Diabetic Ketoacidosis with Hyponatremia

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ABSTRACT

Hyponatremia is a common electrolyte abnormality caused by an excess of total body water when compared to total body sodium content. Hyponatremia can be classified according to its osmolality, volume status, and urine osmolality. Diabetic ketoacidosis with its high blood glucose will usually cause dilutional hyponatremia. This paper will also discuss classification of hyponatremia and the development of hyponatremia in diabetic ketoacidosis or in hyperglycemic patients.

Keywords: Diabetic ketoacidosis, hyperglycemic, hyponatremia.

ABSTRAK

Hiponatremia adalah gangguan elektrolit karena total air tubuh yang berlebih dibandingkan dengan total jumlah natrium. Hiponatremia dapat diklasifikasikan menurut osmolalitas, status volume tubuh, dan osmolalitas urin. Ketoasidosis diabetik dengan kadar gula darah tinggi biasanya akan menyebabkan hiponatremia dilusional. Pada laporan kasus ini akan dibahas klasifikasi hiponatremia dan kejadiannya pada pasien ketoasidosis diabetik atau pada pasien hiperglikemia. **Irianto. Diabetic Ketoacidosis with Hyponatremia**

Kata Kunci: Ketoasidosis diabetic, hiperglikemia, hiponatremia.

INTRODUCTION

Diabetic ketoacidosis (DKA) results from insulin deficiency with a relative or absolute increase in glucagon; it may be precipitated by inadequate insulin administration, infection, infarction, surgery, trauma, drugs, or pregnancy.[1] It is a potentially life-threatening complication of diabetes mellitus. This report is a case of diabetic ketoacidosis and severe acidemia presumed to be precipitated by inadequate glycemic control and infection, succeeded by hyponatremia after diabetic ketoacidosis resolution, treated in a private type C hospital in Indonesia.

CASE

A 55-year-old male presented to the emergency department with sudden onset of shortness of breath. He has a history of type-2 diabetes and took metformin thrice a day. He was fully alert but tense, blood pressure 160/100 mmHg, heart rate 110 beats/min, respiratory rate 30 breaths/minute with bilateral slight rhonchi, temperature 36,5°C, body weight of 45 kg and dehydrated with dry mucous membrane. The electrocardiogram (ECG) showed sinus rhythm at the rate of 96 beats per minute without any significant findings. Laboratory investigations revealed an elevated white blood count of 20.330 mm3/μL. Urea was slightly elevated at 45:9 mg/dL. Metabolic acidosis was found with arterial pH of 6.884 and bicarbonate of 3.5 mmol/L. Arterial pO2 and PCO2 levels were 113.2 mmHg and 19.3 mmHg respectively. Random blood glucose was high - indicated levels above 600 mg/dL with a ketone serum of 2.7. His sodium, potassium and chloride levels were 131.1 (the normal reference range of sodium is 130-150 mmol/L in our laboratory values), 4.33 and 85 mmol/L respectively. The chest X-ray showed a normal heart contour and a bilateral infiltration in the perihilar lung region, interpreted as bronchopneumonia.

He was diagnosed with diabetic ketoacidosis. This was his second episode of diabetic ketoacidosis. Treatment started with an initial 1 L intravenous normal saline using two IV lines, followed by a 0.1 unit/kg insulin bolus and a 50 mEq potassium replacement over 6 hours. Additional normal saline was also given to a total of 4.5 L over 6 hours, followed by fluid maintenance of 2000 L per 24 hours. The Empirical intravenous antibiotic levofloxacin 500 mg once daily was started to treat bronchopneumonia.

Insulin was given initially with 0.1 unit/kg bolus and maintained with 0.1 unit/kg/h. Blood glucose was checked hourly. He was given an additional 0.1 unit/kg bolus of insulin because his blood glucose didn't decrease more than 50 mg/dL. With fluid resuscitation and continuous insulin drips, his blood glucose started to fall adequately; 25 mEq bicarbonate was given twice through a peripheral IV line.

His shortness of breath was subsiding after his blood glucose reached 200-250 mg/dL. Oral feeding was started while his insulin was maintained at 2 units/hour to slowly solve his acidosis. Repeated serum electrolytes showed normal potassium levels (4.05 mmol/L) and hyponatremia (127.8 mmol/L). The symptoms were gradually resolved.

The hyponatremia was treated with 500 mL

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Table 1. Blood glucose levels during initial DKA treatment

Time	Initial	1st hour	2nd hour	3rd hour	4th hour	5th hour	9th hour	12th hour
Blood Glucose	>600	> 600	444	414	358	238	210	167
Inter-vention	1 liter of normal saline	4.5 unit insulin bolus and 4.5 unit/h maintenance	4.5 unit/h insulin	4.5 unit/h insulin + 4.5 unit bolus	4.5 unit/h insulin	2 unit/h insulin	2 unit/h insulin	1 unit/h insulin

Table 2. Sodium levels in this patient during hospital stay.

Time	Initial	12 hours	36 hours	4 days
Sodium	131.1	127.8	128.9	132.1
Blood Glucose	>600	167	344	187
Intervention	DKA protocol	500 cc of hypertonic saline for 24 hours	Optimizing blood glucose	Discharge

3% hypertonic saline over 24 hours, but the sodium level was only slightly increased to 128.9 mmol/L. On physical examination he was euvolemic, the jugular veins were flat, no edema, wet mucous membrane with normal vital signs. After the stabilization of blood glucose, the patient was discharged. The patient came a week after discharge with no symptoms and then lost to follow-up.

DISCUSSION

Diabetic ketoacidosis (DKA) was diagnosed based on findings of shortness of breath with acidosis, high blood glucose, and ketosis.[2] Bronchopneumonia from his physical and radiographic findings. Since there were no other significant findings, bronchopneumonia was presumed as the trigger of his DKA. The patient's poor glycemic control may also contribute to the condition. The initial sodium level was 131.1 mmol/L; it was in the lower reference range. But, after diabetic ketoacidosis management, hyponatremia was developed.

Hyponatremia is a common finding in diabetic ketoacidosis (DKA).[3] DKA causes a hyperosmolar state driven by the osmotic force of hyperglycemia in the intravascular space; water from body cells was 'pulled' into the intravascular space, resulting in more water than sodium in the intravascular space, causing dilutional hyponatremia.[3] Usually, diabetic ketoacidosis hyponatremia is treated by fluid administration to reduce the hyperosmolar state in the intravascular space, which helped water to move back to the intracellular compartment resolving the dilutional hyponatremia found in DKA.

Hyponatremia is a common electrolyte abnormality caused by an excess of total body water when compared to total body sodium content. Hyponatremia represents an imbalance in the ratio where total body water is more than total body sodium. Symptoms of hyponatremia can range from anorexia, nausea and vomiting, fatigue, headache, and muscle cramps to altered mental status, agitation, seizures, and even coma.[4] Severe symptoms (altered mental status, agitation, seizures) with sodium levels <125 mmol/L need to be given immediate hypertonic saline, while in asymptomatic to mild symptoms of hyponatremia we should evaluate serum osmolality, volume status, urine sodium, and urine osmolality.[5]

First, we should evaluate the serum osmolality of the hyponatremic patient. True hyponatremia patients are hypotonic (below 275 mOsm/L) because hyponatremia represents excess of free water. If it is normotonic (275 mOsm to 290 mOsm/kg), consider pseudohyponatremia. If hypertonic (above 290 mOsm/L) assess for hyperglycemia, mannitol or sorbitol usage, and other hypertonic fluid administration.[4,5]

Then, evaluate the patient's volume status. Evaluate vital signs, orthostatic (lying and upright blood pressure difference), jugular venous pressure, skin turgor, mucous membrane, peripheral edema, blood urea nitrogen, and uric acid levels. Hypovolemic patients with hyponatremia indicate low water and salt levels, euvolemic indicates increased total body water with normal sodium levels and hypervolemic indicates increased total body water.[4,5]

Urine sodium is useful in determining the need for isotonic saline administration. Traditionally, urine sodium of 20 or 30 mmol/L is used as a cutoff value to differentiate whether a patient will respond to an isotonic saline infusion or not. Low urine sodium, especially below 10 mmol/L, indicates extrarenal loss of fluid, which will respond to isotonic saline infusion. Urine sodium greater than 20 or 30 mmol/L suggests renal loss of urine, which usually requires another intervention.[4,6]

Urine osmolality (measured with an osmometer) is useful for the determination of urine diluting ability, which actually reflects vasopressin (antidiuretic hormone, ADH) activity. A value below 100 mOsm/kg suggests that the kidneys' ability to excrete maximally diluted urine remains intact. In this case, the cause of hyponatremia is either primary polydipsia or other underlying psychiatric disorders, such as schizophrenia, which are associated with massive water intake. However, hyponatremia with decreased Uosm can also be observed in individuals who consume large amounts of fluid but small amounts of salt and protein. This results in a limited amount of solutes being excreted and leads to decreased water excretion. This phenomenon has been reported in ill-nourished heavy drinkers (beer potomania syndrome) but also in the elderly with increased water and low solute intake (for example, tea and toast diet). A value above 100 mOsm/kg is usually present in syndrome of inappropriate antidiuretic hormone, hypothyroidism, adrenal insufficiency and drug usage.[4,5,7]

All in all, hyponatremia is largely a symptom, not a disease. Hyponatremia findings can help us in cluing where the pathology is, and usually the resolution of the pathology will also resolve the hyponatremia. We also need to determine whether it is true hyponatremia or it is pseudohyponatremia from the electrolyte testing method and additional lab testing described above to guide the treatment of hyponatremia.

Electrolyte measurement is usually done by direct or indirect testing. Most commonly with indirect ion selective electrode (ISE) testing, which after a dilution step, does not take into account the real percentage of plasma water in sodium concentrations determination.[5] Indirect ISE measures electrolyte content in total plasma volume as opposed to direct ISE which measures electrolyte content in the plasma water. Total plasma contains water and solid components (protein and lipids), a variation of proteins and lipids may cause errors in the reported electrolyte results from the indirect ISE method.[6]

In our case, we don't measure serum osmolality, but calculate serum osmolality with the Smithline-Gardner formula formula: 2(Na) + glucose/18 + BUN/2.8,[10] which result in 311.8 mOsm/L. The diagnosis is hypertonic hyponatremia caused by diabetic ketoacidosis/hyperglycemia, the treatment is to treat the DKA and stabilize the blood glucose, and the hyponatremia usually will resolve.[5]

CONCLUSION

We reported a case of a 55 year-old patient with diabetic ketoacidosis. The diagnosis was made by history, physical examination and laboratory findings. The diagnosis was a simple DKA with mild hyponatremia after its resolution with unknown causes.

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