

Intravenous Dextrose for Children With Gastroenteritis and Dehydration: A Double-Blind Randomized Controlled Trial

Jason A. Levy, MD; Richard G. Bachur, MD; Michael C. Monuteaux, ScD; Mark Waltzman, MD

Study objective: We seek to determine whether an initial intravenous bolus of 5% dextrose in normal saline solution compared with normal saline solution will lead to a lower proportion of hospitalized patients and a greater reduction in serum ketone levels in children with gastroenteritis and dehydration.

Methods: We enrolled children aged 6 months to 6 years in a double-blind, randomized controlled trial of patients presenting to a pediatric emergency department. Subjects were randomized to receive a 20 mL/kg infusion of either 5% dextrose in normal saline solution or normal saline solution. Serum ketone levels were measured before and at 1- and 2-hour intervals after the initial study fluid bolus administration. Primary outcome was the proportion of children hospitalized. Secondary outcome was change in serum ketone levels over time.

Results: One hundred eighty-eight children were enrolled. The proportion of children hospitalized did not differ between groups (35% in the 5% dextrose in normal saline solution group versus 44% in the normal saline solution group; risk difference 9%; 95% confidence interval [CI] -5% to 22%). Compared with children who received normal saline solution, those who received 5% dextrose in normal saline solution had a greater reduction in mean serum ketone levels at both 1 hour (mean Δ 1.2 versus 0.1 mmol/L; mean difference 1.1 mmol/L; 95% CI 0.4 to 1.9 mmol/L) and 2 hours (mean Δ 1.9 versus 0.3 mmol/L; mean difference 1.6 mmol/L; 95% CI 0.9 to 2.3 mmol/L).

Conclusion: Administration of a dextrose-containing bolus compared with normal saline did not lead to a lower rate of hospitalization for children with gastroenteritis and dehydration. There was, however, a greater reduction in serum ketone levels in patients who received 5% dextrose in normal saline solution. [Ann Emerg Med. 2013;61:281-288.]

Please see page 282 for the Editor's Capsule Summary of this article.

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INTRODUCTION

Background

Acute gastroenteritis accounts for approximately 2 million outpatient pediatric visits and 10% of all pediatric hospital admissions annually.¹ For patients with mild to moderate dehydration, oral rehydration therapy is the preferred method for fluid replacement.^{2,3} For severe dehydration or when oral rehydration therapy fails, intravenous fluid therapy is warranted. Traditional teaching suggests that, once the patient is stabilized, fluid deficit replacement by the intravenous route should be undertaken slowly during 24 to 48 hours.⁴ Currently, however, rapid intravenous rehydration and discharge has become common, albeit with significant practice variation with regard to fluid rate and composition.^{5,6} More recent data suggest that dextrose may play an important role in intravenous rehydration.^{7,8}

Importance

In an emergency department (ED)-based retrospective case-control study of children receiving rapid intravenous rehydration,⁷ we found an inverse association between dextrose

administration and return visits requiring admission.

Specifically, children returning to the ED for admission received less intravenous dextrose at the initial visit than those who did not return. Additionally, our experience suggests that children with gastroenteritis and dehydration frequently have metabolic acidosis because of elevated serum ketone levels, which may lead to persistent nausea, poor oral intake, and vomiting. We hypothesized that an initial intravenous bolus of dextrose-containing solution (triggering an increase in endogenous insulin) would facilitate faster resolution of ketoacidosis and therefore more rapid clinical improvement.

Goals of This Investigation

We conducted a randomized trial to determine whether an initial intravenous bolus of 5% dextrose in normal saline solution compared with normal saline solution without dextrose would lead to a lower proportion of hospitalized children and a greater reduction in serum ketone levels in children with gastroenteritis and dehydration.

Editor's Capsule Summary*What is already known on this topic*

The optimal intravenous fluid for pediatric rehydration is unclear.

What question this study addressed

Which fluid choice is superior at reducing hospital admission: normal saline solution or normal saline solution with 5% dextrose?

What this study adds to our knowledge

In this randomized trial of 188 dehydrated children aged 6 months to 6 years, the frequency of hospitalization was similar with either fluid. Serum ketone levels declined more quickly in the group with dextrose, but this may lack clinical importance.

How this is relevant to clinical practice

Intravenous rehydration in children aged 6 months to 6 years is similarly effective with normal saline solution with or without 5% dextrose.

prepared all intravenous solution bags in the hospital pharmacy. Each bag was concealed by an opaque cover to maintain blinding and all ED staff and caregivers were blinded to treatment assignment.

Interventions

After enrollment but before fluid administration, demographic and clinical data related to dehydration were collected prospectively on a standardized data collection form. The treating attending physician assessed the physical examination findings related to dehydration and assigned a general appearance score on a 5-point scale (with 1=obtunded to 5=alert and active). A peripheral intravenous catheter was required for all enrolled patients for administration of intravenous fluids. At the catheter placement, study personnel measured an initial bedside serum glucose level and bedside serum ketone level (Precision Xtra Blood Glucose and Ketone Monitoring System; Abbott Laboratories, Libertyville, IL). The caregiver, treating physician, and nurse were blinded to these results unless the bedside glucose level was less than 40 mg/dL or greater than 200 mg/dL, at which point the patient was removed from the study protocol. Patients were subsequently randomized to receive the 20 mL/kg study fluid bolus (normal saline solution or 5% dextrose in normal saline solution) administered during 45 to 60 minutes. Study enrollers, the medical team, and caregivers were blinded to the treatment assignment. One hour after initiation of the study fluid bolus, a second blinded bedside blood glucose and ketone level was obtained. Subsequent intravenous or oral fluids (with or without dextrose) were then administered at the discretion of the treating attending physician. Two hours after initiation of the original study fluid bolus, a third blinded bedside glucose and ketone level was obtained. At ED disposition or 3 hours after initiation of the study fluid bolus (whichever occurred first), the treating attending physician rescored the general appearance on the 5-point scale.

For all hospitalized patients, the attending physician completed a standardized checklist indicating all reasons for admission.

Decisions to obtain other diagnostic studies, administer medications (including antiemetics), or perform other medical care were directed by the clinical team caring for the patient, and these data were collected prospectively by the study enroller.

For patients discharged from the ED, blinded study personnel telephoned caregivers 48 to 72 hours after discharge to ask standardized questions about the need for further unscheduled medical care, defined a priori as care sought for ongoing vomiting or refusal to drink and excluding previously scheduled or recommended appointments. If caregivers could not be reached in this period, additional calls were attempted up to 5 days after enrollment. All admitted patients were treated and discharged at the discretion of the inpatient medical team. Follow-up for all patients was augmented by reviewing inpatient diagnostic studies and hospital medical records.

MATERIALS AND METHODS**Study Design and Setting**

We performed a prospective, double-blind, randomized controlled clinical trial comparing 5% dextrose in normal saline solution and normal saline solution for rapid intravenous rehydration among children with gastroenteritis and dehydration. Subjects were enrolled from November 2007 through December 2010 at Boston Children's Hospital, an urban, tertiary care pediatric hospital with approximately 60,000 ED visits per year. The institutional Committee on Clinical Investigation approved the study (<http://www.ClinicalTrials.gov> Trial Number NCT01343758).

Selection of Participants

Eligible children included those aged 6 months to 6 years who had symptoms of gastroenteritis and received intravenous fluids for dehydration as determined by the treating attending physician. A convenience sample of children was identified when a study enroller (research assistant, registered nurse, or study investigator) was available and written informed consent was obtained. Subjects were excluded if they had a history of chronic illness or disorder of glucose metabolism, symptoms for more than 7 days, received intravenous fluids in the previous 12 hours, or were suspected to have a comorbid condition such as pneumonia, urinary tract infection, or appendicitis.

Subjects were randomized to receive an initial 20 mL/kg fluid bolus of either normal saline solution or 5% dextrose in normal saline solution. An independent statistician performed computerized randomization in blocks of 10. One pharmacist

Outcome Measures

The primary outcome was the proportion of patients admitted to the hospital. Secondary outcome was the change in serum ketone concentration in each patient over time (calculated as the initial serum ketone value minus the 1- and 2-hour ketone values). For patients discharged from the ED, we also evaluated the need for unscheduled medical care defined above. Because the efficacy of a dextrose-containing bolus is premised on an underlying metabolic acidosis, we chose a priori to analyze the subset of children with abnormal serum bicarbonate levels ($\text{HCO}_3 \leq 20$ mmol/L) with respect to hospitalization and need for unscheduled medical care. Data assessing the change in general appearance score (5-point scale score at 3 hours minus initial score), ability to tolerate oral fluids, length of stay, and the volume of oral fluid consumed were collected prospectively and described.

Primary Data Analysis

Because we have an educational and quality improvement initiative around oral rehydration therapy, we anticipated that most children who receive intravenous rehydration in our ED are either severely dehydrated or have previously failed oral rehydration. We therefore estimated that 40% of eligible children who received intravenous fluid would require hospitalization. To detect a change in proportion of patients hospitalized from 40% to 20% with power of 80% and an α of .05, 91 patients in each treatment group were required.

We calculated descriptive statistics for baseline demographic and clinical characteristics across the 2 treatment groups. For each patient, a dehydration score was calculated after patient discharge, using prospectively collected data from the standardized data collection form. This score is based on validated clinical findings from previously published data⁹ and was included in the analysis of baseline characteristics for each treatment group.

For our primary outcome, we calculated the difference in proportion of patients hospitalized between treatment groups with 95% confidence intervals (CIs). To determine whether any baseline demographic or clinical factors were potentially inducing bias, we adopted a published approach recommended for randomized clinical trials.^{10,11} For each factor, we considered the magnitude of imbalance between treatment groups and the prognostic importance of each factor in question relative to our primary outcome. Any factors considered predictive of our primary outcome and that demonstrated any but a negligible difference between treatment groups were included in a multivariate logistic regression model, along with treatment assignment, in the prediction of hospitalization. The univariate and multivariate results were compared to assess the bias, if any, induced by these factors.

For our secondary outcome, we assessed the difference between treatment groups for within-patient change of serum ketone level over time. We used a generalized estimating equation with the gaussian distribution and identity link. Working covariance structures were specified with Huber-White

robust estimators of variance to account for repeated serum ketone measurements in each patient. We modeled ketone level as the dependent variable and treatment group, time (ie, coded categorically as baseline, 1-hour testing, and 2-hour testing), and the treatment group-by-time interaction as the independent variables. A statistically significant interaction term was interpreted as evidence of a treatment effect. This term tests whether the change in the outcome variable over time in subjects receiving 5% dextrose in normal saline solution differs from the change over time in those receiving normal saline solution. That is, the interaction tests the effect of 5% dextrose in normal saline solution over time, over and above the effect of normal saline solution.

Data analysis was performed with Stata (version 10.0; StataCorp, College Station, TX). All analyses were carried out with an intention-to-treat principle, and all statistical tests were 1-tailed, with an α level set at .05.

A patient safety and monitoring plan was in place during the trial. We measured bedside glucose at enrollment and at 1 and 2 hours subsequent to study fluid initiation. Additionally, we measured urine output in all patients to monitor for osmotic diuresis related to potential hyperglycemia.

RESULTS

Characteristics of Study Subjects

Study enrollers identified and approached 231 eligible patients, 32 of whom declined participation. Five patients were withdrawn after consent but before randomization and study fluid administration because of either inability to obtain intravenous access ($n=4$) or a change in diagnosis ($n=1$) at consent. Six patients were excluded as part of the study protocol because of an initial serum glucose level less than 40 mg/dL. A total of 188 subjects were randomized to treatment, 94 to normal saline solution and 94 to 5% dextrose in normal saline solution (Figure 1). Baseline characteristics and ED treatment variables for each group are reported in Tables 1 and 2, respectively. No clinically meaningful differences between the groups were observed. During the entire ED visit, the 5% dextrose in normal saline solution group received significantly more intravenous dextrose, approximately equal to a 20 mL/kg bolus of a 5% dextrose-containing solution (Table 2). Fifty-five percent of subjects randomized to the normal saline solution group received some intravenous dextrose during their ED visit.

Parents of 18 children declined repeated blood sampling, and no data exist on repeated serum ketone levels for these patients. Five children were found to have diagnoses other than gastroenteritis after randomization and study fluid administration; 2 patients with urinary tract infection, 2 patients with pneumonia, and 1 patient with glomerulonephritis and acute renal failure. Data from these patients were included in all analyses.

Main Results

Primary outcome was the proportion of children hospitalized. In the 5% dextrose in normal saline solution

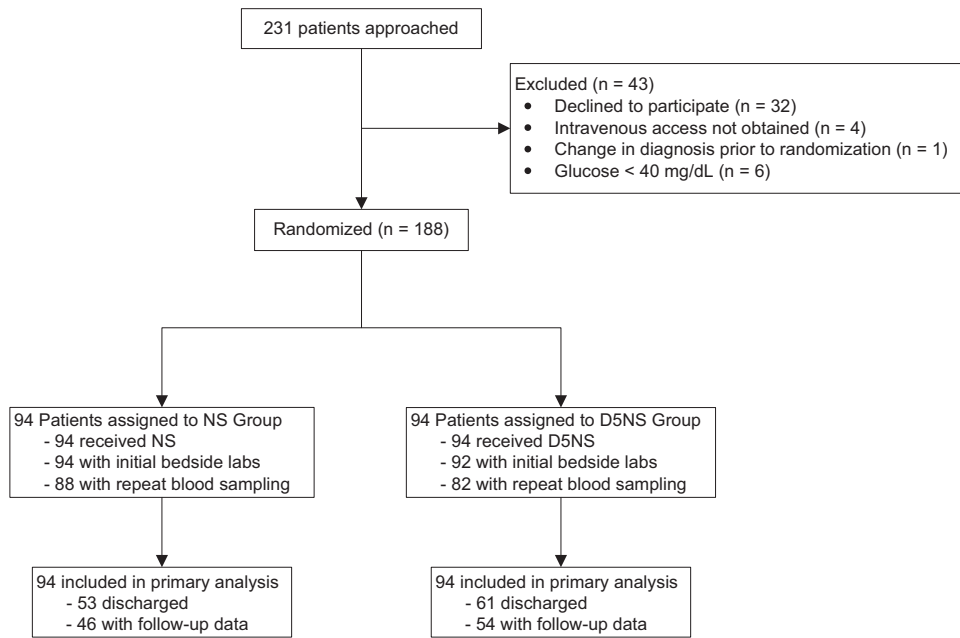


Figure 1. Enrollment and outcomes. D5NS, 5% dextrose in normal saline solution; NS, normal saline solution.

Table 1. Baseline characteristics of subjects in both treatment groups.

Clinical Characteristics	Normal Saline Solution Group (n=94)	5% Dextrose in Normal Saline Solution Group (n=94)
Age, y*	2.0 (1.1–3.2)	2.4 (1.4–3.9)
Triage heart rate, beats/min [†]	127 (23)	126 (17)
Dehydration score ⁹	4 (3–5)	4 (3–5)
Score >2, %	95	89
Score >3, %	82	76
Duration of symptoms in days, %		
<1	18	28
1–2	29	25
3–4	41	30
≥5	12	17
General appearance score	3 (3–4)	3 (3–4)
Laboratory measurements at enrollment	(n=86)	(n=87)
Glucose, mg/dL	80 (23)	85 (22)
Bicarbonate, mmol/L	18 (3.7)	19 (3.6)
Ketone, mmol/L	3.7 (1.6–4.7)	2.9 (1.1–4.4)
Bicarbonate <20 mmol/L, %	79	66
Blood urea nitrogen:creatinine >20, %	99	99

*All median scores are given with interquartile ranges.
[†]All mean values are given with SD.

group, 35% of patients were hospitalized compared with 44% in the normal saline solution group (risk difference 9%; 95% CI –5% to 22%). In the subset of acidotic patients (n=123), 53% in the normal saline solution group were hospitalized compared with 46% in the 5% dextrose in normal saline solution group (risk difference 7%; 95% CI –10% to 25%). These results did

Table 2. Details of ED management.

Treatment Variable	Normal Saline Solution Group (n=94)	5% Dextrose in Normal Saline Solution Group (n=94)
Received ondansetron, %	78	80
Total intravenous fluids, mL/kg*	30 (10)	28 (9)
Total intravenous dextrose, g/kg	0.28 (0.40)	1.27 (0.34)
Reasons for admission (N=74), %		
Refusing oral fluids	34	24
Lethargy/somnolence	29	18
Persistent vomiting	17	30
Severe dehydration	15	21
Abnormal sodium	7	9
Return visit or transfer	7	6
Family request	5	15
Primary physician request	5	0
Abnormal serum bicarbonate level	42	42

*All mean values are given with SD.

not change in a logistic regression model controlling for clinical variables listed in Table 1.

Secondary outcome was change in serum ketone levels over time. Baseline mean serum ketone level was 3.2 mmol/L (normal ≤0.2 mmol/L) for all patients, and there was no difference in baseline level between treatment groups. In a between-group comparison, there was a significant difference in mean serum ketone concentration at both 1 and 2 hours after the initial study fluid bolus (Figure 2). In a repeated-measures analysis within randomization group, serum ketone level reduction (initial ketone level minus repeated level) was greater in the 5% dextrose in normal saline solution group at both the 1-hour (mean Δ 1.2 versus 0.1 mmol/L; mean

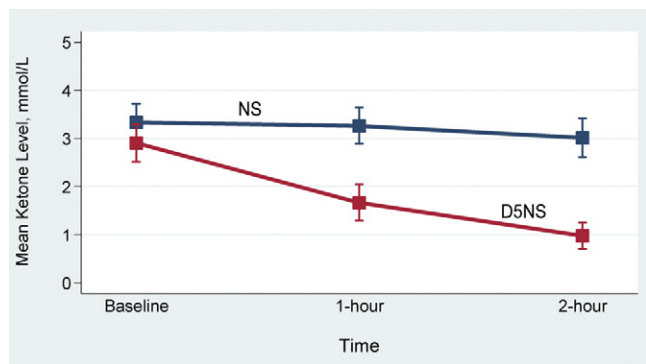


Figure 2. Mean serum ketone concentration over time in patients treated with normal saline solution (NS) versus 5% dextrose in normal saline solution (D5NS). Bars represent 95% CIs. Baseline represents ketone level at initiation of the study fluid bolus. Mean ketone levels at the 1- and 2-hour intervals were significantly lower in the 5% dextrose in normal saline solution group.

difference 1.1 mmol/L; 95% CI 0.4 to 1.9 mmol/L) and 2-hour (mean Δ 1.9 versus 0.3 mmol/L; mean difference 1.6 mmol/L; 95% CI 0.9 to 2.3 mmol/L) measurements (Figures 3 and 4).

Among the entire sample, 61% of patients (114/188) were discharged from the ED after intravenous rehydration, and telephone follow-up was obtained for 88% (100/114) of those subjects. Among these children, 17% required unscheduled medical care in the 5% dextrose in normal saline solution group compared with 24% in the normal saline solution group (risk difference 7%; 95% CI -9% to 23%). In the subset of discharged children with metabolic acidosis for whom follow-up was obtained ($n=55$), 11% in the 5% dextrose in normal saline solution group required unscheduled medical care versus 30% in the normal saline solution group (risk difference 19%; 95% CI -2% to 40%).

Among other measured clinical variables, there were no clinically meaningful differences between treatment groups (Table 2).

There were no adverse events in either treatment group. Twenty-six children were hypoglycemic (glucose <60 mg/dL) before receiving the study fluid. None of the children who received a 5% dextrose in normal saline solution bolus were hypoglycemic 1 hour after initiation of the study fluid bolus, whereas all of the children in the normal saline solution group remained hypoglycemic at 1 hour, with an additional 12 children becoming hypoglycemic in this treatment arm. Six children were hypoglycemic at the 2-hour measurement, 3 from each treatment group. Children who received a 5% dextrose in normal saline solution bolus had higher mean serum glucose levels at 1 and 2 hours after initiation of treatment. There was no difference in mean urine output between groups during the 3-hour study period (Table 3).

LIMITATIONS

In our study, hospitalization was at the discretion of the treating physician and may have been influenced by nonclinical factors such as primary care physician request, parental preference, individual attending physician practices, patient social factors, hospital volume, and time of day. Reasons for admission were recorded on a standardized data sheet, however, and there were no clinically meaningful differences between groups.

Although we prospectively collected data related to signs and symptoms of dehydration and assigned a score based on previously validated data,⁹ this score was calculated subsequent to enrollment, and there was no minimum score required for eligibility in the study. The assessment of level of dehydration is inexact and individual signs of dehydration are imprecise, but combinations of factors improve diagnostic accuracy.¹² We chose to include children with dehydration requiring intravenous rehydration as assessed by the treating physician. Consequently, some patients may not have been truly dehydrated. Still, 79% of subjects (148/188) had a dehydration score greater than or equal to 3; 92% (173/188), greater than or equal to 2. Finally, of patients for whom serum chemistry tests were conducted, 72% were acidotic (bicarbonate level concentration ≤ 20 mmol/L) and 99% had a ratio of blood urea nitrogen to creatinine greater than 20, lending some objective evidence for the enrollment of dehydrated children.

As is standard in our department, most children in our study received some intravenous dextrose regardless of the study fluid administered. We did not regiment oral or intravenous fluid therapy after the study fluid bolus and allowed the treating physicians to administer additional intravenous fluids at their discretion. Subjects in the 5% dextrose in normal saline solution group, however, received (on average) approximately 1 g/kg more intravenous dextrose than those in the normal saline solution group. The reduction in serum ketone levels was still greater in the 5% dextrose in normal saline solution group even at 2 hours post-therapy, at which point most patients in both groups had received an additional hour of fluid therapy with a solution containing 5% dextrose at a rate of 1.5 to 2 times maintenance.

DISCUSSION

Gastroenteritis with dehydration is one of the most common reasons for evaluation in the pediatric ED. Currently, oral rehydration therapy is the preferred and most effective method of rehydration. In cases of severe dehydration or when oral rehydration therapy fails, intravenous fluid therapy is often undertaken.

Although many pediatric EDs use rapid intravenous rehydration routinely for all levels of dehydration, there is no standard method or universally accepted published guideline. Thus, considerable variation exists both in practice and in the literature. Rosenstein and Baker¹³ demonstrated that in a single pediatric ED, fluid resuscitation volume ranged from 14 to 103 mL/kg and that patients received a variety of intravenous fluid

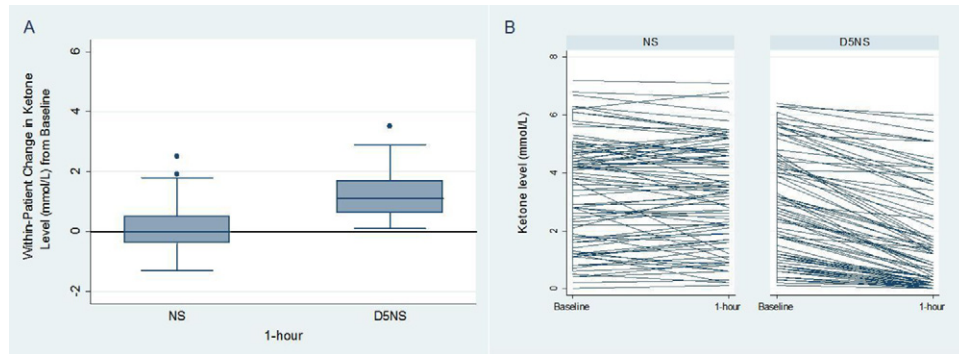


Figure 3. One-hour change in serum ketone levels in each treatment group. *A*, Summary change in serum ketone concentration over time in patients treated with normal saline solution (NS) versus 5% dextrose in normal saline solution (D5NS). Zero line represents no change after the study fluid bolus. A decrease in serum ketone level is represented by a positive change (initial level minus repeated level), and an increase in serum ketone level is represented by a negative change. The upper whisker is the upper adjacent value, defined as the 75th percentile + $1.5 \times (75\text{th percentile} - 25\text{th percentile})$. Likewise, the lower is the lower adjacent value, defined as the 25th percentile - $1.5 \times (75\text{th percentile} - 25\text{th percentile})$. *B*, Intraindividual changes in ketone level from baseline to 1-hour assessments, stratified by treatment assignment.

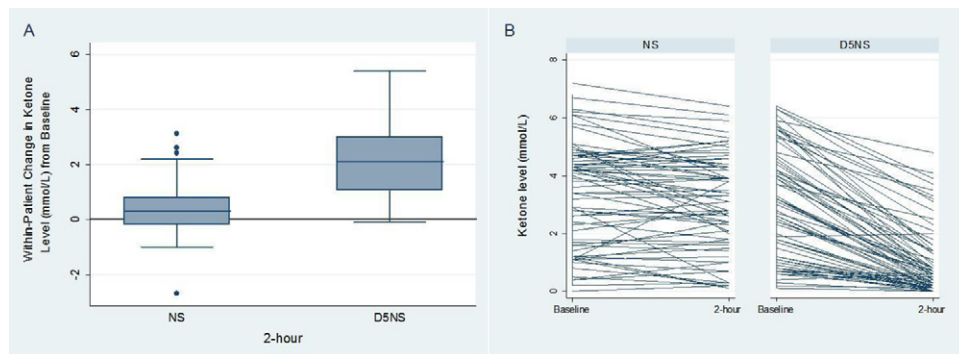


Figure 4. Two-hour change in serum ketone levels in each treatment group. *A*, Summary change in serum ketone concentration over time in patients treated with normal saline solution (NS) versus 5% dextrose in normal saline solution (D5NS). Zero line represents no change after the study fluid bolus. A decrease in serum ketone level is represented by a positive change (initial level minus repeated level), and an increase in serum ketone level is represented by a negative change. The upper whisker is the upper adjacent value, defined as the 75th percentile + $1.5 \times (75\text{th percentile} - 25\text{th percentile})$. Likewise, the lower is the lower adjacent value, defined as the 25th percentile - $1.5 \times (75\text{th percentile} - 25\text{th percentile})$. *B*, Intraindividual changes in ketone level from baseline to 2-hour assessments, stratified by treatment assignment.

solutions. Moineau and Newman¹⁴ reported similar variability with respect to variation in fluid amount. In a systematic review of rapid intravenous rehydration, Gorelick⁶ found that the composition of fluids differed considerably. In a recent survey, Freedman et al⁵ reported wide ranges with respect to both intravenous fluid volume and rate of administration for moderately dehydrated children.

Previous studies suggest that infants and young children develop metabolic acidosis even at moderate levels of dehydration.^{12,15-17} Furthermore, in a case-control study,⁷ we previously demonstrated an association between ED return visits requiring admission and the amount of intravenous dextrose administered in children who had been discharged after rapid rehydration. We believe that, similar to those of children with

diabetic ketoacidosis, elevated serum ketone levels in dehydrated children likely contribute to ongoing symptoms of nausea, vomiting, and malaise. We therefore hypothesized that children who received a dextrose-containing intravenous bolus would have a greater reduction in serum ketone levels (through increased endogenous insulin release), leading to a lower rate of hospitalization and improved clinical outcomes.

In our study population, subjects had significantly elevated serum ketone levels above the range considered predictive of diabetic ketoacidosis.¹⁸ We found that children who received an initial bolus of 5% dextrose in normal saline solution had a greater reduction of their serum ketone levels compared with those who received a normal saline solution bolus without dextrose. This difference continued to hold true even at 2 hours,

Table 3. Secondary outcome measures.

Therapy	Normal Saline Solution Group (N=94)	5% Dextrose in Normal Saline Solution Group (N=94)
Change in general appearance score*	1 (0–1)	1 (0–1)
Tolerated oral fluids, %	75	76
Volume of oral fluids, mL [†]	90 (14–170)	118 (30–200)
Length of stay in ED (min)	280 (246–361)	288 (238–349)
Urine output, mL [†]	44 (75)	35 (60)
Glucose at 1-hour (mg/dL)	70 (57–86)	272 (221–361)
Glucose at 2-hour (mg/dL)	106 (87–172)	154 (121–221)

*Change in general appearance score equals the value on a 5-point scale at 3 hours subtracted from the value at enrollment (detailed in text).

[†]All median scores are given with interquartile ranges.

*All mean values are given with SD.

when most subjects (78%) in both groups had received additional dextrose-containing fluids at a rate of 1.5 to 2 times maintenance.

Although our hypothesis around serum ketone level reduction did hold true, we did not find a clinically important difference in admission rate. Forty-four percent of patients in the normal saline solution group were hospitalized compared with 35% in the 5% dextrose in normal saline solution group, with overlapping CIs. In the subset of patients who were acidotic and discharged, 30% of patients in the normal saline solution group required unscheduled medical care compared with only 11% in the 5% dextrose in normal saline solution group. Although this planned analysis was not subject to hypothesis testing and may have been due to chance alone, our findings may have clinical relevance and warrant further study. As a theoretic rationale for this effect, we would hypothesize that some patients in the normal saline solution group who had metabolic acidosis and were discharged may have had short-lived improvement while in the ED as a result of antiemetic treatment but had return of symptoms once home because of persistent ketoacidosis. Those in the 5% dextrose in normal saline solution group had greater reduction of their serum ketone levels, which in turn may have contributed to the persistent resolution of symptoms and thus the less frequent need for unscheduled medical care.

In our study population, 38 children were hypoglycemic at either the initial or 1-hour glucose check. Three subjects in each treatment arm were hypoglycemic at 2 hours. For the 3 children in the 5% dextrose in normal saline solution group, 2 received no additional oral or intravenous fluids after the study fluid bolus. According to an anticipated endogenous insulin surge and subsequent potential hypoglycemia after an intravenous bolus of dextrose, we would recommend continuing dextrose administration either orally or intravenously after the initial bolus is administered to prevent this uncommon occurrence.

Among dehydrated children requiring intravenous rehydration, administration of a dextrose-containing fluid bolus

appears to be safe and led to a greater reduction in serum ketone levels compared with a bolus of normal saline solution. This did not translate, however, into a clinically significant reduction in a need for hospitalization. Further studies are needed to determine the optimal fluid regimen for rapid intravenous hydration in children presenting with gastroenteritis, dehydration, and metabolic acidosis.

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Author affiliations: From the Division of Emergency Medicine, Children's Hospital Boston, Harvard Medical School, Department of Pediatrics, Boston, MA.

Author contributions: JAL, RGB, and MW conceived the study and designed the trial. MW obtained partial funding. JAL supervised the conduct of the trial and data collection. JAL and MW recruited patients. JAL managed the data. JAL, RGB, and MCM provided statistical advice and aided with data analysis. JAL drafted the article, and all authors contributed substantially to its revision. JAL takes responsibility for the paper as a whole.

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Address for correspondence: Jason A. Levy, MD, E-mail jason.levy@childrens.harvard.edu.

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IMAGES IN EMERGENCY MEDICINE

(continued from p. 278)

DIAGNOSIS:

Ludwig's angina. Ludwig's angina is a life-threatening infection of the submandibular space, with a mortality rate approaching 10%.¹⁻³ It generally arises from a dental infection; however, it can be precipitated by trauma or foreign body or spread from other local infections. The course is rapid, with cellulitis spreading through the connective tissues of the oral cavity floor (Figures 1 and 2), and frequently includes abscess formation.^{1,4}

Ultrasonography may reveal cobblestoning of the soft tissues and a complex hypoechoic collection if abscess is present (Figure 3). CT may demonstrate edema and swelling of the soft tissues and visualization of abscesses (Figure 4).^{5,6} Airway compromise is a paramount concern, and asphyxiation is the most common cause of death.^{1,7} Management consists of airway stabilization and intravenous antibiotics. Surgery is considered when medical therapy fails or when abscesses exist.^{1,5}

The patient had an oral surgery consultation, received intravenous clindamycin, and was taken to the operating room for abscess drainage.

Author affiliations: From the Department of Emergency Medicine, St. Luke's/Roosevelt Hospital Center, New York, NY.

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