Letter

http://dx.doi.org/10.6065/apem.2016.21.2.111
Ann Pediatr Endocrinol Metab 2016;21:111-112

Annals of Pediatric Endocrinology & Metabolism
apem

The Author Reply: Endocrine and metabolic emergencies in children: hypocalcemia, hypoglycemia, adrenal insufficiency, and metabolic acidosis including diabetic ketoacidosis

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To the editor:

I thank Dr. Rosival, who provided the interests and comments on my paper "Endocrine and metabolic emergencies in children ...."[1]. Dr. Rosival questioned about absolute and relative insulin deficiency of the patients with diabetic ketoacidosis (DKA), and why bicarbonate therapy should be sparing for patients with DKA.

1. Insulin deficiency

Dr. Rosival commented on serum insulin level of the patients with DKA citing several references. Of course, the patient who shows normal concentration of serum insulin could be developed full-blown DKA. Even if insulin levels were checkable, it meant relative insulin deficient status, because those levels of insulin itself couldn't overcome the overwhelming hyperglycemic effects of counter regulatory hormones (cortisol, epinephrine, growth hormone, and glucagon) during stress (infection, trauma, and surgery)[1]. The basic underlying mechanism for DKA is a reduction in the net effective concentration of circulating insulin, coupled with a concomitant elevation of counter regulatory stress hormones[2]. In patients with DKA, the deficiency in insulin can be absolute, or it can be insufficient relative to an excess of counter regulatory hormones[2].

Also, a lot of new patients have been diagnosed as type 1 diabetes mellitus initially through DKA due to absolute insulin deficiency[3]. DKA has been increasingly noted in newly diagnosed obese type 2 diabetic patients[4].

2. Bicarbonate therapy

Bicarbonate therapy should be considered in severe acidosis (pH<6.9) due to DKA[1]. Acidosis of the patients with DKA is reversible by fluid and insulin replacement. Insulin stops further ketoacids production and allows ketoacids to be metabolized, which generates bicarbonate. Treatment of hypovolemia improves tissue perfusion and renal function, increasing the excretion of organic acids. Controlled trials have shown no clinical benefit from bicarbonate administration[5-8], and there are well recognized adverse effects of bicarbonate therapy, including paradoxical central nervous system acidosis[9] and hypokalemia from rapid correction of acidosis[9]. Failure to account for the sodium being administered and appropriately reducing the NaCl concentration of the fluids can result in increasing osmolality[9]. Nevertheless, there may be selected patients who may benefit from cautious alkali therapy. These include patients with severe acidemia (arterial pH<6.9), in whom decreased cardiac contractility and peripheral vasodilatation can further impair tissue perfusion, and patients with life-threatening hyperkalemia[10]. Therefore, routine bicarbonate administration for patent with DKA is not recommended unless the acidosis is profound.

Received: 19 June, 2016
Accepted: 24 June, 2016

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ISSN: 2287-1012(Print)
ISSN: 2287-1292(Online)

I was trying to describe condensing a bunch of essential facts of the clinically important contents of endocrine emergencies into a short paper[1], which is readable than tedious longer one. It has, therefore, short explanation of pathophysiology of DKA. Dr. Rosival pointed out 2 issues (insulin and bicarbonate) and led us to consideration of the core pathogenesis of DKA.

Conflict of interest

No potential conflict of interest relevant to this article was reported.

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