Case Report

THYROID STORM PRESENTING AS ACUTE ABDOMEN AND NORMOTHERMIA

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هدف الدراسة : تعد عاصفة الغدة الدرقية من أكثر المضاعفات شيوعاً للمرضى الذين يعانون من مرض الدراق لاسيما أثناء أو بعد إجراء العمليات الجراحية للغدة الدرقية. في هذه الحالة الوصفية نستعرض إحدى الحالات النادرة لأحد المرضى الذين حضروا إلى قسم الطوارئ بمستشفى الخرج وهو يشتكي من ألم حاد بالبطن وبعد إجراء الفحوصات السريرية والمخبرية تبين أن المريض يعاني من ارتفاع هرمونات الغدة الدرقية ، وتم تشخيص الحالة وعلاجها وقد أبدت تحسناً واضحاً وتم خروج الحالة وهي في حالة صحية جيدة وشخصت تشخيصاً نهائياً كحالة نادرة من حالات (العاصفة الدرقية).

الكلمات المرجعية :

Thyroid storm is a known complication of thyroid surgery. Nowadays, it is commonly seen in thyrotoxic patients. In this case report we discuss a patient who presented with acute abdomen and normothermia and was discovered to have thyrotoxicosis, a rare feature of thyroid storm.

***Key Words:** Thyroid storm, acute abdomen.*

INTRODUCTION

Thyroid storm used to be a common complication of toxic goiter surgery during intra and postoperative period. At present, thyroid storm is more commonly seen in thyrotoxic patients or in patients who are undiagnosed with some intercurrent illness like infection, tooth extraction, diabetic ketoacidosis, hypoglycemia, trauma, bowel infection, emotional stress, toxemia of pregnancy, pulmonary embolism, cerebral or myocardial infarction or even drug reaction.[1]

Incidence of thyroid storm is about 10% in hospital admissions with thyrotoxicosis, and the male to female ratio is 1:10. This fulminating condition carries a mortality of 20 to 30%.[2,3]

Burch and Wartofsky have devised a diagnostic point scale to distinguish between thyrotoxicosis, impending storm and an established storm. According to their score system, a score below 25 is unlikely to represent thyroid storm, a score of 25 – 44 is suggestive of an impending crisis, and a score of 45 or greater is highly suggestive of thyroid storm.

CASE REPORT

A forty-year-old healthy Non-Saudi male working as a farm laborer was brought to the Emergency Room (ER) with a history of severe diffuse abdominal pain and repeated vomiting of 10-12 hours' duration. The pain started after his usual meal after work. There was no other relevant present or past history.

Clinical examination in the Emergency Room revealed that the patient was in severe pain and dehydrated . Vital signs were as follows: Pulse 120 beats /min regular, BP 80/60mmHg and Temp 36.5^{0}C. Heart: sinus tachycardia, Chest: normal vesicular breathing, Abdomen: tender right hypochondrium and para-umbilical regions with guarding and feeble bowel sounds. Initial investigations showed marked leucocytosis and hypoglycemia (Table 1).

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Table 1: *Relevant investigation at selected intervals carried out for the patient .*

Investigat -ion	1st day	2nd day	7th day	20th day	Normal Values
WBC	36.11	28.26	18.35	6.93	3.5-10 x 10/ul
Glucose	2.9	9.2	6.2	5	>10 mmol/l
Urea	7.2	11.4	7.7	5.3	1.8 - 7.1 mmol/l
Creatinine	142	154	121	95	53 - 124 umol/l
Sodium	140	143	130	138	133 -152 mmol/l
Potassium	2.98	5	3.75	3.66	3.5 - 5.6 meq/l
T.bilirubin	1.31	2.52	1.46	1.1	0.2 - 1.1 mg/dl
AST	21	639	46	29	Up to 37 u/l
ALT	40	794	195	25	Up to 40 u/l
LDH	359	1355	895	180	240 - 480 u/l
CK	536	6684	168	40	Up to195 u/l
S. amylase	32	44	162		Up to220 u/l

WBC=White blood cell, AST=Aspartate transaminase enzyme, ALT=Alanin transaminase enzyme, LDH=Lactic acid dehydrogenase enzyme,
CK=Creatinine phosphokinase enzyme

The patient was admitted as an acute abdomen for possible exploratory laparotomy. Upon correcting the dehydration, the blood pressure became 120/70mmHg but his general condition continued to deteriorate with the tachypnea (40/min), the abdominal pain became more severe and he was restless and agitated. The patient was shifted to ICU with a pulse rate of 160/min, intermittent atrial fibrillation (AF), raised jugular venous pulse (JVP), systolic murmur over left parasternal region and a scratchy sound over left 2nd and 3rd space with bilateral basal crepitations. Arterial blood gases (ABG) showed hypoxia (PO_2 55mmHg with O_2 mask) and metabolic acidosis (pH 7.15), so the patient was intubated and connected to ventilator.

Six hours after admission, an examination of the neck revealed a minimal enlargement of both lobes of thyroid gland with audible bruit on the right side. Diagnosis of thyroid storm was considered and a blood sample was sent for thyroid hormonal assay. Treatment was started immediately with oral neomercazole 60mg daily followed by lugol's iodine 0.5ml 4 times daily, injection hydrocortisone 100mg daily IV 6 hourly. Inspite of two doses of digoxin injection, he continued to be tachycardiac with atrial fibrillation (AF). He was given a cautious dose of IV propranolol 0.5mg slowly, which brought heart rate down, but he developed transitory hypotension .

In view of his acute abdomen, the patient underwent exploratory laparoscopy, which showed minimal fluid in the Morison's Pouch and right para colic gutter. Laparoscopy was converted into open laparotomy, which proved to be negative. Post operatively, the patient continued to receive same medications. By the second day the patient's condition remained critical with a tremendous increase in AST, ALT, LDH and CK enzymes (Table 1). In the course of the next four days, however, the patient started showing signs of clinical and biochemical improvement as tachycardia settled, no longer manifested signs of pulmonary congestion and showed a steady improvement of the deranged biochemical tests (Table 1). Thyroid hormones results were available at that time : free T3 4.20 pg/ml (N: 2.6-5.10), free T4: 57.12 pmol/l (N: 11.8-24.6), TSH: 0.02 mU/L (N: 0.23-3.80). Blood culture, urine culture, serological tests for brucella, typhoid and VDRL were negative. Gastroscopy showed severe monilial esophagitis, and colonoscopy revealed normal findings.

The patient's recovery was uneventful except the bouts of loose motion he had 6-7 times a day for 5-6 days, a temperature of 38^0C for one day and severe oral candida infection which responded well to mycostatin. He was extubated on the 5th day and slowly resumed his normal diet and started gaining weight. The signs of heart failure were no longer present and sinus rhythm reverted to normal. He was discharged after 30 days of hospital stay, was advised to continue on neomercazole and was given an outpatient appointment. Eight weeks later, the patient came for his appointment. His general condition was good and his thyroid function tests revealed the following results , free T4: 20.1 pmol/l (N: 11.8-24.6), TSH: 0.13 mU/L (N: 0.23-3.80).

DISCUSSION

Thyroid storm is a decompensated state of thyrotoxicosis that can be fatal unless recognized and aggressively treated.[5,6] This was not a known case of thyrotoxicosis, nor was there a history of any complaints suggestive of thyrotoxicosis. Moreover, his thyroid gland was minimally enlarged and could have been missed in a patient who was critically ill. In this case, however, our patient's score on the HB Burch's diagnostic point scale, was 70 points, which was well above the diagnostic criteria of thyroid storm.[4]

Our patient presented as a case of acute abdomen and it was difficult to postulate whether the abdominal symptoms were due to thyrotoxic storm as one of its rare manifestations or due to some other precipitating causes since the interrelationship of the gut and thyroid is well known.[2,7]

Hyperthermia with a temperature of 40^0C is a salient feature of thyroid storm as a result of defective thermoregulation and thyroid hormones that potentiate calorogenic effects of other hormones like catecholamines. Excessive heat energy so produced if not dissipated, causes a rise in temperature.[5] This case, however, was unexplainably normothermic. This is the second reported case so far, of thyrotoxic storm with normothermia.[7] This patient also had metabolic acidosis that might have been due to an accumulation of lactic acid (not measured in our case) as a result of increased tissue oxygen supply resulting from cardiovascular decompensation.[8,9] Elevated liver and muscle enzymes along with bilirubin, urea and creatinine which returned to normal after treatment, supports the view that the patient also had multi organ dysfunction.

Thyroid storm is precipitated by many factors, some of which are emotional stress, hypoglycemia and occult infection. All of these could be relevant in our patient.[3] The patient was an expatriate, away from his family and consequently more prone to emotional stress. He presented with definite hypoglycemia (RBS: 2.9 mmol/dl). The possibility of an occult infection was suggested by the marked leukocytosis.

Our case report highlights atypical and rare features of thyrotoxic storm, such as normothermia, acute abdomen, and acidosis and multi-organ dysfunction. Moreover, the storm can occur in a patient with a minimally enlarged thyroid gland, which could be missed clinically. These abnormal features may be deceptive in the making of the proper diagnosis unless one is aware that thyroid storm could present with varied clinical features. When thyroid storm is suspected clinically, the appropriate treatment should not be withheld or delayed until there is laboratory confirmation of hyperthyroidism. A timely diagnosis and intervention would prevent a fatal outcome.

ACKNOWLEDGMENT

I appreciate Dr. Aggarwal S. Kumar and Dr. Mohammed Abul-Fattah Ali for their help in typing the draft of this report and linguistic revision.

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