International Journal of Research in Medical Sciences
Pratikto BC et al. Int J Res Med Sci. 2023 May;11(5):1792-1795
www.msjonline.org
pISSN 2320-6071 | eISSN 2320-6012

DOI: https://dx.doi.org/10.18203/2320-6012.ijrms20231357

Case Report

A case report: management of hypernatremia in diabetic ketoacidosis patient with type II diabetes mellitus

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Received: 11 March 2023
Revised: 11 April 2023
Accepted: 12 April 2023

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ABSTRACT

Diabetic ketoacidosis (DKA) is a life-threatening hyperglycemic crisis. DKA rarely presents with hypernatremia, so it is not a common case. We report an unusual case of a 47-year-old woman with type 2 diabetes mellitus (T2DM) admitted with blood glucose 654 milligrams/deciliter and serum sodium 147 millimoles/liter. Patient was transferred to ICU for further management. Patient was given initial bolus of normal saline and switched to D5-1/4 saline for correction of hypernatremia along with insulin therapy. The patient gradually recovered. This report reminds us about how we choose the right treatment to correct the hypernatremia in patient with DKA and also reminds that monitoring the electrolyte levels in hyperglycemia is relevant in the management of diabetes.

Keywords: Case report, DKA, Hypernatremia, T2DM

INTRODUCTION

Diabetic ketoacidosis or DKA is a complication of type 1 and T2DM which is caused by decreased insulin levels in the blood due to increased levels of glucose produced by the liver and kidneys. DKA complications appear more frequently in patients with T2DM, usually children. However, this does not rule out the possibility for patients with T2DM to have complications from DKA. DKA is an acute complication of diabetes mellitus where morbidity and mortality continue to increase.[1] A study in the United States showed that there were 220,340 cases of DKA in the United States and 37.9% of them died.[2] In Indonesia, studies on DKA are still rare and can only be found in studies at certain hospitals. Based on a study at Soetomo general hospital in East Java, there were 316 DKA patients during the period January-December 2017 with 66.67% being women. There was an increase in cases of DKA in Indonesia from 63-71% in 2015-2017, and caused 40-57.14% of deaths.[3]

In a study from January 1, 2017 to December 31, 2019 which took place at the Wangaya Hospital Denpasar medical record installation, 33 medical record data were obtained which could be used as research samples. In 33 patients with DKA with T2DM, 11 people (33.3%) were found to be 46-55 years old. More were found in female patients, there were 18 people (54.5%). Most of the random blood sugar levels found in patients were in the range of 451-550 mg/dL, there were 9 people (27.3%). The highest blood pH levels were found in the range of 7 to 7.24, there were 14 people (42.4%).[4]

DKA continues to be a severe medical emergency requiring intensive care unit (ICU) admission in most cases.[5] DKA consists of a biochemical triad of hyperglycemia, ketonemia, and high anion gap metabolic acidosis together with electrolyte derangements including dilutional or hyperosmolar hyponatremia. The management of DKA includes close monitoring, adequate and timely fluid resuscitation, insulin therapy, and electrolyte replacement to avoid adverse effects that

could be fatal.[6] Water deficit from inadequate water intake and free water loss that supersedes electrolyte loss through diarrhoea and vomiting can cause hypernatremia, which is a rare condition associated with DKA. Its etiology remains unclear, DKA usually is accompanied by normal sodium or hyponatremia.[7] Hypernatremia is associated with high mortality due to associated comorbid conditions. Here we discuss a unique case about a 47-years-old female presenting with hypernatremia in DKA with T2DM and the management for this condition. This report serves as a reminder of the importance of a management approach in patients with DKA with a rare manifestation of hypernatremia.

CASE REPORT

A 47 years old female patient was brought to the emergency room (ER) with complaints of general weakness, feeling very thirsty, nausea, and decreased appetite. The patient had a history of T2DM with oral medication but the patient didn't consume it regularly. The awareness of the patient was compos mentis. Her blood pressure was found to be 151/70 mmHg, heart rate of 134 beats/minute, respiratory rate of 24 breaths/minute, axilla temperature of 36.5° Celsius, and oxygen saturation of 99% on room air. On physical examination, epigastric tenderness was found. Other than that, those were normal. Initial laboratory results showed leucocytosis (30.08×103/µL), high Haemoglobin count (16.2 g/dL), high haematocrit level (50.8 %), high random blood sugar level (654 mg/dL), low blood pH (7.18), low bicarbonate level (3 mEq/L), high sodium level (147 mmol/L), high potassium level (5.7 mmol/L), high chloride level (115 mmol/L), and high ketone level in urine (+4). Chest x-ray was normal.

She was diagnosed with T2DM, DKA, and Hypernatremia. The patient initially received 2 liters of 0.9% saline solution at the ER, insulin intravenous drip 4 units/hours, esomeprazole 40 mg every 24 hours, cefoperazone 1 gram every 12 hours. Then, the patient was admitted to the ICU. Once the blood glucose was 244 mg/dL, we stopped the insulin intravenous infusion and changed to long-acting insulin (Aspart) subcutaneously 8 units before a meal, with 3 meals per day. By day 2 at ICU, the blood glucose increased to 339 mg/dL, the sodium level also increased to 160 mmol/L, the potassium level decreased to 3.4 mmol/L and the chloride level increased to 120 mmol/L. We changed the Intravenous Fluid Drops (IVFD) to dextrose 5%, sodium chloride 0.225% 2000 cc/24 hours. We increased the insulin Aspart (subcutaneously) 16 units before a meal, with 3 meals per day.

By day 3 at ICU, the blood glucose was 287 mg/dL, the sodium level was 154 mmol/L, the potassium level was 4.1 mmol/L, and the chloride level was 122 mmol/L. We increased the insulin Aspart (subcutaneously) 20 units before a meal, with 3 meals per day. We added 4 units of insulin glargine (subcutaneously) at night. By day 4, The patient was moved to the ward in the hospital. The blood glucose was 236 mg/dL, the sodium level was 157 mmol/L, the potassium level was 3.4 mmol/L, and the chloride level was 117 mmol/L. We added Furosemide 20 mg IV every 24 hours for 2 days.

On day 6 of hospitalization, the blood glucose was 235 mg/dL, the sodium level was 141 mmol/L, the potassium level was 2.7 mmol/L, and the chloride level was 101 mmol/L. We stopped giving the furosemide. The patient was given KCL 25 mEq into 500 cc of NaCl 0,9% 20 drops per minute. On day 7 of hospital stay, the blood glucose was 235 mg/dL, the sodium level was 137 mmol/L, the potassium level was 2.8 mmol/L, and the chloride level was 104 mmol/L. The patient was given KCL 50 mEq into 500 cc of NaCl 0,9% 20 drops per minute. The patient was given 8 units of insulin Glargine.

On day 8 of hospitalization, the blood glucose was 230 mg/dL, the sodium level was 142 mmol/L, the potassium level was 3.1 mmol/L, and the chloride level was 102 mmol/L. The patient was given 10 units of insulin glargine. By day 9, the patient was able to tolerate oral intake, had no complaints, vital signs were normal, physical examination was normal, the blood glucose level was controlled, and the imbalance electrolytes level resolved. The patient was eventually discharged without any further events on day 9 on subcutaneous insulin aspart 20 units before a meal, with 3 meals per day, 12 units of insulin glargine, omeprazole 20 milligrams 2 times a day, potassium chloride 600 milligrams 3 times a day.

DISCUSSION

T2DM is a disease that is caused by defective insulin secretion and insulin resistance, followed by hyperglycemia condition.[8] Glycosuria, dehydration and acetoacetic acid and beta-hydroxybutyric acid production can be the result from hyperglycemia due to insulin deficiency resulting in anion-gap acidosis which can cause changes in mental status. The patient's consciousness is determined by the severity of dehydration and acidosis, the patient may present in conscious state or progress to lethargy as well as even coma.[9]

Patients with DKA who have hyperglycemia condition, have electrolyte imbalances due to osmotic fluid shift and osmotic diuresis. The dilution effect of hyperosmolar status caused by an increase in blood glucose which makes the movement of water from the intracellular space to the extracellular space, results in low serum sodium in DKA patients. The osmotic force of glycosuria that drives the sodium by osmotic diuresis contributes to a volume depletion status. Excessive water losses relative to the osmotic loss of sodium through the urine and by vomiting can cause hypernatremia in DKA.[7]

An elevated level of serum sodium (Hypernatremia) can be connected to multiple processes, including hypotonic fluid loss, hypertonic fluid gain, general water depletion. The increased extracellular osmolality drives water outward from cells, resulting in cell shrinking. Long-term damage such as impairments manifest in various organ systems can happen, although an ionic osmolytic response serves to counter these dangerous effects.[10] Our patient's initial measured sodium level of 147 mEq/L that later progressed to 160 mEq/L indicated an urgent intervention.

Management of hypernatremia requires two approaches: (1) the underlying cause is identified and resolved and (2) the established hypertonicity (hyperosmolarity) is corrected by considering onset time (acute vs. chronic), the severity of neurologic symptoms, and volume status. Prevention of further water loss or hypertonic sodium gain is important by identifying the underlying cause of hypernatremia and initiating treatment. In symptomatic hypernatremia with acute onset (within 48 hours), a more aggressive rapid correction of plasma sodium (falling by 1–2 mmol/L/hr for the first 6-8 hours, restoring a sodium level of 145 mmol/L within 24 hours) improves the prognosis with decreasing the risk of cerebral edema.[11]

In moderate-to-severe DKA, treatment can be initiated with an IV bolus of regular insulin, 0.1 units/kg followed within 5 minutes by continuous IV infusion of 0.1 units/kg/hour. These doses of insulin decrease serum glucose by 2.8-3.9 mmol/L per hour. If glucose does not fall by this, from the initial value in the first hour, the insulin infusion rate should be doubled every hour until a steady decline in serum glucose of this magnitude is achieved. Alternatively, subcutaneous rapid-acting insulin is an initial injection of 0.3 units/kg, followed by 0.1 units/ kg every hour until the serum glucose is less than 13.9 mmol/L. The insulin dose is then decreased to 0.05 to 0.1 units/kg and administered every one or two hours until the resolution of the ketoacidosis.[6]

In our case, we gave the patient initial insulin intravenous drip 4 units/hours. Once the blood glucose was around 200-250 mg/dL, we stopped the insulin intravenous infusion and changed to rapid acting insulin (Aspart) subcutaneously 8 units before a meal, with 3 meals per day. The patient also was given long-acting insulin (Glargine) with 4 units subcutaneously at night. We increased the rapid acting insulin dose according the increased blood glucose.

Acute hypernatremia with DKA patients have high morbidity and mortality.[9] Precaution to avoid risking cerebral edema, from overcorrecting acute hypernatremia is important.[6] The treatment for hypernatremia in DKA consists of infusing 0.9% normal (isotonic) saline, at a rate of 15-20 milliliters per kilogram per hour or 1-1.5 liters during the first hour, to maintain effective plasma osmolarity. Subsequently, fluids should be changed to 0.45% sodium chloride as the corrected sodium concentration may be in excess of 145 mmol/liter.[12]

Hypernatremic range at presentation with hyperglycemia indicates excessive water deficit that must be corrected with 5% dextrose in water, and diuretics should be administered to promote sodium excretion and maintain a negative fluid balance. In a patient with low or normal serum and DKA, normal saline is the fluid of choice.[11]

Infusion of hypotonic solutions will eventually be needed regardless of whether the early phase of treatment aims at maintaining or decreasing the corrected sodium. Frequent measurement of the relevant serum biochemical values, and repeated calculations of the corrected sodium and blood glucose after each measurement, and monitoring urine volume are imperative.[13]

However, in patients with hypernatremia and DKA, we do not want to increase the serum sodium. Our patient initially received 2 liters of 0.9% saline solution at the ER. Hence, we changed the solutions with less sodium content, such as dextrose 5%, sodium chloride 0.225%, which is more appropriate to decrease the serum sodium. The patient also was given diuretics. We only measured the electrolytes levels once a day to cut the cost of health care. Once daily monitoring of electrolytes gave us a clear picture of electrolyte imbalance and correction for our patients.

CONCLUSION

This case description wants to highlight the importance of early recognition, treatment strategy, possible complication, and outcome of DKA with T2DM. This case report concludes that there is significant association of sodium with DKA in type 2 diabetic individuals. It is also observed that the risk of diabetic complications may be as a result of a significant role of electrolyte imbalance. Diabetic individuals need to regularly monitor the serum electrolytes, because the electrolyte imbalance can be found in uncontrolled diabetes patients. Monitoring on serum glucose concentration, hemodynamic, and electrolyte levels become very important. Fluid replacement therapy and insulin treatment are proven to be crucial steps to control hypernatremia in DKA. Therefore, we hope that our case report will be a reminder on the management for the physicians in handling the patients with hypernatremia in DKA with type 2 diabetic patients.

ACKNOWLEDGEMENTS

Author would like to thanks to internal medicine department, Wangaya general hospital, Denpasar, Bali, Indonesia for reviewing our work and general support.

Funding: No funding sources
Conflict of interest: None declared
Ethical approval: Not required

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Cite this article as: Pratikto BC, Suryana K. A case report: management of hypernatremia in diabetic ketoacidosis patient with type II diabetes mellitus. Int J Res Med Sci 2023;11:1792-5.