Brief Communication

EUGLYCEMIC KETOACIDOSIS

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SUMMARY

The purpose of this ariticle is to highlight the importance of not relying exclusively on blood glucose measurements when assessing sick type I diabetics. Urinary ketones and venous bicarbonate are essential in making the diagnosis of relatively low blood glucose plus ketoacidosis, that we call euglycaemic ketoacidosis.

KEY WORDS: Euglycaemic Ketoacidosis.

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INTRODUCTION

The condition is defined as diabetic ketoacidosis with a bicarbonate level of less than 10mEq/L and a glucose level of less than 16.7mmol/l or 300mg% along with ketonemia or ketonuria, Euglycemic ketoacidosis occurred in 30% of patients admitted to the hospital on account of diabetic ketoacidosis.[1] True euglycaemic ketoacidosis (initial blood glucose 10mmol/l (180mg%) or less) is rare, occurring in 0.8–1.1% of all episodes depending on the defining plasma bicarbonate concentration.[2] Clinically Euglycaemic ketoacidosis is usually manifested by vomiting. In addition some patient can present with, abdominal pain dysuria, productive cough, thirst, nausea.

The cause of preserved "euglycemia" could be greater urinary loss of glucose triggered by counter-regulatory hormones or decreased rate of hepatic glucose production observed during a fast. It has also been demonstrated that the key path physiologic determinant is the quantity of food ingested prior to development of diabetic ketoacidosis: When patients are well fed, their liver contains large amounts of glycogen, which primes it to make glucose and to suppress ketogenesis. However, when patients have been vomiting and unable to eat, the liver is depleted of glycogen and primed to produce ketones. Thus, patients with euglycemic ketoacidosis are usually in the fasting state before they become ill. Because gastroenteritis, nausea, and vomiting often precede euglycemic ketoacidosis, it is not uncommon in association with excessive vomiting and continued insulin administration. The clinical picture and biochemical profile were typical of diabetic ketoacidosis, although if only the blood glucose level at presentation had been taken into account the unwary would have missed the diagnosis.[3]

MANAGEMENT

Management of euglycaemic ketoacidosis with low-dose continuous intravenous infusion of insulin together with adequate fluid replacement is effective. The principles of treatment are to correct the fluid and electrolyte loss and to re-establish carbohydrate metabolism. Sufficient exogenous glucose must also be given to restore normal cellular utilization and thus

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reverse the ketoacidosis. Normal saline should therefore be avoided and dehydration corrected with 5% dextrose/saline and 5% dextrose. When the blood glucose is low in the absence of clinical dehydration 10% dextrose may be required. It has been recommended that the dose of insulin should be proportional to the blood glucose level. Response to therapy is assessed very largely by the serum bicarbonate and ketones.

Conclusions: This article explains the reasons for relatively low blood glucose levels encountered in some patients presenting with Diabetic Ketoacidosis (DKA). Cases of true euglycaemic DKA are rare. This communication emphasizes that a normal, or more accurately near-normal, blood glucose level does not exclude the diagnosis of DKA.

REFERENCES

1. De P, Child DF. Practical Diabetes International 2001;18(7):239-40.
2. Munro F, Campbell IW, McCuish AC,. Duncan LJP. Euglycaemic ketoacidosis BMJ 1973;2(5866):578–80.
3. Burge MR, Hardy KJ, Schade DS- Short term fasting as mechanism for the development of euglycemic ketoacidosis during periods insulin deficiency. J Clinl Endocrin Metabolism 1993;76(5):1192-8.