Case Report

Type 1 renal tubular acidosis in a patient of Type 1 diabetes mellitus: Is it coincidence or coexistence?

Muzafar Naik, Tariq Bhat, Mubarik Naqash, Manzoor Wani, Nazir Dar, Younis Shah

Department of General Medicine, Sher-I-Kashmir Institute of Medical Sciences Medical College and Hospital, Srinagar, India

ABSTRACT

A 26-year-old male patient suffering from Type 1 diabetes mellitus got admitted with abdominal pain and high blood sugars. On further evaluation, he was found to have normal anion gap metabolic acidosis without ketonuria and urinary pH was alkaline. The patient was diagnosed as Type 1 renal tubular acidosis (RTA) (distal RTA) and was managed by alkali replacement in addition to control of blood sugars. The association of Type 1 RTA with Type 1 diabetes mellitus has been rarely reported in the literature. The association needs a different attention as diagnosis and management of diabetic ketoacidosis in such cases will be tricky. The case presented here is the first of its kind from our part of the world and second as far as English literature is concerned.

Key words: Type 1 diabetes mellitus, Type 1 renal tubular acidosis, normal anion gap metabolic acidosis

Introduction

Type 1 diabetes mellitus (T1DM) is an autoimmune disorder and individuals with this disorder are prone to develop high anion gap metabolic acidosis with ketonuria, known as diabetic ketoacidosis (DKA). We report a case of normal anion gap metabolic acidosis without ketonuria during the management of hyperglycemia in a patient of T1DM. The patient was diagnosed to have distal renal tubular acidosis (dRTA). Although T1DM and dRTA are both associated with autoimmune disorders, their coexistence has been very rarely reported. Our case represents the second of its kind to the best of our knowledge and emphasizes the need to screen for dRTA in patients of T1DM, justified by the fact that different treatment strategy will be required for the management of diabetic ketoacidosis in T1DM associated with dRTA.

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Case Report

Our patient was a 26-year-old male, a known case of T1DM for 6 years on insulin, who presented to our outpatient department (OPD) with complaints of epigastric pain, postprandial fullness, and nausea. The patient was not compliant with the treatment and was coming erratically for follow-up to the OPD. On examination, the patient was conscious, oriented, dehydrated, and had mild pallor. Examination revealed a pulse rate of 60/minute, blood pressure (BP) of 100/60 mm Hg, respiratory rate (RR) of 17 breaths/minute, and temperature of 98.6°F. He had bilateral posterior subcapsular cataract and acne on face. Systemic examination revealed tender epigastrium and absent deep tendon reflexes. Rest of the systemic examination was normal.

Investigations revealed the following: hemoglobin (Hb) 10.2 g/dL; total leukocyte count (TLC) 5.3 × 10^9/L; differential leukocyte count (DLC): N 71%, L 20%, M 7.5%; platelet 273 × 10^9/L; erythrocyte sedimentation rate (ESR) 24/1st h; urea 37 mg/dL; creatinine 1.09 mg/dL; bilirubin 1.34 mg/dL; aspartate transaminase (AST) 34 U/L; alanine transaminase (ALT) 50 U/L; alkaline phosphatase (ALP) 450 U/L; total protein 5.9 g/dL; and albumin 3.7 g/dL. Initial blood sugar was 284 mg/dL. Arterial blood gas analysis revealed the following: pH 7.20, pO2 72 mm Hg,

Corresponding Author: Dr. Muzafar Naik, Department of General Medicine, SKIMS MCH Bemina, Srinagar, India. E-mail: muzafarnaik@rediffmail.com

sO2 95 mm Hg, pCO2 36 mm Hg, Na^+ 137 mEq/L, K^+ 2.5 mEq/L, $HCO3^-$ 12.1 mEq/L, Cl^- 116 mEq/L (100–112 mEq/L). Urine for ketone bodies was negative. His HbA1C was 11.8%. ECG and chest X-ray were normal. 24-hour urinary protein was 100 mg/dL. The USG abdomen was normal. Patient was initially managed with IV normal saline and potassium replacement. After initial resuscitation with IV fluids, and potassium replacement, the patient's blood sugar stabilized to 172 mg/dL random; however, metabolic acidosis and hypokalemia persisted. Then, the possibility of dRTA was thought of. Anion gap was calculated as Na − [HCO3 + Cl], which was normal: 137 − [12.1 + 116] = 8.9 mEq/L. Furthermore, urine pH was 6.0 even at a serum pH of 7.20. The patient was diagnosed as dRTA in view of normal anion gap metabolic acidosis with hypokalemia with inability to acidify urine in the presence of systemic metabolic acidosis. Meanwhile, the patient started tolerating oral intake and was started on oral feeds with pre-meal subcutaneous regular insulin. Subsequently, the patient was put on oral sodium bicarbonate tablets at a dose of 2 mEq/kg and oral potassium replacement. His metabolic acidosis and hypokalemia improved markedly. He was discharged with a final diagnosis of T1DM with dRTA.

Discussion

T1DM is a less common type of diabetes found in younger age group. It is thought to be caused by interaction of genetic, environmental, and immunological factors, which leads to the pancreatic beta cell destruction.[1] T1DM is associated with many autoimmune diseases as almost 15–30% patients have autoimmune thyroid disease, 4–9% have celiac disease, and 0.5% have Addison's disease.[2,3] T1DM is also a part of autoimmune polyglandular syndrome type 1 and 2.[4] Other uncommon associated autoimmune diseases include pernicious anemia, juvenile rheumatoid arthritis, psoriasis, vitiligo, etc.[2]

Type 1 RTA or dRTA is a rare disease which can be either inherited,[5] sporadic,[6] endemic,[7] or acquired secondary to a variety of conditions. The most common causes of secondary dRTA are autoimmune disorders like Sjögren syndrome, autoimmune thyroiditis, chronic active hepatitis, primary biliary cirrhosis, systemic lupus erythematosus, hypothyroidism, and vasculitis.[8-11] It has been seen that in some patients dRTA may be possibly caused by autoantibody against renal collecting duct.[12,13]

The clinical spectrum of dRTA in T1DM is similar to that of diabetic ketoacidosis complicating T1DM, except for urine pH >5.5 in the former and presence of ketonuria in the latter. However, the management of acidosis in dRTA is contrary to that in diabetic ketoacidosis due to the controversial role of alkali therapy in the latter situation. Although T1DM and dRTA are both associated with autoimmune disorders, their coexistence has rarely been reported.[14] Whether this coexistence is purely coincidental or is because of common autoimmune pathogenesis is unclear. Interestingly, till date, diabetic ketoacidosis complicating T1DM associated with dRTA has not been reported in literature as the diagnosis and management of such a case will be challenging because the results of most clinical trials do not support the routine use of bicarbonate replacement,[15,16] as in one study in children which showed that its use was associated with increased risk of cerebral edema.[17] On the other hand, soda bicarbonate is the cornerstone therapy for management of hypokalemia due to dRTA, and without correction of hypokalemia, the use of insulin is contraindicated in diabetic ketoacidosis.

Therefore, it is necessary to first explore the possible mechanism involved in dRTA in T1DM in order to draw a definite association of the two autoimmune disorders and secondly plan a treatment strategy during diabetic ketoacidosis in a patient of T1DM associated with dRTA.

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