

DKA TREATMENT PROTOCOL-DENVER

The Pediatric Diabetes Group has offices at The Barbara Davis Center, an outpatient diabetes treatment center and a basic science/clinical research facility. It is part of the University of Colorado at Denver and Health Sciences Center and is also the outpatient diabetes unit for The Children’s Hospital. It is located on the Fitzsimons campus at 1775 Aurora Court (Colfax and Aurora Court – two blocks west of Colfax & I-225).
<http://www.uchsc.edu/misc/diabetes/index.html>.

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NOTE: Diabetic ketoacidosis is a life-threatening condition. One of the diabetes physicians listed above should be notified regarding treatment of any child. The overall mortality for a child in the USA with DKA is 2%. Those with severe DKA have a much higher risk for morbidity and mortality. Meticulous attention to the details of therapy and the child's clinical course can decrease this risk.

A patient who is unresponsive to vocal commands or presents with hypotension is rare and requires close physician monitoring. Urgent critical care and diabetes consultation should be obtained.

I. DKA Definition:

A state of absolute or relative insulin deficiency resulting in hyperglycemia, dehydration and accumulation of ketone bodies in the blood with subsequent metabolic acidosis (pH < 7.30; serum bicarbonate < 15 mmol/L). The severity of DKA is defined by the venous pH. Severe DKA is defined by a pH <7.15 and usually will require treatment in the ICU. Moderate DKA is defined by a pH of 7.15-7.25 and may usually be treated on the ward. A pH >7.25 is mild DKA and **usually** can be treated in the ED over a 4-6 hour time period, or on the floor, if admission is otherwise required.

II. Causes of DKA:

- A. Initial presentation of type 1 diabetes mellitus
- B. Missed insulin injections
- C. Inadequate insulin dosage in a known diabetic patient
- D. Emotional stress/ trauma/surgery without adequate insulin adjustment
- E. Intercurrent illness/infection without appropriate dose adjustment

III. Clinical Presentation

A. History (key points)

1. Classic triad = polydipsia, polyuria, and weight loss (polyphagia is unusual in children)
2. Vomiting/abdominal pain
3. Increased, difficult, or deep respirations
4. Symptoms of infection/flu (may be similar to those of DKA)
5. Illness in family members or close friends
6. In a known diabetic:
 - *when and how much insulin was last taken?
 - *missed shots?
 - *emotional stress as clues to missed shots?

B. Physical exam

1. Vital signs
2. Hydration status/peripheral perfusion/hypovolemic shock?
3. Acetone-fruity breath
4. Kussmaul respirations
5. Neurologic status
6. Signs of infection

C. Initial labs-stat

1. For diagnosis: blood glucose and urine ketones.
A simple urine dipstick and/or a meter glucose level in an ED or office may make a diagnosis and save a life. If abnormal, obtain consultation. "THINK ABOUT IT - DO IT!"
2. Serum glucose, electrolytes including Na⁺, K⁺, HCO₃ and BUN, venous pH and PCO₂. [Arterial PCO₂ less than 20 mmHg may be an important predictor of cerebral edema in severe DKA. (pH <7.0) (19)].
3. Serum osmolality*, calcium, phosphorus.
*serum osmolality should be calculated in all, and measured in severe DKA and/or dehydration; however, this will give a slightly low value because serum Na⁺ will be factitiously low:
serum Osmolality (mOsm/L)=2(Na+K) + glucose/18 + BUN/2.8
Na correction for glucose:
Corrected Na= measured Na + (serum glucose-100)(1.6)/100

4. Appropriate cultures and/or UA if infection is suspected from H&P. Delay chest film until hydration is normalized.

D. Follow-up lab

1. **First four hours** (or until glucose and electrolytes stable): **q1hour** serum glucose, electrolytes, and venous pH in severe DKA.
2. **When glucose and electrolytes stable: q2 hour** venous pH and electrolytes until the pH is above 7.3 or the HCO₃ is above 15 mEq/L. Note: continue to check bedside blood glucose q1hour while on insulin drip.
3. Other studies (osmolality, calcium, phosphorus, etc.) as indicated.
4. **Flow sheet of I & O, lab values**; catheterization may be necessary in the critically ill child, but ask to void hourly for I&O.

IV. Management

Theory: Any treatment plan for DKA should be based on the underlying pathophysiology. Hyperglycemia and ketoacidosis induce important alterations in organ physiology. Hyperglycemia causes an osmotic diuresis and eventually leads to dehydration, electrolyte depletion, and hypertonicity. Metabolic acidosis is partially compensated by hyperventilation and hypocapnia. These effects in turn cause changes in renal, CNS, and cardiovascular system functioning.

Considering the above, the first therapeutic step is to restore extracellular fluid volume which has been depleted through osmotic diuresis and vomiting. Insulin must be given to allow normal carbohydrate utilization and to stop ketogenesis. Serum hyperosmolality should be normalized gradually and intracellular stores of potassium replenished. Severe acid/base disturbances need to be corrected both for homeostatic reasons and to permit optimally effective insulin action. Normal glycogen and fat stores, and protein synthesis also need to be restored over time.

Medications which may alter mental status should be given with extreme caution. Agitated patients may have impending circulatory collapse or CNS catastrophe, which may be precipitated or masked by medications that alter mental status, e.g., Inapsine.

V. Complications in treating DKA

A. Dehydration/shock

In the presence of severe dehydration, the tendency is to want to correct the dehydration very rapidly – which can be VERY DANGEROUS (see cerebral edema below). However, decreased vascular volume and impending circulatory collapse also must be addressed and continued excessive urine output needs to be considered during the first several hours of therapy. Five percent albumin (10 ml/kg over 30 min) or other colloid should be given if severe shock is present or if there is still evidence of shock one hour after receiving saline (crystalloid). Measured replacement is suggested in VI (B). In patients with severe dehydration or in patients with severe mental status changes, intravascular pressure monitoring to follow hydration status is indicated.

Disposition for ED, ICU, or Ward should not be made until initial laboratory values return.

B. Criteria for ICU Admission

1. Severe DKA, including long duration of symptoms, impaired circulation,

- depressed level of consciousness.
2. Children at increased risk for cerebral edema.
 3. Under the age of 5.

C. Hypokalemia - Hyperkalemia:

Correction of acidosis results in intracellular movement of K⁺. Resultant hypokalemia may lead to muscular weakness (including diaphragmatic fatigue in an already exhausted patient) which may result in a respiratory arrest. **(Hypokalemia or hyperkalemia may lead to cardiac arrhythmias or cardiac arrest.)**

D. Hypoglycemia

While on a continuous IV infusion of insulin, the patient is at risk for hypoglycemia. Hourly glucose levels and addition of dextrose to the IV solution when the blood glucose falls to 250 mg% should prevent the problem. It is appropriate to use 10% dextrose if glucose levels are <150 mg/dl on D5 and HCO₃ not yet 15-17mg% and, therefore not yet appropriate to discontinue IV insulin. For acute hypoglycemia, the insulin infusion may be discontinued for 15 minutes, then recheck the serum glucose and restart insulin with a higher concentration of dextrose. If the patient can tolerate oral glucose, 2-4 ounces of juice may be given as well.

E. Cerebral edema

The major cause of death in childhood DKA. Children, in contrast to adults, develop cerebral edema if their rehydration is undertaken too rapidly, even in hyperosmolar non-ketotic coma. The etiology of cerebral edema is still unknown, but may result from unfavorable osmotic gradients (excessive free water) and /or cerebral anoxia. Recent evidence suggests a greater likelihood if the serum sodium concentration fails to rise as the serum glucose falls. Mahoney, et al. (19) found: cerebral edema to be more likely with an arterial pH <7.1, and PCO₂ <20, and in children receiving more than 50 cc/kg of fluid in the first 4 hours of treatment. Usually seen in patients who are less than 15 years old who are severely dehydrated, very acidotic, and very hyperosmolar. Newly diagnosed patients who are < 5 years old seem to be at greatest risk.

Clinically the patient may complain of headaches or have a change in mental status hours after therapy for DKA has begun. In some there is a premonitory period when development of cerebral edema could be suspected if there is a change in arousal or behavior, severe headache, incontinence, pupillary changes, seizures, bradycardia, or disturbed temperature regulation. Early intervention before respiratory arrest is essential. Most often, the patient's lab values are improving as she/he appears to be worsening clinically.

Treatment includes **decreasing fluids** (<70cc/kg/day) and giving **Mannitol** (1 gm/kg over 30 minutes), elevating the head of the bed, **intubation and hyperventilation** until a pCO₂ level of 30-35 mmHg is reached may be necessary, although many already have a pCO₂ less than 30 mmHg due to hyperventilation. Hypocapnea causes cerebral vasoconstriction. Mannitol may need to be repeated depending on the clinical condition of the patient. Dexamethasone should not be given.

Treatment **should not be delayed** until after the radiographic studies have been obtained. The absence of demonstrable cerebral edema or CT scan does not preclude the diagnosis (8,9).

VI. General Management (cookbook)

A. Mistakes and how to avoid them

There are **two major mistakes** one can make in diagnosing and managing DKA:

- 1) Failure to recognize the problem (e.g., the diagnosis of new onset diabetes, cerebral edema, hypokalemia, etc.), and
- 2) Failure to react to the situation, whether the problem is due to the natural course of the disease, or secondary to therapy.
 - a. Keep a **flow sheet** for fluids, insulin, vital signs, lab values, etc.
 - b. Record all **intake and output** meticulously.
 - c. EKG monitor for K⁺ changes if severe acidosis or elevated K⁺.
 - d. Urinary catheter only if unconscious. If conscious, ask patient to void every hour. In the young child, weigh the diapers hourly.
 - e. Intracath for frequent blood draws when hydration allows.
 - f. **Check pupils and sensorium** hourly (for cerebral edema).

B. Fluids

1. **Initial volume expansion** = 10 to 20cc/kg (300-600cc/m²) of a physiologic solution (such as saline or lactated Ringers solution) over the first one to two hours.

This may need to be repeated if the patient is severely dehydrated and/or if urine output is massive. However, **the initial bolus re-expansion should never exceed 40 cc/kg as a total fluid dose for the first four hours of treatment.**

2. 24 hour fluid therapy

a. **Replacement:** Use estimates of dehydration based on physical exam varying from **5 to 10% of body weight for mild to severe losses. Deficits should be replaced evenly over 48 hours.** Remember to subtract the quantities given in the first hours of re-expansion from the 24 hour totals. Follow urine output to be certain initial estimates are adequate. Replacement of urine output (“cc” for “cc”) is generally not required, since excessive urine output should resolve within the initial 2 to 4 hours of therapy as the hyperglycemia resolves. Total fluid replacement should not exceed 4 L per square meter per 24 hours.

b. Maintenance

body weight (kg)	24 hour fluid maintenance requirements
up to 10	100 ml/kg
10 to 20	1000 ml + 50 ml/kg over 10 kg
>20	1500 ml + 20 ml/kg over 20 kg

c. Special additional losses

Additional replacement may be required where there is severe vomiting, etc.

3. **Monitoring fluid requirements** In the severely dehydrated child, or the one with mental status compromise, monitoring fluid administration with a CVP and/or arterial pressure monitoring may be required to ensure adequate fluid replacement.

C. Insulin

1. **No insulin should be given until a blood glucose level has been obtained.** Blood sugar can be checked at the bedside with a glucose meter.
2. Insulin therapy can be **started immediately, but must be started no later**

than immediately after the initial rehydration bolus. The serum glucose level falls fairly rapidly during volume re-expansion with or without insulin. **An initial IM or IV insulin bolus should not be given** since this increases the rate of initial glucose fall without decreasing the time required to correct the acidosis.

3. Continuous **IV regular insulin is given at a dose of 0.1u/kg per hour.** Because insulin will bind to the walls of the IV tubing, the tubing is first washed with 50 ml of the insulin solution. IV insulin provides a relatively smooth decline in blood glucose levels with a predictable time to expect a blood sugar of 300mg%.
4. Aim to have blood glucose level decrease by 50-100 mg %/hr.
5. **When blood glucose falls to 250mg% add dextrose to the IV solution.** The IV solution can be changed to $\frac{3}{4}$ or $\frac{1}{2}$ normal saline at this time.
6. Aim to keep glucose between 150-300 mg % by addition of 5 to 15% Dextrose. Unless a patient is truly hypoglycemic, however, the insulin drip should not be decreased to less than 0.05u/kg/hr as insulin is essential for preventing continued ketogenesis, IV insulin should not be discontinued until the HCO₃ >15 mEq/L

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D. Electrolytes

1. Potassium: K⁺ is a special problem because high urinary losses occur in association with normal serum levels caused by the intracellular exodus of K⁺ in the presence of acidosis. Vomiting may also contribute to hypokalemia. **Total body potassium is usually depleted, but serum levels may be normal or high. As acidosis is corrected, K⁺ is driven back into the cells and there is usually a fall in serum K⁺ in spite of large K⁺ replacements. Low or high serum potassium levels can be a cause of cardiac arrhythmias, which can be fatal.**

- a. **Potassium must never be given until the serum potassium level is known.**
- b. Once the serum potassium is known to be normal or low, and after voiding is observed, **generally after the first hour of fluid resuscitation, all IV fluids should include 20-40 mEq/L of potassium.** If the serum potassium is high, it is best to wait to add K⁺ to the IV until the K⁺ begins to decrease. The potassium may be in the form of KCl, KAc, K₂HPO₄ or a combination of these supplements, no more than half of the potassium replacement should be given as PO₄. **Do not give K⁺ as a rapid IV bolus or cardiac arrest may result. Severe hypokalemia may lead to respiratory arrest due to muscle dysfunction.**
- c. EKG strips (Lead II) may give the best indication of total body K⁺ deficit or change.

2. Sodium: Initial serum Na⁺ is frequently low for several reasons: 1) Depletion secondary to urinary losses/vomiting, 2) Hyperglycemia creates an osmotic dilution of extracellular solute so that for each 100 mg% increase in glucose above a 100 mg% baseline, there is an expected decrease of 1.6 mEq/L of Na⁺, 3) Hyperlipidemia displaces water in the lab method used, causing serum Na⁺ to be factitiously low .

Laboratory hyponatremia will correct with resolution of hyperglycemia and

ketonemia. Total body Na⁺ deficit is approximately 10 mEq/kg based on a dehydration estimate of 10%. Because of the factitious Na deficit, the sodium deficit is not usually calculated. It is important to follow the serum sodium during therapy to make certain the level is rising. A falling serum sodium may be associated with cerebral edema and impending herniation. Usually initial treatment with isotonic saline, or lactated Ringers followed by ½ or ¾ physiologic saline will adequately replace Na⁺ deficit.

3. Phosphorus: Uncontrolled diabetes causes an increased urinary excretion of phosphorus.

- a. Serum phosphorus may, like potassium, be elevated initially in diabetic acidosis, only to fall rapidly during therapy.
- b. Hypocalcemic tetany has occurred with excessive phosphorus administration.**
- c. Clinical problems due to low phosphorus are not proven, but there is some evidence that neurologic disturbances may respond to raising the serum phosphorus level when it is very low (<1mg/dl) (normal adult phosphate =3-5mg/dl).
- d. On theoretical grounds, a low phosphorus may lead to a low red cell 2, 3 DPG causing a shift of the O₂ dissociation curve to the left, creating a relative tissue hypoxia.
- e. Treatment may be administered as KH₂ PO₄ at 10-20 mEq/L in IV solutions (see potassium therapy above).

4. Calcium: Hyperglycemia also causes increased urinary calcium loss. Because of the large calcium reservoir in bone, serum calcium usually remains normal. Excessive phosphorus administration may result in hypocalcemia due to suppressed PTH.

E. Osmolality: Hyperosmolality always accompanies DKA. During treatment, serum osmolality may decrease more rapidly than CNS osmolality, resulting in fluid shifts into the CNS. This may cause life threatening cerebral edema. Excessive, rapid fluid administration may increase the risk for cerebral edema.

1. Serum osmolality should not be rapidly decreased or CNS damage may result.
- 2. If the serum osmolality is very high (greater than 320 mOsm/L), the elevated blood glucose and dehydration should be corrected cautiously with special attention to neurological status. However, severe dehydration must be steadily corrected to prevent circulatory collapse.**
- 3. An 18 mg% rise in blood glucose yields a 1 milliosmol rise in serum osmolality.**
- 4 The osmolality can be calculated by: $2(\text{Na}+\text{K}) + \text{glucose}/18+\text{BUN}/2.8$. Measured or calculated serum osmolality should be followed if the initial osmolality is >320 mOsm/L.**
5. Hyperosmolar, non-ketotic coma is different than diabetic ketoacidosis. It should not be treated as outlined in this paper. Fortunately, this condition is rare in childhood. When it occurs in a pediatric aged patient attention still needs to be given not to decrease the osmolality too rapidly. **Children, in contrast to adults, develop cerebral edema if their rehydration is undertaken too rapidly, even in hyperosmolar non-ketotic coma.**
6. Overweight or obese children may make estimation of dehydration difficult. A measured serum osmolality should always be obtained in the overweight patient with either DKA or hyperosmolar, non-ketotic conditions.

7. Monitoring intravascular pressure, cardiac rhythm, and osmolality is essential for patients with HHS.

F. Acid-base: The cause of the acidosis is ketogenesis from insulinopenia. Correction of this will reverse the acidosis.

Recent reports (16-18 & 25) confirm that bicarbonate therapy is not necessary even in severe DKA (pH <7.1).

Arguments against the use of bicarbonate revolve around 4 issues:

- 1) Bicarbonate therapy causes a paradoxical CNS acidosis and decreases CNS oxygenation. Bicarbonate crosses the blood-brain barrier slowly, but the CO₂ formed (from HCO₃ + H⁺ → H₂O + CO₂) crosses rapidly into the CNS forming H₂CO₃, thereby **accentuating rather than reducing the CNS acidosis.**
- 2) Use of bicarbonate will lead to a more rapid initial correction of acidosis with resultant intracellular movement of K⁺ and hypokalemia. Potassium replacement requirements are 2 to 3 times greater in patients treated with bicarbonate.
- 3) Rapid infusion of bicarbonate and correction of acidosis may shift the oxygen dissociation curve to the left, thereby decreasing oxygen delivery to the tissues.
- 4) Several reports (including Glazer, et al, NEJM 344:264, 2001) have found a greater likelihood of cerebral edema when HCO₃ is given.

G. Ketones: Acetoacetate and a small amount of acetone are measured by the urinary “dipstick” reactions as used by the clinician or laboratory. Beta-hydroxybutyrate is not measured by this method and is usually the major ketoacid in DKA. As treatment is begun, B-OH butyrate is oxidized to acetoacetate so ketonuria may appear to worsen initially. Urinary determinations are markedly affected by hydration status and urine output. A bedside meter, the Precision Xtra, is now available which can be used to estimate serum B-OH butyrate levels. Serum B-OH levels >3.0 mmol/L are usually indicative of severe DKA (nl = <0.6mmol/L) Serum ketones usually disappear at or about the same time the venous pH reaches a level of 7.30. Following bedside B-OH butyrate levels may be helpful. They can be extremely useful, if available at home, in determining if an ill child requires ED therapy. Repeating urine ketones is not necessary.

VII. Following the patient

1. Use a flow sheet.
2. VS q one hour until stable.
3. Accurate record of hourly fluid intake and output.
4. Neurologic checks every hour (at least pupil size and sensorium) until metabolically stable.
5. Lab tests: Bicarbonate (bicarbonate < 10 mm/L), glucose, K⁺ every hour for 4 hours, hourly pH until pH >7.1; then every 2-4 hours until HCO₃=15, and the pH is >7.3. Also do a blood sugar at the bedside with each blood draw.

VIII. The next day, or when ketoacidosis is resolved and the patient is ready to eat, routine diabetes care can be initiated or resumed. **IV dextrose must be discontinued when the insulin infusion is discontinued.**

A. Diet: Order a constant carbohydrate appropriate for age - 1000 calories for 1st year of life. Add in 100 calories/year for each year thereafter up to 2500 K Cal/24 hours. **May need to increase this by 25-50% if significant weight loss has occurred.** The diet is given as 3 meals and 3 snacks, the dietitian will provide the same grams of carbohydrate

at each meal and snack. The dietary staff will help to determine the appropriate caloric content of the diet.

B. Insulin: The $1/2$ life of IV insulin is 6 minutes in blood and 30 minutes in tissue so insulin infusion need not be discontinued until sub-q insulin is given.

1. Sub-q insulin dose

- a. known diabetic - return to usual dose the morning after the acidosis is corrected. Do not waste a day using a sliding insulin scale. The usual dose may need to be supplemented with additional rapid acting insulin (lys-pro,aspart or glulisin insulin) since there is some insulin resistance following an episode of DKA.
- b. new diabetic - start on recombinant human insulin
In hospital: $3/4 - 1$ u/kg/day = total insulin dose
 $2/3$ of dose in AM $1/3$ of dose in PM
 $1/4 - 1/3$ Lys-pro $3/4 - 2/3$ NPH before breakfast
& $1/4$ to $1/2$ Lys-pro $1/2$ to $3/4$ NPH before supper
In established patients and in select new onset patients, glargine insulin may be used at night instead of NPH. Glargine cannot be mixed with other insulins due to pH incompatibility.
- c. monitoring the dose: obtain glucose levels at times when different insulins are expected to peak or an insulin dose is to be given(e.g. before meals, at bedtime and at 2 AM) or anytime there are hypoglycemic symptoms.

C. Potassium: Since patients are often total body K^+ depleted, continued supplementation with oral K may be necessary for several days.

SUMMARY OF SUGGESTIONS FOR THE MANAGEMENT OF KETOACIDOSIS

A. At the onset:

1. Weigh the patient prior to starting treatment.
 2. Do pulse and blood pressure and capillary filling time on the patient yourself as an estimate of shock rather than relying on someone else's values.
 3. Administer plasma or other colloid to a patient in shock.
 4. Do not administer more than 20 cc's/kg as total bolus without careful consideration of fluid status and neurological status. More than 40cc's/kg should never be given in the first 4 hours.
 5. Start insulin treatment after the blood sugar and urine ketones are determined and initial resuscitation fluid bolus has been given.
 6. Always know the blood glucose prior to starting insulin therapy.
 7. Know the blood pH and serum potassium prior to starting potassium therapy.
 8. Ask the lab to check for lactic acid when the sodium level is very low.
- Na correction for elevated glucose: $Na = \text{lab Na} + (1.6)(\text{serum glucose}-100)/100$**
9. Determine osmolality in a patient with a very high blood glucose (above 800 mg/dl), or severe dehydration and aim to decrease the osmolality gradually (10 mOsm/hr).

The osmolality can be calculated by:

$$2(\text{Na}+\text{K}) + \text{glucose}/18+\text{BUN}/2.8$$

B. During the treatment:

10. Have the nurses run 50 ml of insulin solution through the IV tubing and buretrol prior to starting insulin therapy.
11. Keep an **accurate flow sheet** including lab values, precise intake and output records.
12. Check blood glucose levels every one hour initially and then every two hours with the goal to decrease the value at a rate of 50 to 100 mg/dl/hour.
13. **Add glucose to the IV when the blood glucose level falls below 250 mg/dl** so that insulin therapy can be continued to help stop hepatic ketone formation and promote hepatic glucose uptake. The IV fluid at that time may be changed to 1/2 or 3/4 physiological saline. **Usually 1.5 times maintenance fluid rate is appropriate at that time, but calculation of deficit, ongoing losses, and maintenance requirements should be done.**
14. Check neurologic status at regular intervals as a sign of possible early cerebral edema.

C. When ready to discontinue IV therapy:

15. Make sure the HCO₃ is above 15 or the pH is above 7.3 prior to discontinuation of IV fluids.
16. Make sure the sodium and potassium are normal prior to discontinuation of IV fluids.
17. Discontinue IV insulin when the subcutaneous insulin is administered.
18. **Stop the IV glucose when the insulin drip is stopped** so that the blood glucose levels do not become high and cause diuresis.
19. Weigh the patient prior to discharge to have a baseline for possible future problems and calculation of insulin dose.

For previously diagnosed patients:

20. Oral fluids should be tolerated prior to discharge.
21. Make sure the patient is checking in with a diabetes care provider at a set time after discharge.
22. Make an appointment for the patient to see their diabetes-care-provider in the week after treatment, so that education and changes in therapy can be given to help prevent future similar episodes.

References:

1. Chase HP, Rainwater NG: Missed insulin injections: A common syndrome. *Pract Diabetol* 8:20-23, 1989.
2. Bureau MA, Begin R, Berthiaume Y, Shapcott D, Khoury K, Gagnon N: Cerebral hypoxia from bicarbonate infusion in diabetic acidosis. *J Pediatr* 96:968, 1980.
3. Fisher JN, Kitabchi AE: A randomized study of phosphate therapy in the treatment of diabetic ketoacidosis. *J Clin Endo Metab* 57:177, 1983.
4. Krane EJ, Rockoff MA, Wallman JK, Wolfsdorf JL: Subclinical brain swelling in children during treatment of diabetic ketoacidosis. *New Eng J Med* 312:1147, 1985.
5. Harris GD, Fiordalisi I, Harris WL, Mosovich LL, Finberg L: Minimizing the risk of brain herniation during treatment of diabetic ketoacidemia: A retrospective and prospective study. *J Pediatr* 117:22-31, 1990.
6. Hoffman, WH, Steinhart, CM, Gammal, TE, et al.: Cranial CT in children and adolescents with diabetic ketoacidosis. *AJNR* 9:733-739, 1988.
7. Durr, JA, Hoffman, WH, et al.: Correlates of brain edema in uncontrolled IDDM. *Diabetes* 41:627-32, 1992.
8. Pinhas-Hamiel O, Dolan LM, Zeitler PS: Diabetic ketoacidosis among obese African-American adolescents with NIDDM. *Diabetes Care* 20(4):484-6, April 1997.
9. Finberg, L: Fluid Management of Diabetic Ketoacidosis. *Pediatric Review* 17(2):46, 52, February 1996.
10. Finberg, L: Why Do Patients With Diabetic Ketoacidosis Have Cerebral Swelling, and Why Does Treatment Sometimes Make It Worse? *Pediatric Adolescent Medicine* 150(8):785-6, August 1996.
11. 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. *Annals Emergency Medicine* 31(1):41-8, January 1998.
12. Okuda Y, Adroque HJ, Field JB, Nohara H, Yamashita K: Counterproductive Effects of Sodium Bicarbonate in Diabetic Ketoacidosis. *Journal of Clinical Endocrinology and Metabolism* 81(1):314-20, January 1996.
13. Viallon A, Zeni F, Lafond P, Venet C, Tardy B, Page Y, Bertrand JC: Does Bicarbonate Therapy Improve the Management of Severe Diabetic Ketoacidosis? *Critical Care Medicine* 27(12), December 1999.
14. Mahoney CP, Vlcek BW, DelAguila M: Risk Factors for developing Brain Herniation During Diabetic Ketoacidosis. *Pediatric Neurology* 21:721-727, 1999.
15. Hoffman WH, Locksmith JP, Burton EM, Hobbs E, Passmore GG, Pearson-Shaver AL, Deane DA, Beaudreau M, Bassali RW: Interstitial Pulmonary Edema in Children and Adolescents with Diabetic Ketoacidosis. *Diabetes Complications* 12(6):314-20, Nov-Dec 1998.
16. Holsclaw DS Jr, Torcato B: Acute Pulmonary Edema in Juvenile Diabetic Ketoacidosis. *Pediatric Pulmonology* 24:438-443, 1997.
17. Buyukasik Y, Ileri NS, Haznedaroglu IC, Karaahmetoglu S, Muftuoglu O, Kirazli S, Dundar S: Enhanced Subclinical Coagulation Activation During Diabetic Ketoacidosis. *Diabetes Care* 21(5):868-869, May 1998.
18. Vanelli M, Chiari G, Ghizzoni L, Costi G, Giacalone T, Chiarelli F: Effectiveness of a Prevention Program for Diabetic Ketoacidosis in Children. *Diabetes Care* 22(1):7-9, January 1999.
19. Duck SC, Wyatt DT: Factors Associated with Brain Herniation in the Treatment of Diabetic Ketoacidosis. *Journal of Pediatrics* 113:10-14, 1988.
20. Glaser N, Barnett P, McCaslin I, Nelson D, Trainor J, Louie J, Kaufman F, Quayle K, Roback M, Malley R, Kuppermann N: Risk Factors for Cerebral Edema in children with

diabetic ketoacidosis. The Pediatric Emergency Medicine Collaborative Research Committee of the American Academy of Pediatrics. *N Engl J Med*, 344(4):264-9, January 2001.

21. Roberts MD, Slover RH, Chase HP: Diabetic Ketoacidosis with Intracerebral Complications. *Pediatric Diabetes*, 2:109-114, 2001.
22. Dunger et al: European Society for Pediatric Endocrinology/Lawson Wilkins Pediatric Endocrine Society Consensus on Diabetic Ketoacidosis in Children and Adolescents. *Pediatrics* 113(2):e133-e140, February 2004.
23. Felner E, White P: Improving Management of Diabetic Ketoacidosis in Children. *Pediatrics* 108(3):735-740, September 2001.
24. Glaser NS, Wootton-Gorges SL, Marcin JP, Buonocore MH, Dicarolo J, Neely EK, Barnes P, Bottomly J, Kuppermann N. Mechanism of cerebral edema in children with diabetic ketoacidosis, *J Pediatr*. 2004 Aug;145(2):164-71.
25. Muir AB, Quisling RG, Yang MC, Rosenbloom AL. Cerebral edema in childhood diabetic ketoacidosis: natural history, radiographic findings, and early identification, *Diabetes Care*. 2004 Jul;27(7):1541-6.
26. Wolfsdorf J, Glaser N, Sperling MA. Diabetic Ketoacidosis in Infants, Children, and Adolescents. A consensus statement from the American Diabetes Association. *Diabetes Care* 2006 May;29(5):1150-1159.
27. Edge JA, Yvonne R, Bergomi A, Murphy NP, Ford-Adams, ME, Ong KK, Dunger DB. Conscious level in children with diabetic ketoacidosis is related to severity of acidosis and not to blood glucose concentration. *Ped Diab* 2006; 7:11-15.
28. Fiordalisi I, Novotny WE, Holbert D, Finberg L, Harris GD, and the Critical Care Management Group. An 18-year prospective study of pediatric diabetic ketoacidosis: an approach to minimizing the risk of brain herniation during treatment. *Ped Diab* 2007; 8 (3):142-149.

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