Case Report

Diabetic Ketoacidosis in Hashimoto's Thyroiditis with Thyrotoxic Goiter in Adult Female Patient – A Case Report

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Abstract

Thyroid diseases and diabetes mellitus are the two most common endocrine disorders encountered in clinical practice. Diabetes and thyroid disorders have been shown to mutually influence each other and associations between both conditions have long been reported [1, 2]. On one hand, thyroid hormones contribute to the regulation of carbohydrate metabolism and pancreatic function, and on the other hand, diabetes affects thyroid function tests to variable extents. We reported a case of 40 years old woman with a history of type 2 diabetes mellitus and hyperthyroidism who presented to the emergency with complaints of fever, nausea, vomiting, abdominal pain. Patient was drowsy with dry skin and mucous membranes. Kussmaul breathing was present. Blood glucose levels were 658 mg/dl, urine was positive for ketone bodies and metabolic acidosis with an anion gap of 56. An arterial blood gas showed Ph 7.1, PaO2 105.3, PaCO2 22, and HCO3 6.1 meq/l. ECG showed tachycardia. T3 = 181.5 ng/dl, T4 = 12.5 micro g/dl, TSH= 0.013 micro u/l. Further examination revealed Hashimoto's Thyroiditis with Thyrotoxic Goiter. This is a rare case of Diabetic Ketoacidosos in Hashimoto's Thyroiditis with Thyrotoxic Goiter in adult female patient with thyrotoxicosis.

Key words

Diabetic Ketoacidosis (DKA), Thyrotoxic Goitre, Hashimoto's Thyroiditis.

Introduction

Thyroid diseases and diabetes mellitus are the two most common endocrine disorders encountered in clinical practice. Diabetes and thyroid disorders have been shown to mutually influence each other and associations between both conditions have long been reported [1, 2]. Diabetic ketoacidosis (DKA) is an acute complication of diabetes mellitus, that occurs more commonly in type 1 diabetics and whose incidence has not decreased in the last few years in spite of medical breakthroughs [3, 4]. The most common triggering factors of diabetic ketoacidosis are infection, insulin therapy omission, and the onset of the disease, but if these are excluded, other less frequent etiologies must be ruled out before classifying it as idiopathic [5].

In addition, thyroid hormones have a hyperglycemic counter-regulatory effect and it is well known that hyperthyroidism worsens metabolic control in diabetic patients. The association of diabetic ketoacidosis with thyrotoxicosis has been well described [6], but recently there have been few case reports described in the literature [7, 8].

Case report

40 years old female patient, a known case of type 2 diabetes mellitus since 3 years and hyperthyroidism since 1 year on carbimazole 20 mg one tablet thrice daily and Inderal 10 mg one tablet twice a day, presented with complaints of low grade fever, nausea, vomiting, abdominal pain of two days duration. Polyurea and polydypsia were present. Patient was drowsy with dry skin and mucous membranes, thinly built, with no history of recent illnesses, or skipped doses of insulin. On examination Kussmaul breathing was observed, PR 120 bm, BP 140/90 mmHg. Cardiovascular system and Respiratory system examination was normal. CNS examination revealed tremors and proximal myopathy. Random blood Glucose levels were 658 mg/dl. ECG showed tachycardia, urine ketone bodies were positive. An arterial blood gas showed Ph 7.1, PaO2 105.3, PaCO2 22, and HCO3 6.1 meq/l. metabolic acidosis with an anion gap of 56.

On investigations, complete blood picture showed Hb-11g%, RBC count 4.2 mill/cumm, WBC count 7500 cells/cumm with Neutrophils - 80%, Lymphocytes - 24%, Eosinophils - 2%, Basophils - 0%, Monocytes - 2%, Platelet count was normal. Urine was positive for ketone bodies. X-ray chest was normal. USG Abdomen was normal. Patient remained in tachycardia, with ECG showing sinus tachycardia, despite adequate fluid resuscitation. Tremors were present, Temperature was 38.6^0C. On neck examination, there was a goitrous swelling, right lobe- 3x2 cm hard in consistency, left lobe 2.5x2 cm hard in consistency and there was no hoarseness of voice. USG of thyroid showed Rt. lobe - 3.4 x 1.8 cm., Lt. lobe 3.6 x 1.7 cm. Isthmus normal in size, normal in echotexture with normal vascularity, there were no enlarged lymph nodes in bilateral cervical regions, great vessels and strap muscles of neck was normal, Impression - diffuse thyromegaly. Thyroid profile showed T3 = 181.5 ng/dl, T4 = 12.5 micro g/dl, TSH= 0.013 micro u/l. Anti TPO Antibodies = 331.1 U/ml (REF values = < 60 – Negative, > 60 – Positive)., Anti Thyroglobulin Anti Bodies = < 15.0 I u/ml (< 34 – Negative, > 34 – Positive), Anti ds DNA = 98.2 U/ml (ref. values= negative-VE – 0-200, + 201 – 300, ++ - 301 – 800 ,++++ - ≥ 801). FNAC of thyroid revealed Hashimoto's Thyroiditis. Liver function test and Renal function test were normal.

A diagnosis of Diabetic Ketoacidosis in Hashimoto's Thyroiditis with Thyrotoxic Goiter was made. Standard Treatment for Diabetic Ketoacidosis was given along with Propranolol 40 mg every 8 hours, Propylthiouracil 100mg every 8 hours was initiated. Patients symptoms subsided, sugars were brought under control with insulin infusion, tachycardia improved. Patient made steady improvement.

Discussion

DKA is a complex disordered metabolic state characterized by hyperglycemia, ketoacidosis, and ketonuria. DKA usually occurs as a consequence of absolute or relative insulin deficiency that is accompanied by an increase in counter regulatory hormones (i.e., glucagon, cortisol, growth hormone, and epinephrine).

Effects of thyroid hormones on glucose homeostasis

Severe hyperthyroidism worsens glycemic control in diabetic patients through several mechanisms, such as

1. An increase of intestinal glucose absorption,
2. A decrease in insulin secretion, and
3. A decrease in the peripheral use of glucose due to insulin resistance.

Several reports documented a higher than normal prevalence of thyroid dysfunction in the diabetic population. Particularly, Perros, et al. demonstrated an overall prevalence of 13.4% of thyroid diseases in diabetics with the highest prevalence in type 1 female diabetics (31.4%) and lowest prevalence in type 2 male diabetics (6.9%) [9]. Recently, a prevalence of 12.3% was reported among Greek diabetic patients [10] and 16% of Saudi patients with type 2 diabetes were found to have thyroid dysfunction [11]. Thyroid disorders remain the most frequent autoimmune disorders associated with type1 diabetes. This was shown in a cross sectional study involving 1419 children with type1 diabetes mellitus, where 3.5% had Hashimoto's thyroiditis [12]. Thyroid hormones affect glucose metabolism via several mechanisms.

Hyperthyroidism has long been recognized to promote hyperglycemia [13]. During hyperthyroidism, the half-life of insulin is reduced most likely secondary to an increased rate of degradation and an enhanced release of biologically inactive insulin precursors [14, 15]. In untreated Graves' disease, increased proinsulin levels in response to a meal were observed in a study by Bech, et al. [16]. In addition, untreated hyperthyroidism was associated with a reduced C-peptide to proinsulin ratio suggesting an underlying defect in proinsulin processing [17]. Another mechanism explaining the relationship between hyperthyroidism and hyperglycemia is the increase in glucose gut absorption mediated by the excess thyroid hormones [18, 19].

Endogenous production of glucose is also enhanced in hyperthyroidism via several mechanisms. Thyroid hormones produce an increase in the hepatocyte plasma membrane concentrations of GLUT2 which is the main glucose transporter in the liver, and consequently, the increased levels of GLUT-2 contribute to the increased hepatic glucose output and abnormal glucose metabolism [20, 21]. Additionally, increased lipolysis is observed in hyperthyroidism resulting in an increase in FFA that stimulates hepatic gluconeogenesis. The increased release of FFA could partially be explained by an enhanced catecholamine-stimulated lipolysis induced by the excess thyroid hormones [22]. Moreover, the non-oxidative glucose disposal in hyperthyroidism is enhanced resulting in an overproduction of lactate that enters the Cori cycle and promotes further hepatic gluconeogenesis.

The increase in GH, glucagon and catecholamine levels associated with hyperthyroidism further contributes to the impaired glucose tolerance [23-25]. It is well known that diabetic patients with hyperthyroidism experience worsening of their glycemic control and thyrotoxicosis has been shown to precipitate diabetic ketoacidosis in subjects with diabetes [26, 27].

Ghrelin is an orexigen secreted from the fundus of the stomach. It has been shown to exert several diabetogenic effects including decreasing secretion of the insulin sensitizing hormone adiponectin [28]. Ghrelin levels are lower in obese subjects and those with type 2 diabetes, states associated with hyperinsulinemia [29]. Reduced ghrelin levels were observed in hyperthyroid patients [30, 31], and these levels rose to normal values after pharmacological

treatment of hyperthyroidism [32-34]. Hyperthyroidism, being a state of negative energy balance should result in an increase in ghrelin levels. Interestingly, ghrelin levels in thyroid dysfunction states seem to correlate with insulin resistance rather than food intake and energy balance [35]. Hyperthyroidism is associated with insulin resistance and hyperinsulinemia suppresses ghrelin levels [36].

Conclusion

This is a rare case presentation of diabetic ketoacidosis in hashimoto's thyroiditis with thyrotoxic goiter in adult female patient. The presence of diabetic ketoacidosis should be suspected in a case of thyrotoxicosis and vice versa, especially in females. Thyrotoxicosis can precipitate DKA and, delay in diagnosis will result in poor prognosis and outcome. A question arises here, whether hashimoto's thyroiditis further precipitates, early dysregulation of carbohydrate metabolism, and may land in DKA.

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