Iraqi Journal of Veterinary Sciences

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The superiority of combined ringer's lactate and gelatin resuscitation in mitigating renal histopathological injury post-hemorrhagic shock: a rabbit model study

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Article information

Article history:
Received November 24, 2025
Accepted March 17, 2026
Published April 6, 2026

Keywords:
Acute kidney injury
Gelatin
Histopathology
Fluid resuscitation
Hemorrhagic shock

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Abstract

Background: Hemorrhagic shock (HS) is a leading cause of morbidity and mortality and frequently precipitates multi-organ dysfunction, particularly AKI. Aggressive fluid resuscitation is essential; however, the optimal fluid type to restore hemodynamic stability while protecting organs remains uncertain. Objective: This study compared crystalloid resuscitation using Ringer's Lactate (RL) with combined crystalloid-colloid resuscitation (RL-gelatin) in terms of organ function and renal histopathology after hemorrhagic shock in a rabbit model. Methods: Rabbits were allocated to four groups: Negative Control (KN; no HS, no resuscitation), Positive Control (KP; HS without resuscitation), KP1 (HS with RL), and KP2 (HS with RL-gelatin). HS was induced by withdrawing 35% of the estimated blood volume. Hematologic indices; and renal function were measured from the pre-hemorrhagic phase (T15) through post-resuscitation (T230), followed by renal histopathology and injury scoring. Results: The KP group developed severe organ dysfunction, with massive renal histopathologic damage (score = 4.0). Both resuscitation strategies stabilized renal function; however, KP2 had the lowest injury score (1.5) compared to KP1. The KP group showed a significant increase in creatinine (167.62±42.99, P=0.013) and BUN (13.06±4.87, P=0.031) at T230, indicating severe renal dysfunction. In contrast, the KP2 (RL-gelatin) group maintained stable renal function (creatinine: 110.12, P=0.564; BUN 9.30, P=0.398). Although all HS groups showed significant hemodilution (RBC: 3.51±0.50, P=0.002; Hb: 6.00±1.10, P=0.001, Hct: 23.75±3.50, P=0.001. KP1 and KP2 resuscitation were effective in preventing AKI and stabilizing renal function. Conclusions: This study provides experimental evidence that the combination of RL-gelatin provides superior structural renal protection after hemorrhagic shock.

DOI: 10.33899/ijvs.2026.167357.4630, ©Authors, 2026, College of Veterinary Medicine, University of Mosul.
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Introduction

Hemorrhagic shock is the leading cause of disability and death due to blood loss (1). Hemorrhage remains one of the leading causes of preventable death worldwide, accounting for approximately 1.5 million deaths annually (2). Shock can lead to circulatory system disturbances, resulting in reduced oxygen transport to tissues (3) Due to a sharp decline in effective circulation (4), which causes vasoconstriction, leading to hypoperfusion, cellular hypoxia, organ damage, multi-organ failure, and death (5). Hemorrhagic shock must be managed efficiently with blood transfusion (6). However,

in developing countries like Indonesia, blood banks are not widely available, and direct blood donation procedures are complicated, requiring time to match the donor with the recipient, along with the high costs associated with this method. Therefore, an alternative, such as fluid therapy, is needed.

Fluid therapy is a crucial intervention for the early treatment of hemorrhagic shock to save lives (7), and is used in therapy to restore blood volume (8). Fluid resuscitation is critical in early management, aiming to prevent ischemia and necrosis (9). Crystalloid and colloid fluids are widely used for resuscitation, with RL being a commonly used solution. Other fluid options include albumin and gelatin as protein colloids (10). Given the limited data available on the use of gelatin, this study focuses on investigating the impact of gelatin-based fluid therapy in the management of hemorrhagic shock.

Colloid solutions, such as high molecular weight hydroxyethyl starch, have been associated with an increased risk of mortality and acute kidney injury (11). Intravenous gelatin solution can also contribute to acute kidney injury in a rabbit model (12). Most fluid resuscitation research relies on rodent models, whereas rabbit models offer closer physiological parallels to humans and companion animals. Furthermore, reports on RL-gelatin efficacy regarding organ structural protection remain scarce in this model. This study aims to fill this gap by evaluating the synergistic effect of RL-gelatin on renal histopathology as a clinical approach.

The novelty of this study evaluates a resuscitation protocol that combines RL-gelatin as a non-HES alternative using a rabbit model. Unlike previous rodent-based studies focusing on coagulation or pulmonary inflammation, this research specifically demonstrates superior renal histopathological protection. These findings highlight gelatin role in directly preserving renal structural integrity. To the best of our knowledge, this is the first study to evaluate the renal protective effects of combined RL-gelatin in a rabbit model of hemorrhagic shock.

Materials and methods

Ethical approval

This research has been reviewed and approved by the Hasanuddin University Research Ethics Committee, Faculty of Medicine, RSPTN Hasanuddin University, under Ethical Approval Recommendations Number: 584/UN4.6.4. 5.31/ PP36/ 2023, at August 25, 2023.

Object of research

Male New Zealand White rabbits (2.0–2.8 kg; 5–8 months) were used. All procedures were approved by the Ethics Committee of the Faculty of Medicine, Hasanuddin University (No. 584/UN4.6.4.5.31/PP36/2023), and adhere to animal welfare standards and the 3Rs to minimize pain and animal use. The rabbits were housed in cages measuring 60 × 42 × 50 cm and were provided with standard food and water ad libitum.

Protocol of research

The 24 rabbits were divided into four groups (n=6), satisfying Federer's formula, (t-1) (n-1) ≥ 15, ensuring adequate statistical power and experimental reliability: Negative Control (KN), no hemorrhage; Positive Control (KP), hemorrhage without resuscitation; Treatment 1 (KP1), resuscitation with RL; and Treatment 2 (KP2), resuscitation with a combination of RL-gelatin. Anesthesia was induced with acepromazine 0.75 mg/kg, xylazine 3 mg/kg, and ketamine 70 mg/kg intramuscularly (IM) (13) and maintained with intravenous (IV) ketamine at one-third of the induction dose. Hemorrhage was induced manually via the auricular artery using a 26G IV catheter. To ensure rapid and continuous blood flow, the ear was positioned vertically at a 60^0–90^0 angle, allowing the catheter to remain straight and preventing kinking that could lead to flow obstruction and the saphenous vein/cephalic vein as a location for resuscitation. The experiment lasted 230 min and comprised three phases (Figure 1): (1) pre-hemorrhagic, 15 min (T15); (2) hemorrhagic, 65 min; 15 min of bleeding followed by 50 min of maintaining the hemorrhagic state with blood loss targeted at 35% of total blood volume (estimated as 6% of body weight); and (3) post-hemorrhagic, 150 min; 30 min of fluid therapy followed by 120 min of maintaining resuscitation (14). Following the protocol, rabbits were euthanized using an overdose of ketamine and necropsied for histopathological sampling.

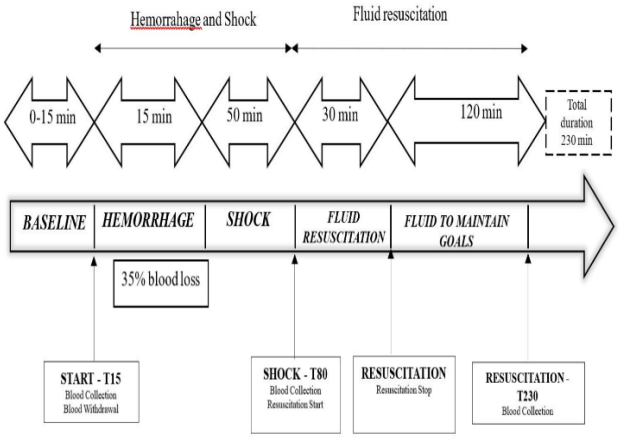

Figure 1: Overview of the study design and blood-sampling schedule for a single 230-min treatment cycle. Samples were collected at pre-hemorrhagic (T15), hemorrhagic (T30), sustained hemorrhage (T80), post-hemorrhagic/post-therapy (T110), and resuscitation maintenance (T230).

Fluid therapy

Fluid therapy using RL and synthetic colloid Gelatin Polysuccinate (Gelofusal®). The dose is calculated from the maintenance volume ((30 x BW (kg)) + 70), deficit replacement (% dehydration x BW (kg) x 1000ml x 80%) and the volume of fluid lost (2 x estimated loss), and the combination of crystalloid (RL) and colloid using a 2:1 ratio (15).

Blood laboratory examination

Hematological parameters (RBC, Hb, HCT, MCV) were measured using an Auto Hematology Analyzer (CC-3200 Vet®). Clinical chemistry analytes (creatinine, BUN, glucose, Na^+) were measured using an Automed Chemistry Analyzer (SMT-120V®). Blood samples (0.5 mL) were collected into EDTA microtainer tubes and immediately analyzed at three time points: T15, T80, and T230 (14). Blood was sampled from the auricular artery due to the technical difficulty of consistent venous access, as rabbit peripheral veins are highly prone to rapid collapse.

Histopathology

Kidney samples were fixed in 10% neutral buffered formalin, dehydrated through a graded series of ethanol, cleared in xylene, and embedded in paraffin wax. Sections were then cut at 2-3 µm thickness and stained with Hematoxylin and Eosin (H&E) for microscopic examination. Tissue injury was assessed using a modified semiquantitative scoring system across five categories: hemorrhage, necrosis, brush-border rupture, obliteration of Bowman's space, and hypertrophy. Each category was graded from 0 to 4, represents the percentage of damage: 0 (none), 1 (<25%), 2 (26-50%), 3 (51-75%), 4 (76-100%) (16,17).

Statistical Analysis

Data were analyzed using IBM SPSS Statistics, version 27. Sources of variation were treatment group and time. Data were analyzed statistically using a two-factor ANOVA (time and group) (18), followed by Tukey's post-hoc test. A value of $P < 0.05$ was considered statistically significant. Data normality was assessed using the Shapiro–Wilk test. Histopathological outcomes were summarized descriptively.

Results

The KN group showed consistent stability across all vital parameters (Figure 2) throughout the observation phases. No significant statistically fluctuations were observed in HR, RR, and temperature at the T15, T30, T80, T110, and T230 phases. All groups subjected to hemorrhage showed a downward trend in HR and temperature parameters at the T80 phase, and an increase in respiratory parameters at T80, reflecting the adverse effects of hemorrhage. However, in the KP1 and KP2 groups, which received resuscitation with RL or its combination, a stabilization trend occurred. Although both groups exhibited physiological improvements, the KP2 group demonstrated a better observational response, with vital parameters tending to return closer to normal values, indicating the effectiveness of the resuscitation combination in supporting the recovery process. While no statistically significant differences ($P > 0.05$) were found between resuscitation groups**.** thermoregulatory function declined drastically in the KP group, dropping from 38.09 ± 0.41°C (T15) to 36.10 ± 1.49°C (T230). Interestingly, the KP2 group exhibited an even more pronounced reduction, reaching 34.80 ± 0.80°C at T230. which may be associated with the acute metabolic response during the shock phase.

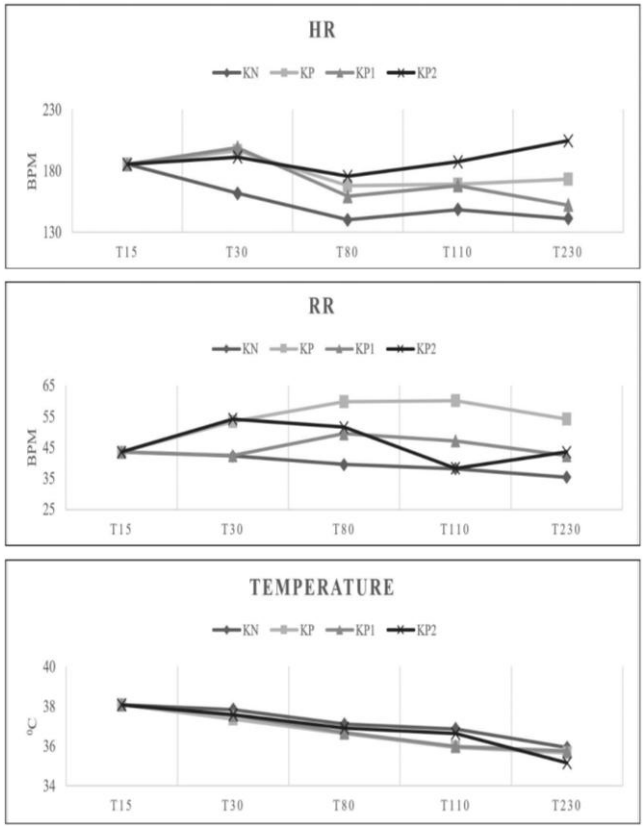

Figure 2: This graph displays the parameters of heart rate (HR), respiratory rate (RR), and temperature (y-axis) against the duration of time (x-axis) in the pre-hemorrhagic phase (T15), hemorrhagic phase (T30), maintain hemorrhagic phase (T80), resuscitation phase (T110) and maintain resuscitation (T230).

Renal histopathological injury scores (Figure 3) were highest in the KP group (score 4.0), whereas the KN group showed no detectable injury (score 0), with cells appearing normal. A score of 4.0 in the KP group reflects the massive

structural damage, characterized by coagulative necrosis of glomerular cells, extensive interstitial hemorrhage, and widespread inflammatory cell infiltration within the tubulointerstitial area. Furthermore, severe tubular damage was evident through the diffuse loss and rupture of the brush border, indicating profound acute tubular injury. Resuscitation with RL (KP1, score 3.0) reduced injury relative to KP, while the combination of RL-gelatin (KP2, score 1.5) produced the most substantial reduction. Consistent with these scores, KP2 sections were dominated by preserved glomerular architecture with only focal hemorrhagic changes, supporting a protective effect of gelatin in limiting further renal structural injury.

Creatinine levels (Table 1) significantly increased (P=0.013) in the shock group without resuscitation (KP) from T15 (104.33±15.30) to T230 (167.62±42.99), indicating the development of severe acute injury. In contrast to the KP group, both resuscitation groups (KP1 and KP2) did not show a significant increase in creatinine levels. This suggests that resuscitation interventions are effective in maintaining renal perfusion. Furthermore, BUN also significantly increased in the KP group (T230: 13.06±4.87). However, KP1 (RL) exhibited only a minimal increase in BUN (P=0.006), highlighting that crystalloids (RL) were less effective in protecting the kidneys from early damage (early dysfunction) due to hemorrhagic shock compared to the KP2 group. The combination of RL-gelatin in KP2 showed no significant increase, indicating that this intervention is effective in maintaining renal perfusion. These findings are consistent with the renal histopathological results in the KP2 group.

The Na+ parameters (Table 2) generally did not show significant differences between time points or between groups. However, during the hemorrhagic phase, there was a trend of decreasing levels, which then increased after resuscitation, although this change was not significant. In contrast, the glucose parameter showed significant dynamics. At T80, all shock groups (KP, KP1, KP2) demonstrated an increase in glucose, reflecting a stress and shock response. Post-resuscitation (T230), both the KP1 (RL) and KP2 (RL-gelatin) groups showed a significant decrease in glucose from T80 (KP1: P=0.040; KP2: P=0.023), approaching baseline levels (T15). This decrease indicates recovery of tissue perfusion and improved glucose absorption, particularly in the KP2 group, which exhibited the lowest levels at T230 (11.83±3.95).

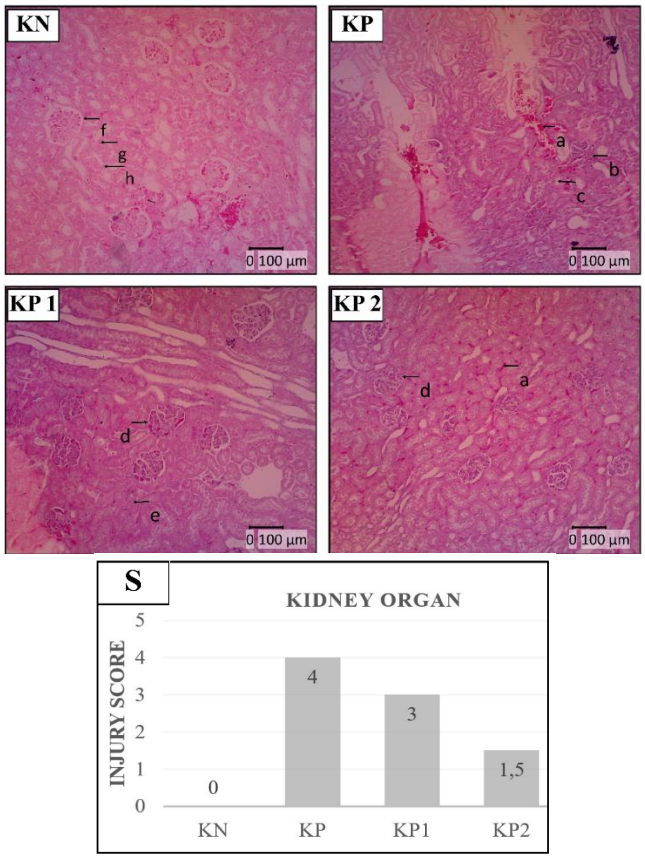

Figure 3: Histopathology of rabbit kidney, HE staining, 100x magnification; Treatment Groups; KN, KP, KP1, and KP2. Image Captions; Hemorrhage (a), Necrosis (b), Brush Border Rupture (c), Bowman's Space Obliteration (d), Hypertrophy (e), Glomerulus (f), Proximal tubule (g), Distal tubule (h). S; Score of histopathological damage to the kidney.

Table 1: Comparison of changes in Creatinine and BUN in the pre-hemorrhagic, hemorrhagic, and post-hemorrhagic/post-therapy phases at different time points

Creatinin Parameters					BUN Parameters				
Group	T15 (N=6)	T80 (N=6)	T230 (N=6)	p-Value	Group	T15 (N=6)	T80 (N=6)	T230 (N=6)	p-Value
KN	89.42±44.96	111.38±50.47	109.15±47.74	0.688	KN	8.02±1.58	9.56±1.65	11.25±2.33	0.225
KP	104.33±15.30	141.10±31.30	167.62±42.99*	0.013	KP	8.51±3.80	10.57±4.3	13.06±4.87*	0.031
KP1	90.10±36.63	120.32±44.63	106.33±68.32	0.608	KP1	7.07±1.13	8.20±1.08	9.55±1.17*	0.006
KP2	105.65±41.33	132.03±48.93	110.12±43.98	0.564	KP2	7.45±2.06	8.85±2.24	9.30±2.80	0.398

Time points in the table rows show significant differences (P<0.05) with respect to T15 (*) as baseline and times showing differences with respect to T80 (#) as hemorrhagic period.

Table 2: Comparison of changes in Na+ and Glucose in the pre-hemorrhagic, hemorrhagic, and post-hemorrhagic/post-therapy phases at different time points

Na+ Parameters					Glucose Parameters				
Group	T15 (N=6)	T80 (N=6)	T230 (N=6)	p-Value	Group	T15 (N=6)	T80 (N=6)	T230 (N=6)	p-Value
KN	133.23± 20.72	141.29± 2.82	141.23± 3.46	0.363	KN	14.59± 4.75	15.27± 7.39	14.70± 8.16	0.983
KP	143.67± 2.94	142.75± 2.03	143.22± 1.59	0.784	KP	11.19± 3.51	15.17± 4.68	16.56± 5.11	0.133
KP1	142.07± 2.74	124.10± 40.63	141.38± 4.23	0.354	KP1	10.66± 3.29	14.96± 3.03	10.94± 2.44	0.040
KP2	138.68± 7.86	139.00± 3.96	141.28± 3.10	0.667	KP2	13.72± 3.27	17.91± 3.09	11.83± 3.95#	0.023

Time points in the table rows showed significant differences (P<0.05) with respect to T15 (*) as baseline and times showing differences with respect to T80 (#) as hemorrhagic period.

All major hematologic parameters (RBC, Hb, Hct, and MCV; Figure 4) decreased significantly at T230 across all shock groups (KP, KP1, KP2), consistent with post-resuscitation hemodilution and severe blood-volume loss. The most pronounced declines in RBC, Hb, and Hct were observed in the resuscitated groups—KP1 and especially KP2—corroborated by significant increases in mean corpuscular volume (MCV) in both groups. These findings underscore the strong hemodilutional effect of fluid resuscitation, particularly with RL (KP1); moreover, RL-gelatin (KP2) did not appear to offer any advantage in maintaining hemoglobin or hematocrit concentrations.

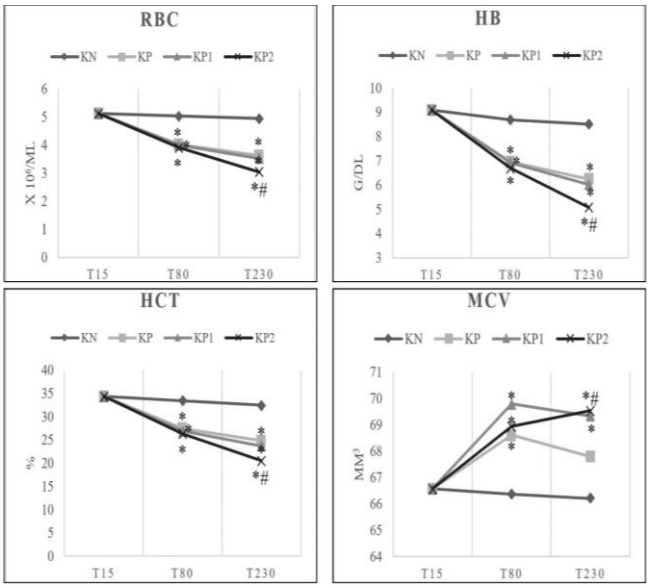

Figure 4: This graph displays the differences between groups and between time periods in RBC, HB, HCT, and MCV parameters (y-axis) against time duration (x-axis) in the pre-hemorrhagic (T15), hemorrhagic (T30), Maintain Bleeding (T80) and post-hemorrhagic/post-therapy (T110), Maintain Resuscitation (T230) phases. The times showing significant differences (P<0.05) against T15 (*) are baseline and the times showing significant differences (p<0.05) against T80 (#) are the maintained hemorrhagic period.

Discussion

Heart rate (HR) measurement during the hemorrhagic phase (T30) indicated an increase. This finding is consistent with reports from other studies that also documented elevated HR during hemorrhage (19). The resulting tachycardia has the potential to increase myocardial workload, which in turn leads to excessive myocardial energy expenditure (20). Another study reported the same thing, namely that the initial HR of bleeding increased, but the HR slowly decreased if the duration of bleeding was longer, and after resuscitation there was a gradual increase (1) in contrast to other reports of resuscitation using Bicarbonate Ringer solution showing a decrease in HR after resuscitation (21). Furthermore, all groups experiencing shock exhibited a progressive decline in body temperature. Hemorrhagic shock in the animal model led to hypothermia (22).

Hemorrhagic shock causes a failure of oxygen distribution to the tissues. In this study, the shock phase was characterized by compensatory tachypnea (increased RR) up to T80. Hemorrhagic shock results in inadequate oxygen delivery (5), After resuscitation, normalization of RR in the treatment group indicates improved oxygen distribution and adequate tissue oxygen intake, although it does not directly measure oxygen delivery. In line with other studies, it has been shown that after resuscitation, oxygen distribution improved, leading to enhanced tissue oxygenation (23).

During the hemorrhagic phase, blood urea nitrogen (BUN) and creatinine levels in all groups showed an increase. However, following resuscitation, creatinine levels decreased, while BUN levels increased. These findings are consistent with results reported by previous studies using different therapies; Specifically, hypertonic 7.5% therapy showed a decrease and improvement following resuscitation (17). In the hemorrhagic phase, sodium (Na) levels showed a decrease in concentration; however, immediately after resuscitation, Na levels increased, approaching normal range values. This finding is consistent with the results reported by previous studies (24).

The histopathological findings of the kidneys in the KP2 group, which received combined resuscitation with RL-

gelatin, demonstrated less cellular damage. This contrasts with other studies, where the use of intravenous gelatin alone was shown to contribute to acute kidney injury (12). Similarly, another study reported a reduction in kidney damage using a different combination therapy, hydroxyethyl starch and RL (15). Another report indicated that resuscitation with a hypertonic solution (7.5%) resulted in less liver damage (23). The KP group with the most severe damage exhibited necrosis, hemorrhaging, inflammation, and rupture of the brush border. Hemorrhaging led to an uncontrolled state, damaging parenchymal organs (25), through a necrotic process caused by oxygen deficiency to tissues (26), with insufficient oxygen delivery to meet metabolic demands, resulting in hypoxia (5).

Resuscitation in cases of hemorrhagic shock involves rapid control of the source of bleeding and effective restoration of tissue perfusion, oxygenation, and cellular metabolism (5). Aggressive treatment protocols, increased fluid requirements, and early management of hyperkalemia are essential and should be closely monitored to improve survival, reduce acute kidney injury, and prevent early death due to hyperkalemia-associated arrhythmias (27). Acid-base balance is more stable when gelatin in balanced electrolyte solutions is used as a replacement for saline, and no serious adverse drug reactions associated with gelatin were observed (28).

Decreased hematological values in both RBC, Hb, and Hct indicators in the KP, KP1, and KP2 groups. Similar results were observed during the hemorrhagic phase, where the Hb value prior to hemorrhage was 7.1±0.7, showing a decrease 1 hour post-hemorrhage to 6.3±1.1 (19), Six hours after hemorrhage, a further decline in RBC, Hb, and Hct was also observed (29). Comparable findings were noted for Hct and Hb, with baseline values of 36.1±1.3 and 12.3±0.5, which decreased following RL resuscitation at 85 minutes to 23.0±2.5 and 6.9±2.0, respectively (30). Additional studies have reported similar trends during the hemorrhagic phase and post-resuscitation, where Hct levels continued to decline (15,31).

During the hemorrhagic phase, a decrease was observed; however, following resuscitation, RBC, Hb, and Hct levels continued to decline further. This is attributed to hemodilution. The reduction in capillary density caused by hemodilution (32). Similar results reported the use of gelatin in indicators on Hct and Hb showed a decrease caused by hemodilution due to blood dilution (28). The KP2 group showed a more pronounced decline compared to KP1, likely influenced by the use of gelatin colloid. Colloid solutions remain in the intravascular space longer than crystalloid solutions, thereby being more effective in maintaining osmotic pressure (33).

The principal limitation of this study lies in the animal model. Extrapolation of findings from rabbits to other veterinary species (dogs, cats, or horses) should be undertaken cautiously, given substantial interspecies variation in response to hemorrhagic shock, fluid metabolism, and risk of complications. Although the histopathological protection afforded by the RL-gelatin combination is a robust and novel finding, its effectiveness requires validation through comparative studies. Moreover, the present investigation focuses primarily on renal outcomes; analyzes of injury to other vital organs (e.g., lung or liver) and a longer observation period to assess long-term morbidity and mortality would provide a more comprehensive assessment of the benefits of this combined resuscitation strategy.

Conclusion

This study concludes that hemorrhagic shock without resuscitation (KP) induces severe multiple-organ dysfunction, characterized by extensive renal histopathologic injury and marked elevations in serum creatinine and blood urea nitrogen (BUN). Fluid resuscitation, either with RL (KP1) or RL-gelatin (KP2), prevents acute kidney injury and stabilizes renal function. Notably, the combined RL–gelatin regimen (KP2) most effectively restored vital signs and provided the greatest structural renal protection (lowest kidney-injury score, 1.5). Nevertheless, gelatin conferred no advantage in mitigating post-resuscitation hemodilution, as evidenced by declines in hematologic parameters.

Acknowledgment

The author expresses gratitude to Gadjah Mada University for supporting the implementation of the research.

Conflict of interest

There is no conflict of interest.

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تفوق إنعاش محلول مركب اللاكتات والجيلاتين في التخفيف من الإصابة النسيجية المرضية الكلوية للصدمة ما بعد النزف: دراسة على نموذج الأرنب

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الخلاصة

الصدمة النزفية هي السبب الرئيسي في معدل الاصابة ومعدل النفوق وكثيرا ما خلل متعدد الأعضاء، وخاصة ضرر الكلى الحاد. ومع ذلك، فإن نوع السائل الأمثل لاستعادة استقرار الدورة الدموية مع حماية الأعضاء لا يزال غير مؤكد. قارنت هذه الدراسة الإنعاش باستخدام محلول لاكتات مقابل الإنعاش البلوري الغرواني المشترك (محلول لاكتات مع الجيلاتين) من حيث معالجة الاعضاء والغير النسجي الكلوي بعد صدمة النزف في نموذج الأرنب. تم تقسيم الأرانب لأربع مجموعات: السيطرة السلبية (بدون صدمة نزفية وبدون إنعاش)، السيطرة الإيجابية (صدمة نزفية وبدون إنعاش)، مجموعة معاملة ١ (صدمة نزفية مع انعاش بمحلول لاكتات)، و مجموعة معاملة ٢ (صدمة نزفية مع انعاش بمحلول لاكتات مع الجلاتين). تم تحفيز التهاب الغدد العرقية المقيح عن طريق سحب ٣٥ ٪ من حجم الدم المقدر. تم قياس وظائف الكلى من مرحلة ما قبل النزفية (الوقت ١٥) مرحلة ما بعد الإنعاش (الوقت ٢٣٠)، تبعها أمراض الأنسجة الكلوية ودرجات الضرر. في مجموعة السيطرة الايجابية حصل فقدان وظيفة شديد للاعضاء، مع ضرر نسيجي كلوي هائل (الدرجة ٤). في كل من استراتيجيات الإنعاش استقرت وظيفة الكلى؛ ومع ذلك، كانت أدنى درجة إصابة ١,٥ في مجموعة المعاملة ٢ مقارنة مع مجموعة المعاملة ١. أظهرت مجموعة السيطرة الايجابية زيادة كبيرة في الكرياتينين (١٦٧,٦٢±٤٢,٩٩، إ=٠,٠١٣) و نيتروجين اليوريا في الدم (١٣,٠٦±٤,٨٧، إ=٠,٠٣١) عند الوقت ٢٣٠، مما يشير إلى ضعف كلوي حاد. في المقابل، حافظت مجموعة المعاملة ٢ (لاكتات مع الجيلاتين) على وظيفة الكلى مستقرة (الكرياتينين: ١١٠,١٢، إ=٠,٥٦٤ ؛ نيتروجين اليوريا في الدم: ٩,٣٠، إ=٠,٣٩٨). على الرغم من أن جميع مجموعات الصدمة النزفية أظهرت تخفيفا كبيرا للدم (كريات الدم الحمر: ٣,٥١±٠,٥٠، إ=٠,٠٠٢؛ الهيموكلوبين: ٦,٠٠±١,١٠، إ=٠,٠٠١، الهيماتوكرت: ٢٣,٧٥±٣,٥٠، إ=٠,٠٠١. مجموعة المعاملة ١ ومجموعة المعاملة ٢ كانت فعالة في منع ضرر الكلى الحاد واستقرار وظيفة الكلى. في الاستنتاجات، تقدم هذه الدراسة أدلة جديدة على أن الجمع بين اللاكتات والجيلاتين يوفر حماية متميزة لهيكلية الكلى بعد صدمة النزف.