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ORIGINAL RESEARCH

Effect of Normal Saline and Balanced Salt Solution on Resuscitation of Patients with Peritonitis: A Prospective Randomized Study

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ABSTRACT

Background: This study aimed to evaluate differences in metabolic parameters following resuscitation with either normal saline or Plasmalyte A in patients undergoing surgery for perforated peritonitis.

Methods: In this prospective, randomized study, 50 adult patients (≥18 years) of either gender presenting with peritonitis and undergoing emergency exploratory laparotomy were enrolled. Patients were randomly allocated into two groups of 25 each: Group A received Plasmalyte A for resuscitation; Group B received normal saline for resuscitation. We recorded baseline measurements, including blood pressure, heart rate, SpO_2, and arterial blood gases (ABG). We then assessed these parameters one hour after fluid resuscitation, preoperatively, intraoperatively (at incision), and postoperatively.

Results: The mean age in Group A was 38.56 ± 17.18 years, and in Group B, 39.80 ± 15.93 years (p = 0.792). Group B showed a notable intraoperative decrease in mean arterial pressure (MAP)

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(73.21 ± 6.95 mmHg) compared with the preoperative period (79.38 ± 7.35 mmHg). Group B also showed a significant intraoperative drop in pH ($p < 0.001$) and a corresponding increase in intraoperative and postoperative PCO_2 levels compared with Group A. Group B also showed a persistent increase in serum sodium levels throughout the study period.

Conclusion: Compared to normal saline, Plasmalyte A offers superior preservation of metabolic stability in patients undergoing large-volume resuscitation. Consequently, it is highly recommended as the crystalloid of choice in these clinical settings.

Keywords: Electrolyte imbalance, normal saline, Plasmalyte A

INTRODUCTION

Proper intravenous fluid administration is a critical component of perioperative care and helps maintain stable intravascular volume. Patients frequently experience volume depletion as a result of preoperative fasting, surgical bleeding, evaporative and urinary losses, third-spacing, and capillary leaks induced by inflammatory responses. To support hemodynamic stability without causing interstitial fluid overload, clinicians must titrate these fluids carefully.[1]

While 0.9% normal saline is the most frequently administered crystalloid, its high-volume use can be problematic. Providing more than 1 liter daily introduces excessive sodium and chloride, which can lead to hypernatremia and hyperchloremia. Furthermore, normal saline's acidic nature, combined with high sodium and chloride concentrations and increased bicarbonate excretion, often triggers hyperchloremic metabolic acidosis. In critically ill patients, this complication is linked to elevated rates of morbidity and mortality.[2,3]

In contrast, Plasmalyte A is a balanced, sterile, and isotonic crystalloid designed to closely mirror the electrolyte profile of human plasma Plasmalyte A contains metabolizable anions (acetate and gluconate), which are converted to bicarbonate equivalents and contribute to its alkalinising effect. Acetate is metabolized throughout the body independently of hepatic function. Research indicates that resuscitation with Plasmalyte A yields superior biochemical and hematologic outcomes, shorter hospital stays, and fewer complications. Additionally, Plasmalyte A can be safely co-administered with packed red blood cells. Including magnesium instead of calcium in balanced solutions helps mitigate compatibility issues, reinforcing their suitability for perioperative fluid therapy.[4]

In cases of perforation peritonitis, patients routinely present with hypovolemia driven by massive fluid shifts into the peritoneal cavity, gastrointestinal losses, reduced oral intake, and fever-induced insensible losses.[5,6] Despite its clinical relevance, the optimal choice of resuscitation fluid in such cases remains uncertain. Despite these severe volume deficits, the ideal resuscitation fluid for this condition is still debated. However, although balanced crystalloids are associated with lower chloride exposure and a reduced risk of hyperchloremic metabolic acidosis, evidence on their effects in patients with perforation peritonitis undergoing emergency laparotomy remains limited. These patients represent a distinct high-risk population with significant gastrointestinal losses, third-

spacing, systemic inflammation, and major emergency surgery. Therefore, this study aimed to compare the effects of Plasmalyte A and 0.9% saline on perioperative metabolic parameters, particularly serum electrolytes and pH, while also assessing their effects on perioperative haemodynamic changes.

MATERIAL AND METHODS

This prospective, randomized trial began after clearance from the institutional ethics committee and formal registration with the Clinical Trials Registry-India (CTRI/2022/07/044247). The study cohort comprised 50 adult patients of either gender aged 18 years or above presenting with peritonitis and planned for emergency exploratory laparotomy. Patients having pre-existing metabolic disorders, diabetic ketoacidosis, low GCS<14, and patients who recently encountered trauma were excluded from the study. The study adhered to Good Clinical Practice standards and the 2013 revision of the Declaration of Helsinki.

Upon arrival at the emergency department, all participants underwent a comprehensive medical history evaluation, along with thorough physical and systemic examinations. The study's objectives and methodology were clearly explained to the patients, and written informed consent was obtained. In the operation theatre, standard ASA monitoring, such as electrocardiogram (ECG), non invasive blood pressure (NIBP), and haemoglobin oxygen saturation (SPO_2), was applied, and baseline haemodynamic parameters were established. Baseline blood pressure (systolic blood pressure, diastolic blood pressure, and mean arterial pressure), heart rate, SPO_2, and arterial blood gas were recorded (T0).

Using a computer-generated randomization sequence with a 1:1 allocation ratio, participants were randomly assigned to Group A or Group B. Allocation concealment was maintained using sequentially numbered, opaque, sealed envelopes, opened only after enrolment. This resulted in 25 participants in each group. Group A (n = 25) received Plasmalyte A for fluid resuscitation, while Group B (n = 25) received 0.9% normal saline. The assigned crystalloid solution (Plasma-Lyte A or 0.9% normal saline) was initiated for fluid resuscitation in the preoperative room. An initial fluid volume of 30 mL/kg was administered within the first 3 hours, followed by additional 250 mL boluses as clinically indicated and guided by haemodynamic response and ongoing fluid requirements. During the first hour, approximately one-third of the initial 30 mL/kg volume was administered, with further fluid guided by the patient's haemodynamic response. One hour after initiation of fluid resuscitation, systolic blood pressure, diastolic blood pressure, mean arterial pressure, heart rate, SpO_2, and arterial blood gas parameters were reassessed (T1). All patients were managed with fluid resuscitation alone, and no vasopressor support was initiated preoperatively. Once mean arterial pressure exceeded 65 mm Hg, patients underwent emergency laparotomy; intravenous fluids were continued during the operative period as per the assigned group. Patients' urine output was monitored from the beginning of resuscitation till the end of the intraoperative period. General anaesthesia was administered as per the standard protocol. Induction of anesthesia was done with fentanyl 2 mcg/kg and propofol 2 mg/kg, and rapid sequence intubation was done using rocuronium as a neuromuscular blocker.

Maintenance of anaesthesia was achieved with a 50:50 mixture of oxygen and nitrous oxide, with sevoflurane titrated to maintain 1 MAC.

Intraoperative episodes of hypotension were managed with intermittent intravenous boluses of 3 mg ephedrine, while a noradrenaline infusion was initiated for persistent hypotension. At the completion of surgery, residual neuromuscular blockade was reversed with neostigmine and glycopyrrolate. The patients were transferred to the intensive care unit for further monitoring and management. Persistent postoperative hypotension was managed with a noradrenaline infusion.

Further hemodynamic monitoring (Heart rate, respiratory rate, SPO_2, mean arterial pressure, systolic blood pressure, and diastolic blood pressure), Blood gas analysis [BGA] monitoring, and Serum electrolytes (sodium, potassium, calcium, and chloride) were done preoperatively (T2), intraoperatively (at the time of incision) (T3), and postoperatively (T4).

We calculated the sample size based on a previous study[7], using a mean difference of 21 and a standard deviation (SD) of 25 with a 95% confidence interval, 80% power, and an alpha value of 0.05. We set the sample size at 25 per group.

We coded the data and entered them into a Microsoft Excel spreadsheet. Analysis was done using SPSS version 20 (IBM SPSS Statistics Inc., Chicago, Illinois, USA) Windows software program. Descriptive statistics included calculating percentages, means, and standard deviations. We checked data normality before statistical analysis using the Kolmogorov-Smirnov test. We applied the unpaired t-test (for quantitative data comparing two independent groups) and the paired t-test (for quantitative data comparing before-and-after observations). We used the chi-square test to compare all clinical indicators. We set the level of significance at $p \leq 0.05$.

RESULTS

Baseline demographic characteristics were well matched between the two cohorts, with no statistically significant disparities. We compared baseline demographic and clinical characteristics to confirm comparability between the two randomized groups and minimize the potential influence of baseline differences on study outcomes. Mean age was comparable across both groups: 38.56 ± 17.18 years for Group A and 39.80 ± 15.93 years for Group B (p = 0.792). Physical measurements were similarly aligned: Group A had a mean height of 169.04 ± 7.18 cm and a mean weight of 64.4 ± 9.6 kg, while Group B averaged 170.16 ± 6.55 cm in height and 64 ± 9.72 kg in weight (p = 0.567 and p = 0.884, respectively). Consequently, the mean Body Mass Index did not differ significantly between Group A (22.29 ± 2.42 kg/m²) and Group B (21.50 ± 3.49 kg/m²; p = 0.355). Additionally, the ASA physical status classifications were evenly distributed between the two groups (p = 0.44).

Preoperative duration was 3.47 ± 1.66 hours in Group A and 4.44 ± 2.39 hours in Group B, with no statistically significant difference between groups (p = 0.106). The mean duration of surgery was 2.32 ± 1.43 hours in Group A and 2.45 ± 1.84 hours in Group B, respectively (p = 0.624). Tables 1a, 1b, and Tables 2a, 2b present haemodynamic changes between the two groups. Arterial blood gas (ABG) analysis is summarized in Tables 3a, 3b, and 4a, 4b. Tables 5a and 5b present electrolyte changes observed across the groups. The total amount of fluid administered is shown in Table 6.

Table 1a: Comparison of MAP and HR changes in both groups

Time point	Group A		Group B		*p*-value
	MAP mmHg (mean±SD)	HR Beats/min (mean±SD)	MAP mmHg (mean±SD)	HR Beats/min (mean±SD)	
T0	84±15.51	109.16±15.43	84±7.68	110.68±16.462	1.0 (MAP) 0.74 (HR)
T1	86.68±10.69	100.64±14.60	86.4±7.86	105.40±16.062	
T2	82.79±10.55	107.76±15.39	79.40±7.20	118.20±14.212	
T3	83.64±9.53	96.76±14.44	73.21±6.95	114.68±14.86	
T4	87.16±9.79	108.76±9.571	84.50±5.79	107.04±9.258	

Significant

Table 1b: Comparison of P values of MAP and HR at different intraoperative time points

Group A		Group B	
p-value (MAP)	*p*-value (HR)	*p*-value (MAP)	*p*-value
T1 vs T2 p =0.02*	T0 vs T1 p <0.01*	T0 vs T1 p <0.01*	T0 vs T1 p <0.01*
T3 vs T4p= 0.03*	T1 vs T2 p = 0.009*	T1 vs T2 p <0.01*	T1 vs T2 p <0.01*
T1 vs T4 p = 0.15	T2 vs T3 p <0.01*	T2 vs T3 p <0.01*	T2 vs T3 p = 0.12
	T3 vs T4 p <0.01*	T3 vs T4 p <0.01*	T3 vs T4 p = 0.007*
	T1 vs T4 p = 0.90	T1 vs T4 p = 0.83	T1 vs T4 p = 0.22

**Significant*

Table 2a: Comparison of RR and SpO_2 changes in both groups

Time point	Group A		Group B		*p*-value
	RR (Breaths/min) (mean±SD)	SpO_2% (mean±SD)	RR (Breaths/min) (mean±SD)	SpO_2% (mean±SD)	RR/ SpO_2
T0	21.92±2.98	96.36±2.53	22.56±2.84	97.16±2.27	0.44(RR) 0.24 (SpO_2)
T1	20.76±2.93	98.40±1.19	21.80±4.06	98.64±1.15	
T2	20.36±3.03	98.52±1.19	22.04±3.16	98.80±1.04	
T3	15.08±1.29	99.44±0.50	15.96±2.72	98.96±0.35	
T4	20.52±2.14	98.28±1.27	21.20±2.29	97.16±2.26	

Significant

Table 2b: Comparison of P values of RR and SpO_2 at different intraoperative time points

Group A		Group B	
p-value (RR)	*p*-value (SpO_2%)	*p*-value (RR)	*p*-value (SpO_2%)
T0 vs T1 p= 0.008*	T0 vs T1 p<0.01*	T0 vs T1 p= 0.17	T0 vs T1 p= 0.01*
T2 vs T3 p<0.01*	T1 vs T2 p= 0.32	T1 vs T2 p= 0.76	T1 vs T2 p= 0.10
T3 vs T4 p<0.01*	T2 vs T3 p= 0.002*	T2 vs T3 p<0.01*	T2 vs T3 p= 0.07
T1 vs T4 p= 0.03*	T3 vs T4 p<0.01*	T3 vs T4 p<0.01*	T3 vs T4 p=0.03*
		T1 vs T4 p= 0.03*	T1 vs T4 p<0.01*

**Significant*

Table 3a: Comparison of P^H, PO_2 and PCO_2 in both groups

	Group A			Group B			*p*-value
	P^H (mean±SD)	PO_2(mmHg) (mean±SD)	PCO_2(mmHg) (mean±SD)	P^H (mean±SD)	PO_2(mmHg) (mean±SD)	PCO_2(mmHg) (mean±SD)	PH/PO_2/ PCO_2
T0	7.42±0.07	90.18±8.88	31.34±6.42	7.44±0.06	92.85±12.65	30.36±6.20	0.35 (PH) 0.39 (PO_2) 0.58 (PCO_2)
T1	7.42±0.05	109.28±17.39	32.01±5.64	7.40±0.054	114.21±15.85	31.70±5.64	
T2	7.42±0.05	111.60±19.66	31.82±6.35	7.40±0.070	119.00±16.40	41.01±4.94	
T3	7.36±0.03	313.93±89.55	37.75±5.84	7.29±0.045	347.93±103.72	39.10±5.16	
T4	7.41±0.05	104.67±22.59	35.85±17.75	7.36±0.033	129.00±18.26	32.73±5.20	

Significant

Table 3b: Comparison of P value of P^H, PO_2 and PCO_2 at different intraoperative time points

Group A			Group B		
p-value (P^H)	*p*-value (PO_2)	*p*-value (PCO_2)	*p*-value (P^H)	*p*-value (PO_2)	*p*-value (PCO_2)
T0 vs T1 p=0.73	T0 vs T1 p <0.01*	T0 vs T1 p= 0.28	T0 vs T1 p <0.01*	T0 vs T1 p= 0.01*	T0 vs T1 p=0.006*
T1 vs T2 p=0.61	T1 vs T2 p=0.55	T1 vs T2 p = 0.86	T1 vs T2 p= 0.60	T1 vs T2 p= 0.06	T1 vs T2 p <0.01*
T2 vs T3 p <0.01*	T2 vs T3 p <0.01*	T2 vs T3 p <0.01*	T2 vs T3 p <0.01*	T2 vs T3 p <0.01*	T2 vs T3 p <0.01*
T3 vs T4 p <0.01*	T3 vs T4 p <0.01*	T3 vs T4 p= 0.63	T3 vs T4 p <0.01*	T3 vs T4 p= 0.03*	T3 vs T4 p <0.01*
T1 vs T4 p=0.80	T1 vs T4 p=0.005*	T1 vs T4 p= 0.24	T1 vs T4 p <0.01*	T1 vs T4 p <0.01*	T1 vs T4 p=0.009*

**Significant*

Table 4a: Comparison of HCO_{3-} and Base Excess (BE)

Time point	Group A		Group B		*p*-value
	HCO_{3-} mmol/L (mean±SD)	BEmEq/L (mean±SD)	HCO_{3-} mmol/L (mean±SD)	BEmEq/L (mean±SD)	HCO_3/ BE
T0	20.04±2.83	-5.18±3.31	20.36±2.94	-5.10±2.28	0.689 (HCO_{3-}) 0.921(BE)
T1	20.45	-3.82±2.44	19.40±2.28	-4.22±2.04	
T2	20.63	-4.41± 2.65	19.40±2.36	-6.64±2.28	
T3	19.36	-4.27± 2.58	16.86±2.24	-7.99±2.66	
T4	20.46	-3.91± 2.49	19.90 ±2.38	-4.33±1.51	

Significant

Table 4b: Comparison of P values of HCO_{3-} and Base Exces (BE) at different intraoperative time points

Group A		Group B	
p-value (HCO_{3-})	*p*-value (BE)	*p*-value (HCO_{3-})	*p*-value (BE)
T0 vs T1 p >0.05	T0 vs T1 p <0.01*	T0 vs T1 p= 0.007	T0 vs T1 p= 0.01*
T1 vs T2 p >0.05	T1 vs T2 p >0.05#	T1 vs T2 p= 0.98	T3 vs T4 p= 0.007*
T2 vs T3 p >0.05	T2 vs T3 p >0.05#	T2 vs T3 p <0.01*	T1 vs T4 p =0.20
T3 vs T4 p >0.05	T3 vs T4 p >0.05#	T3 vs T4 p <0.01*	T3 vs T4 p =0.56
T1 vs T4 p >0.05	T1 vs T4 p >0.05#	T1 vs T4 p=0.67	T1 vs T4 p=0.34

**Significant; #non-significant*

Table 5a: Comparison of Serum Na^+, K^+, Ca^{++}, Cl^- levels in both groups

Time Point	Group A				Group B				*p*-value
	S Na^+ mEq/L (mean±SD)	S K^+ mEq/L (mean±SD)	S Ca^{++} mEq/L (mean±SD)	S Cl^- mEq/L (mean±SD)	S Na^+ mEq/L (mean±SD)	S K^+ mEq/L (mean±SD)	S Ca^{++} mEq/L (mean±SD)	S. Cl^- mEq/L (mean±SD)	
T0	135.16±4.03	3.72±0.58	1.11±0.09	106.66±3.42	132.07±3.55	3.94±0.62	1.11±0.09	103.22±3.02	0.06 (Na^+) 0.20 (K^+) 0.88 (Ca^{++}) <0.001 (Cl^-)*

(Continued)

Time Point	Group A				Group B			
	S Na^+ mEq/L (mean±SD)	S K^+ mEq/L (mean±SD)	S Ca^{++} mEq/L (mean±SD)	S Cl^- mEq/L (mean±SD)	S Na^+ mEq/L (mean±SD)	S K^+ mEq/L (mean±SD)	S Ca^{++} mEq/L (mean±SD)	S. Cl^- mEq/L (mean±SD)
T1	135.93±3.67	3.82 ± 0.61	1.14±0.06	106.51±2.18	134.95±3.32	3.84 ± 0.57	1.12±0.05	105.88±2.18
T2	137.02±3.87	3.75 ± 0.55	1.11±0.06	106.61±1.74	135.72±3.18	3.78 ± 0.54	1.11±0.09	105.91±2.01
T3	136.97±2.79	3.57 ± 0.52	1.00±0.60	106.55±1.98	142.66±4.49	3.84± 0.59	1.08±0.11	110.66±3.15
T4	137.49±2.50	3.83± 0.59	1.04±0.41	107.08±1.36	143.04±4.87	4.04 ± 0.63	1.14±0.07	110.38±2.92

**Significant*

Table 5b: Comparison of Serum Na^+, K^+, Ca^{++}, Cl - levels in both groups

Group A				Group B			
p-value (S Na^+)	*p*-value (S K^+)	*p*-value (S Ca^{++})	*p*-value (S Cl^-)	*p*-value (S Na^+)	*p*-value (S K^+)	*p*-value (S Ca^{++})	*p*-value (S. Cl^-)
T0 vs T1 p=0.13	T0 vs T1#	T0 vs T1 p= 0.07	T0 vs T1#	T0 vs T1 p<0.00*	T0 vs T1 p=0.00*	T0 vs T1 p= 0.38	T0 vs T1 p<0.00*
T1 vs T2 p=0.03*	T1 vs T2#	T1 vs T2 p =0.03	T1 vs T2#	T1 vs T2 p= 0.16	T1 vs T2 NS#	T1 vs T2 p=0.59	T1 vs T2 p<0.00*
T2 vs T3 p<0.00*	T2 vs T3#	T2 vs T3 p =0.34	T2 vs T3#	T2 vs T3 p<0.00*	T2 vs T3 NS#	T2 vs T3 p=0.41	T2 vs T3 p<0.00*
T3 vs T4 p =0.32	T3 vs T4#	T3 vs T4 p =0.78	T3 vs T4#	T3 vs T4 p =0.71	T3 vs T4 NS#	T1 vs T4 p=0.13	T3 vs T4 p<0.00*
T1 vs T4 p=0.00*	T1 vs T4#	T1 vs T4 p =0.37	T1 vs T4#	T1 vs T4 p<0.00*	T1 vs T4 NS#		T1 vs T4 p<0.00*

**Significant; #NS: non-significant (p>0.05)*

Table 6: Comparison of preoperative and intraoperative fluid administration between groups

	Group A (mL) (mean±SD)	Group B (mL) (mean±SD)	*p*-value
Preop resuscitation (3 hrs)	2050±29	2180±291.60	0.119
Intraoperative fluid replacement	1494±133	1568±14	0.051

Urine output from the resuscitation period to the end of surgery was significantly higher in Group A than in Group B (506±150.96 ml vs 411±101.84 ml; p = 0.012). In the present study, 4 patients in Group B (normal saline) required noradrenaline support,whereas none in Group A (Plasmalyte A) required vasopressor support. This difference was statistically significant (p=0.038). The mean intraoperative blood loss in Group A was 385 ± 121.5 ml, compared to 432 ± 149 ml in Group B; however, this difference was not statistically significant (p = 0.266).

DISCUSSION

In the present study, arterial blood gas (ABG) analyses showed that the Plasmalyte A group maintained better acid-base balance, with minimal pH fluctuations during the perioperative phase. Conversely, the normal saline (NS) group experienced a marked drop in pH from the preoperative to the intraoperative period. Although their pH levels partially recovered postoperatively, they failed to return to baseline or preoperative values (p<0.001). These findings align with existing literature[8-12], which shows that administering normal saline in various clinical settings is associated with a decline in pH, commonly attributed to hyperchloremic metabolic acidosis. The chloride load is dose-dependent, with larger volumes of NS delivering greater amounts of chloride. Excess chloride increases serum chloride, reduces the strong ion difference, and consequently lowers bicarbonate and pH, resulting in hyperchloremic metabolic acidosis. However, acidosis depends not only on fluid volume but also on fluid composition. In our study, Group B received a slightly greater total fluid volume than Group A (3748 ± 423 mL vs. 3544 ± 415 mL); therefore, the greater acidosis in the NS group more likely reflects NS's higher chloride content than fluid volume alone. Additionally, prior studies indicate that patients receiving NS often require higher doses of vasopressors for circulatory support compared to those given balanced crystalloids. This elevated need for vasoactive medications, coupled with the administration of large fluid volumes, likely worsens the acidotic state in NS-treated patients.[13]

Because of its buffered, alkalinizing composition, Plasmalyte A promotes greater acid-base homeostasis and serves as a safer alternative to unbalanced crystalloids in patients with pre-existing or developing acidosis.[14] Conversely, Plasmalyte A achieves a physiological pH of approximately 7.4 by incorporating acetate and gluconate buffers. It also substitutes calcium with magnesium; this lack of calcium ensures safety during concurrent red blood cell transfusions. Additionally, its balanced formulation reduces the risk of interstitial edema compared with traditional isotonic saline.[17,15]

In the present study, patients administered normal saline exhibited a marked elevation in the partial pressure of carbon dioxide (PCO_2) during both the intraoperative and postoperative phases. Conversely, the patient receiving Plasmalyte A (Group A) experienced a significantly milder increase in PCO_2. This smaller increase in Group A may be attributed to better haemodynamic stability in the Plasmalyte A group, which facilitates optimal tissue perfusion and enhances systemic carbon dioxide clearance. As ventilation was standardised across groups, the observed difference in $PaCO_2$ was unlikely to be attributable to differences in ventilatory parameters. Although saline-induced metabolic acidosis may have contributed to the altered acid–base status, the increase in $PaCO_2$ should not be attributed solely to metabolic acidosis.

Bicarbonate concentrations remained largely consistent among patients administered Plasmalyte A in our study. In contrast, the NS group experienced a substantial reduction in bicarbonate throughout the perioperative phase, with the most severe decline noted intraoperatively ($p<0.001$). These results align with prior research demonstrating that NS infusions significantly lower serum bicarbonate.[8,11,12,16] The primary mechanism driving this phenomenon is the elevation of plasma chloride, which forces a compensatory reduction in bicarbonate to preserve electrochemical neutrality, thereby inducing iatrogenic hyperchloremic metabolic acidosis. In contrast, balanced solutions like Plasmalyte A feature physiological chloride levels alongside metabolizable organic anions (acetate and gluconate) that generate bicarbonate in vivo. This unique formulation yields a neutral to slightly alkalinizing impact on the body's acid-base status, explaining the divergent pH and bicarbonate outcomes observed between the two fluid regimens.[8,11,12,16] Furthermore, while both groups exhibited a perioperative reduction in base excess, the decline was distinctly more profound in the NS group. This exaggerated drop likely reflects the cumulative acidifying effects of 0.9% saline and the larger fluid volumes administered, findings that closely echo existing literature. The greater decline in bicarbonate and base excess in the NS group may reflect the combined effects of fluid volume and composition, as Group B (NS) received a slightly greater total volume than Group A (Plasma-Lyte A) (3748 ± 423 vs. 3544 ± 415 mL). However, the relatively modest volume difference, together with NS's higher chloride content, suggests that chloride load and fluid composition likely contributed substantially to the observed hyperchloremic metabolic acidosis.[8,10,12]

Electrolyte analysis revealed that patients receiving NS experienced a sustained rise in serum sodium relative to their baseline measurements. This abrupt sodium elevation reflects the high sodium load of 0.9% saline (154 mEq/L) and aligns with earlier investigations.[7,8,16,17] The NS group also exhibited both hyperkalemia and hypercalcemia. These ionic disturbances likely result from metabolic acidosis, where sudden increases in hydrogen ion concentrations prompt transcellular movement of intracellular potassium and calcium into the extracellular fluid compartment.

Regarding serum chloride, concentration remained relatively stable in the Plasmalyte A group, showing only a slight postoperative rise. Meanwhile, the NS group displayed a consistent upward trajectory in chloride levels at all measured intervals, though these variations did not achieve statistical significance. This tendency toward hyperchloremia directly mirrors the excessive chloride burden of NS (154 mEq/L) versus the more physiological chloride content of Plasmalyte A (98 mEq/L). These results corroborate established literature linking normal saline infusions to subsequent hyperchloremia.[8,10–12,16,17] Prior comparative studies have consistently linked NS administration to hyperchloremic acidosis. The observed changes in MAP should be interpreted cautiously, as haemodynamics in patients with perforation peritonitis are influenced by multiple factors, including hypovolaemia, systemic inflammation, fluid shifts, blood loss, and vasopressor use. Although differences in chloride exposure may contribute to haemodynamic effects, a direct causal relationship between serum chloride and MAP cannot be established from our study. Elevated plasma chloride concentrations depress both the cardiac index and the left ventricular

stroke work index, thereby lowering overall MAP.[9]

Vasopressor requirements were notably elevated in Group B (NS), whereas no patients in Group A (Plasmalyte A) needed vasopressor assistance. This difference may reflect several factors, including preoperative clinical condition, total fluid volume administered, and fluid composition. Group B received a slightly greater total fluid volume (3748 ± 423 vs. 3544 ± 415 mL), while the higher chloride load associated with NS may have contributed to hyperchloremic metabolic acidosis and haemodynamic instability. Therefore, the increased vasopressor requirement in the NS group should be interpreted as multifactorial rather than solely attributable to the type of intravenous fluid. Evidence indicates that these pH imbalances provoke a widespread release of pro- and anti-inflammatory mediators—namely interleukin-6 (IL-6), interleukin-10 (IL-10), and tumor necrosis factor-alpha (TNF-α)—which can significantly increase the risk of septic shock.[18,19]

In the present study, hemodynamically assessment was done in both groups. Notably, patients administered normal saline exhibited a substantial intraoperative decline in MAP, dropping from a preoperative baseline of 79.38 ± 7.35 mmHg down to 73.21 ± 6.95 mmHg. Conversely, those resuscitated with Plasmalyte A demonstrated superior hemodynamic stability, maintaining a much more consistent MAP throughout the surgical intervention. The observed MAP changes should be interpreted cautiously, as haemodynamics in perforation peritonitis are multifactorial and may be influenced by hypovolaemia, systemic inflammation, blood loss, anaesthetic effects, and fluid shifts, rather than fluid type alone.

Preoperative duration was comparable between the Plasmalyte A and normal saline groups ($p = 0.106$). Urine output was significantly higher in the Plasma-Lyte group compared with the normal saline group (506 ± 150.96 mL vs. 411 ± 101.84 mL; $p = 0.012$), despite comparable preoperative duration between the groups ($p = 0.106$). This finding may suggest better preservation of renal perfusion with Plasma-Lyte; however, urine output is influenced by several factors, including preoperative hydration status, haemodynamic condition, vasopressor use, and total fluid administration, and therefore should not be attributed solely to fluid composition. The normal saline group had a significantly higher requirement for vasopressor support (16% vs. 0%; $p = 0.038$). This finding may be associated with the greater acid–base disturbances observed with normal saline; however, the underlying mechanism cannot be established from our study, as inflammatory mediator levels were not assessed."[19]

CONCLUSION

We conclude that Plasmalyte A was associated with better preservation of perioperative metabolic and haemodynamic stability compared with normal saline in patients undergoing laparotomy. Plasmalyte A was associated with less disturbance in pH and electrolyte balance, along with a lower requirement for inotropic support. Although the NS group received a slightly greater total fluid volume, the observed differences may reflect the combined effects of fluid composition, volume administered, and preoperative patient condition. Therefore, Plasmalyte may be considered a suitable balanced crystalloid for patients requiring perioperative fluid resuscitation, although these findings should not be interpreted as evidence that fluid composition alone accounts for the observed outcomes.

LIMITATIONS

This study is limited by its small sample size and single-center design, which may affect the generalizability of the findings. Additionally, potential confounding factors could have influenced the observed metabolic abnormalities. To robustly validate these results, a large-scale, randomized, multicenter controlled trial is warranted to further evaluate metabolic changes associated with fluid resuscitation in patients with peritonitis.

AUTHORS' CONTRIBUTIONS

Conceptualization and design: PK, R, SM

Literature search: PK, R, SM, KK

Data collection: PK, R, SV, A

Data analysis: PK, R, KK, SV, A

Manuscript drafting and critical revision: PK, R, KK

Proofreading and approval of the manuscript: PK, KK

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DECLARATION OF CONFLICTS OF INTEREST

There are no conflicts of interest to declare.

ETHICAL CLEARANCE

Approved by the Biomedical Research Ethics Committee (BREC), PT BD Sharma Postgraduate Institute of Medical Sciences, UHS, Rohtak, India (No: EC/NEW/INST/2020/874); Approval date: April 2, 2021.

The study was registered with the Clinical Trials Registry – India.

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