 2018

Published: 11 May 2018

ISSN: 2475-9406

EDITORIAL

The ideal resuscitation fluid is one that produces a sustained increase in intra vascular volume without any change in chemical composition of body fluid or any adverse metabolic effect [1]. For effective resuscitation by crystalloids, usually rapid transfusion of large volume of Ringer's lactate is required which often results in extreme elevation of total body sodium and water content (interstitial edema especially if associated with hypoproteinemia/albuminemia). Due to edema, if there is compression of venous drainage at thoracic inlet it may exaggerate edema over head and neck area [2].

Resuscitation end point (target goal)

Though best clinical guideline for adequate resuscitation is urine output 1-2ml /Kg/ hour. Also, commonly in practice systolic arterial pressure (SAP) '≥ 100 mmHg OR ≥ 80% baseline value' is taken as resuscitation end point [3]. The mean arterial pressure (MAP) is a better determinant of organ perfusion than SAP. Hence, it is important to take MAP ≥ 60 mmHg as end point in case of hypovolemia without significant increase in capillary permeability, but in SIRS or sepsis it is better to take all of following as end point guidelines: CVP 8-12 mm Hg (if available/not contraindicated in burn patients), MAP ≥ 65 mm Hg, Urine output ≥ 0.5 mL/kg/hr, and Central venous oxygen pressure (Scvo2) ≥ 70% (The 2016 Surviving Sepsis Guidelines) [4,5].

Though there is low quality of available evidence, it is better to normalize lactate (Lactate ≥ 4 mmol/L) in patients with elevated lactate levels as a marker of tissue hypo perfusion [4]. For the purpose of convenience of description the subject will be discussed in three heads-

Fluid volume

Parkland and Brooke formula are commonly used as guide to initial fluid resuscitation. For more than 25 years in my practice, use of modified Brooke formula (2ml/Kg/% body surface area burn crystalloid + daily requirement during first 24 hour (first half in first 8hours and next half in next 16 hours); 1ml/Kg/% body surface area burn crystalloid + daily requirement during next 24 hour) as initial guide to fluid resuscitation has given quite satisfactory result. Albumin transfusion is avoided in first 24 hours. This volume may be changed in major amputee cases according to the corrected total surface area burn ($C_{TBSA=}$TBSA x (100/100-surface area of amputated part) [6]. Response to same resuscitation fluid volume may vary from patient to patient due to several reasons. Even if other variables are kept constant, only burn depth index in same size of burn in two different but comparable patients may considerably influence the response to fluid resuscitation [7]. First degree burn is subtracted from TBSA burn for the purpose of fluid calculation. It is better to limit TBSA burn to 50% for the purpose of fluid calculation in very extensive burn (>50% TBSA burn) and later modify fluid volume as per response in next 3 hours.

In absence of any complication of excess fluid transfusion, if target end point is not reached with modified Brooke formula, relatively larger volume (up to 30%) with or without inotrops/vasopressors may be considered. In case of resuscitation end point not reached within 48 hours or in cases of SIRS/sepsis 30ml/kg crystalloid is transfused for 6 hours and reassessed for re-fixation of new target end point and rate of transfusion. Hypovolemic shock due to delayed resuscitation may require increased fluid volume by 30% for adequate resuscitation; but if stage of irreversible shock is attained, patient may not respond to fluid resuscitation. To avoid mechanical blockade of renal tubules due to myoglobinuria and hemoglobinuria in extensive 3rd degree burn and electric burn, urine output is maintained at 100-150ml/hour by transfusing more fluid and if required by use of diuretics (osmotic). To avoid precipitation of myoglobin and hemoglobin, urine is made alkaline by intravenous soda bicarbonate at frequent intervals. In response to burn trauma, excess circulating catecholamine may cause renal vessel spasm with reduced urine output in spite of adequate fluid transfusion. In these cases low dose dopamine (3-5 microgram/kg/minute) can produce desirable effect by increasing renal perfusion.

Leak of transfused fluid due to increased capillary permeability may lead to subcutaneous compartment syndrome [8,9] with tissue ischemia and conversion of superficial burn to deep burn [10]. To reduce resuscitation fluid volume albumin transfusion or bolus resuscitation may be considered. The bolus resuscitation with albumin or saline (FEAST- Fluid expansion as supportive therapy) in pediatric population has shown to increase death rate significantly [11,12]. Large volume fluid transfusion in extensive burn patients, semi-recumbent position and elective mechanical

ventilation with increased airway pressure and higher PEEP within permissible limits may be helpful in protecting from pulmonary edema. Incisions/eschorotomy/punctured wounds over burnt area and intermittent negative pressure dressings (e.g. Limited access dressing) [13], may protect from problems of excessive subcutaneous fluid collection. In burn patients abdominal compartment syndrome (intra-abdominal pressure > 30 cm H_2O) associated with large volume resuscitation may increase mortality by 70-100% [14]. Eschorotomy (H shape) over abdomen with two vertical limbs of H on either side of abdomen helps to relieve the intra-abdominal pressure. Fluid overload may increase interstitial edema of kidney and compress capillaries with increased intra tubule pressure leading to reduced filtration gradient with consequent acute kidney injury (AKI). Hence, use of diuretics at the point of onset of oliguria after desired volume transfusion may be beneficial in reducing kidney interstitial edema and possible excretion of proinflammatory chemicals in urine with obvious advantages. In cases of inhalation injury and renal inefficiency, it is better to do arterial blood gas analysis (ABG) at repeated intervals to diagnose and treat acid base balance abnormalities.

If MAP target is not achieved by initial fluid as per standard fluid formula, CVP measurement is indicated and established resuscitation protocols are used to change the course of the treatment. Use of vasopressor (noradrenaline) in hypovolemia along with altered capillary permeability may produce tissue ischemia due to vasoconstriction; and consequent oxidative stress may lead to exaggerated inflammatory cascade leading to poor prognosis. Hence, once CVP target is achieved by crystalloid or colloid and MAP target is not achieved, and cardiac cause/ other causes of hypotension is excluded; serum albumin/albumin + globulin level correction should be considered. Vasopressor and inotrops may be required as adjunct therapy (noradrenaline 0.4 mg/hour to 0.8 mg/hour noradrenaline base (0.8 mg/hour to 1.6 mg/hour noradrenaline tartrate); i.e 40 mg/litre noradrenaline base (80 mg/litre noradrenaline tartrate) at the rate of 0.16 to 0.33 ml/min; addition of vasopressin 0.03units/minute has been recommended to reduce nor-adrenaline dose. If MAP target is not achieved phenylephrine 200-300microgram/minute may be added [15]. Once MAP target is achieved, Scvo2 level should be checked to determine end of resuscitation. If Scvo2 target is not achieved, packed red blood cells should be transfused to raise hematocrit by 30%. Once target end point of resuscitation is achieved further step down therapy is started. If alteration of course fails, transition to comfort care is planned.

The resuscitation failure is considered to exist when at least one major complication of fluid overload (pulmonary edema, abdominal compartment syndrome, subcutaneous compartment syndrome, multi-organ dysfunction and failure) appears during resuscitation [15]. Failure to resuscitate by standard protocol fluid volume are usually due to delayed resuscitation, cardiac dysfunction because of carbon monoxide poisoning, coronary artery disease, and old age; occurrence of sepsis, MODS and MOF.

Use of stroke volume optimization is a better guide to fluid therapy than central venous pressure (CVP) and urine output measurement. Clinically low stroke volume may not be precisely indicated clinically by tachycardia, reduced cardiac output urine output, reduced SpO2, and hypotension. Development of newer technologies for estimation of instant blood volume in critical patients may be a better idea in stroke volume stabilization [16,17]. Also, instant blood volume measurement along with CVP may prove to be a better guide to remove accumulated excess transfused volume by continuous renal replacement therapy (CRRT)/use of diuretics (in case of normal kidney function) in cases of subcutaneous and abdominal compartment syndrome, pulmonary edema and AKI.

ROLE OF COLLOID TRANSFUSION

Large amount of albumin (4%) transfusion or saline as resuscitation fluid has been shown to be associated with significant increase in rate of death/ organ failure [18,19]. Albumin transfusion as early resuscitation fluid in children at 2 year age with brain injury has been shown to significantly increase in death as compared to saline as resuscitation fluid [20]. Serial serum protein study [21] to calibrate/grade the capillary pore size, may be a better guide for albumin transfusion to reduce the chances of albumin leak. Albumin transfusion should be avoided in first 24 hours. It is better to consider albumin transfusion if serum albumin level is less than 20gram/L. It is safer to transfuse 5% albumin 0.5–1 g/kg/% burn per day till serum albumin reaches at the level of 30gram/L [21]. It will be better to transfuse larger protein molecule (e.g. globulin) if capillary pore size is larger than albumin size [22].

MANIPULATION OF CAPILLARY PERMEABILITY AS A PRIMARY GOAL OF RESUSCITATION

Following burns and trauma platelet degranulation, and local complement activation (in response to oxidative damage following ischemia and reperfusion to endothelial cell membrane, neutrophil adhesion to endothelium and by apoptotic endothelium) leads to inflammatory response causing local vasodilatation, increased capillary permeability, and further recruitment and activation of neutrophils leading to reperfusion injury. This response is further amplified by release of TNF-alpha and interleukins from activated neutrophils [1,2]. In addition to oxidative stress, supra-physiological loads of glucocorticoids (exogenous/ endogenous), subsequent infection (bacterial degradation/toxins) and other similar insults e.g. shock, surgical trauma (multi hit theory) leads to an inflammatory cascade (acute phase protein synthesis, up regulation of inflammatory adhesion molecules and pro-inflammatory cytokine) [3-5]. When these molecules and cytokines are absorbed in systemic circulation, it produces systematic inflammatory response syndrome/ sepsis (in presence of infection) that ultimately may lead to MODS/ MOF with poor prognosis. Hence, Oxidative damage, ischemia, reperfusion, change in capillary permeability (with loss of fluid, electrolyte and serum protein leading to shock), are main factors that serve as guide to fluid therapy. Apart from this any preexisting disease (kidney, lungs, heart) or trauma to other organs also regulate fluid resuscitation.

Also, as the changed capillary permeability persists for an unspecified period, the controversy in resuscitation may reduce if we study it and target capillary pore normalization [22] as primary therapeutic goal and use current resuscitation goal as supportive therapy goal. Also, the accumulated excess

fluid along with proinflammatory cytokines and other harmful chemicals should be actively removed at the earliest. Hence, more accurate method of capillary pore calibration, instant blood volume estimation and removal of excess accumulated fluid (e.g. CRRT/ active use of appropriate diuretics) needs to be developed/studied. Drugs like high doses of vitamin C (2g per day), antihistaminic and Aprotinin that prevents end organ damage due to hypotension may be investigated in manipulating capillary pore [10].

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Cite this article

Kumar P (2018) Burns Fluid Resuscitation with Considerations for Changing Target Goals. JSM Burns Trauma 3(1): 1038.