THYROID STORM WITH ACUTE MYOCARDIAL INFARCTION: A CASE REPORT

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ABSTRACT
Thyroid storm is hyperthyroid-related medical emergency with high mortality rate. Acute myocardial infarction cause even higher mortality in thyroid storm. Early recognition and immediate treatment are important to prevent mortality. Therefore, a case of adult female with thyroid storm and myocardial infarction was presented. A woman, 46 years old, complained pressing chest pain felt from middle chest to the pit of the stomach and spread to the left back. She also had got dyspnea and palpitated for 2 days. Patients had history of uncontrolled hypertension for 4 years, cardiac problem for 3-4 years and a lump in the neck for 5 years. Physical examination found tachycardia, fever, and anemia. A mass was palpated in the anterior region of neck moving upward while swallowing with a diameter approximately 6 cm, having rubbery consistency and undefinable border. Physical examination also found fine tremor on the extremities. Meanwhile, the laboratory examination found increased cardiac marker, high thyroid hormone level and low TSH. Electrocardiograph (ECG) showed ischemic on inferior and anterior area. In addition, Chest radiography showed cardiomegaly with CTR of 58%. Echocardiography found EF of 65%. Therefore, the patient was diagnosed as Thyroid Storm (TS) with very high risk NSTEMI and anemia. As a treatment, the patient was given prophylthiouracyl (PTU), lugol solution, propranolol, intravenous injection of metilprednisolone, ASA, clopidogrel, atorvastatin, lisinopril and ISDN 5 mg. On the day 11th, she was discharged.

Keywords: Acute, Coronary Syndrome, Acute Myocardial Infarction, Non-ST Elevation Myocardial Infarction, Thyroid Storm

I. INTRODUCTION
The thyroid storm (TS) is a hyperthyroid-related medical emergency characterized by decompensation of one or more organ systems. The mortality rate of thyroid ranges from 20 to 30%. Rapid recognition and treatment are essential to limit morbidity and mortality.¹

Acute coronary syndrome (ACS) is the commonest cardiovascular disease. Non-ST elevation myocardial ischemia (NSTEMI) is one of type of ACS defined by an elevation of cardiac biomarkers in the absence of ST elevation and presence of elevated cardiac enzymes.

Approximately 70% of ACS is NSTEMI. Patients with NSTEMI typically have more comorbidities, both cardiac and noncardiac, than STEMI. NSTEMI is a recognized diagnosis that has an unacceptable mortality rate when it goes unrecognized.¹²

Thyroid hormones (TH) possibly result in decreased systemic vascular resistance, increased blood volume, increased contractility, and increased cardiac output. Although angina pectoris occurs occasionally in patients with thyrotoxicosis, myocardial infarction (MI) is rarely reported. Higher mortality has been found in overt hyperthyroidism with cardiovascular and cerebrovascular disease. On the contrary, TS can be precipitated by an acute illness such as a MI, infection, or other stress. Acute myocardial infarction (AMI) and hyperthyroidism can be fatal if it left undiagnosed or treated incorrectly.¹³ Therefore, a case of TS accompanied by AMI is presented in this case report.
II. CASE REPORT

HISTORY TAKING

A 46 years old female was admitted to hospital through the Emergency Room with a chief complaint of chest pain. Patients had a chief complaint of heavy chest pressure at the middle chest and the pit of the stomach, radiating to the left back and arm since 1 day before admission. During hospitalization patient was treated in ICCU room and cardiology low care room before been referred for an internal medicine subdivision endocrinology opinion and therapy because the results obtained thyroid function serological examination that shows a hyperthyroid. Patient was unable to communicate since 3 days before being referred, got fever since 2 days before being referred, and accompanied with diarrhea. In addition, she also had dyspnea and palpitated for 2 days.

Patients had history of uncontrolled hypertension for 4 years, cardiac problem for 3-4 years and a lump in the neck for 5 years. Meanwhile, familial medical illness was denied.

PHYSICAL EXAMINATION

Based on physical examination, patient was apathetic (GCS 446), had increased heart rate (112 x/minute), fever (38.5°C). Blood pressure on the right upper arm was 130/90 mmHg. Head examination resulted anemic conjunctiva. A mass was palpated in the anterior region of neck moving upward while swallowing with a diameter approximately 6 cm, having rubbery consistency and undefinable border. Hence, the examination also found fine tremor on the extremities.

LABORATORY EXAMINATION

The laboratory test resulted the hypochromic anemia (Hb 10.4 g/dL; Hct 32%; MCH 26.4 pg; MCHC 31.8 g/dL), slightly increased BUN (35 mg/dL), hypoalbuminemia (3.1 g/dL), increased cardiac marker (CKMB 43,7 IU/L; Troponin T 584,5 ng/L), high thyroid hormone level (FT3 > 8,0 pg/mL; FT4 > 24,86 ng/dL) and low TSH (0.005 mIU/L). Electrocardiograph (ECG) showed sinus rhythm 89 times per minute, clock-wise rotation of horizontal axis, ischemic on inferior and anterior area. ECG while at Internal Medicine HCU room was sinus tachycardia 105x/minute, with normal axis. Plain chest radiography showed cardiomegaly with CTR of 58%. Echocardiography found diastolic dysfunction, left ventricular concentric remodeling, EF of 65%.

DIAGNOSIS

The patient was diagnosed with TS with very high risk NSTEMI and anemia.

THERAPY

The patient was planned to have TSH, FT4, CBC, and hemostasis test. The patient was admitted to the ICCU. She was suggested to have total bed rest and given O2 8 lpm with simple mask. She was given prophylthiouracyl (PTU) loading dose 400 mg once per day orally, continued by 200 mg 6 times per day orally, lugol solution 6 drops per 6 hours 1 hour after PTU, propanolol 10 mg per 6 hours orally, intravenous injection of metilprednisolone 25 mg three times per day. ASA 100 mg per 24 hours orally, clopidogrel 75 mg once per day, atorvastatin 40 mg per 24 hours, lisinopril 5 mg per 24 hours and ISDN 5 mg three times per day orally.

DISEASE PROGRESSION

After her mental status improved on 4th day, she complained of palpitation and general weakness and the TSH level got lower. Consequently, she got moved to the regular ward. The methylprednisolone started to be tapered off on the day 6th as the complains improved. On the 11th day, she was discharged and planned to visit the endocrine and cardiac outpatient clinic in Dr. Soetomo Hospital.

III. DISCUSSION

Patients with hyperthyroidism can develop a life-threatening complication called TS. The mortality rate of this condition ranges from 10% to 75%. Multiple organ failure and infection are the most common cause of death, followed by congestive heart failure, respiratory failure, arrhythmia, disseminated intravascular coagulation, gastrointestinal
perforation, hypoxic brain syndrome, sepsis, diabetic ketoacidosis, hypoglycemia, hyperosmolar coma, pulmonary embolism, thyroid hormone overdose, withdrawal of anti-thyroid medications, iodinated contrast medium ingestion, vascular accidents, surgery, stress, parturition, eclampsia, trauma and MI. However, 25% to 43% of the patients have no identifiable factor.[6]

Diagnostic criteria for TS were first proposed in 1993 and subsequently widely adopted as Burch-Wartofsky Point Scale (BWPS). These criteria include hyperpyrexia, tachycardia, arrhythmias, congestive heart failure, agitation, delirium, psychosis, stupor and coma, as well as nausea, vomiting, diarrhea, hepatic failure, and the presence of an identified precipitant. Points are awarded in the BWPS system based on the severity of individual manifestations, with a point total of ≥ 45 consistent with TS, 25-44 points classified as impending TS, and < 25 points making TS unlikely.[4,7]

The cause of myocardial ischemia and infarction in thyrotoxic patients remains unclear. Researcher predicts temporary occlusion of a major coronary artery, severe coronary artery spasm or a direct metabolic effect to the myocardium may play role. Supraventricular tachycardia or atrial fibrillation secondarily cause MI as well.

Thyroid hormone (TH) affects cardiac muscle via the sympathetic nervous system and direct effect on the catecholamine-independent myocardial contractility. Excessive TH can increase cardiac oxygen demands by producing a hypermetabolic state and cause relative myocardial ischemia due to insufficient blood supply.[8,9]

Treatment should be initiated once TS is suspected to decrease the mortality and morbidity. The treatment includes supportive care, inhibition of new hormone synthesis, inhibition of thyroid hormone release, peripheral β-adrenergic receptor blockade, preventing peripheral conversion of T4 to T3, identifying and treating precipitating factors. TS is treated as another acute disease, which is initial assessment according to airway, breathing, circulation and disability assessment.[6,10]

Thionamides, such as methimazole or propylthiouracil (PTU), are used as primary treatment of thyrotoxicosis. They decrease the synthesis of TH production by preventing organification, trapping of iodide to iodine and inhibiting coupling of iodothyrosines. Hence, these drugs must be instituted first, and only given at least 1 hour later.

Iodine Lugol solution, potassium iodide or ipodate can be given to stop thyroid hormone release. Iodine blocks the release of pre-stored hormone and decreases iodide transport and oxidation in follicular cells. If iodine is contraindicated, such as due to hypersensitivity, lithium ought to be consumed. Lithium inhibits thyroid hormone release from the thyroid gland as well as decreases thyroid hormone synthesis.[6,10]

Glucocorticoid, for example Hydrocortisone, dexamethasone, blocks the peripheral conversion of T4 to T3, which is responsible for 80% of T3 present in the circulation.

Propranolol is given intravenously in slow 1–2 mg boluses and could be repeated every 10–15 min until improvement is achieved. Orally, propranolol therapy begins at 20–120 mg per dose or 160–320 mg per day in divided doses.

A vigorous search for an infectious source should be done in febrile thyrotoxic patients. This could be done with blood, urine, throat, and sputum cultures. A chest radiograph should be done to rule out chest infection. In addition, an electrocardiogram can also be done to rule out MI, ischemia, or arrhythmia.[6,10]

Management of TS with loading dose of 400 mg of PTU followed by maintenance dose of 100-200 mg of PTU every 4 hours, or Loading dose 40 mg thyrosol, followed by 10-20 mg thyrosol, iodides 1 hour after initiation of PTU every 4 hour, 6 drops of Lugol’s solution every 6 hours. 6 drops of lugol’s solution every 6 hours, 10-40 mg of propanolol orally every 6 hours or 0.5-1 mg of propanolol intravenously every 3 hours, 100 mg of hydrocortisone or 2 mg dexamethasone intravenously every 8 hours, clinical improvement within 24 hours and resolved TS within 6 days. However, improvement in TS usually occurs within 24-72 hours. In addition, the formula of 7-1-5 indicates the drugs, especially the corticosteroids, are administrated at 07.00 am, 01.00 pm, and 05.00 pm. Once haemodynamic, thermoregulatory and neurological stability has been achieved, the patient was given the maintenance therapy. The antithyroid treatment should be continued as the maintenance therapy until euthyroidism is achieved.[11,12]
Acute coronary syndrome (ACS) is a spectrum of conditions compatible with acute myocardial ischemia and/or infarction that are usually due to an abrupt reduction in coronary blood flow. NSTEMI is one of ACS causes defined by an elevation of cardiac biomarkers in the absence of ST elevation. The term “possible ACS” is often assigned during initial evaluation if the ECG is unrevealing and troponin data are not yet available.\(^2\,^13\)

The pathogenesis of ACS is the sudden imbalance between myocardial oxygen consumption and demand mostly caused by coronary artery obstruction. Acute coronary insufficiency, noncoronary factors, nonischemic myocardial injury and other multifactorial factors may cause myocardial oxygen supply-demand mismatch as well.\(^14\)

The typical clinical presentation of ACS is retrosternal pressure or heaviness (angina) radiating to the left arm, neck or jaw and lasting several minutes or occasionally persistent. These symptoms are possibly accompanied by dyspnea, diaphoresis, nausea, vomiting, palpitations and syncope. Atypical symptoms can also occur including epigastric pain, recent onset indigestion, stabbing chest pain, chest pain with pleuritic symptoms, or increasing dyspnea.\(^2\)

The patient complained of chest pain felt from middle chest to the pit of the stomach and spread to the left back accompanied with limp, which led to ACS.

History that may lead to MI, such as age, diabetes mellitus, hypertension, smoking, family history, angina, dyspnea, aspirin intake, CAD, and dyslipidemia, has to be taken and evaluated. The presence of tachycardia, heart failure or hemodynamic indicates early diagnosis and prompt treatment. It is also important to identify clinical circumstances that may precipitate or exacerbate NSTEMI, such as anemia, infection, fever and metabolic or thyroid disorders. Non-cardiac causes of chest pain and non-ischemic cardiac disorders, such as pulmonary embolism, aortic dissection, pericarditis, valvular heart disease, or extra cardiac causes must be excluded.\(^15\)

Electrocardiogram of NSTEMI may include ST depression, transient ST-elevation, or new T-wave inversion. ST depression of >2 mm carries an increased mortality risk. Inverted T waves, especially if greater than or equal to 2 mm (0.2 mv) also indicate NT-STEMI. Q waves suggesting prior MI indicate a high likelihood of IHD. In fact, normal ECG does not exclude ACS and occurs in 1% to 6% of such patients. Comparison with the previous ECGs is required.\(^2\,^15\)

Electrocardiography (ECG) in this case showed sinus rhythm 89 x/minute, normal frontal axis, horizontal axis CWR, ischemic on inferior and anterior area, leading to NSTEMI or UA.

Cardiac troponins (CTN) are the most sensitive and specific biomarkers for NSTEMI. Troponin levels usually increase after 3-4 hours and remain elevated for up to 2 weeks. Elevated CTN values signal a higher acute risk and an adverse long-term prognosis. CK-MB is less sensitive and specific for the diagnosis of NSTEMI.\(^16\)

Based on laboratory tests conducted, the result showed increase in CKMB and Troponin T, leading to NSTEMI.

Echocardiography and Doppler examination are recommended after hospitalization to assess the global left ventricular function, wall motion abnormality, pericardial effusion and causes of chest pain other than MI.\(^2\)

The goals of NSTEMI treatment are the immediate relief of ischemia and the prevention of MI and death. Stable patients with definite or probable NSTEMI consequently should be admitted to an inpatient unit for bed rest with continuous rhythm monitoring and careful observation for recurrent ischemia. Patients with continuing angina, hemodynamic instability, uncontrolled arrhythmias, or a large MI should be admitted to a coronary care unit. Fibrinolytic (thrombolytic) therapy was proven harmful for NSTE-ACS.\(^1\,^2\)

Patients with NSTEMI with continuing ischemic pain should receive sublingual nitroglycerin (0.3 mg to 0.4 mg) every 5 minutes for up to 3 doses, after which an assessment should be made about the need for intravenous nitroglycerin if not contraindicated. Intravenous nitroglycerin is indicated for patients with NSTEMI for the treatment of persistent ischemia, HF, or hypertension. It is also reasonable to administer morphine sulfate intravenously if there is continued ischemic chest pain despite the nitroglycerin.\(^17\,^18\)
Oral beta-blocker therapy should be initiated within the first 24 hours in patients assuming no continuing or frequently recurring ischemia in NSTE-ACS. Otherwise, nondihydropyridine calcium channel blocker (CCB), such as verapamil or diltiazem, should be given alternately. Additionally, high-intensity statin therapy is also strongly recommended for all NSTE-ACS.[19,20]

Aspirin and clopidogrel should be given to all patients with NSTE-ACS without contraindications as soon as possible after presentation, and continued by the maintenance dose. Clopidogrel should be maintained for at least 12 months unless there is an excessive risk of bleeding.[21]

IV. CONCLUSION
A 46 years old female was reported to experience TS with NSTEMI. The combination of AMI and TS are dangerous if left undiagnosed or treated incorrectly. The patient was treated with clopidogrel, aspirin, atorvastatin, and Ramipril for her MI while the TS was treated by administering prophylthiouracyl (PTU), lugol solution, propanolol 10 mg and metilprednisolone using TC-41668.24.6 and 7-1-5formula. Her condition was improved then discharged after 11 days of treatment.

Conflict of Interest
There is no conflict of interest in this research.

Ethical Clearance
Not required for a case report.

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