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Case Report

# A Thyroid Storm in a Context of Diabetic Ketoacidosis: Case Report

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**Abstract:** The association of a thyroid storm and diabetes ketoacidosis is relatively rare. We report two cases.The first one is a 20 years old woman, with no history of diabetes, who came with ketoacidosis, a history of non-medicated thyrotoxicosis, and she was complaining about fever, a thyrotoxicosis syndrome, a vascular goiter, and exophthalmos. Her general condition had improved significantly after the treatment in emergency and under maintenance therapy.The second one is a 54 years old female, with a case of diabetes mellitus, who presented a ketoacidosis. But despite the control of the situation, the patient's consciousness level became disturbed and she presented hyperthermia. The thyroid function tests revealed hyperthyroidism. Thus, thyroid storm was evocated and managed. The patient finally passed away despite the prompt medical care. In these two cases, a thyroid storm, associated with Graves' disease covered by diabetic ketoacidosis was the final diagnostic.Thereby, for patients having diabetic ketoacidosis we should seek an eventual thyroid storm, and vice versa: in fact, there are some symptoms in common. But there are scores to help systematize the diagnosis. The management is acutely controlled based on antithyroid drugs in high doses, betablockers and corticosteroids. We should not disregard the search for and treatment of precipitating factors: ketoacidosis in our cases. And identify the thyroid underlying disease: most commonly Graves' disease.

**Keywords:** Thyroid storm, diabetic ketoacidosis, endocrine emergencies, antithyroid drugs, Graves' disease.

## INTRODUCTION

Thyroid storm (TS) is a condition, nowadays rare, but still having huge consequences. The mortality rate related to it remains high, but considering it as an endocrine emergency, a specific management clear and codified, can improve the outcome.

It defined by a very high level of circulating thyroid hormone in the organism. So, thyroid storm is an acutely exaggerated manifestation of the thyrotoxic state, associated to fever, dehydration and manifestations of organ decompensation. Graves' disease seems to be the most common underlying condition, but the other etiologies of thyroid storm can be incriminated.

Among patients hospitalized having thyroxicosis less than 10% may be complicated by thyroid storm. Whereas the mortality may approach 20-30% in case of default of support (Jameson L *et al*., 2001).

Also, thyroid storm can be associated to another endocrine metabolic emergency, much more common. In fact, diabetic ketoacidosis (DKA) concerns 5 out of 1000 diabetic patients per year in Taiwan (Liu CC *et al*., 2010).

It corresponds to a hyperglycemia associated to the presence of acetone and decrease blood pH arising from deficient insulin activity. And it is also an emergency whom management is established and systematized.

We report two cases of thyroid storm developed in a context of diabetic ketoacidosis.

## Observation

We report the case of a 20 years old female, 7 months prior to presentation, neck swelling without signs of compression was observed, and associated with palpitations, tremor and exophthalmos, without diarrhea or thermophobia, evoluted with an important weight loss. In the last two months, she presented polyuria and polydipsia, and in the past two weeks, an important

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asthenia as well as palpitations. The presentation complicated with profuse sweating, exaggerated tremor in a context of fever and alteration of the general condition. Three days before presentation, she developed nausea, vomiting and abdominal pain. She was first hospitalized in intensive care unit, because of her low consciousness level.

When first examined, the patient has dehydrated, with heart rate at 120/min, blood pressure at 120/60 mmHg, temperature at 39°C, high glucose level at 4,2 g/l and positive urinary acetone, exophthalmos, moderate diffuse goiter with audible bruit without node or lymphadenopathy.

Biochemically, blood glucose was 3,55 g/l, the result of the serum electrolytes showed a sodium 134 mmol/l, potassium 3,17 mmol/l, bicarbonates 5,7 mmol/l, thyroid stimulating hormone (TSH) <0,05 µUI/l, free thyroxin (T4) 35 pmol/l and free triiodothyronine (T3) 2,34 pg/ml.

She was treated with a massive physiological saline infusion for correction of dehydration et ketoacidosis, accompanied by initiation of insulin therapy. Her general condition had improved significantly as well as her biological parameters.

Thus, the diagnostic of thyrotoxic crisis was established, managed by administration of hydrocortisone, propranolol 60 mg per day, and carbimazole 60 mg per day.

Further investigations were required including thyroid ultrasonography, which revealed a diffuse swelling of the thyroid gland and increase internal blood flow, and antibodies: TSH receptor antibody positive and TPO antibody negative.

Thus, the final diagnostic was a thyroid storm, associated with Graves' disease covered by diabetic ketoacidosis.

The general condition had improved with treatment by antithyroid drugs and insulin therapy.

**Observation 2**

We report the case of a 54-years-old female with a case of type2 diabetes mellitus, and hypertension. She stopped her insulin injections a month ago. A week before presentation, she developed epigastric pain, vomiting, and weight loss.

After examination, the patient's vital signs were pulse rate 105 beats/min, blood pressure 180/110 mmHg, body temperature 38,5°C. She also presented a high glucose level 2,41g/l, positive urinary acetone and signs of dehydration with a diffuse goiter.

Laboratory data revealed blood glucose 2,16 g/l, sodium 139 mmol/l, potassium 4,48 mmol/l, bicarbonates 11 mmol/l. The diabetic ketoacidosis was managed by fluid replacement and insulin with monitoring of clinical and biological parameters. However, the patient still suffered from progressive dyspnea and her neurological status became disturbed: she presented confusion and hyperthermia 41°C. The brain scan did not reveal any abnormality, neither with the lumbar puncture, hemocultures and ECBU and the C-reactive protein.

We further checked the thyroid function, and the data revealed thyroid stimulating hormone (TSH) 0,011 µUI/l, free thyroxin (T4) 95 pmol/l and free triiodothyronine (T3) 15 nmol/l. A heteronodular goiter at the thyroid ultrasonography, and a sinus tachycardia 122 beat/min at the electrocardiogram.

Thus, the thyroid storm diagnostic was established, with the indication of intravenous hydrocortisone, carbimazole, and propanolol.

Following her neurological status, the patient was hospitalized in an intensive care unit.

During her stay, she presented a hypernatremia 166 mmol/l complicated by an alteration of her conscious level. The correction of the hydroelectrolytic disorders and the treatment previously installed was continued. The patient finally passed away despite the prompt medical care.

**DISCUSSION**

Thyroid storm (TS) corresponds to a condition of organs failure, due to excessive thyroid hormones in the organism. Diabetic ketoacidosis is a severe metabolic disorder arising from deficient insulin activity. The coexistence of these two conditions is rare (Bridgman JF *et al*., 1980).

Thyroid storm diagnostic is clinical. Its manifestations consist in fever higher than 39°C (when a patient with thyrotoxicosis presents fever, TS is suspected), profuse sweating with a rise in the insensible losses and which can cause dehydration, heart failure that is manifested by tachyarrhythmia, most commonly from atrial origin, neurological signs (delirium, psychosis) and less frequently liver failure, which can arise from cardiac involvement or caused by the excess of thyroid hormones (Carroll R *et al*., 2010).

These signs were present for the two patients. Essentially fever, consciousness disturbance and tachycardia.

Thus, thyroid storm requires an emergency supporting in an intensive care unit, to manage the life-threatening emergencies: cardiac, respiratory and neurological ones. General supportive care will deal

with hyperthermia using antipyretics, fluid resuscitation, hydroelectrolyic replacement and nutritional support. Non invasive or intubated ventilation can be prescribed. If the patient is agitated, a sedative (haloperidol, benzodiazepine) can be given. The management includes also a thyroid-specific therapy. Its goal is to decrease thyroid hormone production, to block their release in the blood and to reduce their peripheral effects (Carroll R *et al*., 2010). The therapeutic management looks like thyrotoxicosis one, the only difference is the higher doses of drugs and the more frequent administration.

**This Therapeutic Management Is Based On 5 Items:**

- Block thyroid hormone synthesis using antithyroid drugs
- Block their release using the iodide
- Block T4 to T3 conversion
- Betablockers to reduce peripheral effects of excess thyroid hormone
- Block enterohepatic circulation

**Generally**,

Antithyroid drug, carbimazole or propylthiouracile (PTU), is administrated per os, using a nasogastric tube if necessary. PTU is preferred because of its fastest onset of action and its inhibition of T4 to T3 conversion. However, the FDA evoked recently that PTU can have liver toxicity (Malazowski S *et al*., 2010 ; FDA, 2010). That is why, many experts recommend using carbimazole, thus, only beta-blockers and corticosteroids are used to inhibit T4 to T3 conversion. The loading dose of PTU is 600 mg followed by a dose of 200-250 mg every 4-6 hours. Using carbimazole the dose is 20-30 mg every 4-6 hours.

Iodine is administrated at least one hour after antithyroid drugs to block thyroid hormone release. This situation can seem paradoxal, but we can explain it by Wolff-Chaikoff effect. In fact, administration of high doses of iodine stop TH release during 2 weeks. These doses are 5 drops per os of potassium iodide or 5-10 drops of lugol's solution every 6-8 hours.

Beta-blockers must be administrated at once (after eliminated contraindication: asthma, COPD…) to stop adrenergic effects of a high rate of thyroid hormones. Propanolol, which is non cardioselective, is given with the dose of 1-2mg/min on IV, until the patient's hemodynamic parameters are stable or if the dose of 10 mg is reached, after that switching to oral administration: 40-60 mg every 4-6 hours. In case of contraindication, cardioselective betablocker (metoprolol, atenolol) can be used.

Corticosteroids inhibit the T4 into T3 conversion and are administrated as hydrocortisone 100 mg IV or IM, or 2 mg of dexamethasone IV every 6 hours, until the resolution of the thyroid storm.

In some exceptional situations, when the usual treatment is non effective, other options can be considered: such as plasmapheresis or thyroidectomy (Nayak B *et al*., 2006).

Emergency treatment should not disregard the analysis of the precipiting illness. Searching the underlying cause factor and managing it is the second step after having eliminated a life-threatening element: stabilization of the hemodynamic, respiratory and neurological condition.

The main causes are infection, pregnancy, trauma, surgery emergency, others are less commonly reported such as diabetes ketoacidosis (DKA).

The association between diabetes and thyrocoxicosis is well known. However, the coexistence of DKA and TS is rare and potentially fatal. That is why, it is important to keep it in mind. The diagnostic of diabetes ketoacidosis should make us look for thyrotoxicosis signs and DKA should be sought in patients with thyrotoxicosis (Osada E *et al*., 2011; Yeo K *et al*., 2007). The prognosis depends on earliness diagnosis and management.

But in practice, TS diagnostic can be delayed in patients with DKA, because fever and signs of thyrotoxicosis can be subdued in this case (Kitabchi AE *et al*., 2004 ; Kunishige M *et al*., 2001), and because the rate of thyroid hormones is relatively low.

However, in our second case, the common symptoms between DKA and TS caused diagnostic's delay, essentially tachycardia, vomiting, abdominal pain, consciousness disorder, and no signs orienting towards etiology (goiter, exophthalmos). When the situation did not improve, other investigations and different diagnostics were evoked. In the first case, the thyrotoxicosis syndrome was known since months, thus, the diagnostic was quickly established.

In 1993, Burch and Wartofsky established a scoring system for thyroid storm (Burch HB *et al*., 1993; Satoh T *et al*., 2016), to codify and identify its probability. The Japan Thyroid Association, published in 2008, another diagnostic criterion, very simple and practical as compared to the Burch and Wartofsky's one.

It is not always easy to define which one occurs first: diabetes or Grave's disease. However, glucose intolerance and diabetes are linked to hyperthyroidism (Cooppan R *et al*., 1980), thus, Grave' disease increases glucose intestinal absorption and neoglucogenesis. Thereby, thyroid hormones are associated to triggering and aggravating a diabetes situation.

## CONCLUSION

Thyroid storm represents a rare endocrine emergency condition, which is potentially fatal.

Its main etiology is Graves' disease and can be revealed by an infection, a pregnancy or a ketoacidosis. In the two cases we reported, ketoacidosis was the precipiting factor. We notice that it is a rare association, and it needs codified management in an intensive care unit.

That is why, it seems important to think about the association between these two diseases when ketoacidosis or thyroid storm are evoked.

The prognosis varies depending on whether or not we detected and treated it with sufficient earliness.

**CONFLICT**: The authors declare they have no conflict of interest.

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