Early treatment and intensive care of children with burn injury

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Major burn injury remains a significant cause of morbidity and mortality in pediatric patients. The treatment of burned children differs substantially from that of adults not only because of the different body proportions but also because of the metabolic processes involved, hormonal responses, the immunological profile, the degree of psychological maturation and healing potential. After assessing the overall physiological status of the child, accurate assessment of the burn injury and appropriate fluid resuscitation are of great importance. The severity of burn injury is characterized by the depth of the burn, total body surface area (TBSA) that is involved, the location of burn injury and the presence or absence of inhalation injury. Early excision and grafting, adequate nutrition, alleviation of the hypermetabolic response, treatment of hyperglycaemia, and physical therapy improve survival and outcomes in children with severe burns.

Key words: burn injury, children, fluid resuscitation.

INTRODUCTION

Major burn injury is the fifth leading cause of unintentional child injury related death. Flame and scald burns, caused by spilling of boiling liquids, constitute the major mechanisms of burn injury in the pediatric population. Children at the age of 4 and younger are at greatest risk.

All children should be considered for the possibility of abuse, neglect, or an unsafe environment that may necessitate social services intervention.

Young children have much greater ratio of body surface area to weight compared with adults, the skin is thinner, they have greater percentage quantity of body water in relation to weight, lower tolerance of a rapid variation of body liquid volumes, greater risk of onset of hypothermia, and more accentuated metabolism. All those clinical differences determine children’s response to the heat trauma that is quite specific and imposes different treatment from the moment of the burn.

INITIAL ASSESSMENT

Before assessing the extent and depth of burn, the overall physiological status of the child must be assessed.

Airway is the first priority in the management of a burn injured child. Pediatric airway is much smaller and more easily occluded by edema. Early intubation is preferable to an emergency cricothyroidotomy, especially in an edematous, burn injured neck.

Evaluation for the possibility of inhalation injury and carbon monoxide intoxication is very important. Clinical markers for inhalation injury include: respiratory distress, hypoxemia, hoarseness, stridor, wheezing, oropharyngeal blistering, tongue swelling, carbonaceous sputum and singed eyebrows and nasal hairs. Diagnosis is made by bronchoscopy and standard care protocols include bronchodilatators, nebulised heparin, nebulised acetylcysteine and racemic adrenaline for the extreme mucosal oedema. The use of corticosteroids is contraindicated.

Any child with an isolated flame injury that presents obtunded is presumed hypoxic from carbon monoxide intoxication but may also need to be evaluated for the possibility of concomitant closed head injury. Pulse oximetry and arterial blood gas measurements are not accurate for assessing carboxyhemoglobin levels and can be misleading. A blood carboxyhemoglobin level measures the degree of carbon monoxide intoxication and helps guide the therapy. Treatment with high concentrations of inspired oxygen displaces carbon monoxide from Hgb.

A prolonged oxygen requirements in the face of normal carboxyhemoglobin level may signify acute lung injury from smoke inhalation. Acute lung injury results from toxin induced injury to the lung parenchyma that impairs
alveolar function and usually manifests 24 h to 48 h after the initial insult. 7

After assessment of the airway and respiratory status, fluid resuscitation is the next priority in the initial stages of treatment. For more severe burns (>10% TBSA) intravenous rehydration with an emergency solution and formula (valid for the first 2 hours): 20 ml/kg/h of Ringers lactate is necessary.

It is very important to perform analgesia immediately without any prejudices regarding the use of opioids which at the recommended doses do not create any problems even for very small children.

**BURN ASSESSMENT**

Assessing the extent and depth of a burn helps in determining initial triage and management. Extent of injury is expressed as a calculated % TBSA. Percentage of TBSA is best estimated using the Lund-Browder chart that accounts for childhood changes in body proportion with growth. 8 In the pediatric population, the surface area of a child’s hand approximates 1%TBSA over a wide range of ages. 6

Modern burn classification system classifies burns by increasing depth: superficial, superficial partial, deep partial, full and deep full thickness, also known as subdermal.

The severity of burn injury is characterized by the TBSA involved, depth, location (burns involving the face, hands, feet, or perineum are considered major burn injuries), child’s age and concomitant diseases, presence of inhalation injury, and associated trauma.

**PATHOPHYSIOLOGY**

Pathophysiologic abnormalities associated with burn injury include metabolic derangements, neurohumoral responses, massive fluid shifts, sepsis, and the systemic effects of massive tissue destruction. Significant burns compromise the immune function and the ability of the skin to regulate temperature and retain moisture. Surface colonization and infection of burn wounds coupled with local and systemic immune dysfunction make infectious complications one of the leading causes of death for children with extensive burn injury.

Heat injury activates the release of inflammatory and vasoactive mediators responsible for local vasoconstriction and systemic vasodilatation, as well as increased transcapillary permeability. Released mediators include complement proteins, kinins, histamine, bradykinin, serotonin, prostaglandins, and oxygen derived free radicals and neuropeptides. Fluid shift from the microcirculation is enhanced by decrease in oncotic pressure because protein is lost to the interstitial space.

Due to these pathophysiological changes generalized dehydration, hyperkalemia, hyponatremia, metabolic acidosis and hemokoncentration occur. Failure to restore cardiac output with fluid resuscitation leads to inadequate tissue perfusion, progressive organ dysfunction, circulatory collapse and death.

Systemic dysfunction impacts all organ systems, and after 3 to 5 days from burn injury children develop a hypermetabolic state, with an increase in resting energy expenditure, increase of cardiac output, tubular dysfunction, encephalopathy etc.

The precise underlying cause is not well understood but it is most likely induced by stress and inflammation, and persistent increases in catecholamines, glucocorticoids, glucagon and dopamine secretion that trigger the hypermetabolic response and subsequent catabolism. 9

Hypermetabolism results in gluconeogenesis, insulin resistance and hyperglycaemia, increased lipolysis, and protein catabolism. Hyperglycaemia in patients with burns is associated with increased frequency of infections, sepsis, incidence of pneumonia, and mortality. 9 Lipolysis and free fatty acids contribute to morbidity and mortality after burn injury through fatty infiltration of various organs. Findings from pathology analyses and spectroscopy studies have shown that children with burns have a three to five times increase in hepatic triacylglycerides. 10

**CRYSTALLOID RESUSCITATION**

There is no consensus on the specific formula or the type of fluid to be administered to burn patients. Optimal fluid resuscitation aims to support organ perfusion with the least amount of fluid necessary. Resuscitation formulas are useful as starting guidelines, rather than rigid goals for volume resuscitation and the volume infused should be continually titrated to avoid both under and over resuscitation.

The most widely used Parkland formula for over 40 years, provides 4 ml/kg%TBSA burned of Ringer Lactate to be given over the first 24 h (half of total volume over the first 8 hours and the rest over the next 16 hours).

Recent studies have found that average volumes administered to contemporary burn patients far exceed formula predictions. The most of this fluid creep is attributable to a tendency to maximize preload using invasive monitors over targeting urine output, an increased use of opioids and sedatives that may antagonize the stress response or increase vasodilatation, as well as reluctance to decrease infusion rates when urine output exceeds target goals. 11

Increased fluid administration is associated with adverse outcomes, such as worsening edema formation, pleural effusions, pericardial effusions, pulmonary edema, elevated compartment pressures, acute respiratory distress syndrome (ARDS) and multiple organ failure.

Patients who routinely require additional fluid than volume proposed by Parkland formula are patients with inhalation injuries, secondary injuries including multiple trauma, alcohol and drug use, patients with electrical burns and those in whom resuscitation was delayed.

Limited physiological reserves in children mandate increased vigilance and precision during resuscitation from burn injuries. In centers experienced with pediatric burns, formulas have been developed that include maintenance fluid based on body surface area in addition to estimated needs based on burn size.
Glucose homeostasis is an important parameter in children. Hepatic glycogen stores in young children are depleted after 12 to 14 hours of fasting. It is therefore important to provide sufficient glucose substrates during first 24 hours of resuscitation.

**COLLOID RESUSCITATION**

Considerable controversy persists as to the role and type of the colloid in burn resuscitation.

The advantages of using colloids for fluid resuscitation are reduced oedema, reduced incidence of compartment syndrome, reduced gastrointestinal oedema and incidence of ileus. The disadvantages are potentially higher costs, possible viral transmission and increased febrile incidence with fresh frozen plasma, leakage from the vascular compartment, thereby elevating the extracellular osmolality. The issue of pulmonary extravascular water during the resuscitation period is also contradictory. Holm et al. confirmed that increases in lung water and capillary permeability are rare, even in the presence of an inhalation injury. Many burn centers report that they never use colloids in their initial resuscitation, during the first 24 hours, after which capillary integrity may be sufficiently restored to allow manipulation of intravascular oncotic pressure. Others have reported successful resuscitation with plasma, albumin, and high molecular weight glucose polymers such as dextran and hydroxyethylstarch.

Except for transient loss of capillary integrity, nonburn tissues soon regain the ability to sieve plasma proteins. This physiologic argument has prompted some clinicians to adopt a "middle of the road" approach, whereby colloids are administered later in the second half of the first 24 hours. The most common colloid used is albumin, followed by HES and fresh frozen plasma.

**RESUSCITATION ENDPOINTS**

Hourly urine output as the primary index of optimum resuscitation contrasts with more sophisticated monitoring devices to assess the moment to moment physiologic state available in modern burn centers. For instance, abnormal arterial lactate and base excess values correlate with the magnitude of injury and their failure to correct over time predicts mortality. There are no prospective studies to support the use of these parameters to guide fluid resuscitation. Increasing preload and cardiac index with aggressive volume administration has no benefits. Invasive monitoring with central venous catheters or pulmonary artery catheters may still be occasionally indicated for patients with inadequate response to standard treatment.

**ADJUNCTS TO FLUID RESUSCITATION**

Antioxidant therapy

Membrane lipid peroxidation and oxygen-derived free radicals are major components of burn shock pathophysiology. Treatment with high dose ascorbic acid reduces edema and fluid requirements during resuscitation.

**Treatment of the hypermetabolic response**

Since hypermetabolism is a major contributor to poor outcome after burn, treatment of hypermetabolic response is beneficial for patient outcomes.

Early enteral nutrition alleviates catabolism, however, overfeeding in the form of excess calories or protein, or both, is associated with hyperglycemia, carbon dioxide retention, fatty infiltration of organs, and azotemia. Accurate calculation of the caloric requirements is imperative.

Nutrition that is high in glucose, protein and amino acids and low in fat with some unsaturated fatty acids is recommended and should be initiated within 12 h after injury.

Plasma concentration of vitamins and trace elements are substantially decreased for prolonged periods after the acute burn injury because of increased urinary excretion and substantial cutaneous losses. Replacement of these micronutrients reduces morbidity in patients with severe burns.

Early excision and grafting have been the biggest advances in burn care in the past few decades and they substantially reduce basal energy expenditure, mortality and costs. Increasing ambient room temperature from 25 C to 33C also reduces resting energy expenditure in patients with serious burns.

Physical therapy is a crucial intervention that can promote muscle protein synthesis, increase body mass, build endurance and prevent contractures of the burn wound. It is recently shown that a burn is not over once burn wounds are healed, and that profound pathophysiologic responses persist for a substantially longer time, as seen by a 3 year increase in energy requirements, catecholamines, urine cortisol, serum cytokines, and impairment in glucose metabolism and insulin sensitivity.

**CONCLUSION**

The ultimate goal of intensive burn care is to keep the patient alive, an outcome that is dependent on coverage of burn wounds, maintenance of organ function, control of infection and sepsis, and alleviation of hypermetabolism.

The fluid status of burned children is a dynamic process that requires careful reevaluation and therapeutic adjustments.

Recognizing the basic elements of burn physiology in children is important for implementing a successful resuscitation.

**SUMMARY**

**INICIJALNI TRETMAN I LEČENJE PEDIJATRIJSKIH PACIJENATA SA TEŠKIM OPEKOTINAMA**

Teške opekotine predstavljaju značajan uzrok morbidi-teta i mortaliteta u pedijatrijskoj populaciji. Njihovo lečenje se značajno razlikuje od lečenja adultnih pacijenata ne samo usled različitih proporcija tele već i zbog razlika u metaboličkim procesima,
hormonalnom statusu, imunološkom profilu, stepenu fiziološke maturacije i potencijalu za zarastanje opećenih površina.
Nakon procene opštega stanja deteta, određivanje težine opekotine i adekvat na terapija tečnostima su od najvećeg značaja. Težinu opekotine određuje njena dubina, površina tela koju zauzima, lokalizacija, kao i postojanje inhalacione povrede.
Rana ekcizija opećenih površina i grrafting, adekvatna nutritacija, regulisanje glikemije i fizikalna terapija poboljšavaju preživljavanje i krajnji ishod teških opektina u pedijatrijskoj populaciji.

Ključne reči: opekotine, deca, reanimacija tečnostima.

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