DOI: https://doi.org/10.53350/pjmhs221621082

ORIGINAL ARTICLE

Comparison of Ringer's Lactate Versus 0.9% Normal Saline Solution in the Management of Diabetic Ketoacidosis

JAHAN SARDAR[1], ABDUL BAQI[2], MUHAMMAD NADEEM[3], MEDRARULLAH KHAN[4], KALIM ULLAH KHAN[5], SOBIYA MOHIUDDIN OMAR[6], NAHEED MUMTAZ[7]

[1]*Assistant Professor Medicine, Kuwait Teaching Hospital, Peshawar Medical College, Peshawar*
[2]*Resident Internal Medicine, Mercy Saint Vincent's Medical Center, Toledo, Ohio, 43606*
[3]*Assistant Professor, General Medicine, Department of Medicine, Lady Reading Hospital, Peshawar*
[4]*Medical officer, RHC Putwar Peshawar*
[5]*Assistant professor Medicine Department NMC/QHAMC Nowshera*
[6]*Department of Microbiology, Jinnah University for Women, Karachi*
[7]*Department of Eastern Medicine, Faculty of Medical and health sciences, University of Poonch Rawlakot Azad Kashmir*
Corresponding author: Kalim ullah khan, Email: kalim83@yahoo.com

ABSTRACT

Background: Diabetic ketoacidosis (DKA) is a life-threatening diabetic condition characterized by metabolic and homeostatic disturbances. KDA is a common condition in people with diabetes, especially in children and adolescents

Objective: To compare ringer's lactate versus 0.9% normal saline solution in the management of diabetic ketoacidosis

Methodology: The study design was randomized control trial carried out at the Medical 'A' ward Khyber teaching hospital (KTH) Peshawar for a period of six months after synopsis approval. Patients were divided into two groups, groups A, receive 0.9% normal saline infusion and group B receive ringers lactate infusion. 82 subjects were included in each group. SPSS software version 20 was used for analyzing data.

Results: In group B subjects, the serum bicarbonates level was higher than group A subjects (p=0.091). The Anion gap and blood PH level increases non-significantly in both the groups over a period of time (p=0.061). The mean hospital stay in group A and group B was 13.11 hours and 11.5 hours respectively (p=0.0031). The fluid quantity required in group A was more than group B (p=0.0031)

Conclusion: When prescribing fluid, clinicians should always be cautious since any fluid may be dangerous if dosed wrongly. The ringer's lactate was shown to be superior to 0.9 % normal saline in terms of alleviating DKA quicker in our study.

Keywords: Diabetic ketoacidosis; 0.9 % normal saline; ringer's lactate

INTRODUCTION

Diabetic ketoacidosis (DKA) is a life-threatening diabetic condition characterized by metabolic and homeostatic disturbances. KDA is a common condition in people with diabetes, especially in children and adolescents. A DKA affects 15 to 20% of persons with type 1 diabetes [1-3]. DKA is claimed to be the cause of more than 100,000 hospital admissions annually in the United States, and it accounts for 4–9% of all diagnoses given to diabetic patients upon discharge from hospitals [4]. According to the EURODIAB research, 8.6% of 3250 diabetic individuals were admitted to the hospital for DKA in the previous studies [5]. There were 12.9 cases per 100,000 people annually in a Danish research, and the fatality rate was 4.3% [6]. The mortality rate correlated with DKA is less than 5%, provided that standardized written recommendations are followed [7, 8]. With a fatality rate of 26–29 percent in Africa, the mortality rate is unacceptably high [9]. DKA is more common in type 1 diabetics, although it may also happen in type 2 diabetics at times of acute stress, like trauma or infections [10]. There are many ways to mange DKA currently. These include replacing lost fluid, correcting hyperglycemia with insulin, correcting electrolyte losses, figuring out what caused DKA, and giving insulin to keep DKA from occurring again [11]. In DKA, normal saline (0.9% NaCl) has typically been used as a replacement fluid, and this is reflected in current recommendations [11-13]. The injection of significant amounts of saltwater (0.9% NaCl) may, however, contribute to the development of metabolic acidosis, according to current research findings [14]. In saline, the excessive Chloride ion administration is the primary cause of the acidifying action. Endotoxemia and surgical patients are both known to have hyperchloraemic metabolic acidosis [15-17]. Hyperchloraemia is more common in DKA patients as therapy progresses, with the most rapid development occurring during the period when fluid (saline) is administered at the fastest rate. The acidifying impact of chloride masks the cure of ketoacidosis, with ketones being the predominant source to acidosis initially and chloride later in the therapy of DKA [18].

Locally no data available in studies regarding the comparison of Ringer lactate and 0.9% Normal saline in the acute resuscitation of diabetic ketoacidosis. The goal of our research is to see whether Ringer's lactate solution is better than normal saline infusion as a main resuscitation fluid in diabetic ketoacidosis patients in terms of acidosis resolution time.

MATERIALS AND METHODS

The study design was randomized control trial carried out at the Medical 'A' ward Khyber teaching hospital (KTH) Peshawar for a period of six months after synopsis approval. Sample Size was164, calculated by using software stata 12.0. The inclusion criteria for our study was patients of both the sex and age 20-60 years having plasma glucose level less than 13.9 mmol/L, arterial pH of less than 7.3, serum bicarbonate of less than 15 mmol/L whereas the exclusion criteria was patients having biochemical abnormalities like sodium level of more than 150 mmol/L Potassium more than 6.2 mmol/L, chloride more than 113 mmol/L, patients with multiple co-morbidities e.g. stroke and ischemic heart diseases and extremely critical ill state. Informed consent was signed from all the study subjects. Patients were divided into two groups A, receive 0.9% normal saline infusion and B receive ringers lactate infusion. 82 subjects were included in both the groups. Detailed history and clinical examination and routine examination were done for all the subjects. Group A patients receive 0.9 % normal saline up to 4-6 L/day while group B patients receive Ringer lactate in the same dose. The patient's blood sugar was checked every hour, and their serum electrolytes and anion gap were checked every two hours. DKA was deemed resolved in this research when serum bicarbonate was more than 18 mEq/l. Blood sugar and electrolyte levels were measured at the conclusion. SPSS software version 20 was used for analyzing data. Mean (standard deviation) were computed for numerical variables whereas frequency (percentage) were computed in both groups for categorical variables. For comparison of both the group patients, Chi-square test was employed by taking p value of less than 0.05 as significant.

RESULTS

In group A, the number of patients were 20 (24.29%), 35 (42.68%), 20 (24.29%) and 7 (8.53%) in age group 20-30 years, 31-40 years, 41-50 years and 51-60 years respectively while in group B, the number of patients were 18 (21.96%), 34 (41.46%), 23 (28.05%) and 77 (8.53%) in age group 20-30 years, 31-40 years, 41-50

years and 51-60 years respectively. (Figure 1) The number of males and females in group A were 50 (60.98%) and 32 (39.02%) respectively while in group B, males were 52 (63.41%) and females were 30 (36.59%). (Figure 2) The baseline serum electrolytes of both the groups are given in Table 1. By comparing both the groups, a statistically significant increase was observed in serum sodium and chloride in group A subjects (p=0.003). The mean level of chloride in group A subjects after 8 hours was higher (115meq/l) than group A subjects. The level of serum potassium in group B subjects increased significantly as compared to group A subjects (p=0.001). In group B subjects, the serum bicarbonates level was higher than group A subjects (p=0.091). The Anion gap and blood PH level increases non-significantly in both the groups over a period of time (p=0.061). (Table 1) The mean hospital stay in group A and group B was 13.11 hours and 11.5 hours respectively (p=0.0031). (Figure 3) The fluid quantity required in group A was more than group B (p=0.0031). (Figure 4)

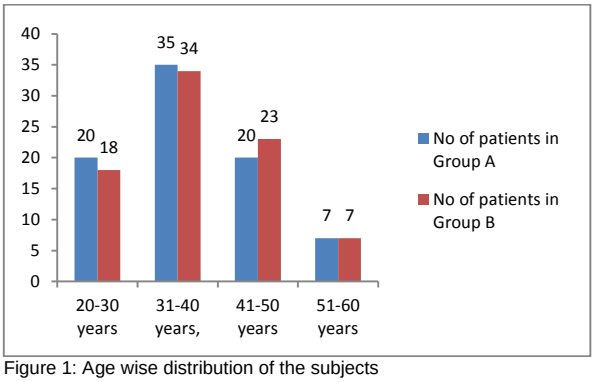

Figure 1: Age wise distribution of the subjects

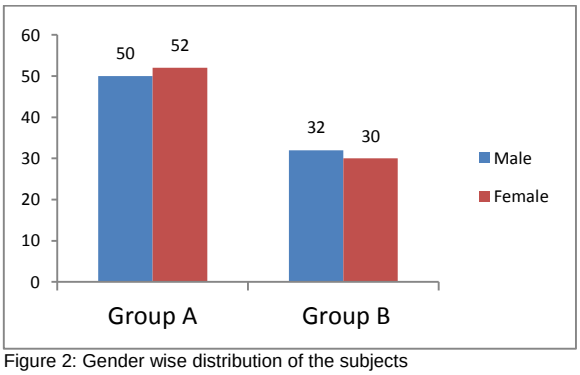

Figure 2: Gender wise distribution of the subjects

Table 1: Baseline and after 8 hours serum electrolyte, anion gap and blood ph of both the group

Parameter	Sub-category Mean (SD)	Group A Mean (SD)	Group B Mean (SD)	P value
Serum sodium	Baseline	135.11 (2.23)	136.01 (1.99)	0.003
	After 8 hours	139.22 (4.16)	138.05 (3.27)	
Serum chloride	Baseline	103.5 (0.75)	104 (0.22)	0.003
	After 8 hours	114 (2)	107 (1.21)	
Serum potassium	Baseline	5.76 (0.21)	4.56 (0.33)	0.001
	After 8 hours	4.60 (0.12)	5.20 (0.55)	
Serum bicarbonate	Baseline	10.36 (1.55)	10.95 (2.23)	0.091
	After 8 hours	16.11 (1.24)	17.33 (2.01)	
Anion gap	Baseline	9.95 (0.66)	10.01 (0.99)	0.061
	After 8 hours	12.55 (0.75)	12.23 (0.86)	
Blood ph	Baseline	7.12 (0.01)	7.12 (0.01)	0.061
	After 8 hours	7.28 (0.09)	7.29 (0.08)	

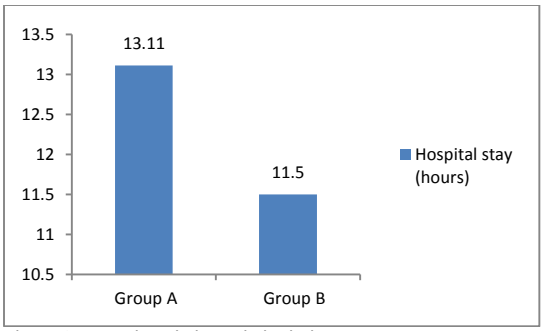

Figure 3: Mean hospital stay in both the groups

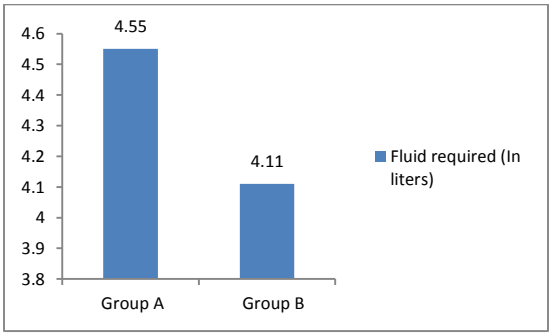

Figure 4: Fluid required in both the groups

DISCUSSION

Correction of the acidotic condition with intravenous fluids is the first and most important step in the treatment of diabetic ketoacidosis. Crystalloids are chosen over colloids as intravenous fluids because of their cost effectiveness. The many crystalloids available each have their own set of benefits and drawbacks. When normal saline is injected, it quickly distributes amongst the extracellular compartments and persists in the body for a long period. While the majority of the administered volume is located in the interstitial region, intravascular volume increase may last up to 6 hours in normovolaemic individuals following normal saline infusion [19, 20]. Normal saline has a greater chloride content, therefore giving it intravenously raises the level of chloride in the blood, which causes renal vasoconstriction and lower GFR (glomerular filtration rate) [16]Hyperchloremic acidosis occurred in 10 of the 82 individuals in our research who received normal saline and was afterwards rectified in all of the patients. Lactated Ringer's solution, for example, is neither isotonic nor exactly balanced, despite the fact that it is routinely employed. Some investigations found that infusing lactated Ringer's solution with an osmolarity of 273 mOsmol/L and a calculated osmolality of 254 mOsmol/kg resulted in a slight reduction in plasma osmolality [17, 18]. There is a frequent misconception that ringer lactate is safe for all patients since it is so near to serum. However, lactic acidosis may occur if a patient has liver disease and the liver is unable to break down the lactate. Furthermore, if the patient's serum pH is higher than 7.5, bicarbonate will develop when lactate breaks down, resulting in alkalosis. Because excessive amounts of saline might produce hyperchloremic acidosis, the American College of Surgeons' Committee on Trauma advises lactated Ringers. Large quantities of RL were shown to contribute to cerebral edema in the same study [21]. In our study, by comparing both the groups, a statistically significant increase was observed in serum sodium and chloride in group A subjects (p=0.003). The mean level of chloride in group A subjects after 8 hours was higher (115meq/l) than group A subjects. The level of serum potassium in group B subjects increased significantly as compared to group A subjects (p=0.001). In group B subjects, the serum bicarbonates level was higher than group A subjects (p=0.091). The Anion gap and blood PH level

increases non-significantly in both the groups over a period of time (p=0.061). The mean hospital stay in group A and group B was 13.11 hours and 11.5 hours respectively (p=0.0031). The fluid quantity required in group A was more than group B (p=0.0031). In accordance with our study, another study reported comparable results [22]. In a study carried out by Van Zyl D G et al. on diabetic ketoacidosis fluid management showed that the median time to achieve 7.32 ph, 0.9% normal saline take 683 minutes and Ringer's lactate group take 540 minutes [23] however in our study to reach a pH of 7.32 it takes 600 minutes for patients who took ringer lactate and 730 minutes for patients who took 0.9% NS, hence the findings were extremely similar to the studies done before.

CONCLUSIONS

When prescribing fluid, clinicians should always be cautious since any fluid may be dangerous if dosed wrongly. Although the differences in immediate effectiveness between crystalloid and colloid solutions are minor, the differences in long-term safety seem to be more important. However, because of the expense, crystalloids now play a significant part in fluid management. Patients with diabetic ketoacidosis, which is a life-threatening emergency, should be treated as soon as possible with a better intravenous fluid that will relieve the situation as quickly as possible. The crystalloid was shown to be superior to 0.9 % normal saline in terms of alleviating DKA quicker in our study. However, further randomized controlled studies should be undertaken to generalize and highlight which crystalloid is more effective in the treatment of DKA.

REFERENCES

1. FAICH GA, FISHBEIN HA, ELLIS SE. The epidemiology of diabetic acidosis: a population-based study. Am J Epidemiol. 1983;117(5):551-8.
2. Umpierrez GE, Kelly JP, Navarrete JE, Casals MM, Kitabchi AE. Hyperglycemic crises in urban blacks. Arch Intern Med. 1997;157(6):669-75.
3. Mudly S, Rambiritch V, Mayet L. An identification of the risk factors implicated in diabetic ketoacidosis (DKA) in type 1 and type 2 diabetes mellitus. South African Family Practice. 2007;49(10):1.
4. Palumbo P, Melton III LJ. Peripheral vascular disease. Diabetes in America. 1995:401.
5. Ellemann K, Soerensen JN, Pedersen L, Edsberg B, Andersen OO. Epidemiology and treatment of diabetic ketoacidosis in a community population. Diabetes Care. 1984;7(6):528-32.
6. Henriksen OM, Røder ME, Prahl JB, Svendsen OL. Diabetic ketoacidosis in Denmark: incidence and mortality estimated from public health registries. Diabetes Res Clin Pract. 2007;76(1):51-6.
7. Kitabchi AE, Umpierrez GE, Murphy MB, Barrett EJ, Kreisberg RA, Malone JI, et al. Management of hyperglycemic crises in patients with diabetes. Diabetes Care. 2001;24(1):131-53.
8. Wagner A, Risse A, Brill H-L, Wienhausen-Wilke V, Rottmann M, Sondern K, et al. Therapy of severe diabetic ketoacidosis. Zero-mortality under very-low-dose insulin application. Diabetes Care. 1999;22(5):674-7.
9. Otieno C, Kayima J, Omonge EO, Oyoo GO. Diabetic ketoacidosis: risk factors, mechanisms and management strategies in sub-Saharan Africa: a review. East Afr Med J. 2005;82(12).
10. Rheeder P, Stolk R, Grobbee D. Ethnic differences in C-peptide levels and anti-GAD antibodies in South African patients with diabetic ketoacidosis. QJM. 2001;94(1):39-43.
11. Kitabchi AE, Umpierrez GE, Miles JM, Fisher JN. Hyperglycemic crises in adult patients with diabetes. Diabetes Care. 2009;32(7):1335-43.
12. Wallace T, Matthews D. Recent advances in the monitoring and management of diabetic ketoacidosis. QJM. 2004;97(12):773-80.
13. Eledrisi MS, Alshanti MS, Shah MF, Brolosy B, Jaha N. Overview of the diagnosis and management of diabetic ketoacidosis. The American journal of the medical sciences. 2006;331(5):243-51.
14. Morgan TJ, Venkatesh B, Hall J. Crystalloid strong ion difference determines metabolic acid-base change during in vitro hemodilution. Crit Care Med. 2002;30(1):157-60.
15. Kellum JA, Bellomo R, Kramer DJ, Pinsky MR. Etiology of metabolic acidosis during saline resuscitation in endotoxemia. Shock (Augusta, Ga). 1998;9(5):364-8.
16. Prough DS, Bidani A. Hyperchloremic metabolic acidosis is a predictable consequence of intraoperative infusion of 0.9% saline. The Journal of the American Society of Anesthesiologists. 1999;90(5):1247-9.
17. Waters JH, Miller LR, Clack S, Kim JV. Cause of metabolic acidosis in prolonged surgery. Crit Care Med. 1999;27(10):2142-6.
18. Taylor D, Durward A, Tibby SM, Thorburn K, Holton F, Johnstone IC, et al. The influence of hyperchloraemia on acid base interpretation in diabetic ketoacidosis. Intensive Care Med. 2006;32(2):295-301.
19. Stoneham M, Hill E. Variability in post-operative fluid and electrolyte prescription. The British journal of clinical practice. 1997;51(2):82-4.
20. Greenfield RH, Bessen HA, Henneman PL. Effect of crystalloid infusion on hematocrit and intravascular volume in healthy, nonbleeding subjects. Ann Emerg Med. 1989;18(1):51-5.
21. McFarlane C, Lee A. A comparison of Plasmalyte 148 and 0.9% saline for intra-operative fluid replacement. Anaesthesia. 1994;49(9):779-81.
22. Self WH, Evans CS, Jenkins CA, Brown RM, Casey JD, Collins SP, et al. Clinical effects of balanced crystalloids vs saline in adults with diabetic ketoacidosis: a subgroup analysis of cluster randomized clinical trials. JAMA network open. 2020;3(11):e2024596-e.
23. Van Zyl DG, Rheeder P, Delport E. Fluid management in diabetic-acidosis—Ringer's lactate versus normal saline: a randomized controlled trial. QJM: An International Journal of Medicine. 2012;105(4):337-43.