

A New Protocol for Diabetic Ketoacidosis: The Same Mistakes

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Dear editor of the Portuguese Journal of Pediatrics, I have been waiting for the new protocol for the treatment of diabetic ketoacidosis from the Portuguese Society of Pediatrics with great expectation. Unfortunately, I am concerned that the protocol¹, which was published on the official site of the Sociedade Portuguesa de Pediatria on May 20, 2019, has some serious mistakes. Some of them are potentially fatal if the recommendations are followed to the letter.

I have decided to write this letter because I have witnessed several patients being treated according to the referred protocol over the last year without sound evidence supporting many of its recommendations. Some of them even go against best practice for this condition.

I wanted to draw attention to the mistakes that I consider more serious with a personal commentary justifying the reason why the recommendation is incorrect:

1. In **point 6.1 Fluid therapy** “Dehydration varies between 5% and 7% in moderate diabetic ketoacidosis and between 7% and 10% in severe situations. This should be corrected over 24 to 48 hours (72 hours in case of cerebral edema), together with basal needs using normal saline or **sodium chloride (NaCl) 0.45%** or Ringer’s lactate.”

Commentary: Hypotonic solutions increase the risk of cerebral edema and should be avoided in conditions that have a high risk of cerebral edema, as is the case with diabetic ketoacidosis. It is dangerous to recommend a hypotonic solution (NaCl 0.45%) as the initial fluid therapy. There are many good alternatives on the market for **isotonic solutions** (e.g. Plasma-Lyte 148, Ringer’s lactate) that would be much more adequate and would prevent the hyperchloremic acidosis associated with normal saline. The exception would be a patient with hyperkalemia since most of these solutions have potassium in their composition. In such a case, normal saline would be the safest initial solution.

2. In the same **point 6.1 Fluid therapy** “In order to prevent an abrupt decrease in blood glucose levels, when the insulin intravenous (IV) infusion is started, NaCl 0.9% or NaCl 0.45% **should contain 5% dextrose**”.

Commentary: It makes no sense to start a solution with 5% dextrose in a patient with severe hyperglycemia. I do not know of any protocol or guideline that recommends it. Most guidelines suggest adding dextrose to the fluid therapy when the blood glucose levels decrease to less than 250-300 mg/dL or when it decreases more than 100 mg/dL in one hour.

For example, a child with a blood glucose level of 800 mg/dL according to this protocol would start a solution with 5% dextrose at a very high rate. I have witnessed cases where this was done and then the blood glucose levels would not decrease or even increased.

I find it very useful to have two solutions with simultaneous perfusion, one with dextrose and another one without. Depending on the rate of the blood glucose level decrease, we can adjust the rates of both solutions to achieve a steady and safe decrease in the blood glucose levels. It is much better than all or nothing.

3. In **point 6.2 Potassium** “**40 mmol/L** added to the IV solution bag”.

Commentary: Although there are some recommendations that limit the potassium concentration to 40 mmol/L for peripheral IV infusion, it is possible, and many times necessary, to use higher concentrations. It is safe to administer a concentration of up to **60-80 mEq/L of potassium** in a peripheral vein, particularly in patients with hypokalemia.

4. In **point 6.4 Bicarbonate** “Because of the high risk of hypokalemia, the administration of bicarbonate is not recommended, except in the presence of severe acidosis (**venous pH < 6.9**) or potentially fatal hyperkalemia with an evident compromise of the cardiac contractility”.

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Commentary: pH for itself is not an indication for bicarbonate administration. If the patient has a pH < 6.9 and is hemodynamically stable, **bicarbonate administration should be avoided** because it has been shown to increase the risk of cerebral edema.

5. In **point 7.1 Cerebral edema** “In the presence of respiratory failure secondary to neurological compromise, intubate the patient, being careful not to hyperventilate and keeping a partial pressure of carbon dioxide (pCO₂) > 35 mmHg”.

Commentary: Invasive ventilation in a patient with diabetic ketoacidosis is extremely challenging and complex. Nonetheless, **it is mandatory to hyperventilate the patient and to try to keep a pCO₂ similar to the one the patient had in spontaneous ventilation** to avoid a dramatic decrease in pH and the worsening of the acidosis. For example, in a patient with pH 7.15 and pCO₂ of 15 mmHg that was ventilated according to this protocol to a pCO₂ of 35 mmHg, pH would immediately decrease to 6.78.

I hope that these commentaries might contribute to the better care of patients with diabetic ketoacidosis

and that my colleagues who treat these patients have a critical vision of the present protocol.

The most severe complications of diabetic ketoacidosis are mainly iatrogenic, namely cerebral edema, hypokalemia, hypoglycemia, and vascular thrombosis. The more serious the presentation is, the more careful we should be in its treatment.

Fortunately, most patients with diabetic ketoacidosis have an excellent short-term prognosis. *Primum non nocere*.

Note: The references (1 to 5) to the protocol have been translated to English by the author.

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Conflicts of Interest

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