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Comparison of the effects of RL, RL combined with HES 6%, and RL combined with gelofusine in advanced resuscitation of patients with hemorrhagic shock

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Abstract

***Objective*: To compare the resuscitative effects of Ringer's lactate (RL), RL combined with hydroxyethyl starch (HES) 130/0.4 6% (RLH), and RL combined with gelofusine (RLG) on blood lactate and neutrophil gelatinase-associated lipocalin (NGAL) levels in patients with hemorrhagic shock.**

***Design*: A quantitative, analytical, prospective cohort study.**

***Setting*: A tertiary care hospital operating room setting where patients experienced intraoperative hemorrhagic shock.**

***Patients and participants*: Patients who met clinical criteria for hemorrhagic shock and received fluid resuscitation with RL, RLH, or RLG. Consecutive sampling was applied, with baseline and 12-hour post-resuscitation measurements collected.**

***Interventions*: Fluid resuscitation using one of three regimens: RL alone, RL+HES 130/0.4 6% with a 2:1 ratio, or RL+gelofusine with a 2:1 ratio.**

***Measurements and results*: Blood lactate and NGAL were measured before (T0) and 12 hours after resuscitation (T1). All three regimens produced significant reductions in lactate and NGAL levels ($p<0.001$). The most significant decrease in lactate was observed in the RL group (-3.30±0.88 mmol/l), followed by RLG (-3.10±0.57 mmol/l) and RLH (-2.61±0.91 mmol/l), with no statistically significant difference among groups ($p=0.067$). NGAL also declined significantly across all groups, with RL showing the largest decrease (-6288.63±14218.55 ng/ml), followed by RLG (-2030.73±2383.25 ng/ml) and RLH (-978.84±765.31 ng/ml). However, intergroup differences were not significant ($p=0.655$).**

***Conclusions*: RL, RLH, and RLG were all effective in improving microcirculatory markers in hemorrhagic shock, as reflected by significant decreases in lactate and NGAL levels. These findings indicated that all three strategies offered comparable biochemical benefits during early resuscitation from hemorrhagic shock.**

Keywords: Fluid resuscitation, gelofusine, hydroxyethyl starch, hemorrhagic shock, Ringer's lactate.

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Introduction

Hemorrhagic shock is a subtype of hypovolemic shock in which intravascular blood loss and cellular alterations induced by hypoxia lead to progressive tissue and organ dysfunction, ultimately resulting in death once a critical threshold is exceeded. (1,2) Approximately 60,000 individuals in the United States and 1.9 million people worldwide die each year due to hemorrhage and its consequences. (1,2) However, epidemiological data on the prevalence of hemorrhagic shock in Indonesia are currently unavailable. (3,4)

The management of hemorrhagic shock is highly challenging and complex. (2) Treatment is fundamentally centered on timely, rapid, and definitive control of the source of bleeding, accompanied by adequate replacement of blood loss. Fluid and blood replacement aims to prevent ischemia–reperfusion injury, optimize tissue oxygenation, and improve microcirculatory dynamics. (5) Despite ongoing advances, fluid resuscitation strategies in hemorrhagic shock remain the subject of considerable debate, as no clear consensus has yet been reached regarding the optimal type, volume, or therapeutic targets needed to improve patient outcomes. (1,2)

Isotonic crystalloid resuscitation has been used for decades, dating back to early historical management of hemorrhagic shock. However, isotonic crystalloids provide no intrinsic benefits beyond a temporary increase in intravascular volume. (2) Currently, crystalloids remain the most frequently administered intravenous fluids in Intensive Care Units, with Ringer's lactate (RL) and normal saline (NaCl 0.9%) being the most commonly used. Nevertheless, both solutions present notable clinical limitations. (6)

Colloid solutions offer advantages over crystalloids in terms of hemodynamic stabilization, as the enhanced oncotic and osmotic properties of colloids provide substantial benefits at the level of the microcirculation. Their ability to expand plasma volume with smaller infused volumes helps reduce the risk of ongoing or recurrent bleeding that may result from elevated pressure or volume within the macrocirculation. (5) Commonly administered colloids include human plasma derivatives (albumin) and semisynthetic colloids (starches, gelatins, and dextrans). (7)

A clinical investigation demonstrated that RL combined with hydroxyethyl starch (HES) 130/0.4 6% reduced syndecan-1 levels more effectively than RL alone, suggesting improved endothelial protection. (8) Evidence from network meta-analyses also indicates that colloids—including HES and gelofusine—may attenuate lactate accumulation more effectively than crystalloids following hemorrhagic shock. (9) Although studies on the combination of RL with gelofusine remain limited, its pharmacological characteristics, such as faster elimination and a lower risk of accumulation, imply potential benefits for tissue perfusion. (10,11)

Given the pivotal role of lactate and neutrophil gelatinase-associated lipocalin (NGAL) in evaluating microcirculatory recovery and early kidney injury, direct comparison of RL, RL combined with HES 130/0.4 6% (RLH), and RL combined with gelofusine (RLG) is clinically relevant. However, human studies assessing these combinations and their effects on microcirculatory biomarkers remain scarce. Therefore, this study aimed to compare the resuscitative effects of RL, RLH, and RLG on blood lactate and NGAL levels in patients with hemorrhagic shock, to identify the most effective strategy to improve microcirculation and optimize early resuscitation outcomes.

Materials and methods

Study design

This study employed a single-blind experimental design comparing the resuscitative effects of three fluid strategies—RL, RLH (2:1), and RLG (2:1)—on blood lactate and NGAL levels in patients with hemorrhagic shock.

Sample size calculation

The minimum sample size was calculated for numerical analytic comparison across three independent groups. Using α=5%, β=10%, standard deviation (SD)=8.5, and a clinically significant NGAL difference of 10 ng/ml, each group required 15 participants. Thus, a total of 45 subjects were targeted.

Setting and study period

The study was conducted in the Intensive Care Unit (ICU) of Wahidin Sudirohusodo Hospital, Makassar, and in the laboratory of Hasanuddin University Hospital. Data collection took place from April 2025 until the required sample size was achieved. The inclusion criteria were 1) Major hemorrhage causing hemorrhagic shock, 2) Lactate >2 mmol/l, 3) Partial pressure of carbon dioxide (pCO2) gap >6 mmHg, 4) Age 18–65 years, and 5) Provided informed consent. The exclusion criteria were 1) Contraindications to study fluids, 2) History of renal failure, heart failure, congenital heart disease, or hematologic disorders. The dropout criteria were death before completion of the final blood sampling procedure.

Ethical approval

Ethical clearance was obtained from the Biomedical Research Ethics Committee of the Faculty of Medicine, Hasanuddin University. All eligible patients received verbal explanations and signed informed consent forms before enrollment. Participants retained the right to withdraw at any time.

Research procedures

Participants were then allocated to one of three

groups based on the fluid regimen administered: RL alone, RLH, or RLG. Fluid administration volumes were adjusted based on the estimated magnitude of blood loss. Upon enrollment, baseline measurements were obtained at time point T0. Arterial blood was drawn for lactate analysis, and venous blood was collected for NGAL analysis. Following administration of the assigned resuscitation fluid, repeat measurements of lactate and NGAL were performed 12 hours later at time point T1. NGAL samples were centrifuged, and serum concentrations were analyzed by enzyme-linked immunosorbent assay (ELISA) according to the manufacturer's protocol. Lactate concentrations were determined using standard arterial blood gas analysis. The study variables consisted of the type of fluid administered as the independent variable and lactate, NGAL, and pCO2 gap as the dependent variables. Controlled variables included patient age and baseline lactate and pCO2 gap values.

Statistical analysis

Statistical analyses were performed using IBM SPSS Statistics version 29.0.1.1. Numerical data were summarized as mean±standard deviation and analyzed using nonparametric tests, such as the Mann–Whitney U test or the Wilcoxon signed-rank test, where appropriate. In contrast, categorical data were expressed as frequencies and percentages and analyzed using chi-square testing.

Results

Sample characteristics

In this study, the sample characteristics were divided into three groups: the RL group, the RLH group, and the RLG group (**Table 1**). The mean age in the RL group was 45.07±10.51 years, in the RLH group was 42.60±9.70 years, and in the RLG group was 44.07±12.61 years. Sex distribution showed that the RL group consisted of 6 females (40.0%) and 9 males (60.0%); the RLH group included 5 females (33.3%) and 10 males (66.7%); whereas the RLG group consisted of 11 females (73.3%) and 4 males (26.7%). The mean pre-intervention lactate level was 5.78±1.12 mmol/l in the RL group, 5.85±0.82 mmol/l in the RLH group, and 6.02±0.87 mmol/l in the RLG group. Following the intervention, mean lactate levels decreased to 2.48±0.77 mmol/l in the RL group, 3.24±0.84 mmol/l in the RLH group, and 2.92±0.82 mmol/l in the RLG group. Pre-intervention pCO2 gap values were 6.53±1.12 mmHg in the RL group, 5.27±1.10 mmHg in the RLH group, and 5.93±0.70 mmHg in the RLG group. Post-intervention, the pCO2 gap decreased in all three groups to 5.33±0.72 mmHg in the RL group, 4.53±0.74 mmHg in the RLH group, and 5.07±0.59 mmHg in the RLG group. Mean pre-intervention NGAL levels were 11740.17± 24496.84 ng/ml in the RL group, 2431.83±2603.12 ng/ml in the RLH group, and 6506.93±16218.56 ng/ml in the RLG group. After the intervention, NGAL levels also declined, reaching 5451.53± 16486.16 ng/ml in the RL group, 1452.99±2080.54 ng/ml in the RLH group, and 4476.20±15006.78 ng/ml in the RLG group. Subsequent analyses were conducted using bivariate testing based on the normality results. In this study, lactate demonstrated a normal distribution ($p>0.05$), whereas NGAL exhibited a non-normal distribution ($p<0.05$).

Changes in blood lactate levels

Table 2 demonstrates the changes in blood lactate levels before and after resuscitation using the three fluid regimens in patients with hemorrhagic shock. In the RL group, mean lactate levels decreased significantly from 5.78±1.12 mmol/l to 2.48±0.77 mmol/l, with a delta of -3.30 ($p<0.001$; 95% CI 2.81–3.78). In the RLH group, lactate levels declined from 5.85±0.82 mmol/l to 3.24±0.84 mmol/l, yielding a delta of -2.61 ($p<0.001$; 95% CI 2.10–3.12). Meanwhile, in the RLG group, the mean lactate level decreased from 6.02±0.87 mmol/l to 2.92±0.82 mmol/l, with a delta of -3.10 ($p<0.001$; 95% CI 2.78–3.42). These findings indicate that all three resuscitation fluids significantly reduced blood lactate levels in patients with hemorrhagic shock, with the most significant reduction observed in the RL group.

Changes in NGAL levels

Table 3 illustrates the changes in NGAL levels before and after resuscitation using the three fluid regimens. In the RL group, mean NGAL levels decreased from 11740.17±24496.84 ng/ml to 5451.53±16486.16 ng/ml, with a delta of -6288.63±14218.55 ($p<0.001$; 95% CI -1589.33 to 14162.60). In the RLH group, NGAL levels also showed a significant reduction, decreasing from 2431.83±2603.12 ng/ml to 1452.99±2080.54 ng/ml, with a delta of -978.84±765.31 ($p<0.001$; 95% CI 555.02–1402.66). Meanwhile, in the RLG group, mean NGAL levels declined from 6506.93± 16218.56 ng/ml to 4476.20±15006.78 ng/ml, with a delta of -2020.73±2383.25 ($p<0.001$; 95% CI 710.93–3350.54). These results demonstrate that all three resuscitation fluids significantly reduced NGAL levels. However, the magnitude of reduction varied across the groups.

Comparison of post-resuscitation lactate and NGAL levels

Based on **Table 4**, the comparison of delta lactate levels after resuscitation demonstrated relatively similar reductions among the three groups: RL -3.30±0.88, RLH -2.61±0.91, and RLG -3.10±0.57, with a difference that approached statistical significance (p=0.067). These findings indicate that all three resuscitation fluids exert comparable effects on lactate levels, although there was a tendency for RL and RLG to produce slightly greater reductions than RLH. Meanwhile, for the delta NGAL variable, all three groups showed substantial variability in the magnitude of decline, with the RL group exhibiting the most significant reduction (-6288.63±14218.55), followed by RLG (-2030.73±2383.25), and RLH (-978.84±765.30). However, these differences were not statistically significant (p=0.655), indicating that the type of resuscitation fluid did not significantly influence post-resuscitation changes in NGAL levels.

According to **Table 5**, post hoc Bonferroni analysis showed that the comparison of delta lactate levels between the RL and RLH groups approached statistical significance, with a mean difference of -0.68 (95% CI: -1.42 to 0.04; p=0.074), suggesting a trend toward a difference. In contrast, the comparison between RL and RLG showed a minor mean difference of -0.19 (95% CI: -0.92 to 0.54; p=1.000), indicating no significant difference. Likewise, the comparison between RLH and RLG yielded a difference of 0.49 (95% CI: -0.24 to 1.22; p=0.304), which was not statistically significant. Overall, although a trend was observed between the RL and RLH groups, there was no statistically significant difference among the three fluid regimens in terms of lactate reduction following resuscitation in patients with hemorrhagic shock.

Based on **Table 6**, post hoc Mann–Whitney analysis demonstrated no significant differences in delta NGAL levels among the fluid groups. The comparison between RL and RLH yielded a mean difference of -5309.79 (95% CI: -12899.59 to 2280.00; p=0.412), while the comparison between RL and RLG revealed a mean difference of -4257.89 (95% CI: -11847.69 to 3331.89; p=0.724). Similarly, comparisons between RLH and RLG also showed nonsignificant results—both RLH vs RLG (1051.89; 95% CI: -6537.90 to 8669.69; p=0.304) and RLG vs RLH (-1051.89; 95% CI: -8641.69 to 6537.90; p=0.539). Overall, these results indicated that the type of resuscitation fluid used did not significantly affect changes in NGAL levels.

Discussion

The present study evaluated the comparative effects of RL, RL combined with HES 130/0.4 6%, and RL combined with gelofusine on lactate and NGAL levels following resuscitation in patients with hemorrhagic shock. All three fluid regimens demonstrated significant reductions in lactate and NGAL, indicating effective restoration of tissue perfusion and attenuation of early kidney injury markers. These findings aligned with the expected pathophysiology of hemorrhagic shock, wherein massive blood loss leads to intravascular volume depletion, reduced cardiac output, impaired tissue perfusion, and a shift toward anaerobic metabolism, resulting in elevated lactate production. Elevated lactate is a sensitive indicator of systemic oxygen supply—demand imbalance and hepatic clearance dysfunction, and high lactate levels correlate with poor prognosis in shock states. (10,12)

Following resuscitation, all treatment groups exhibited marked reductions in lactate, demonstrating successful restoration of macrocirculatory and microcirculatory flow. Although the RL and RLG groups showed numerically greater reductions in delta lactate than the RLH group, these differences did not reach statistical significance. This result contrasted with network meta-analysis findings by Wu et al., which suggested that colloids, including HES and gelofusine, may outperform crystalloids in mitigating lactate accumulation after hemorrhagic shock. (9) The lack of a significant difference in the present study may be attributable to variations in patient volume status, heterogeneity of blood loss, timing of intervention, or sample size limitations.

Regarding NGAL, all groups exhibited significant declines post-resuscitation, although the magnitude varied considerably, and no intergroup differences were statistically significant. NGAL plays an important role in the inflammatory response and tissue injury during hemorrhagic shock, as hypoperfusion-induced hypoxia triggers the release of inflammatory mediators and promotes neutrophil activation. (13,14) NGAL is closely associated with acute kidney injury (AKI), and its kinetics may reflect both renal hypoperfusion and systemic inflammation. Elevated NGAL has been demonstrated in abdominal trauma patients with hemorrhagic shock and correlated with injury severity and organ dysfunction. (15,16)

Despite the biologic plausibility that colloids, particularly HES, may alter endothelial permeability or renal stress, the NGAL reductions observed in the RL, RLH, and RLG groups did not differ significantly. The absence of a significant benefit of RLH over RL alone warrants careful interpretation. Prior studies have demonstrated that RL combined with HES may better preserve endo-

thelial glycocalyx integrity, as indicated by lower syndecan-1 levels following resuscitation. (8,17) However, concerns regarding HES-associated renal toxicity remain substantial, supported by mechanistic and clinical evidence indicating potential risks of AKI. In contrast, gelofusine, although a colloid with weaker oncotic effects than HES, has been reported to enhance intravascular volume more efficiently than crystalloids and potentially improve organ perfusion without the same risk profile. (10,11)

The present findings suggest that, in acute hemorrhagic shock resuscitation, neither colloid combination produced superior biomarker improvement compared with RL alone. It is possible that the short observation window (12 hours) was insufficient to capture differential effects on renal biomarkers such as NGAL, whose peak elevation may occur within variable time frames (0–24 hours), as noted in previous studies. (14,18) From a physiological standpoint, the comparable improvements across all groups reinforce the principle that timely restoration of circulating volume, regardless of fluid type, remains the cornerstone of hemorrhagic shock management. RL, as a balanced crystalloid, provides rapid intravascular expansion but diffuses quickly into the interstitial space. In contrast, gelofusine maintains intravascular retention for a longer period due to its colloid-osmotic properties, potentially reducing total fluid requirements. Synthetic colloids, including HES, exert a more potent volume-expanding effect but carry a risk of renal and coagulation complications. (17,19,20) The absence of outcome differences in this study may indicate that early microcirculatory improvement is predominantly driven by restoration of perfusion pressure rather than by fluid composition.

The findings of the current study must be interpreted in light of several limitations. First, the single-center design limits generalizability, as differences in population characteristics, clinical practice patterns, and hemorrhage etiologies could influence outcomes. Second, the sample size, although adequate for the primary endpoints, may be underpowered to detect more subtle differences in NGAL dynamics or endothelial markers. Third, NGAL was measured only once post-resuscitation, whereas its temporal pattern may require serial measurements to elucidate renal or inflammatory trajectories fully. These limitations are consistent with the constraints acknowledged in similar clinical investigations and highlight the need for multicenter, larger-scale trials with serial biomarker assessment to strengthen the evidence base.

In summary, this study demonstrates that RL, RLH, and RLG all effectively improved lactate and NGAL levels after resuscitation in hemorrhagic shock, with no significant differences among them. While theoretical advantages of colloid combinations exist, particularly in endothelial protection and volume expansion, the present findings suggest that RL alone provides comparable biochemical recovery in the early phase of resuscitation. These insights add to the growing body of literature emphasizing individualized fluid selection based on patient physiology, risk profile, and clinical context rather than a universal preference for crystalloid or colloid solutions.

Table 1. Sample characteristics

Variable	Group		
	RL	RLH	RLG
Age (years), mean±SD	45.07±10.51	42.60±9.70	44.07±12.61
Gender, n (%) - Female - Male	 6 (40.0) 9 (60.0)	 5 (33.3) 10 (66.7)	 11 (73.3) 4 (26.7)
Lactate (mmol/l), mean±SD - Pre - Post	 5.78±1.12 2.48±0.77	 5.85±0.82 3.24±0.84	 6.02±0.87 2.92±0.82
pCO2 gap (mmHg), mean±SD - Pre - Post	 6.53±1.12 5.33±0.72	 5.27±1.100 4.53±0.74	 5.93±0.70 5.07±0.59
NGAL (ng/ml), mean±SD - Pre - Post	 11740.17±24496.84 5451.53±16486.16	 2431.83±2603.12 1452.99±2080.54	 6506.93±16218.56 4476.20±15006.78

Legend: RL=Ringer's lactate; RLH=RL combined with HES 130/0.4 6%; RLG=RL combined with gelofusine; SD=standard deviation; pCO2=partial pressure of carbon dioxide; NGAL=neutrophil gelatinase-associated lipocalin.

Table 2. Changes in blood lactate levels

Group	Lactate (mmol/l), mean±SD		Delta	p-value	95% CI	
	Pre	Post			Upper	Lower
RL	5.78±1.12	2.48±0.77	-3.30	<0.001	2.811	3.78
RLH	5.85±0.82	3.24±0.84	-2.61	<0.001	2.10	3.12
RLG	6.02±0.87	2.92±0.82	-3.10	<0.001	2.78	3.42

Legend: SD=standard deviation; CI=confidence interval; RL=Ringer's lactate; RLH=RL combined with HES 130/0.4 6%; RLG=RL combined with gelofusine.

Table 3. Changes in NGAL

Group	NGAL (ng/ml), mean±SD		Delta	p-value	95% CI	
	Pre	Post			Upper	Lower
RL	11740.17±24496.84	5451.53±16486.16	-6288.63±14218.55	<0.001	-1589.33	14162.60
RLH	2431.83±2603.12	1452.99±2080.54	-978.84±765.31	<0.001	555.02	1402.66
RLG	6506.93±16218.56	4476.200±15006.78	-2020.73±2383.25	<0.001	710.93	3350.54

Legend: NGAL=neutrophil gelatinase-associated lipocalin; SD=standard deviation; CI=confidence interval; RL=Ringer's lactate; RLH=RL combined with HES 130/0.4 6%; RLG=RL combined with gelofusine.

Table 4. Comparison of changes in lactate levels and NGAL

Variable	Group			p-value
	RL	RLH	RLG	
Delta lactate value	-3.30±0.88	-2613±0.91	-3.10±0.57	0.067[a]
Delta NGAL value	-6288.63±14218.55	-978.84±765.3	-2030.73±2383.25	0.655[b]

Legend: NGAL=neutrophil gelatinase-associated lipocalin; RL=Ringer's lactate; RLH=RL combined with HES 130/0.4 6%; RLG=RL combined with gelofusine.
[a]One-way ANOVA analysis; [b]Kruskal-Walis.

Table 5. Post hoc comparison of lactate levels

Group		Mean differences	p-value[a]	95% CI	
				Upper	Lower
RL	RLH	-0.68	0.074	-1.42	0.04
	RLG	-0.19	1.000	-0.92	0.54
RLH	RL	0.68	0.074	-0.04	1.42
	RLG	0.49	0.304	-0.24	1.22
RLG	RL	0.19	1.000	-0.54	0.92
	RLH	-0.49	0.304	-1.22	0.24

Legend: CI=confidence interval; RL=Ringer's lactate; RLH=RL combined with HES 130/0.4 6%; RLG=RL combined with gelofusine.
[a]Post hoc Bonferroni.

Table 6. Comparison of changes in NGAL

Group		Mean differences	p-value[a]	95% CI	
				Upper	Lower
RL	RLH	-5309.79	0.412	-12899.59	2280.00
	RLG	-4257.89	0.724	-11847.69	3331.89
RLH	RL	5309.79	0.412	-2280.00	12899.59
	RLG	1051.89	0.304	-6537.90	86.69
RLG	RL	4257.89	0.724	-3331.89	11847.69
	RLH	-1051.89	0.539	-8641.69	6537.90

Legend: CI=confidence interval; RL=Ringer's lactate; RLH=RL combined with HES 130/0.4 6%; RLG=RL combined with gelofusine.
[a]Post hoc Mann-Whitney.

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