

## Case Report

### Approach to A Rare Case of Diabetic Ketoalkalosis

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#### Abstract

Diabetic Ketoacidosis (DKA) is one of the most common complications of Type 1 Diabetes Mellitus (T1DM). DKA is an insulin-deficient state during which ketonemia and ketonuria develop due to the inability to suppress lipolysis. The reason for insulin deficiency can be endogenous, exogenous or an increase in secretion of counter-insulin hormones depending on the type of diabetes. Patients with DKA usually present with nausea, vomiting, shortness of breath, abdominal pain, weight loss, dry mouth, polyuria and/or polydipsia. According to the American Diabetes Association, DKA is defined as hyperglycemia (glucose >300 mg/dL or glucose >250 mg/dL in pregnancy), acidemia (arterial Ph <7.35 or serum HCO<sub>3</sub><sup>-</sup> <15mEq/L) and ketonuria and/or ketonemia. DKA is the most frequent cause of increased anion gap metabolic acidosis. However, in 30% of cases, DKA is a mixed acid-base disturbance and the most common association is vomiting-dependent metabolic alkalosis. Other causes may be hypercortisolism or concomitant diuretic use.

A 31-year-old female patient, who was 10 weeks pregnant with her first child, diagnosed with type 1 diabetes mellitus known since the age of 12 presented with nausea, vomiting, and inguinal/pubıc pain for 5 weeks. During this period, she had experienced symptomatic hypoglycemia several times and her blood pressure had presented constantly hypotensive. The patient, who missed insulin doses due to severe nausea and vomiting, admitted to the Istanbul Faculty of Medicine Obstetrics and Gynecology with the same complaints. The patient, who had a

hyperglycemic course, was hospitalized in another hospital with the same complaints on December 14th. Upon observing Ketone 3+ and neutrophilic leukocytosis in complete urinalysis, and pH 7.31, HCO<sub>3</sub> 12, Blood Glucose levels as 450 in venous blood gas measurements on December 15, she was followed up with insulin infusion for 36 hours under the case of the Diabetic Ketoacidosis, the total admitted dose is unknown.

In the texts requested on admission, TSH 0.0005, TRAK (-), high fT<sub>4</sub>, normal fT<sub>3</sub>, were found. Thyroid doppler USG showed increased vascularization. Propylcil was started as 5 mg 1 + 1/2 tablets. Her current regimen consisted of propyl thiouracil (50mg tb 1 + 0.5 tb ), novorapid 3 \* 4U, Levemir 1 \* 12U. Patient with pH 7.55, HCO<sub>3</sub> 27, Blood Glucose 413, hbA<sub>1c</sub> 7,7, AG 21, pCO<sub>2</sub> 29 at the blood tests, and Ketone 3+ at complete urinalysis in her admission to Istanbul Faculty of Medicine Emergency Internal Medicine on December 25 with the diagnosis of metabolic alkalosis secondary to hyperemesis gravidarum, diabetic ketoacidosis and accompanying respiratory alkalosis, insulin infusion and hydration therapy was taken over by us for further examination and treatment.

Family history was unremarkable. In the physical examination, her general condition was good, she was conscious, oriented, and cooperating. S<sub>1</sub>, S<sub>2</sub> was natural. S<sub>3</sub>, S<sub>4</sub>, additional voice, no murmur was heard. There was no venous fullness. Both hemithorax participated equally in respiration. Skin appearance was pale. Oropharynx was natural; there was dryness of the mucous membranes; tongue papillae were also natural. In cardiovascular examination; Heart Rate: 100 / min (rhythmic), Blood pressure arterial 110/70 mmHg. Fever: 36.7 °C. Respiratory Rate: 16 / min. No pathology was detected in other system examinations.

The patient, who had vaginal bleeding once in the follow-up in the emergency department, was evaluated as abortion imminence by the Istanbul Faculty of Medicine Gynecology and Obstetrics Department; follow-up and bed rest was recommended. With the current Thyroid Function Tests results, the treatment of Propylcil 5 mg tb 3x1 was rearranged in the emergency department.

Metabolic alkalosis due to persistent vomiting was detected when blood gas and other tests were evaluated. Diabetic ketoacidosis was associated with increased anion gap metabolic acidosis compared to corrected Na<sup>+</sup> values due to the development of pseudohyponatremia due to hyperglycemia. A compensatory respiratory alkalosis was found to be present ever since the beginning of pregnancy. DKA protocol was applied to the patient because of high blood glucose and urinary urine level of +3 ketone and +3 glucose. Intravenous hydration starting from of the patient with 0.1 IU / kg (60 Kg, 6 IU) dose bolus short-acting insulin (Humulin -R) was started after insulin infusion. Once blood glucose was <200 mg, insulin infusion was reduced in half and 5% dextrose fluid was added. Potassium replacement was also performed. Diabetic Ketoalkalosis was considered in the patient, who continued to vomit despite the treatment given for vomiting and metabolic alkalosis was detected in the control venous blood gases.

In the evaluation and treatment of acid- base disorders, the patient's clinical and metabolic characteristics should be considered holistically. Ph, PCO<sub>2</sub>, HCO<sub>3</sub> and anion deficit should be taken into consideration in blood gas evaluation in order to avoid complicated acid- base complications. Renal insufficiency, ketoacidosis (diabetic, alcoholic, hunger), lactic acidosis, and intoxications (salicylate, ethylene glycol, etc.) should be considered in the diagnosis of increased anion deficit metabolic acidosis. Diabetic ketoacidosis is a common complication of type 1 diabetes mellitus in the presence of endogenous, exogenous insulin deficiency or increased secretion of hormones that counteract insulin. DKA is an important cause of recurrent hospitalization and morbidity. DKA in increased

anion gap metabolic acidosis and diabetic ketoacidosis connected vomiting result of metabolic alkalosis and diabetic in ketoacidosis metabolic acidosis compensate for respiratory alkalosis is observed. In our patient with DKA during pregnancy, increased anion gap metabolic acidosis was observed in connection with DKA compensation by respiratory alkalosis, hyperemesis gravidarum and the DKA associated vomiting resulted in the development of metabolic alkalosis. As a result, diabetic ketoalkalosis has been considered.

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