RIFLE CRITERIA FOR ACUTE KIDNEY INJURY IN BURN PATIENTS: PREVALENCE AND RISK FACTORS

CRITÈRES DE RIFLE ET DÉFAILLANCE RÉNALE AIGUË CHEZ LES BRÛLÉS : PRÉVALENCE ET FACTEURS DE RISQUE

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SUMMARY. Acute kidney injury is one of the severe complications after burns. The purpose of this study was to identify prevalence, risk factors to the development of acute kidney injury (AKI) in burn patients and mortality, using RIFLE classification: risk (R), injury (I), failure (F), loss (L), and end-stage kidney disease (E). This 3-year retrospective study was conducted in burn patients admitted to the Dr. Soetomo Hospital Burn Center between January 2018 and September 2020. Burn patients aged >18 years old and diagnosed with acute kidney injury during hospitalization were enrolled in this study. Factors influencing AKI and its mortality were assessed using bivariate and multivariate logistic regression analysis. Eighty-nine burn patients were available for analysis, and 18 (20%) of them developed AKI according to the RIFLE classification: risk in 6 (33%), injury in 7 (39%) and failure in 5 (28%). Patients with AKI had a significantly higher age and % of TBSA than those without AKI (p-value <0.05). Age more than 60 years old was significantly associated as a risk factor to develop AKI (OR=25.553, p value=0.014). The mortality rate of patients with AKI was 83% (15 deaths from 18 patients), with the overall mortality of patients 16.8%. Chi-square analysis indicated inhalation injury, % of TBSA, and age as risk factors for mortality (p-value < 0.05). The conclusion of our study was that the incidence of AKI in burn patients was relatively high. Older age as a risk factor to develop AKI and inhalation injury, TBSA, and age were associated with mortality.

Keywords: AKI, burn patients, RIFLE, prevalence, mortality

RÉSUMÉ. *La défaillance rénale aiguë (DRA) est une des complications graves des brûlures. Cette étude a pour d'évaluer sa prévalence, les facteurs de risque de sa survenue et sa mortalité, en utilisant la classification RIFLE : R (Risk- Risque), I (Injury- Lésion), F (Failure- Défaillance), L (Loss- Perte de fonction), E (End stage- Terminale). Cette étude rétrospective a concerné les patients hospitalisés dans le CTB de l'hôpital Dr Soetomo durant 3 ans (janvier 2018- décembre 2020). Elle a concerné tous les patients de plus de 18 ans ayant subi une DRA. Les facteurs de risque ont été évalués par analyses uni- et multivariées. Quatre- vingt- dix- neuf patients étaient éligibles, dont 18 ont développé une DRA (6- 33%- R ; 7- 39%- I et 5- 28%- F). Les patients avec DRA étaient plus âgés et brûlés plus extensivement, en particulier, l'âge de plus de 60 ans avait un OR de 25,553 ; p= 0,014). La mortalité des patients avec DRA était de 83% (15 morts sur 18) alors que la mortalité globale était de 16,8%. L'analyse par C² montrait que l'âge, la surface brûlée et l'inhalation de fumée étaient des facteurs de mortalité (p<0,05). L'incidence de DRA est relativement élevée et corrèle avec l'âge quand la mortalité globale reste liée à l'âge, la surface brûlée et l'inhalation de fumées.*

Mots-clés : *défaillance rénale aiguë, brûlure, classification RIFLE, prévalence, mortalité*

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Introduction

Burns cause not only local effects but also systemic effects. The local impact of burns are redness of skin, swelling and pain.[1] The systemic impact of burns decreases intravascular volume, increases vascular resistance, decreases cardiac output, and leads to ischemia and metabolic acidosis. These systemic effects usually occur when the total body surface area burned is more than 20%, and they cause severe damage to cardiovascular function, which is called hypovolemic shock. Hypovolemic shock occurs due to reduced intravascular plasma volume, which causes fluid displacement to non-functional body spaces.[2,3] The most apparent clinical condition due to hypovolemic shock is damage to the kidney filtration system known as acute kidney injury (AKI).[4]

AKI is a condition where there is a sudden decrease in kidney function.[5] Hypovolemic shock reduces renal perfusion and is followed by reduced glomerular filtration rate (GFR). This process reduces creatinine clearance and oliguria or decreases urine output (urine production <200ml/12 hours). Several studies stated that the incidence of AKI in burn patients still varies depending on the definition used. Clinicians can use several guidelines to define AKI, including KDIGO, AKIN and RIFLE. To establish the definition of AKI, the Acute Dialysis Quality Initiative Group initiated the RIFLE criteria.[6] RIFLE criteria indicate three stages of renal injury based on serum creatinine levels and/or urine output which reflects a reduction in GFR followed by duration and severity of decreased urine output. The RIFLE criteria consist of risk (R), injury (I) and failure (F), along with two outcomes, loss (L) and end-stage kidney disease (E). RIFLE criteria have been used in several studies in burn settings to define AKI and to correlate AKI with hospital outcomes.[7,8]

AKI is a common complication in burn patients, with an incidence and mortality of 30% and 80%, respectively. However, the incidence rate of AKI depends on the burn population studied and the definition of AKI used. Burns related to kidney injury are usually classified as early AKI (0-3 days after injury) or late AKI (≥4 days after injury). Early burn AKI is generally caused by hypovolemia, low renal perfusion, direct cardiac suppression resulting from the release of TNF-alpha, and denatured protein deposition. In contrast, late AKI is often caused by sepsis, multi-organ failure (MOF), and the use of nephrotoxic drugs.[9]

According to a meta-analysis study by Folkestad et al., the risk factors for AKI in burn patients are old age, chronic hypertension, diabetes mellitus, a high percentage of TBSA, a high score of ABSI (Abbreviated Burn Severity Index), a high score of APACHE (Acute Physiology And Chronic Health Evaluation) II scores, and SOFA (Sequential Organ Failure Assessment) score. The presence of inhalation trauma, rhabdomyolysis, surgery, sepsis, and use of mechanical ventilators were also reported as risk factors to developing AKI in burn patients.[10] So far, there is no study in Indonesia regarding the prevalence and risk factors for AKI in burn patients. Therefore, based on this background, the purpose of our study is to identify the prevalence and risk factors affecting the incidence and mortality of AKI among burn patients using RIFLE criteria in Indonesian populations. A good understanding of the incidence and risk factors for AKI in burn patients can help clinicians and pharmacists provide appropriate management therapy to prevent morbidity and mortality.

Materials and methods

Patient selection

This study was an analytic retrospective observational study using medical records of burn patients who were admitted to the Dr. Soetomo Hospital Burn Center, Surabaya-Indonesia between January 2018 and September 2020. This study took place from September to December 2020. Inclusion criteria were burn patients aged >17 years old and diagnosed with acute kidney injury on admission or during hospitalization and completed by the examination of creatinine serum level, blood urea nitrogen, or urine output. Exclusion criteria were patients who had a history of kidney disease and heart failure and if they arrived in our burn center more than 72 hours after burns. In this study, we used the RIFLE criteria to determine the definitions for AKI in burn patients during their hospital stay, as shown in *Table I*. These

Table I - RIFLE criteria for AKI classification

Staging	Serum creatinine	Urine output
Risk (R)	Elevated sCr 1.5-1.9 times from baseline or GFR decrease > 25%	< 0.5ml/kg/h x 6-12 h
Injury (I)	Elevated sCr 2.0-2.9 times from baseline or GFR decrease > 50%	< 0.5ml/kg/h x 12 h
Failure (F)	Elevated sCr 3.0 times from baseline or GFR decrase > 75%, or sCr > 4 mg/dl with minimal acute increase of > 0.5mg/dl	< 0.5ml/kg/h x 24 h or anuria for more than 24 h
Loss (L)	Loss of kidney function more than 4 weeks	
End Stage (E)	Loss of kidney function more than 3 months	

criteria state that an increase in serum creatinine levels up to 50% or more above the baseline is determined as AKI. Serum creatinine level on admission was used as a baseline. In this study, we also divided burn patients with AKI into early- (≤3 days) and late-onset AKI (≥4 days). Baseline serum creatinine level was defined first on admission or the earliest available serum creatinine level in the first 5 days after burns. The lowest creatinine level at the time of admission was used as the baseline for creatinine.

Data analysis

Demographic data including age, sex, percentage of total body surface area, cause of burns, length of stay, and mortality were recorded and presented as percentages in each group (*Table II*).

Table II - Demographic data of burn patients

Variables	With AKI (n=18)	Without AKI (n=71)	p-value
Sex			
Male	11 (61%)	51 (72%)	0.469[a]
Female	7 (39%)	20 (28%)	
Age (years)			
Range	20-81	19-73	0.015[a]
Mean ± SD	52.9 ± 16.6	42.4 ± 13.6	
TBSA (%)			
Range	7- 94	1-73.5	0.001[a]
Mean ± SD	46.4 ± 24.7	28.5 ± 21.4	
Grade of burns			
Grade IIAB	11 (61%)	40 (56%)	0.598[b]
Grade III	7 (39%)	31 (44%)	
Mortality			
Survived	3 (17%)	57 (80%)	0.001[b]
Died	15 (83%)	14 (20%)	
Length of stay (days)			
Range	5-37	3-55	0.551[a]
Mean ± SD	14.9 ± 9.0	16.5 ± 10.2	
Cause of burns			
EIHV	2 (11%)	17 (24%)	
Scald	4 (22%)	4 (6%)	
Fire	12 (67%)	50 (70%)	

[a]Independent t-test [b]Chi-square

We also observed laboratory data including creatinine serum, blood urea nitrogen (BUN) on admission and during hospitalization. Quantity data were represented as mean ± SD. Statistical analysis to analyze differences between groups (AKI vs. non AKI group) was done using the independent t-test.

To correlate % of TBSA and albumin level, we used the Pearson correlation test. Furthermore, bivariate and multivariate logistic regression were used to predict risk factors for AKI and mortality. We used SPSS 20.0 to conduct all statistical analysis. P-value < 0.05 was considered statistically significant.

Results

In this study, we found several complications among burn patients with AKI, as shown in *Table III*. Among 18 AKI burn patients, 6 (33%) patients were early AKI and 12 (66%) patients were late onset AKI.

Table III - Complications among burn patients with AKI

Complications	n (%)
Sepsis	9 (50)
Inhalation Injury	11 (61)
Hypoalbuminemia	16 (89)
Anemia	7 (39)
Thrombocytopenia	2 (11)
Metabolic acidosis	3 (17)
Hyperkalemia	2 (11)
Pneumonia	3 (17)

The correlation between percentage of TBSA and albumin level among burn patients with AKI is shown in *Fig. 1*. The mean albumin level of burn patients with AKI was 2.58 ± 0.33 g/dl.

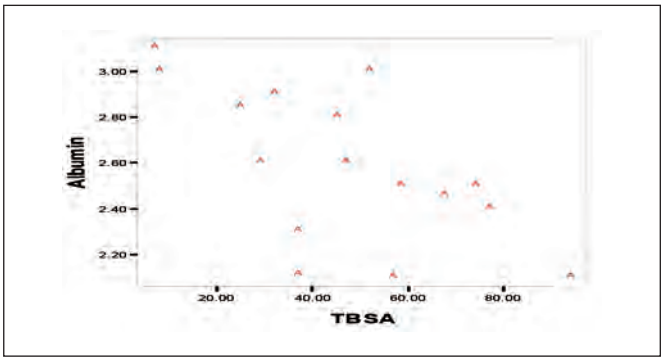

Fig. 1 - Pearson correlation between percentage of TBSA and albumin level

By Pearson correlation, it was found that r = -0.632, p value = 0.008. It showed that the higher the total body surface area, the lower the albumin level. We also divided the serum creatinine and BUN level among burn patients with AKI between survivors and non-survivors on admission, as shown in *Table IV*.

Table IV - Creatinine and BUN level between AKI patients

Variables (mg/dl)	Survived (n=3)	Died (n=15)	P value
BUN	30.33 ± 8.02	34.46 ± 16.70	0.686
Creatinine	2.21 ± 0.85	2.07 ± 0.78	0.783

The association between inhalation injury, age, sex, percentage of TBSA, grade of burns and cause of burns and the development AKI in burn patients by bivariate analysis (Chi-square) is shown in *Table V*.

Table V - Bivariate analysis factors related to developing AKI

Variables	OR	P value	CI 95%
Inhalation trauma	2.954	0.041	1.019-8.567
TBSA (>20%)	4.000	0.031	1.064-15.032
Age (>60 years old)	5.091	0.005	1.534-16.890
Grade of burns (grade III)	0.842	0.749	0.293-2.420
Cause of burns (fire)	0.880	0.820	0.293-2.646
Sex (male)	0.604	0.358	0.205-1.778

Bivariate analysis showed only inhalation trauma, percentage of TBSA, and age with p value less than 0.25. To associate the risk factors for developing AKI in burn patients, we used multivariate logistic regression, as shown in *Table VI*.

Table VI - Risk factors for developing AKI by multivariate logistic regression

Variables	OR	P value	CI 95%
Inhalation trauma	1.929	0.269	0.601-6.187
TBSA (>20%)	0.694	0.742	0.079-6.115
Age (>60 years old)	25.553	0.014	1.905-342.840

We used ROC analysis to predict whether a combination of inhalation trauma, TBSA percentage, and age led to AKI in burn patients. Area under curve (AUC) was 74.5% with p value = 0.000, as shown in *Fig. 2*.

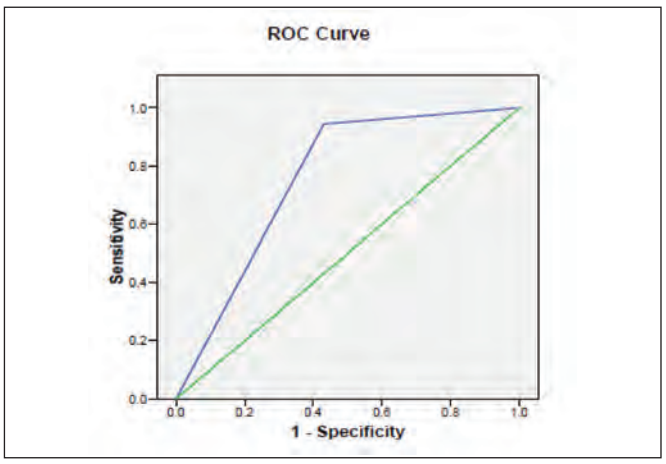

Fig. 2 - ROC curve

We couldn't use multivariate logistic regression to determine the risk factors for mortality in burn patients with AKI due to some variables being constant and to the small number of patients. Therefore, we used bivariate analysis to analyze risk factors for mortality, as shown in *Table VII*.

Table VII - Bivariate analysis associated with mortality

Variables	OR	P value	CI 95%
Inhalation trauma	1.60	0.034	0.935-2.737
Sepsis	1.50	0.058	0.945-2.381
Hypoalbuminemia	5.50	0.180	0.385-78.573
TBSA	28.0	0.011	1.208-648.809
Age	0.57	0.017	0.301-1.08

Discussion

Out of the 89 burn patients, 18 were diagnosed with AKI. In this study, we found that age and TBSA in burn patients with AKI was significantly greater than in burn patients without AKI. Our study was similar to a study by Emami et al., who also found that the percentage of TBSA in burn patients in the AKI group was more significant than in the non-AKI group.[11] In burns with high TBSA, skin layer and blood vessel damage will be even more significant, increasing vascular permeability and shifting intravascular fluid to extravascular, resulting in hypovolemia. Hypovolemia induces oliguria earlier after burns. Sodium retention in collagen fibers and damage to the sodium-potassium pump also participate in causing generalized edema. If fluid resuscitation is not carried out adequately, renal hypoperfusion will occur, leading to azotemia or prerenal AKI. On the other hand, the excessive fluid resuscitation during the oliguria phase decreases the plasma tonicity, which also causes renal edema and is followed by acute kidney injury.[3]

In our study, we used serum creatinine level and BUN rather than output urine production. Serum creatinine is directly proportional to body muscle mass and inversely related to glomerular filtration. Since body muscle mass is usually constant, serum creatinine levels are generally a reliable indicator of GFR. In each multiple, the serum creatinine level represents a 50% decrease in GFR. Although decreased urine volume is often used as a physiological marker in burn patients and widely used in clinical practice, it is not sensitive. The measurement of urine output production based on the difference between glomerular filtration rate and tubular reabsorption makes it possible for patients with a renal injury to still have normal urine production, particularly in the early stages

of renal injury.[12] A study by Emami et al., who analyzed biomarkers that can be used to predict AKI in burn patients, stated that serum creatinine and BUN levels had good predictive values marked by AUC of 0.738 and 0.707, respectively. Furthermore, among serum creatinine, BUN and albumin, only BUN marker was significantly associated with AKI occurrence.[11] Measurement of urine output to define AKI has limitations. Urine output can significantly change by the administration of diuretics to enhance urine flow and this is not considered in the RIFLE classification. In addition, urine output can only be measured in patients using bladder catheter, common in ICU patients but not frequent in non ICU patients.[13]

RIFLE criteria were first used to define and classify AKI in burn patients by Coca et al., who reported that AKI incidence was 27% with a mortality rate of 60%.[14] In our study, the incidence rate of acute kidney injury in burn patients was 20.2% with RIFLE criteria at first diagnosed as AKI: risk in 6 (33%), injury in 7 (39%), and failure in 5 (28%). Our finding was consistent with a study by Chung et al., who reported that the prevalence of AKI in burn patients using the RIFLE criteria was 24%.[15] Sanchez et al. also reported that out of 165 critical burned patients, 32 (19.3%) developed AKI using RIFLE criteria, risk in 6 patients (6.1%), injury in 11 (6.7%) and failure in 11 (6.7%). A lower stratification of RIFLE by Sanchez et al. compared to our study, because total patients for each classification was divided by total number of burned patients (n=165).[16] A different result was reported by Palmieri et al. in severe burn patients: the incidence rate of AKI was 53.3% with the RIFLE category: risk (28.1%), injury (18.8%), and failure (53.3%). This study's results differed from those reported by Palmieri and colleagues, because they used the maximum RIFLE value when patients were diagnosed with AKI. Conversely, in our study we could not observe the maximum RIFLE criteria because some patients died during seven days of hospitalization and we couldn't find serum creatinine data.[17] A recent meta-analysis based on studies utilizing the RIFLE stratification (risk, injury, failure, loss of function, end-stage renal disease)[18] or the definition by Acute Kidney Injury Network (AKIN),[19] or the definition of Kidney Disease: Improving Global Outcomes (KDIGO),[20] reported that the prevalence of acute kidney injury among burn patients admitted to intensive care ranges from 30%-46%.[10] The risk of death was 11 times higher in burn patients with AKI than in burn patients without AKI. Studies comparing diagnostic criteria for AKI in critically ill patients using three criteria, KDIGO, AKIN and RIFLE were conducted by Luo and colleagues. AKI incidence rates using the KDIGO, AKIN and RIFLE criteria were 51%, 38.4% and 46.9%, respectively. Moreover, it was found that there was no difference in cases of mortality between KDIGO and RIFLE, but there were significant differences between KDIGO and AKIN.[21]

In our study, fifty percent of burn patients with AKI also had sepsis. The hemodynamic effect on sepsis may decrease the glomerular filtration rate as arterial vasodilation due to up-regulation of its cytokines triggers nitric oxide (NO) synthesis in blood vessels, and finally leads to septic shock. Furthermore, efferent arteriolar vasodilation occurs in early sepsis or persistent renal vasoconstriction due to activation of the sympathetic nervous system, the renin-angiotensin-aldosterone system, the release of vasopressin and endothelin also causes a decrease in the glomerular filtration rate. Sepsis can trigger endothelial damage, and produces microvascular thrombosis, reactive activation of oxygen species as well as adhesion and migration of leukocytes that can damage renal tubular cells that trigger AKI.[22,23] Sepsis has been reported as a risk factor for the development of AKI in critically ill patients, and 35-50% of kidney injury in the ICU was associated with sepsis.[24-28] Burn patients with AKI had a high incidence of sepsis and sepsis was significantly associated with mortality ($p<0.05$).[17]

Hypoalbuminemia is often found in critical patients, especially in burns. Burns produce hypermetabolic and hypercatabolic responses depending on the extent and depth of the burn.[29] Burns of more than 20% TBSA cause excessive fluid loss, thereby inducing shock and increasing vascular permeability, and decreasing plasma albumin levels from wound exudation. Hypoalbuminemia also increases complications of extravascular fluid shifting, such as edema, abnormal wound healing and sepsis susceptibility.[30,31] We found a significant negative correlation between percentage of TBSA and albumin level. Our study

was similar to the study by Ramos et al., who reported that severe hypoalbuminemia (< 2 g/dL) in 20% of burn patients was significantly correlated with increased TBSA (p=0.001).[32] A study by Aguayo et al. stated that in burn patients who died, albumin levels were significantly lower (1.5g/dl) than in burn patients who survived (2.8g/dl). Lower albumin levels were significantly associated with higher TBSA in burns patients who died (49.7%) than in survivors (14.7%).[33]

In our study, 33% burn patients with AKI developed early AKI. A study by Ho et al. stated that 64.2% of burn patients with TBSA more than 20% developed early AKI within seven days after burns. Older age and resuscitation with high doses of vitamin C or ascorbic acid were significantly associated with the onset of early AKI.[34] High doses of vitamin C are often used in burns for fluid resuscitation because it has a high antioxidant effect and is strongly suspected to prevent capillary leakage.[35] One of the mechanisms that can explain the development of early AKI due to the use of high doses of vitamin C is the formation of oxalate crystals, which are a metabolite of ascorbic acid that settles in the renal tubules. Another study by Buehener et al. reported that AKI from oxalate nephropathy occurred in burn patients who administered high doses of vitamin C for burn resuscitation.[36] Fortunately, in our study, none of the burn patients were given intravenous vitamin C for burn resuscitation.

We found in our study that only older age more than 60 years old was significantly a risk factor associated with AKI in burn patients by multivariate logistic regression. Burn patients over 60 years old were 25 times more likely to develop AKI than those less than 60 years old. A study by Rakkolainen et al. also reported that age (per 10y increase) was one of the risk factors for AKI (OR 1.80, CI95% 1.37-2.37).[37] Another meta-analysis study indicates that old age was one of the independent variables that was significantly associated with AKI in burn patients.[10] In this study, we found that 89% of burn patients with AKI had a TBSA greater than 20%, with a significant mortality rate (83.3%) compared to burn patients without AKI (20%), p-value < 0.05. This study indicates that the presence of AKI harms the prognosis of hospitalized burn patients. A study by Emami et al. stated that the mortality rate of burn patients with AKI was 76.9%.[11] Significant mortality in burn patients with AKI compared to non AKI was also observed by Sanchez et al.[16] A retrospective study stated that in severe burn patients with TBSA >30%, the mortality rate was 88%.[38] Another study by Chung et al. stated that percentage of TBSA was significantly associated with mortality (OR 1.05, p value=0.0067, CI95% 1.03-1.07) in burn patients with AKI compared to those without AKI. In three classifications of RIFLE criteria, RIFLE-injury (OR 2.98, p value=0.028, CI95% 1.12-7.90) and RIFLE-failure (OR 6.73, p value=<0.001, CI 95% 2.79-16.25) were independently associated with mortality, while RIFLE-risk was not. The area under ROC curve for mortality during hospitalization according to the RIFLE criteria was 0.838 compared to the AKIN criteria, which was 0.877 (p=0.0007).[39]

There were some limitations of our study. First, because our study was a single-center retrospective, some aspects were not fully written in the medical records of burn patients because of the incompleteness of data collected. Second, the definition of AKI was determined mainly by the creatinine level, but in most cases we did not find creatinine level before the injury. We used the creatinine level on admission as the baseline. Third, the number of burn patients with AKI who died was too small and some parameters were constant and we could not analyze the factors associated with death by multivariate analysis.

Conclusion

The incidence of AKI in our study was relatively high. Our study demonstrated that older age >60 years old was a risk factor that was significantly associated with the occurrence of AKI. Mortality rate in burn patients with AKI was significantly higher compared to non AKI. Inhalation injury, % of TBSA (>20% TBSA) and age (>60 years) were associated with mortality in burn patients with AKI. Clinicians and pharmacists should elaborate on this to anticipate the occurrence of AKI among burn patients, especially those in older age. Further studies are needed with a larger sample size to evaluate other parameters influencing the occurrence of AKI as well as mortality.

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