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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 volume oriented. Even though mortality remains the problem, survival days markedly increased.

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. $\pm$ 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.

Table 1. Protocols of fluid resuscitation

Period		Formula in the protocol			
I	1998–2000	Parkland formula	First 24 hr.	First 48 hr.	Next 72 hr.
	1. 1998–1999	RL 4mL/TBSA/KgBW	Half of calculated in first 8 hr. Half of calculated in second 16 hr.	Half of first 24 hr. requirements	Maintenance
	2. 2000	RA 4mL/TBSA/KgBW			
II	2001–2003	Low volume resuscitation (maximum to individual intravascular volume)			
	1. 2001–2002	Initial 1000 mL RA + HES 10% 2500–3000 mL/24 hr. for 48 hrs.			Maintenance
	2. 2003	Initial 1000 mL RA + HES 6% 2000–2500 mL/24 hr. for 48 hrs.			
III	2004–2006	Low volume resuscitation (maximum to individual intravascular volume)			
		Initial 1000 mL RA + Gelatin 4% 2000–2500 mL+ NaCl 3% 500 mL/24 hr. for 48 hr.			Maintenance
IV	2007–2010	Balanced fluid resuscitation (maximum to individual intravascular volume)			
		Initial 1000 mL RA + FFP + PRC + TC with ratio of 1:1:1 /24 hr. for three consecutive days			

TBSA: total burned surface area, KgBW: kilogram body weight, RL: lactated Ringer's solution, RA: acetated Ringers' solution, HES: hydroxyethyl starch, FFP: fresh frozen plasma, PRC: packed red cells, TC: platelet

Table 2. Subjects' characteristics, mortality and survival days

	n		Age (yo)	TBSA (%)	Baux score	Delayed (pbh)	Mortality (%)	Survival days (pbd)	
	Total	Study						Min–Max	Mean
1998	108	53	42.7 ± 14.60	40.14 ± 15.73	82.85 ± 21.70	6.66 ± 3.22	23 (43.4)	1–12	3.04 ± 2.30
1999	87	27	38.6 ± 14.90	35.37 ± 17.09	73.89 ± 21.59	8.90 ± 4.00	18 (66.7)	1–17	4.11 ± 1.87
2000	163	58	37.2 ± 18.79	48.07 ± 20.82	85.28 ± 27.96	8.25 ± 4.26	21 (26.2)	1–8	3.38 ± 1.98
2001	175	60	36.3 ± 14.15	41.72 ± 18.12	78.02 ± 21.99	8.16 ± 4.25	32 (53.3)	1–34	4.40 ± 6.13
2002	177	68	38 ± 19.34	46.16 ± 21.32	84.17 ± 27.58	8.22 ± 4.34	39 (57.4)	1–19	5.56 ± 4.78
2003	172	55	36.6 ± 14.68	38.68 ± 11.68	75.30 ± 19.49	8.09 ± 4.49	29 (52.7)	1–7	3.20 ± 1.65
2004	167	50	38.7 ± 15.68	37.55 ± 12.40	76.25 ± 19.17	7.90 ± 4.16	27 (54)	1–9	3.55 ± 2.04
2005	146	44	36.4 ± 15.77	36.55 ± 9.99	72.98 ± 18.41	8.40 ± 4.31	19 (43.2)	2–7	3.73 ± 1.62
2006	101	42	34.9 ± 15.86	41.78 ± 16.48	76.76 ± 23.62	12.09 ± 5.27	13 (31)	7–28	15.76 ± 6.35
2007	126	56	35 ± 17.24	46.52 ± 17.78	81.56 ± 24.22	12.07 ± 5.15	11 (19.6)	14–23	17.81 ± 2.71
2008	146	57	38.2 ± 14.44	42.30 ± 13.08	80.58 ± 21.84	9.19 ± 4.06	4 (7)	17–27	21.25 ± 4.64
2009	139	59	37.8 ± 13.96	43.28 ± 14.48	81.10 ± 20.87	7.10 ± 2.68	7 (11.9)	13–26	19.71 ± 4.19
2010	61	30	36.2 ± 14.75	45.46 ± 16.32	81.70 ± 19.98	7.80 ± 3.21	5 (16.7)	11–31	20.80 ± 7.49
Total	1768	659	37.5 ± 15.91	42.21 ± 16.60	79.70 ± 22.81	8.63 ± 4.43	248 (37.6)	1–34	2.29 ± 6.53

TBSA: total body surface area, pbh: post–burned hours, pbd: post–burned days. Age, TBSA, Baux score, delay expressed by mean ± SD.

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 leaks. 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 significant to the baseline. We also note that all protocols were followed by improved hemodynamic indices, even though there were noted only 40.5% in period 2004–2006 and 37.7% in period of 2007–2010 achieved a standard mean arterial pressure of >65 mmHg. Positive balance of a great number was noted in all protocols that found in line with a large volume delivery, but not in the last period as the focus moved on perfusion, disregarding the volume (table 3).

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 11–31 pbd (20.80 pbd ± 7.49) in 2010.

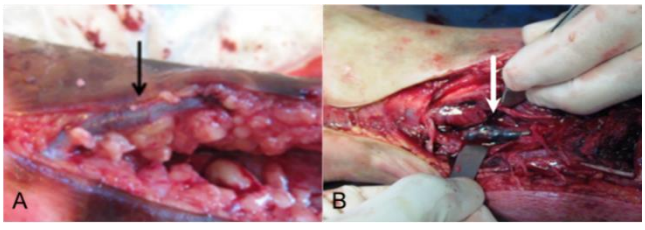


Figure 1. Superficial vein thrombosis (A) and deep vein thrombosis (B) is a common morbidity found and responsible to a pitfall of resuscitation. Large volume fluid replacement leading to massive edema compressing the vessels in the compartment.

Table 2. Hydration status and hemodynamics

Period		Pbh	Dilution						Positive balance			CVP (cmH_2O)			MAP (mmHg)		
			Hemoglobin (g/dL)			Hematocrit (vol%)											
			n	mean ± SD	p	n	mean ± SD	p	n	(mean ± SD)	p	n	mean ± SD	p	n	mean ± SD	p
I	1998–1999	0	71	16.01 ± 2.39	<0.001	76	53.11 ± 8.79	<0.001	72	–	0.001	54	3.29 ± 1.56	<0.001	–	–	–
		24		14.55 ± 2.27			49.53 ± 8.01			3946.73±1636.16			4.23 ± 1.88			–	–
		48		13.87 ± 2.12			46.59 ± 6.90			4345.50 ± 1856.67			6.90 ± 3.51			–	–
	2000	0	25	17.55 ± 1.96	<0.001	35	57.25 ± 6.20	<0.001	52	–	<0.001	33	2.75 ± 1.39	<0.001	–	–	–
		24		16.34 ± 2.41			53.08 ± 6.27			4411.73 ± 2928.43			3.92 ±1.82			–	–
		48		15.54 ± 2.26			51.11 ± 4.34			5116.53 ± 3351.03			6.00 ±3.14			–	
II	2001–2002	0	97	16.84 ± 2.39	<0.001	99	54.37 ± 8.31	<0.0001	114	–	<0.001	83	3.09 ± 1.43	<0.001	–	–	–
		24		14.27 ± 2.38			49.66 ± 7.62			4060.61 ± 2342.38			3.81 ± 1.88			–	–
		48		12.97 ± 2.46			47.40 ± 6.24			4877.12 ± 2814.84			6.54 ± 3.27			–	
	2003	0	51	16.17 ± 2.64	<0.001	53	52.11 ± 9.54	<0.001	49	–	<0.001	20	2.97 ± 1.38	<0.001	–	–	–
		24		12.57 ± 2.17			46.43 ± 8.74			2244.28 ± 1160.18			4.10 ± 2.42			–	–
		48		11.80 ± 2.02			44.39 ± 7.40			3195.06 ± 1366.78			7.22 ± 3.54			–	
III	2004–2005	0	128	16.05 ± 2.54	<0.001	131	53.69 ± 9.64	<0.001	128	2629.42 ± 1569.95	<0.001	89	3.20 ± 1.45	<0.001	42	59.50 ± 10.92	<0.001
		24		14.11 ± 2.35			44.77 ± 9.05			–			4.58 ± 2.05			69.33 ± 9.92	
		48		13.36 ± 2.24			43.47± 10.56			3107.37 ± 2112.13			8.40 ± 3.38			78.26 ± 9.83	
IV	2007–2010	0	69	16.66 ± 2.41	<0.001	56	58.94 ± 9.17	<0.001	202	–	0.086	152	2.24 ± 1.16	<0.001	137	59.40 ± 9.09	<0.001
		24		13.85 ± 2.38			38.75 ± 7.59			633.20 ± 871.34			4.76 ± 1.47			68.45 ± 8.30	
		48		12.78 ± 2.43			37.55 ± 7.32			606.29 ± 905.22			7.17 ± 2.26			76.86 ± 8.85	

Pbh: postburn hours, CVP: central venous pressure, MAP: mean arterial pressure

Table 3. Cellular perfusion in different protocols.

	Period	Pbh	$SvcO_2$ (%)			RBG (mg/dL)			INR			Creatinine		
			n	(mean ± SD)	p	n	(mean ± SD)	p	n	(mean ± SD)	p	n	(mean ± SD)	p
I	1998–1999	0	–	–	–	43	207.81 ± 86.72	<0.001	–	–	–	29	1.77 ± 0.97	0.001
		24		–			173.62 ± 78.01			–			2.92 ± 2.00	
		48		–			145.30 ± 50.15			–			–	
	2000	0	–	–	–	21	232.71 ± 87.11	<0.001	–	–	–	36	2.26 ± 0.74	0.056
		24		–			169.41 ± 64.62			–			2.56 ± 1.24	
		48		–			127.28 ±32.34			–			–	
II	2001–2002	0	–	–	–	45	192.48 ± 85.03	<0.001	44	1.63 ± 0.41	<0.001	85	2.50 ± 0.70	0.445
		24		–			149.97 ± 54.22			–			2.57 ±1.16	
		48		–			138.44 ± 41.92			1.79 ± 0.43			–	
	2003	0	–	–	–	21	154.85 ± 70.30	0.144	20	1.92 ± 0.36	<0.001	34	2.62 ± 0.71	0.188
		24		–			129.66 ± 36.91			–			2.47 ± 1.01	
		48		–			138.47 ± 38.40			2.18 ± 0.33			–	
III	2004–2006	0	35	87.80 ± 4.73	<0.001	68	215.51 ± 109.84	<0.001	71	1.50 ± 0.38	<0.001	68	2.59 ± 0.72	0.050
		24		85.14 ± 3.84			173.92 ± 84.70			–			2.43 ± 0.98	
		48		84.54 ± 5.41			145.70 ± 54.22			1.69 ± 0.36			–	
IV	2007–2010	0	153	85.63 ± 3.85	<0.001	63	278.30 ± 114.99	<0.001	167	1.60 ± 0.34	<0.001	21	2.31 ± 1.40	0.001
		24		83.52 ± 3.22			210.00 ± 88.96			–			1.60 ± 0.72	
		48		80.18 ± 4.58			154.55 ± 68.17			1.27 ± 0.28			–	

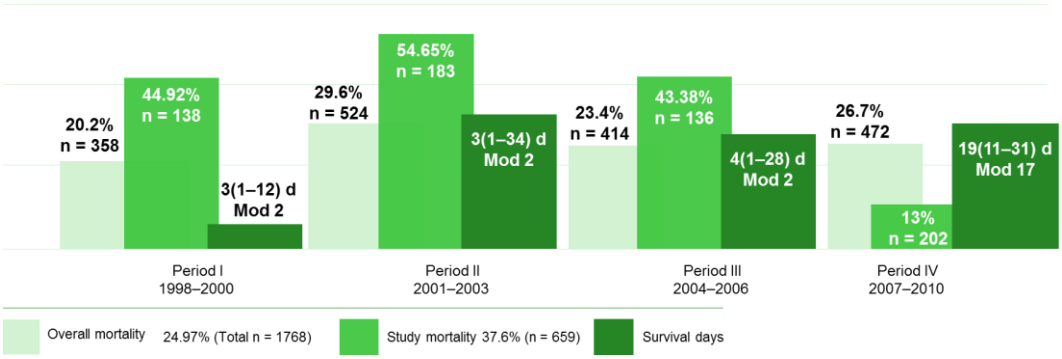
Pbh: postburn hours, $SvcO_2$: central vein oxygen saturation, RBG: random blood glucose, INR: index normalized ratio.

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	76	80.15 ± 23.55	0.299	71	4.92 ± 2.48	0.740	73	-4.71 ± 3.00	0.163
	Yes	62	84.56 ± 26.03		60	5.08 ± 2.88		59	-5.44 ± 2.94	
2001–2003	No	83	68.13 ± 15.39	<0.001	62	3.99 ± 2.29	<0.001	59	-5.46 ± 2.42	<0.001
	Yes	100	25.34 ± 2.53		96	6.00 ± 3.09		95	-9.18 ± 4.63	
2004–2006	No	77	68.70 ± 14.75	<0.001	32	3.31 ± 0.96	<0.001	54	-6.44 ± 3.13	<0.001
	Yes	59	84.02 ± 23.28		44	5.92 ± 2.17		32	-10.71 ± 5.04	
2007–2010	No	175	77.36 ± 19.33	<0.001	130	3.66 ± 1.37	<0.001	147	-6.44 ± 9.40	<0.001
	Yes	27	105.88 ± 21.83		27	7.70 ± 2.42		26	-17.86 ± 5.39	

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

Period	Base deficit			Serum lactate		
	n	Mean ± SD	p value	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		76	4.83 ± 2.17	
2007–2010	173	-8.16 ± 9.82		157	4.36 ± 2.20	



Graphic 1. Mortality and survival days in each period.

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[1,2] 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.[3] 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.[4] Extensive capillary leak is essential as body response to thermal injury. Clinically massive edema,[5,6] unresponsiveness to fluid (sometimes attributed to under resuscitation), low level of serum albumin,[7] deteriorated hemodynamic (low central venous pressure and mean arterial pressure) is obviously found that characterized those with delayed treatment.[8] Furthers, superficial as well as deep vein thrombosis is common findings,[9] 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.[10] 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.[11]

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[12] and adhered endothelial–leukocytes–platelet in inflammatory response, hypoxia,[13] lipid protein complex (LPC) attributed to pernicious effectors in burn formerly known as burn toxin,[14] 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 such a damage to be irreversible should the resuscitation is not provided in an hour15 to two following thermal injury.[16] Adhesion of the three cells (endothelial–leukocytes–platelet) lead to formation of widespread rouleaux formation let red cells oxygen carrier capacity inefficient,[17] intravascular thrombosis plugging microvascular system and let the coagulation system changed manifested as diathesis hemorrhagic or disseminated intravascular coagulation (DIC).[18] Such changes were followed by dysfunction of smooth muscles system in the artery,[19] in all vascular bed, which in accordance with Demling findings, damaged collagen of supporting endothelium following thermal injury leading to delayed edema resorption.[20,21] In this catastrophe, a fallen of systemic vascular resistance let the circulation down.[17] Thus, it much more a distributive– rather that hypovolemic shock. Such conditions representing the features of sepsis syndrome, and this is found much earlier in a nature of septicemia.[11]

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[22,23] 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)[24] there was no improvement. In next two periods, we tried to move on colloids which is attributed to low volume resuscitation.[25] With this colloid, hemodynamic indices 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.[26–28] 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,[29] it was realized that resuscitation fluid restores the volume but not perfusion. Adding more volume in those unresponsive worsen the perfusion,[30] and provokes the reperfusion injury[31] Even though ascorbic acid per drip was the added to the protocol.[13,32,33] 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 hemoconcentration as it not recommended.[34,35] 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.[36]

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.[29] 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

thinking of things that were not certain yet, and if they happen to be later, 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 colloid in nature, although hemodynamic indices showed no significant changes. We also noted improved base deficit and serum lactate that correlates significantly to mortality[38] 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 ration was found decreased significantly. There's no transfusion related acute lung injury noted.[40] We did believe that MOF was found in relation to burn damage rather than blood transfusion. Finally, we could see as this regimen applied, those with critical burns were survived longer; even though mortality remains high.

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 the strength of a study that was a portrait of a real clinical setting found in those delayed presented, which is characterized our population. Future studies are advocated to find out doses, protocol and number of volumes to be delivered.

Conclusion

Those with delayed presented burns should be managed based on problems encountered. The endpoint of resuscitation, namely restored perfusion is not achieved by volume replacement. Study showed that balanced fluid resuscitation is superior than volume replacement. Even though mortality remains the problem, survival days markedly increased.

Conflict of interest

Author disclose there was no conflict of interest.

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