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Problem based management in delayed presented burned in dr. Cipto Mangunkusumo General Hospital, Jakarta

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Problem based management in delayed presented burned in
dr. Cipto Mangunkusumo General Hospital, Jakarta

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Abstract

Introduction. Burn injured victims managed in Burn Unit of dr. Cipto Mangunkusumo General Hospital, Jakarta (RSCM) is dominated by delayed presentation and categorized as the difficult cases. Though had been rescued before being referred to our unit, these cases were characterized by massive edema and minimal to nil responsiveness to standard burn fluid resuscitation and were followed by high mortality.

Method. A retrospective study run on those resuscitated in period of 1998–2010 using different protocols aimed to find out the most suitable formula to treat these subjects. Pediatric,–, chemical– and electrical burns was excluded. Hydration status, hemodynamic– and perfusion indices, complication(s), mortality as well as survival days were variables of interest subjected to statistical analysis. Significance met if p <0.05.

Results. Out of 1768 subjects managed, 659 were enrolled in the study. Mortality in those treated in first period was 44.9% with survival 10.10 pbd ± 7.39, in the second period was 54.6% with survival 8.55 pbd ± 6.39, in the third period was 43.4% with survival 11.34 pbd ± 7.34, and the last period was 13.4% with survival 18.78 ± 6.32 pbd.

Conclusion. In these characteristics, perfusion targeted resuscitation showed to be superior than volum

Keywords: delayed presented burned, balanced fluid resuscitation, mortality, survival days

Introduction

Burn injured victims managed in Burn Unit of dr. Cipto Mangunkusumo General Hospital, Jakarta (RSCM) is dominated by delayed presentation and categorized as the difficult cases. Though had been rescued before being referred to burn unit, these cases were characterized by massive edema and minimal to nil responsiveness to standard burn fluid resuscitation,1 and were followed by high mortality. No evaluation has been reported which is addressed to this characteristic; thus, a review of those resuscitated in period of 1998–2010 using different protocols was run; aimed to find out the most suitable formula to treat these subjects, retrospectively.

Method

We run a review to protocol of fluid resuscitation managed in burned subjects treated in Burn Unit of dr. Cipto Mangunkusumo General Hospital, Jakarta, during period of January 1998 to April 2010. Adult burns with delayed presented were enrolled in a study. Those who presented more than 24 hours and pediatric that has specific problems were excluded. In addition, chemical– and electrical burns with specific characteristic other than fluid resuscitation were also excluded.

Efficacy of different protocols applied to subject’s population in each period as shown in table 1 were reviewed. Baseline data such as subject characteristics represented by age, total burned surface area, Baux score, serum albumin, and duration of onset (time of injury to the treatment), hydration status represented by hemoglobin content and hematocrit, hemodynamic assessment represented by central venous pressure and mean arterial pressure, perfusion (represented by oxygen utilization, random blood glucose and base deficit), organ status and resuscitation morbidity that may have such as acute kidney injury and problem of hemostasis were all variables of interest. Efficacy of resuscitation assessed by comparing those variables found in 24 and 48 hours to the baseline. Instead of mortality, increased of survival days indicates an effective protocol. Data collected from Burn Unit Registration. Statistical analysis using SPSS ver.20 is carried out. Levene statistical analysis used to find out homogeneity of population, and Anova test used to find out correlation of this target population to survival; p value of <0.005 and 95% CI is significant.

Results

There were 1768 subjects managed in burn unit during period of 1998–2010, age in ranged of 1–91 years old (27.648 years + 19.266) with total burned surface area ranged of 2–97% (31.913% + 18.639) with Baux score ranged of 21–180 (59.586 + 26.653). In thirteen years, just 44 (2.5%) were presented directly to the emergency department in less than two hours following injury, while as the remains 1682 (95.1%) were referred from other hospitals and presented in emergency department in ranged of 2–72 hours postburn (9.559 hr. ± 7.455). Forty–two (2.4%) presented >24–72 hours postburn. Six hundred and fifty–five subjects met the criteria of major burn defined (>25% TBSA) and were enrolled to a study. Detail of subject’s characteristic is seen in table 2. Using Levene statistical analysis, we found that age, Baux score, and delayed period in each period were met homogeneity with p value of 0.876, 0.075, and 0.54, respectively.
The other characteristic found in these population was low level of serum albumin ranged of 1.2–2.7 g/dL (1.94 g/dL ± 0.69) during period 1998–2010, representing capillary leak. Focused on the protocols applied, it was shown that all protocols showed efficacy to treat hemoconcentration and improved the hydration status. This efficacy is shown in table 3, indicated by dilution which were found increased in those treated with colloid, both of hydroxyethyl starch and Gelatin. Impact of crystalloid is unable to be analyzed.

Despite volume, the use of vasoactive in the protocol applied during period of 2000–2005 to achieve hemodynamic improvement following volume delivery in those refracted (mostly >40% TBSA) is noticed. Dopamine used in the early phase which was then replaced by dobutamine. Epinephrine and nitroglycerine were also found in the records (data not provided). The use of these vasoactive were no longer found any further after 2005.

Oxygen utilization were restored up to maximum 20% with the application of balanced fluid resuscitation compared to former protocols. Unfortunately, this variable was not a focus in the former periods, let the efficacy in earlier two periods couldn’t be analyzed (table 3). Random blood glucose was found to be controlled effectively in all protocols. It was noticed that in the last period (2007–2010) there were no administration of insulin drip to control hyperglycemia found as it noted in the former protocols (data not provided). Focus on the indices of an important clinical value, Baux score, base deficit and serum lactate showed a significant correlation to mortality (table 5 and 6).

Index normalized ratio (INR) were found increased in those with major burn, particularly of >40% TBSA. In the study showed that INR significantly increased to those treated with colloid, both of starch and Gelatin. Impact of crystalloid is unable to be analyzed since no data available. Acute kidney injury (AKI) as an important issue in burns was noted as much as 66.16% of this population at the presentation. In the care, there was significant increased creatinine by 48 hours postburn. In the second and third period, no significant different found on 48 hours postburn. In contrast to the first period, creatinine significantly fall after fluid correction. Overall mortality in this population was 7–66.7%. There was improvement in survival days in those who died on 1–12 post burned days (pbd) (3.04 pbd ± 2.30) in 1998 to 5.56 ± 4.78 in 2003.

**Figure 1.** Superficial vein thrombosis (A) and deep vein thrombosis (B) is a common morbidity found and responsible to a pitfall of resuscitation. Large volume fluid replacement leading to massive edema compressing the vessels in the compartment.
| Period            | Pbh   | Dilution | Hemoglobin (g/dL) | Hematocrit (vol%) | Positive balance | CVP (cmH₂O) | MAP (mmHg) |
|------------------|-------|----------|-------------------|-------------------|------------------|-------------|------------|
|                  | n     | mean ± SD| p                  | n                 | mean ± SD        | p           | n      |
| I                | 1998–1999 | 0        | 16.01 ± 2.39      |                  | 53.11 ± 8.79     |             |         |
|                  | 24    | 71       | 14.55 ± 2.27      | <0.001           | 49.53 ± 8.01     | <0.001      | 72      |
|                  | 48    |          | 13.87 ± 2.12      |                   | 46.59 ± 6.90     |             |         |
|                  |       |          |                   |                   |                  |             |         |
| II               | 2000  | 0        | 17.55 ± 1.96      | <0.001           | 57.25 ± 6.20     | <0.001      | 72      |
|                  | 24    | 25       | 16.34 ± 2.41      |                   | 53.08 ± 6.27     |             |         |
|                  | 48    |          | 15.54 ± 2.26      |                   | 51.11 ± 4.34     |             |         |
|                  |       |          |                   |                   |                  |             |         |
| III              | 2001–2002 | 0       | 16.84 ± 2.39      | <0.001           | 54.37 ± 8.31     |             |         |
|                  | 24    | 97       | 14.27 ± 2.38      |                   | 49.66 ± 7.62     | <0.0001     | 114     |
|                  | 48    |          | 12.97 ± 2.46      |                   | 47.40 ± 6.24     |             |         |
|                  |       |          |                   |                   |                  |             |         |
| IV               | 2003  | 0        | 16.17 ± 2.64      | <0.001           | 52.11 ± 9.54     |             |         |
|                  | 24    | 51       | 12.57 ± 2.17      |                   | 46.43 ± 8.74     | <0.001      | 49      |
|                  | 48    |          | 11.80 ± 2.02      |                   | 44.39 ± 7.40     |             |         |
|                  |       |          |                   |                   |                  |             |         |
|                  | 2004–2005 | 0      | 16.05 ± 2.54      | <0.001           | 53.69 ± 9.64     |             |         |
|                  | 24    | 128      | 14.11 ± 2.35      |                   | 44.77 ± 9.05     | <0.001      | 128     |
|                  | 48    |          | 13.36 ± 2.24      |                   | 43.47 ± 10.56    |             |         |
|                  |       |          |                   |                   |                  |             |         |
|                  | 2007–2010 | 0     | 16.66 ± 2.41      | <0.001           | 58.94 ± 9.17     |             |         |
|                  | 24    | 69       | 13.85 ± 2.38      |                   | 38.75 ± 7.59     | <0.001      | 202     |
|                  | 48    |          | 12.78 ± 2.43      |                   | 37.55 ± 7.32     |             |         |
|                  |       |          |                   |                   |                  |             |         |

Pbh: postburn hours, CVP: central venous pressure, MAP: mean arterial pressure
Table 3. Cellular perfusion in different protocols.

| Period    | PbH   | SvcO₂ (%) | RBG (mg/dL) | INR   | Creatinine |
|-----------|-------|-----------|-------------|-------|------------|
|           | n     | (mean ± SD) | p     | n     | (mean ± SD) | p     | n     | (mean ± SD) | p     |
| I         |       |           |         |       |           |         |       |           |         |
| 1998–1999 | 0     | –         | –      | 43    | 207.81 ± 86.72 | <0.001 | –      | 1.77 ± 0.97 | 0.001 |
|           | 24    | –         | –      | 173.62 ± 78.01 | <0.001 | –      | 2.92 ± 2.00 | 0.001 |
|           | 48    | –         | –      | 145.30 ± 50.15 |        | –      |          |         |
| 2000      | 0     | –         | –      | 232.71 ± 87.11 |        | –      | 2.26 ± 0.74 | 0.001 |
|           | 24    | –         | –      | 169.41 ± 64.62 | <0.001 | –      | 2.56 ± 1.24 | 0.056 |
|           | 48    | –         | –      | 127.28 ± 42.34 |        | –      |          |         |
| II        |       |           |         |       |           |         |       |           |         |
| 2001–2002 | 0     | –         | –      | 192.48 ± 85.03 | 1.63 ± 0.41 |        | 2.50 ± 0.70 | 0.445 |
|           | 24    | –         | –      | 149.97 ± 54.22 | <0.001 | 44      | <0.001 | 85       | 0.001 |
|           | 48    | –         | –      | 138.44 ± 41.92 | 1.79 ± 0.43 |        |          |         |
| 2003      | 0     | –         | –      | 154.85 ± 70.30 | 1.92 ± 0.36 |        | 2.62 ± 0.71 | 0.188 |
|           | 24    | –         | –      | 129.66 ± 36.91 | 0.144  | 20      | <0.001 | 34       | 0.056 |
|           | 48    | –         | –      | 138.47 ± 38.40 | 2.18 ± 0.33 |        |          |         |
| III       |       |           |         |       |           |         |       |           |         |
| 2004–2006 | 0     | 87.80 ± 4.73 | <0.001 | 215.51 ± 109.84 | 1.50 ± 0.38 |        | 2.59 ± 0.72 | 0.050 |
|           | 24    | 85.14 ± 3.84 | 0.299  | 173.92 ± 84.70 | 0.740  | 73      | -4.71 ± 3.00 | 0.163 |
|           | 48    | 84.54 ± 5.41 |        | 145.70 ± 54.22 |        | 59      | -5.44 ± 2.94 |        |
| IV        |       |           |         |       |           |         |       |           |         |
| 2007–2010 | 0     | 85.63 ± 3.85 | <0.001 | 278.30 ± 114.99 | 1.60 ± 0.34 |        | 2.31 ± 1.40 | 0.001 |
|           | 24    | 83.52 ± 3.22 | 63     | 210.00 ± 88.96 | 0.137  | 167     | -4.44 ± 3.13 | <0.001 |
|           | 48    | 80.18 ± 4.58 | 68     | 154.55 ± 68.17 | 1.27 ± 0.28 |        |          |         |

Pbh: postburn hours, SvcO₂: central vein oxygen saturation, RBG: random blood glucose, INR: index normalized ratio.

Table 4. Indices of mortality

| Period    | Mortal | Baux score | Serum lactate | Base deficit |
|-----------|--------|------------|---------------|--------------|
|           | n      | Mean ± SD  | p value       | n            | Mean ± SD  | p value       | n            | Mean ± SD  | p value       |
| 1998–2000 | No     | 0.80 ± 23.55 | 0.299         | 0.70 ± 2.45  | 0.740       | 0.73          | -4.71 ± 3.00 | 0.163       |
|           | Yes    | 0.86 ± 23.06 |              | 0.58 ± 2.88  |            | 0.59          | -5.44 ± 2.94 |            |
| 2001–2003 | No     | 0.68 ± 15.39 | <0.001        | 0.60 ± 3.09  | <0.001      | 0.59          | -5.46 ± 4.22 | <0.001      |
|           | Yes    | 0.55 ± 23.54 |              | 0.49 ± 2.17  |            | 0.54          | -4.44 ± 3.13 |            |
| 2004–2006 | No     | 0.67 ± 14.75 | <0.001        | 0.55 ± 2.71  | <0.001      | 0.54          | -4.44 ± 3.13 | <0.001      |
|           | Yes    | 0.59 ± 23.28 |              | 0.49 ± 2.17  |            | 0.54          | -4.44 ± 3.13 |            |
| 2007–2010 | No     | 0.77 ± 19.33 | <0.001        | 0.66 ± 1.37  | <0.001      | 0.64          | -4.44 ± 9.40 | <0.001      |
|           | Yes    | 0.80 ± 21.83 |              | 0.70 ± 2.42  |            | 0.66          | -17.86 ± 5.39 | <0.001      |

Table 5. Base deficit and serum lactate as a predictor of mortality

| Period    | Base deficit | n | Mean ± SD | p value | Serum lactate | n | Mean ± SD | p value |
|-----------|--------------|---|-----------|---------|---------------|---|-----------|---------|
| 1998–2000 |              | 132| -5.04 ± 2.98 | <0.001 | 131          | 4.99 ± 2.66 | 0.027 |
| 2001–2003 |              | 154| -7.76 ± 4.32 |         | 158          | 5.21 ± 2.97 |         |
| 2004–2006 |              | 86 | -8.03 ± 4.43 | <0.001 | 76           | 4.83 ± 2.17 |         |
| 2007–2010 |              | 173| -8.16 ± 9.82 |         | 157          | 4.36 ± 2.20 |         |
Discussion

Balanced fluid resuscitation showing the efficacy to restore perfusion in delayed presented burned victim in our burn unit. Vigorous fluid resuscitation to treat hypovolemia advocated by the guidelines were ineffective, vice versa leads to high mortality rate. We do believe that the inappropriateness was not on the protocol as questioned by many parties, but incorrect application. Delayed presented acute burn injury representing the features of difficult cases. Fluid resuscitation in these difficult cases should be constituted based on the pathophysiology of burn shock which is in the past believed to be hypovolemic. Extensive capillary leak is essential as body response to thermal injury. Clinically massive edema, unresponsiveness to fluid (sometimes attributed to under resuscitation), low level of serum albumin, deteriorated hemodynamic (low central venous pressure and mean arterial pressure) is obviously found that characterized those with delayed treatment. Further, superficial as well as deep vein thrombosis is common findings, that only a few surgeons were aware of it.

We found in our previous study (2009–2012) that endothelial lining of moderate sized veins was severely disintegrated. This finding showed disassembled endothelial cell-to-cell junction (both adherens and tight junctions) in burned area and non–burned area, both in critical– and non–critical burns as well. In a study, all enrolled critical burns died.

Damaged endothelium and its’ junction fail to provide primary barrier of endothelial lining thus attributed to endothelial dysfunction were multifactorial etiology. Direct impact of a thermal injury is clearly understood as the causative. Inflammatory mediators and adhered endothelial–leukocytes–platelet in inflammatory response, hypoxia, lipid protein complex (LPC) attributed to pernicious effectors in burn formerly known as burn toxin, product of damaged cells following thermal injury as well as lipopolysaccharide a product of invasive microorganism is responsible to such a damage. Furthers, it is realized that resuscitation fluid restores the barrier of endothelial lining thus attributed to endothelial dysfunction which was Parkland formula the perfusion was not restored. Fluid resuscitation should be carried out by strategy. Massive fluid delivery did not solve the problem. With the administration of a large volume crystalloid as advocated by Baxter which was disseminated as Parkland formula the perfusion was not restored. Although the fluid was titrated in the unit, this large volume resuscitation was followed by third space syndrome which is fatal (massive edema, lung edema, abdominal compartment syndrome). Even though we replaced lactated Ringer’s solution with acetated Ringers’ solution (acetate is metabolized faster than lactate to be used as a source of energy) there was no improvement. In next two periods, we tried to move on colloids which is attributed to low osmotic pressure in intravascular compartment, showed to be improved, but not the perfusion. In addition, morbidity of colloid resuscitation such as deteriorated coagulation system (represented by increased index normalized ratio) as well as acute kidney injury was noted. Nevertheless, mortality increased with this application. In contrast, the longer survival days of those mortal is achieved.

Following a consensus of fluid-electrolytes and acid-base imbalance, it was realized that resuscitation fluid restores the volume but not perfusion. Adding more volume in those unresponsive worsen the perfusion, and provokes the reperfusion injury. Even though ascorbic acid per drip was the added to the protocol, the use of vasoactive was useless in these cases as hypoxic cell would not respond to any vasoactive derived. Thus, we were set the focus on perfusion, disregarding the volume as the target; nor the urine output. There was a doubt at the early date, to deliver red cells in those with hemococoncentration as it not recommended. We found lack of evidence to deliver blood transfusion at the early date as a part of burn resuscitation. The rationale was to improve perfusion, and we did believe that the best oxygen carrier is nothing but blood. Following delivery of 1000 mL lactated Ringer’s solution, we delivered blood component. Initial delivery of 250 mL of fresh frozen plasma and continued with packed red cells of 250 mL. Plasma is essential to maintain osmotic pressure in intravascular compartment, not as the prevention of disordered coagulation system. Red cells required as oxygen carrier and the first buffer protein plays an important role in acid–base homeostasis prior to renal and pulmonary system takes place. This regimen is applied for three consecutive days. The administration of blood component had been considered in advance, and should the immunosuppressive effect be of one consideration, we believed that first things first.

Enfacing problem at the early date was lack of perfusion, namely burn shock. Volume did not solve the problem, but blood. Should we representing the features of sepsis syndrome, and this is found much earlier in a nature of septicemia.
thinking of things that were not certain yet, and if they happen to be late, then we should always find these population were will never survived. In addition, the correlation of blood transfusion and mortality was not supported by any study with level 1–2 of evidence.37

In the application, it was observed in these population that the oxygen utilization was found increased and corrected hypovolemia due to dilutional effect following blood component transfusion which is colloidal in nature, although hemodynamic indices showed no significant changes. We also noted improved base deficit and serum lactate that correlates significantly to mortality36 in our former study.8 The first obstacle we found at the early date come from the blood bank, questioning indications associated with hemoglobin levels. With a good discussion at a time based on the guideline that not to deliver blood based on hemoglobin content,37 then the problem was solved. In the next upcoming period, platelet was added in the regimen to prevent coagulopathy in those with major burns.39

In this regimen, volume restriction was applied to maximum of individual intravascular volume. Should a volume be required, then water is added; the basic principles was sticks to the concept less is more. Perfusion was achieved, and massive fluid administration was avoided.

In further observation, we found mean creatinine decreased significantly. With addition of platelet, index normalized ratio was avoided. More perfusion was achieved, and massive fluid administration was avoided.

There were limitations to a study, indeed. In this retrospective study, there are variables were unavailable, let we couldn’t analyze. It was situation that we are facing of in clinical setting. In contrast, there's variables were unavailable, let we couldn’t analyze. It was a real clinical setting found decreased significantly. There's no evidence.

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