Simultaneous Presentation of Thyrotoxicosis and Diabetic Ketoacidosis Resulted in Sudden Cardiac Arrest

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Abstract. Although many cases of simultaneous presentation of thyrotoxicosis (thyroid storm) and diabetic ketoacidosis have been reported, it is a clinically unusual situation and remains a diagnostic and management challenge in clinical practice. The diagnosis of diabetic ketoacidosis or thyrotoxicosis may be masked leading to serious complications. We report two patients with simultaneous thyrotoxicosis and diabetic ketoacidosis resulted in sudden cardiac arrest, emphasizing early recognition and prompt treatment when these two disease are presented concomitantly.

Key words: Diabetic ketoacidosis, Thyrotoxicosis, Sudden cardiac arrest

DIABETIC ketoacidosis is an acute complication, mostly seen in type 1 diabetic patients [1]. Thyrotoxic crisis is an extreme accentuation of thyrotoxicosis. It is an uncommon but serious complication, usually occurring in association with Graves’ disease. Thyrotoxic crisis is usually of abrupt onset and occurs in patients in whom preexisting thyrotoxicosis has been treated incompletely or has not been treated at all [2]. Both of them are endocrine emergencies. Without appropriate management, they may result in high mortality. Concomitance of these two endocrine emergencies is rare and represents an early diagnostic and management challenge. We reported two patients with thyrotoxicosis and diabetic ketoacidosis resulted in sudden cardiac arrest.

Cases

Case 1: A 22-year-old woman presented with diffuse abdominal pain and vomiting for one day. Fatigue, marked palpitation and restlessness were also found. Graves’ disease was diagnosed 6 months earlier and type 1 diabetes mellitus was diagnosed two months later. However, she had poor compliance of anti-thyroid drug and insulin injection. At emergency department, she was in agitation. Her body temperature was 38.3°C, blood pressure 112/70 mmHg, pulse rate 160 beats/min, and respiratory rate 24 breaths/min. On physical examination, a grade II diffuse goiter was palpated. Bilateral exophthalmoses and dry skin without pigmentation were found. The chest X-ray was normal. The electrocardiogram showed sinus tachycardia. The results of laboratory tests were showed in Table 1. By the scoring of criteria of Burch and Wartofsky, this patient had a score of at least 65, which was highly suggestive of thyrotoxic crisis. She received dexamethasone, propranolol, methimazole and Lugol’s iodine solution. She was also carefully hydrated with normal saline because of dehydration. Simultaneously, insulin infusion with a rate of 0.1 units/kg body weight/h was administered. However, tachycardia and agitation persisted in the next 6 hours. Sudden sinus bradycardia

Received April 17, 2007
Accepted July 23, 2007
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followed by cardiac arrest developed. Cardiopulmonary resuscitation, which included an intravenous bolus of epinephrine 1 mg and atropine 1 mg respectively, was performed. Spontaneous circulation was restored within 2 minutes after resuscitation. All possible causes of sudden cardiac arrest such as myocarditis, sepsis, electrolyte imbalance and acidosis were excluded. She was maintained on anti-thyroid drug and insulin injection. A full recovery was achieved before discharge.

Case 2: A 18-year-old woman presented with a 3-day history of progressive dypsnea. She was diagnosed to have Graves’ disease with methimazole treatment for 4 weeks. She discontinued the medicine because of allergic reaction a week prior to this admission. Then, palpitation, thirst, polyuria and a noticeable weight loss developed. On the day before admission, diffuse abdominal pain, lethargy and progressive dypsnea were complained. At emergency department, she appeared drowsy. Her height and weight were 160 cm and 47 kg respectively. Her body temperature was 35.9°C, blood pressure 64/45 mmHg, pulse rate 120 beats/min and respiratory rate 30 breaths/min. Physical examination revealed dry and cool skin, without pigmentation. Additionally, Grade I diffuse goiter was palpated. The chest X-ray was normal. The electrocardiogram revealed sinus tachycardia. The results of laboratory tests were demonstrated in Table 1. Vigorous intravenous fluid repletion with normal saline was given. Sodium bicarbonate and potassium chloride were optimally administered. However, pulseless ventricular tachycardia occurred suddenly several hours later. Sinus arrest developed after the first defibrillation, followed by recurrent ventricular tachycardia. She was successfully resuscitated after three times of defibrillations. Hypokalemia (serum potassium was 1.6 mmol/l) was found and corrected aggressively. All other etiology investigation, including autoimmune profile, infectious profile and echocardiogram revealed negative findings. Medical treatment with insulin and anti-thyroid agents was continued and she recovered well.

Discussion

There is a general agreement that the incidence of glucose intolerance is increased under the hyperthyroid state [3]. Severe hyperthyroidism worsens glycemic control in diabetic patients through several mechanisms, such as an increase of intestinal glucose absorption, insulin degradation, and enhancement of basal hepatic glucose production, a decrease in both insulin secretion and peripheral use of glucose due to insulin resistance [4–5]. Therefore, the presence of diabetes mellitus or even diabetic ketoacidosis should be sought in patients with hyperthyroidism, and vice versa, particularly in young women. Because one can precipitate the other, and either one may be associated with other endocrinopathies. Type 1 diabetes mellitus is frequently associated with Graves’ disease, in about 30%, and sometimes may constitute the polyglandular autoimmune syndrome type II [6].

Sudden cardiac arrest or death in concomitant diabetic ketoacidosis and thyrotoxicosis has been reported in two cases in the review of literature [7–8]. One was related to prolonged metabolic acidosis from diabetic ketoacidosis [7], and the other was related to the complications of thyroid storm with increased CNS activity and persistent tachycardia resulted in heart failure [8]. The sudden cardiac arrest in our patient 1 was possibly related to heart failure due to persistent tachycardia. The fatal arrhythmia of our patient 2 was related to hypokalemia (Table2). Hypokalemia in hyperthyroidism was attributable to a sudden intracellular shift of potassium (increased sodium-potassium ATPase pump activity) [9–10]. Furthermore, total body potassium deficiency frequently occurred in diabetic ketoacidosis.
Therefore, it may cause profound hypokalemia when the two diseases coexist. Under such circumstances, aggressive potassium repletion must be the main priority to be undertaken during correction of metabolic abnormalities. Intravenous insulin should be withheld until the serum potassium reaches 3.3 mmol/L [11].

The combination of these two endocrine emergencies can occur fulminantly, atypically and is potentially life threatening. Early diagnosis and prompt management are crucial. We emphasize the importance of electrolyte correction and tachycardia management.

### References