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Research Article

EUGLYCEMIA; A HIDEOUT FOR DIABETIC KETOACIDOSIS

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ABSTRACT
An episode of diabetic ketoacidosis is normally initiated with hyperglycemia. However, hyperglycemia may not always be an indicator as ketoacidosis can occur with normal glycemic level as well. An elderly female with Chinese origin and history of type II diabetes, hypertension, dyslipidemia and ischemic heart disease was admitted to hospital with complaint of fever, urinary inconsistency and loose stool. Upon investigation, patient had higher values for capillary glucose, platelets and white blood count and lower values of sodium, potassium and red blood count. Treatment was initiated for gastroenteritis, urinary retention and anemia but patient developed metabolic acidosis which got severe after some time. Patient died later with death note of diabetic ketoacidosis secondary to sepsis. Euglycemic diabetic ketoacidosis is a well defined phenomenon. Patient in this case was however mildly hyperglycemic but was not given insulin. Alongside sepsis, increase in white blood count is also an attribute of dehydration and once diabetic ketoacidosis was ruled in, the rate of fluid resuscitation was not maintained as recommended by guidelines. Outcome of all these factors result in the death of patient. Hereby, we emphasize that guidelines for diagnosis of diabetic ketoacidosis should be applied upon every diabetic admission and followed during the management afterwards.
Keywords: Euglycemia, Diabetic Ketoacidosis, Laboratory Profiles, Diagnosis, Guidelines

INTRODUCTION

Acute complications of diabetes such as hypoglycemia or hyperglycemia are life threatening and require more result oriented medical decisions. DKA is usually triggered by hyperglycemia which may be associated with infection, non-compliance/adherence, or trauma of cardio and cerebrovascular origin. Most guidelines for diagnosis of DKA mention hyperglycemia above 13.9mmol/L[1] though ketosis and acidosis may be present with normal glycemic level[2]. Acute complication disguised in another medically attention seeking state, diverts the capacity of physician to a different treatment. Thus, there should be diagnostic criteria for DKA which should be applied on every diabetic patient in order to get a clear picture. Hereby, we present a case in which patient died attributable to euglycemic DKA secondary to sepsis.

Case Report

A 70-year old Chinese female was admitted seeking medical attention for fever, dysuria and loose stool, for 1 day. Patient had history of type II diabetes, hypertension, dyslipidemia and ischemic heart disease, and was being served with 1gm metformin and 10mg glibenclamide, bd; 5mg amlodipine and 25mg hydrochlorothiazide, od with 25mg metoprolol bd; 40mg simvastatin oN; 10mg isosorbide dinitrate td and 150mg aspirin bd. Signs rendered patient being pale, mildly dehydrated and lethargic. Temperature and blood glucose level (BGL) were recorded to be 38.4^0c and 11.3mmol/dL respectively.

Renal profile revealed hyponateremia (Na^+ = 129mmol/L) and hypokalemia (K^+ = 3.1mmol/L) whereas complete blood count gave an idea about sepsis (WBC = 13.5 $\times$ 10^3/µL), normocytic anemia (RBC = 2.78 $\times$ 10^6/µL, Hb = 9.0g/dL, MCV = 98.3fl) and hemorrhage (HCT = 25.9%) with a high number of platelets (669 $\times$ 10^3/µL). Hence impression was made of urosepsis, gastroenteritis and hemorrhage. Treatment was initiated with NS infusion at a rate of 60ml/h and insertion of closed bag drainage (CBD) catheter. Potassium was then added on from 2nd pint of NS. Cefemipe and omeprazole were given to cover sepsis and gastroenteritis. Tramadol and paracetamol were given to cover fever.

At 11th hour from admission, BGL of patient was reported to be 22mmol/L. When attended, patient was found to have SOB, tachypnoea and acidotic breath. Sample of blood was sent for analysis of ketones and blood gasses. Ketones were found to be 5.6mmol/L. DKA was ruled in and patient was infused with 1 pint each of gelafundin and NS at fast rate. Continuous insulin infusion was started at a rate of 8ii/h and NS was then maintained at 85mL/h.

In the meantime, condition of patient deteriorated. PR and BP went unrecordable at 12th hour. Dopamine, dobutamine and later, noradrenaline were started to manage cardiogenic shock which revived BP up to 162/92. At this time, result of venous blood gas revealed that patient had developed metabolic acidosis (pH = 7.13, Actual HCO_3 = 8.1mmol/L) and hence was given 8.4% $NaHCO_3$. However, patient collapsed, received a CPR and was revived after 10 minutes. Patient was subsequently given IV adrenaline and calcium gluconate. Results from 2nd blood sample for ABG (pH = 6.988, Actual HCO_3 = 7.5mmol/l) and CBC (WBC = 23.7 $\times$ 10^3/µL) revealed that patient had undergone severe metabolic decompensation and dehydration. 2 pints of NS were given at fast rate but patient re-collapsed at 13th hour from admission. Second CPR was given for 20 minutes for which cardiac monitor remained asystole and patient was pronounced dead.

DISCUSSION

Munro FJ et al. found that euglycemia in DKA may vary from as low as 39mg/dL to as high as 299mg/dL[2]. Furthermore they developed a notion that with increase of BGL in DKA, Na+ and K+ decrease to as low as 128mmol/L and 3.5mmol/L respectively2. The loss of electrolytes is induced by hyperglycemia and ketosis when body tries to compensate excess of glucose and ketone bodies via urine3. BGL of patient in this case was lying near the upper threshold of euglucemic DKA and patient also had hyponatremia and hypokalemia at the time of admission which renders condition of the patient more closely to an impending episode of DKA. Apart from these initial findings, which left the patient as a suspect for DKA, the sample was still not sent for "urine full examination microscopy elements" (UFEME) once the patient was inserted with CBD catheter. Ketones would have been detected immediately at that time which

actually had resulted in a lot more different treatment protocol. Bacon DK et al. concluded that leukocytosis may be observed along with increase in temperature as a result of acute dehydration[4]. In this case, patient's number of WBC was high at the time of admission and so was the temperature. However, this increase of WBC was translated as sepsis knowing well that dehydration, which is a definite attribute of DKA, might be the reason of leukocytosis as patient was also found to be dehydrated during initial assessment at the time of admission. There are number of studies which concluded that abdominal pain does have a relation with DKA[2, 3, 5, 6]. This relation though is unexplained but severity of pain is observed more likely in patients with a severe metabolic acidosis[5]. Patient, hereby discussed, also complained for having abdominal pain. All of these initial findings point toward euglycemic DKA.

For the part of management, recommended guidelines suggest that intravenous fluid resuscitation should be initiated at 2000mL/h and 1400mL/h for management of DKA and hyperglycemia and then maintained at a rate of 200mL/h and 140mL/h respectively[1]. Considering that the patient was hyperglycemic, fluid replacement even then was not in accordance with the guidelines to manage hyperglycemia[1, 7]. Moreover, once DKA was ruled in, the infusion rate was still half to the rate recommended by guidelines to manage DKA. Furthermore, it is recommended that patient should be started on constant insulin infusion and managed afterwards with help of 5% or 10% dextrose[3, 7]. Yet, insulin was not injected nor was BGL checked for 11 hours.

Except for the fluid reconstitution and insulin, the rest of the protocol followed was according to guidelines. Patient was given $NaHCO_3$ and potassium replacement for management of severe acidosis and hypokalemia[1, 3, 7]. The use of $NaHCO_3$ remains controversial but it is still recommended if patient undergoes severe metabolic acidosis. Patient was also given gelafundin to overcome hypovolemia[8]. Use of dopamine, dobutamine and adrenaline is also in accordance with recommendations to overcome cardiogenic shock[9, 10].

CONCLUSION

Euglycemic DKA was first reported in 1973 and with help of aforementioned case, it is highlighted that more attention should be paid to its attributes. By following this example, one can prevent a minor error which may even end in death. Symptoms and signs are very important during initial assessment for a diabetic patient as a minute slip-up may lead patient's struggling homeostatic physiochemical state to face a completely different treatment protocol. All initial biochemical and laboratory tests should be carried out to rule in DKA such as for ketones and pH/HCO_3 of blood. We also emphasize that there should be a critical guideline for each patient getting admission to hospital, which may help ruling out diabetic ketoacidosis from similar acute diabetic complications.

This research is registered with "National Medical Research Register (NMRR) Malaysia" and approved by the "Clinical Research Committee (CRC) Malaysia.

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