Intravenous Sodium Bicarbonate Therapy in Severely Acidotic Diabetic Ketoacidosis

Bryson Duhon, Rebecca L Attridge, A Crystal Franco-Martinez, Pamela R Maxwell, Darrel W Hughes

BACKGROUND: The use of intravenous bicarbonate in diabetic ketoacidosis (DKA) may be considered for patients with a pH less than 6.9 according to the American Diabetes Association. The impact of this therapy on resolution of acidosis in patients with DKA is unclear.

OBJECTIVE: To determine whether the use of intravenous bicarbonate therapy was associated with improved outcomes in patients with severe DKA who were seen in the emergency department.

METHODS: This review was conducted from 2007 to 2011 in the emergency department of a tertiary teaching hospital. Adults diagnosed with DKA with an initial pH less than 7.0 were included. Patients were stratified into 2 groups based on receipt of intravenous bicarbonate. The primary study outcome was time to resolution of acidosis, defined as return to pH greater than 7.2. Secondary outcomes included length of stay; continuous infusion insulin use; and intravenous fluid, potassium, and insulin requirements within the first 24 hours of hospital admission, beginning upon admittance to the emergency department. We also conducted a subgroup analysis of patients with an initial pH less than 6.9.

RESULTS: There was no significant difference in time to resolution of acidosis (8 hours vs 8 hours; p = 0.7) or time to hospital discharge (68 hours vs 61 hours; p = 0.3) between patients who received intravenous bicarbonate (n = 44) compared with those who did not (n = 42). The median dose of intravenous bicarbonate was 100 mEq (100-150) for patients who received intravenous bicarbonate. Insulin and fluid requirements in the first 24 hours were significantly higher in patients who received intravenous bicarbonate compared with those who did not (100 units vs 86 units; p = 0.04 and 7.6 L vs 7.2 L; p = 0.01, respectively). There was no significant difference in hours of continuous insulin infusion (27 hours vs 26 hours; p = 0.09) or potassium requirements in the first 24 hours of hospital stay (135 mEq vs 120 mEq; p = 0.84).

CONCLUSIONS: Intravenous bicarbonate therapy did not decrease time to resolution of acidosis or time to hospital discharge for patients with DKA with an initial pH less than 7.0.

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In the US, diabetic ketoacidosis (DKA) is responsible for 500,000 hospital days per year, resulting in an estimated $2.4 billion of medical expense and indirect cost.[1] Severe DKA contributes a significant burden to health systems, despite the fact that it is associated with a low mortality rate (<5%). DKA occurs as a result of insulin deficiency combined with increased counterregulatory hormone production. This leads to mental status changes, severe dehydration, and cardiovascular instability.[2] While fluid resuscitation and insulin therapy help to normalize the body's use of glucose and shut down excess free fatty acid generation and subsequent ketoacids, additional strategies aimed at correcting the severe acidosis associated with DKA are limited.[3]

Intravenous bicarbonate is often used by practitioners for a perceived quicker resolution of acidemia in DKA. Current

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guidelines from the American Diabetes Association (ADA) recommend intravenous bicarbonate by slow infusion in patients with a pH less than 6.9.[4] Previous recommendations advised the use of intravenous bicarbonate in patients with a pH less than 7.0.[3] The recently lowered pH threshold for the use of intravenous bicarbonate reflects the lack of evidence for its use in patients with a pH less than 7.0. To date, peer-reviewed literature lacks an investigation of intravenous bicarbonate use in adults with DKA using a strict inclusion criterion of pH less than 7.0.[2] Available data are focused mainly on patients with an initial pH greater than 7.0 and have shown varying results on time to resolution of acidosis and hospital length of stay. Two previous investigations reported an improvement in acidosis.[5,6] However, these trials also reported a worsening of ketonemia in addition to a lack of sustained benefit past 2 hours of therapy. Apart from a short-term improvement in acidosis reported in the aforementioned investigations, the sustained benefit and impact of intravenous bicarbonate has not been demonstrated.

Our objective was to determine whether the use of intravenous bicarbonate therapy was associated with improved outcomes in patients with severe DKA, with an initial pH less than 7.0, seen in the emergency department. We compared patients who were given intravenous bicarbonate with those who were not. Our primary outcome was time to resolution of acidosis. Secondary outcomes included hospital length of stay; time on continuous infusion insulin; and intravenous fluid, potassium, and insulin requirements in the first 24 hours of hospital stay, beginning upon admission to the emergency department.

Methods

DESIGN

This was a retrospective cohort study performed at University Hospital, University Health System, San Antonio, TX. We compared the use of intravenous bicarbonate versus no intravenous bicarbonate on time to resolution of acidosis (pH >7.2) in patients with severe DKA who presented to the emergency department with initial pH less than 7.0 (venous or arterial blood gas). The study was approved by the institutional review board at The University of Texas Health Science Center at San Antonio as well as the hospital's research department. Given the retrospective design of the study, the need for informed consent was waived by the institutional review board.

STUDY SETTING AND POPULATION

The study was conducted in an urban emergency department with approximately 75,000 adult visits annually. The center serves a predominately Hispanic and white population and is staffed by board-certified emergency medicine faculty.

Adults (age ≥18 years) with severe DKA who presented to the emergency department between January 2007 and July 2011 were eligible for inclusion. The study initiation date of January 2007 correlates to the availability of electronic medical records in the emergency department of our facility. We identified patients using the *International Classification of Diseases, Ninth Revision, Clinical Modification* (ICD-9-CM) code 250.10 related to DKA. Further inclusion eligibility was determined by reviewing laboratory data, physical examination, and vital signs documented in patients' medical records. Severe DKA was defined per ADA evidence-based guidelines as plasma glucose greater than 250 mg/dL, pH less than 7.0, serum bicarbonate less than 10 mEq/L, and positive serum ketones.[3] Patients were excluded if they were pregnant, diagnosed with DKA more than 24 hours after admission, had an out-of-hospital cardiac arrest or trauma, or if initial pH was greater than 7.0. Incarcerated patients were also excluded from analysis; this population was expected to be small and did not warrant the necessary additional review board requirements.

Data collected from medical records included patient demographics, baseline laboratory values, baseline vital signs, outpatient insulin regimens, intravenous bicarbonate dosage, continuous infusion insulin requirements, total insulin requirements, potassium supplementation, fluid requirements, time to resolution of acidosis, and hospital length of stay. Arterial blood gases were most commonly drawn at baseline and repeated at 2-hour intervals until a pH greater than 7.2 was reached. Thus, resolution of acidosis was defined as a pH greater than 7.2. Fluid, electrolyte, bicarbonate, and insulin requirements were collected from the first 24 hours of hospital stay only. Patients who were treated with intravenous bicarbonate most commonly received 100 mEq of sodium bicarbonate as a 2-hour infusion, which was repeated until a pH greater than 7.0 was achieved.

DATA ANALYSIS

Data were collected from patients' medical records by a residency-trained PharmD trained on data abstraction; the information was documented on standardized data collection sheets. Data were analyzed with JMP 9.0.2, 2012 (SAS Institute). Descriptive statistics were used to summarize patient demographics and outcomes. Data are presented as median and interquartile range (IQR) or percentages. Continuous data were analyzed by the Wilcoxon rank sum test. Nominal data were analyzed using χ^2 or Fisher exact test, as appropriate. An a priori α level less than or equal to 0.05 was set to determine statistical significance for all comparisons.

Results

During the study period, 97 patients with severe DKA were identified by ICD-9-CM code, and 86 were included in the final analysis. Reasons for exclusion were out-of-hospital cardiac arrest or trauma (n = 6), incarceration (n = 4), and DKA diagnosed more than 24 hours after hospital admission (n = 1). The population was predominantly female (58%) and Hispanic (64%), with a median age of 35 years (IQR 25-46 years). More patients had type 1 diabetes mellitus (53%) than type 2 (41%); of the patients with type 2 diabetes, 78% were insulin dependent. Mean initial pH and blood glucose level at presentation were 6.96 (IQR 6.91-6.98) and 585 mg/dL (IQR 482-707 mg/dL), respectively. All baseline characteristics were similar between the 2 groups (Table 1).

Of the 86 patients who met all inclusion criteria, 44 received intravenous bicarbonate therapy and 42 did not. Time to resolution of acidosis (pH >7.2) was similar between the group receiving and the group not receiving intravenous bicarbonate (8 hours vs 8 hours; p = 0.72). Hospital length of stay was also similar between treatment groups (68 hours vs 61 hours; p = 0.31). The median dose of intravenous bicarbonate was 100 mEq (100-150) for patients who received intravenous bicarbonate. Patients who received intravenous bicarbonate therapy also received more insulin in the first 12 hours of hospital stay (48 units vs 44 units; p = 0.05), as well as in the first 24 hours of hospital stay (100 units vs 86 units; p = 0.04). Time of continuous infusion insulin did not differ significantly between groups (27 hours vs 26 hours; p = 0.09), but intravenous crystalloid requirements were significantly higher in the intravenous bicarbonate group compared with the no bicarbonate group (7.6 L vs 7.2 L; p = 0.01). Potassium supplementation in the first 24 hours was similar between groups (135 mEq vs 120 mEq; p = 0.84) (Table 2).

Table 1. Baseline Characteristics

Characteristic[a]	Intravenous Bicarbonate (n = 44)	No Bicarbonate (n = 42)	p Value
Age (years)	36 (25-45)	33 (25-47)	0.93
Female	64	51	0.28
Race			
Hispanic	62	68	0.55
white	36	27	0.38
black	0	5	0.22
other	2	0	1.00
Height (cm)	165 (157-173)	165 (160-170)	0.51
Weight (kg)	64 (58-82)	62 (55-70)	0.09
Comorbidities			
type 1 diabetes mellitus	51	56	0.64
type 2 diabetes mellitus	44	37	0.45
type 2 diabetes mellitus, insulin dependent	78	78	0.98
hypertension	18	22	0.62
hyperlipidemia	36	24	0.26
liver disease	4	0	0.50
chronic kidney disease	0	2	0.48
Home insulin regimens, all pts.			
basal insulin	76	76	0.99
dose (units)	35 (24-50)	29 (20-47)	0.18
bolus insulin	48	54	0.58
dose (units)	24 (15-45)	24 (17-38)	0.85
Admission vital signs and laboratory data			
mean arterial pressure (mm Hg)	91 (82-103)	91 (82-107)	0.92
heart rate (beats/min)	120 (106-132)	119 (108-132)	0.5
pH	6.96 (6.88-6.98)	6.97 (6.92-6.98)	0.18
pCO_2 (mm Hg)	22 (17-26)	22 (18-26)	0.54
glucose (mg/dL)	560 (472-711)	597 (490-711)	0.53
potassium (mg/dL)	4.8 (4.5-5.9)	5.1 (4.6-5.6)	0.42

[a]Data reported as mean (interquartile range) or %.

SUBGROUP ANALYSIS

Because the ADA guideline recommendations for intravenous bicarbonate infusions changed during our study from a threshold of pH less than 7.0 to pH less than 6.9, we conducted a subgroup analysis of patients presenting with a pH less than 6.9. In this subgroup, 13 patients received intravenous bicarbonate and 7 patients did not. Time to resolution of acidosis (10 hours vs 12 hours; p = 0.28), hospital length of stay (68 hours vs 70 hours; p = 0.87), and intravenous crystalloid requirement (8.8 L vs 7.3 L; p = 0.09) did not differ significantly between treatment groups. Insulin requirements also did not differ significantly between patients in the first 12 hours (48 units vs 50 units; p = 0.64) or first 24 hours of hospital stay (94 units vs 103 units; p = 0.43).

Discussion

This study was designed to compare the time to resolution of acidosis in patients with severe DKA who received intravenous bicarbonate therapy with that of those who did not receive intravenous bicarbonate. To our knowledge, no previously published studies have assessed intravenous bicarbonate exclusively in patients with an initial pH less than 7.0. This study is one of the largest (n = 86) conducted to evaluate intravenous bicarbonate therapy in severe DKA.

The results of this study suggest no improvement in time to resolution of acidosis or other outcomes associated with DKA therapy in patients with severe DKA who received intravenous bicarbonate therapy versus those who did not. Outcomes between treatment groups were similar in time to resolution of acidosis and hospital length of stay. Additionally, in our subgroup analysis of patients with an initial pH less than 6.9 (n = 20), no significant differences were seen; however, limiting our subgroup analysis to that subgroup reduced power. In light of this, we were not able to detect differences in physiologic consequences of acidemia in this subgroup. The lack of difference in more acidemic patients validates the recent change in the ADA guidelines recommendations, which lowered the pH threshold for intravenous bicarbonate use from 7.0 to 6.9.[3,4]

The insulin requirement in the first 12 and 24 hours of hospital stay was higher in patients who received intravenous bicarbonate therapy. Although the difference would be considered relatively small in clinical practice, this finding has not been observed in previous studies, in which no significant difference was seen in blood glucose decline or insulin requirements.[5-10] Insulin resistance has been shown to increase at a pH less than 7.2.[11] Therefore, therapies resolving severe acidosis more quickly should require less insulin. While we did not apply a specific illness severity scale, patients were similar at baseline in regard to presenting pH, blood glucose level, mean arterial pressure, and pCO_2. This implies that the severity of illness in patients receiving intravenous bicarbonate was not different from that of patients who did not receive intravenous bicarbonate. The seemingly paradoxical difference in higher insulin requirements for patients who received intravenous bicarbonate may be explained by the fact that, because of the rare use of a standardized DKA protocol at our facility, treatment intensity was largely based on physician preference. Additionally, data on patient-reported symptoms, such as severity and duration of illness prior to presentation, were not collected and may have contributed to more aggressive treatment strategies. Regardless, it appears that physicians who used intravenous bicarbonate also treated patients more aggressively with regard to insulin and fluid replacement compared with those who did not use intravenous bicarbonate. Higher insulin usage, as well as increased time on continuous insulin, signifies the need for a higher level of care, which may translate to increased cost. Additionally, higher insulin supplementation increases the risk of hypokalemia and cardiac arrhythmias. In our study, patients receiving intravenous bicarbonate therapy received more potassium supplementation in the first 24 hours, although this was not significantly different from the amount given to patients who received no intravenous bicarbonate.

Table 2. Outcome Comparison

Outcome, mean (IQR)	Intravenous Bicarbonate (n = 44)	No Bicarbonate (n = 42)	p Value
Time to pH >7.20 (hours)	8 (6-11)	8 (6-12)	0.72
Time to hospital discharge (hours)	68 (51-99)	61 (49-85)	0.31
Insulin in first 12 hours (units)	48 (40-58)	44 (34-53)	0.05
Insulin in first 24 hours (units)	100 (79-122)	86 (75-101)	0.04
Hours of insulin drip	27 (23-46)	26 (19-32)	0.09
Potassium use in first 24 hours (mEq)	135 (89-186)	120 (81-175)	0.84
Intravenous crystalloids in first 24 hours (L)	7.6 (6.7-10.2)	7.2 (5.5-8.3)	0.01

IQR = interquartile range.

Previous investigations into the benefit of intravenous bicarbonate therapy have shown mixed results. Studies showing a quicker resolution of acidosis also report a worsening of ketonemia, as well as a lack of sustained benefit past 2 hours of therapy.[5,6] Additionally, patients receiving intravenous bicarbonate therapy required higher amounts of potassium supplementation and showed no benefit in improving hemodynamic stability.[6,12,13] No previous study has shown a significant difference in glucose decline, insulin supplementation, or length of hospital stay. In a previous nonrandomized trial in pediatric patients with DKA, the use of intravenous bicarbonate was the sole variable associated with a greater risk of cerebral edema.[14] Two smaller studies in pediatric patients also found a nonsignificant association of bicarbonate use and cerebral edema.[15,16] No investigation into the variables and risks of cerebral edema in an adult population has been reported. Overall, the literature to support the use of intravenous bicarbonate therapy in any patient with DKA, regardless of severity, is very weak.

To date, no study has strictly looked at the patient population in which intravenous bicarbonate therapy is recommended, a pH less than 6.9. Additionally, no study has investigated the use of intravenous bicarbonate in adults with DKA using an exact inclusion criterion of pH less than 7.0. Therefore, the findings of our study further support the lack of benefit to the use of intravenous bicarbonate in patients presenting with a pH less than 7.0, as well as a pH less than 6.9.

There are several limitations to this study. This was a single-center analysis with a small number of subjects. Additionally, since this analysis was conducted retrospectively, our ability was limited to control for confounders in treatment with regard to concurrent DKA therapy, such as insulin or fluid requirements. We were also unable to collect data regarding ketoanion or lactate levels, as these were not routinely determined on every patient. Furthermore, the timing and extent of initial and follow-up arterial blood gases were not mandated at prespecified intervals or until patients achieved a specific pH level. At our facility, once a baseline arterial blood gas is obtained, repeat levels are regularly drawn at 2-hour intervals until pH greater than 7.2 is achieved, which is considered criteria for admission to the medical ward instead of the intensive care unit. Hence, once pH greater than 7.2 is reached, the interval for obtaining arterial blood gases is commonly extended because of transfer of care. As such, we used a definition of resolution of acidemia (pH >7.2) slightly lower than the ADA definition of complete resolution of acidemia (pH >7.3) to attempt to most accurately depict time to resolution of acidemia.[4] Additionally, we were unable to report time to anion gap resolution, as the timing of electrolyte monitoring was physician driven and highly variable. In addition, use and dosing of intravenous bicarbonate was highly dictated by physician preference. Although patients were similar at baseline in respect to pH, blood glucose, mean arterial pressure, and CO_2, other variables unaccounted for in our study, such as severity and duration of illness prior to presentation, may have led the physician to deem patients receiving bicarbonate therapy more ill, thereby requiring more aggressive treatment. Time in the emergency department or intensive care unit was not universally available on each patient during the study period and, therefore, could not be considered in the analysis.

In this review of 86 patients, the use of intravenous bicarbonate therapy did not improve time to resolution of acidosis or hospital length of stay in severe DKA (pH <7.0). Prospective randomized trials in patients with a pH less than 6.9, using a standardized DKA protocol, would be of value to validate these results.

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References

1. Kim S. Burden of hospitalizations primarily due to uncontrolled diabetes: implications of inadequate primary health care in the United States. Diabetes Care 2007;30:1281-2.
2. Chua HR, Schneider A, Bellomo R. Bicarbonate in diabetic ketoacidosis—a systematic review. Ann Intensive Care 2011;1:1-12. doi: 10.1186/2110-5820-1-23
3. Kitabchi AE, Murphy MB, Umpierrez GE, Kreisberg RA. Hyperglycemic crises in adult patients with diabetes. Diabetes Care 2006;29:2739-48.
4. Kitabchi AE, Miles JM, Umpierrez GE, Fisher JN. Hyperglycemic crises in adult patients with diabetes. Diabetes Care 2009;32:1335-43. doi: 10.2337/dc09-9032.

5. Hale PJ, Crase J, Nattrass M. Metabolic effects of bicarbonate in the treatment of diabetic ketoacidosis. Br Med J (Clin Res Ed) 1984;289: 1035-8.
6. Gamba G, Oseguera J, Castrejon M, Gomez-Perez FJ. Bicarbonate therapy in severe diabetic ketoacidosis. A double blind, randomized, placebo controlled trial. Rev Invest Clin 1991;43:234-8.
7. Okuda Y, Adrogue HJ, Field JB, Nohara H, Yamashita K. Counterproductive effects of sodium bicarbonate in diabetic ketoacidosis. J Clin Endocrinol Metab 1996;81:314-20.
8. Morris LR, Murphy MB, Kitabchi AE. Bicarbonate therapy in severe diabetic ketoacidosis. Ann Intern Med 1986;105:836-40.
9. Lutterman JA, Adriaansen AA, van 't Laar A. Treatment of severe diabetic ketoacidosis. A comparative study of two methods. Diabetologia 1979;17:17-21.
10. Green SM, Rothrock SG, Ho JD, Gallant RD, Borger R, Thomas TL, Zimmerman GJ. Failure of adjunctive bicarbonate to improve outcome in severe pediatric diabetic ketoacidosis. Ann Emerg Med 1998;31:41-8.
11. Walker BG, Phear DN, Martin FI, Baird CW. Inhibition of insulin by acidosis. Lancet 1963;2:964-5.
12. Lever E, Jaspan JB. Sodium bicarbonate therapy in severe diabetic ketoacidosis. Am J Med 1983;75:263-8.
13. Viallon A, Zeni F, Lafond P, et al. Does bicarbonate therapy improve the management of severe diabetic ketoacidosis? Crit Care Med 1999;27: 2690-3.
14 . Glaser N, Barnett P, McCaslin I, et al. Risk factors for cerebral edema in children with diabetic ketoacidosis. N Engl J Med 2001;344:264-9.
15 . Lawrence SE, Cummings EA, Gaboury I, Daneman D. Population-based study of incidence and risk factors for cerebral edema in pediatric diabetic ketoacidosis. J Pediatr 2005;146:688-92.
16 . Edge JA, Jakes RW, Roy Y, et al. The UK case-control study of cerebral oedema complicating diabetic ketoacidosis in children. Diabetologia 2006;49:2002-9.

EXTRACTO

Uso de Bicarbonato de Sodio Intravenoso en Pacientes con Cetoacidosis Diabética Severa

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TRASFONDO: El uso de bicarbonato intravenoso (IV) en cetoacidosis diabética (DKA) puede ser utilizado en pacientes que presenten pH menor de 6.9, según la Asociación Americana de Diabetes (ADA). El impacto de este tratamiento en la resolución de la acidosis en pacientes con DKA no está claro.

OBJETIVO: Determinar si el tratamiento con bicarbonato IV se asocia con mejores resultados en pacientes que se presentan a la sala de emergencia con DKA severa.

MÉTODOS: Este es un estudio retrospectivo que evaluó el período de 2007-2011 en un departamento de emergencia de un hospital de enseñanza a nivel terciario. Se incluyeron los pacientes adultos con diagnóstico de DKA y un pH inicial menor de 7.0. Los pacientes se separaron en dos grupos basado en si recibieron bicarbonato intravenoso. El resultado primario del estudio fue el tiempo para la resolución de la acidosis, definido como el retorno a un pH mayor de 7.2. Los resultados secundarios incluyeron el largo de la estadía, el uso de una infusión continua de insulina, los requerimientos de fluidos intravenosos, potasio y de insulina en las primeras 24 horas luego de admitido al hospital, comenzando con la admisión en el departamento de emergencia. También se realizó un análisis del grupo de pacientes con un pH inicial menor de 6.9.

RESULTADOS: No hubo diferencia en el tiempo de resolución de la acidosis (8 vs 8 horas, p = 0.7) o en el tiempo para ser dado de alta del hospital (68 vs 61 horas, p = 0.3) entre los pacientes que recibieron bicarbonato IV (n = 44) comparado con los pacientes que no lo recibieron (n = 42). La dosis media de bicarbonato fue de 100 mEq (100-150) para los pacientes que recibieron bicarbonato intravenoso. Los requerimientos de insulina y fluidos en las primeras 24 horas fue significativamente más alta en los pacientes que recibieron bicarbonato IV que los que no lo recibieron (100 vs 86 unidades, p = 0.04, y 7.6 vs 7.2 litros, p = 0.01, respectivamente). No hubo diferencias en las horas de infusión continua de insulina (27 vs 26 horas, p = 0.09), requerimiento de potasio en la primeras 24 horas de la estadía en el hospital (135 vs 120 mEq, p = 0.84).

CONCLUSIONES: El tratamiento con bicarbonato intravenoso no disminuyó el tiempo de resolución de la acidosis o el tiempo para ser dado de alta en pacientes con DKA y con un pH inicial de menor de 7.0.

Traducido por Mirza Martínez

RÉSUMÉ

Traitement par voie Intraveineuse du Bicarbonate de Sodium en Présence d'Une Grave Acidocétose Diabétique

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OBJECTIF: Déterminer si l'utilisation du bicarbonate de sodium par voie intraveineuse est associée à un résultat thérapeutique positif chez les patients se présentant à l'urgence avec une grave acidocétose diabétique.

MÉTHODES: Il s'agit d'une étude rétrospective qui a été effectuée de 2007-2011 à l'urgence d'un hôpital d'enseignement de soins tertiaires. Les patients adultes avec un diagnostique d'acidocétose diabétique et une valeur initiale de pH < 7.0 ont été inclus. Les patients ont été stratifiés dans 2 groups selon l'utilisation du bicarbonate de sodium par voie intraveineuse. Le critère d'évaluation principal était le temps pour la résolution de l'acidose définie comme étant un pH de >7.2. Les issues secondaires étaient la durée de séjour, l'utilisation d'une perfusion continue d'insuline, de solutions intraveineuses, de potassium, et des besoins en insuline dans les 24 heures suivant l'admission à l'urgence. Les auteurs ont également effectué une analyse chez un sous groupe de patients avec une valeur initiale de pH de < 6.9.

RÉSULTATS: Aucune différence dans la résolution de l'acidose (8 vs 8 heures, p = 0.7) ou le temps de congé de l'hôpital (68 vs 61 heures, p = 0.3) entre les patients qui ont reçu du bicarbonate de sodium par voie intraveineuse (n = 44) et ceux qui n'en ont pas reçu (n = 42). La dose médiane de bicarbonate de sodium par voie intraveineuse était de 100 mEq (100-150). Les besoins en insuline et en perfusions liquidiennes dans les 24 premières heures étaient significativement plus élevés chez les patients recevant du bicarbonate de sodium par voie intraveineuse lorsque comparé à ceux qui n'en recevaient pas (100 vs 86 unités, p = 0.04, et 7.6 vs 7.2 litres, p = 0.01, respectivement). Aucune différence n'a été observé dans la période de temps pour la perfusion de l'insuline en continue (27 vs à 26 heures, p = 0.09) ou les besoins en potassium dans le 24 premières heures de l'admission à l'hôpital (135 vs 120 mEq, p = 0.84).

CONCLUSIONS: L'utilisation du bicarbonate de sodium par voie intraveineuse ne diminue pas le temps pour la correction de l'acidose ou le temps de congé de l'hôpital pour une acidocétose diabétique chez les patients avec une valeur initiale de pH de < 7.0.

Traduit par Louise Mallet