Thyroid Heart Disease Presenting Thyrotoxic Crisis with Jaundice

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ABSTRACT

Hyperthyroidism can cause thyroid crisis that are a rare endocrinial emergency. Jaundice in thyroid heart disease and hyperthyroid patient was very rare. This case we present a patient with thyroid heart disease presenting thyroid crisis and jaundice. A 49-year-old with complaints of shortness of breath at night accompanied by a feeling of weakness. The patient felt tremor in her upper extremity, lost weight, difficulty sleeping and easily tired in the last 2 months. She said that her eye become yellow in color 10 days ago. On admission the vital signs are blood pressure 130/80 mm Hg, pulse 120x/min, respiration rate 24x/min, O2 saturation 80% on room air, temperature 36.8°C. Physical examination showed icteric, jugular vein pressure was +3 cm and from palpation examination found nodule ±4cm in diameter, well-defined, chewy consistency. Extremities showed tremor and edema. Electrocardiography showed atrial tachycardia and chest x-ray imaging showed cardiomegaly and minimal pleural effusion. Thyroid hormone evaluation showed increase Free T4 and decrease TSH level. Liver function test showed increase bilirubin level. Patient received Propylthiouracil 4x300 mg, Propranolol 4x20mg, normal saline 0.9% Infusion 1000ml/24 hours, Pantoprazole Injection 2x40 mg, Furosemide 10 mg/hour, Spironolactone 1x100mg, Albuforce 2x1 tab, Injection of Ceftriaxone 2x1 gr and referred to RS Soetomo, Surabaya for further treatment Percutaneous Transhepatic Biliary Drainage.

Keywords: thyroid heard disease, Thyrotoxic Crisis, Jaundice, Hyperthyroidism

INTRODUCTION

Hyperthyroidism is a chronic pathological condition when excess thyroid hormone is synthesized and secreted by the thyroid gland.¹ The prevalence of hyperthyroidism in Indonesia approximately was 0.4%.² Thyroid hormone regulates multiple cardiovascular functions. Hyperthyroidism can cause complications such as atrial arrhythmias, weight loss, and both cause hypertension and heart failure called thyroid heart disease.³ There was only 1-2% of cases of hyperthyroidism manifest as thyrotoxicosis.⁴
The other acute complication of hyperthyroidism is a thyroid storm. Thyroid storm is a rare endocrinological condition emergency due to hyperthyroid. Heart failure is the leading cause of morbidity and mortality in patients with thyroid storm.\(^{(5)}\) Thyroid heart disease patient who presents thyroid crisis and jaundice is very rare, especially in Madura.

**CASE ILLUSTRATION**

A 49-year-old Madurese woman presented to the Syamrabu Bangkalan Hospital on January 2022 with complaints of palpitations, and shortness of breath at night accompanied by a feeling of weakness. The patient also complained of a grape-sized lump on the patient’s neck, the first lump appeared about 3 years ago. The lump appeared for the first time when the patient often worked as a household assistant with high activities. The patient never took treatment when a lump appeared on the neck. The patient felt tremors in her upper extremity, lost weight, had difficulty sleeping, and was easily tired in the last 2 months. She said that her eyes become yellow in color 10 days ago. The patient also felt abdominal pain on the right side of the abdomen. The pain was localized, burning, sharp, and intermittent. The patient’s legs have been swollen since a week ago, and he has also complained of diarrhea and nausea without vomiting since 4 days ago. The patient also complained her urine was darker than 2 weeks ago.

On physical examination showed a Glasgow coma scale (GCS) 4-5-6, moderately ill and weak. A vital sign showed that blood pressure: 130/80 mmHg, the pulse rate: 120x/min, respiratory rate: 24x/min, temperature: 36.8°C, O2 saturation 80% on room air, and SpO2 98% with O2 nasal cannula.

Regarding the eyes examination showed exophthalmos and icteric in both eyes. On examination of the local status in the neck region, the jugular vein pressure was +3 cm and from palpation, the examination found a nodule ±4cm in diameter, well-defined, chewy consistency, moves during swallowing, and no tenderness in the right and left thyroid gland (Figure 1).

Thoracic examination revealed symmetrical right and left lung movements, on percussion, there were resonances in both lung fields, the shift of the ictus cordis widened laterally from the left midclavicular ICS, and vesicular breath sounds in both lung fields without additional breath sounds such as crackles and wheezing. Cardiac auscultation examination revealed 1 and 2 single regular heart sounds, Thrill, S3 sound of cor, murmurs. Abdominal distension was found bowel sounds were within normal limits, liver and spleen were palpable enlarged. On extremities showed tremors in both hand fingers and there is edema of the right hand and both feet. During the history and physical examination, the value of the Wayne index criteria was obtained, with complaints in the form of palpitations, tired quickly, preferring cold air, progressive weight loss,
slightly increased appetite, palpable thyroid gland, tremor of the upper extremities was found, with a pulse > 90 beats/minute also found atrial fibrillation.

Hyperthyroid disease scoring can be used based on the symptoms experienced by the patient which uses the Wayne Index criteria. In this patient, the Wayne index criteria were obtained with a scoring range of +35, where if the score was >19 it could be said that hyperthyroidism was toxic, whereas if the score was less than 11, it could be said to be euthyroid. Meanwhile, the condition where the score is 11 to 19 is called the equivocal condition (table 1). The initial use of Wayne’s index score is a clinical index to establish a clinical diagnosis for hyperthyroidism.\(^6\)

<table>
<thead>
<tr>
<th>Symptoms of recent onset and/or increased severity</th>
<th>Score</th>
<th>Signs</th>
<th>Present</th>
<th>Absent</th>
</tr>
</thead>
<tbody>
<tr>
<td>Dyspnea on effect</td>
<td>+1</td>
<td>Palpable thyroid</td>
<td>+3</td>
<td>-3</td>
</tr>
<tr>
<td>Palpitations</td>
<td>+2</td>
<td>Bruit over thyroid</td>
<td>+2</td>
<td>-2</td>
</tr>
<tr>
<td>Tiredness</td>
<td>+2</td>
<td>Exophthalmos</td>
<td>+2</td>
<td></td>
</tr>
<tr>
<td>Preference for heat</td>
<td>-5</td>
<td>Lid retraction</td>
<td>+2</td>
<td>-</td>
</tr>
<tr>
<td>Preference for cold</td>
<td>+5</td>
<td>Lid lag</td>
<td>+1</td>
<td>-</td>
</tr>
<tr>
<td>Excessive sweating</td>
<td>+3</td>
<td>Hyperkinesis</td>
<td>+4</td>
<td>-2</td>
</tr>
<tr>
<td>Nervousness</td>
<td>+2</td>
<td>Hands hot</td>
<td>+2</td>
<td>-2</td>
</tr>
<tr>
<td>Appetite: increased</td>
<td>+3</td>
<td>Hands moist</td>
<td>+1</td>
<td>-1</td>
</tr>
<tr>
<td>Appetite: decreased</td>
<td>-3</td>
<td>Casual pulse rate: &gt;80/min</td>
<td>-</td>
<td>3</td>
</tr>
<tr>
<td>Weight increased</td>
<td>-3</td>
<td>&gt;90/min</td>
<td>+3</td>
<td>-</td>
</tr>
<tr>
<td>Weight decreased</td>
<td>+3</td>
<td>Atrial fibrillation</td>
<td>+4</td>
<td>-</td>
</tr>
</tbody>
</table>

The results of laboratory examinations at the beginning of hospitalized patients on January 28, 2022, obtained Hb 9.7 g/dL (N : 11.7-15.5), Hct 29.1% (N : 35-47), platelets 62 x 10\(^3\)/μL (N : 154-386). Liver function results SGOT 64 U/L (N : 0-37), Albumin 2.9 g/dL (N : 3.4-4.8), total bilirubin >25.00 mg/dl (N : 0.1-1.0), direct bilirubin > 15.00 mg/dl (N 0-0.2), non-reactive HBsAg. Another result of clinical chemistry was fasting glucose 91 mg/dl (N : 70-105), 3 February 2022 the BUN value was 92 mg/dl (N : 4.6-23), creatinine was 2.33 mg/dl (N : 0.25-0.75). Thyroid hormone examination found TSH <0.05 IU/ml (N : 0.25-5), FT4 >7.77 Pg/ml (N : 0.9-1.78). The results of electrolyte laboratory values showed results within normal limits with insignificant increase.

On electrocardiogram (ECG) the results showed atrial fibrillation. On examination of chest x-ray the result showed Cardiomegally and minimum effusion pleura condition.
In this patient, an ultrasound examination of the thyroid showed that the thyroid parenchyma was diffusely enlarged with homogeneous parenchymal intensity and firm boundaries of the regular type accompanied by increased vascularity on Doppler examination.

Based on history and examination, the patient was diagnosed with Thyroid heart disease, thyroid crisis condition with hyperbilirubinemia, the patient was treated with Propylthiouracil 4x 300 mg, Propranolol 4x20mg, normal saline 0.9% Infusion 1000ml/24 hours, Pantoprazole Injection 2x40 mg, Furosemide 10 mg/hour, Spironolactone 1x100mg, Albuforce 2x1 tab, Injection of Ceftriaxone 2x1 gr and referred to RS Soetomo for further treatment Percutaneous Transhepatic Biliary Drainage (PTBD).

**DISCUSSIONS**

Hyperthyroidism is the most common form of thyrotoxicosis, resulting from excess secretion of thyroxine (T4) or triiodothyronine (T3). Grave’s disease is the most common cause; Approximately 60% of hyperthyroidism is caused by Graves’ disease. Hyperthyroidism in Graves’ disease is usually caused by the presence of TSH receptor antibodies that stimulate excessive thyroid activity. The Burch-Wartosfky criteria can help to establish the diagnosis of thyroid crisis based on the existing criteria.\(^\text{3}\) Making a diagnosis of thyroid crisis is difficult because the clinical symptoms resemble infections such as tachycardia, high fever, and sometimes shortness of breath. The etiology of hyperthyroid crisis can be due to excessive iodine intake, sudden discontinuation of antithyroid drugs, infection, trauma, cerebrovascular disease, severe and emotional stress.\(^\text{3}\)

In this case, the patient lives in an area close to the coast, an area where there is a lot of iodine. There was no history of infection, trauma, or cerebrovascular disease. The patient admits to irregularities in taking antithyroid drugs.\(^\text{4}\)

The reported case describes a hyperthyroid crisis with cardiac and hepatic dysfunction in a 49-year-old woman. Theoretically, the diagnosis of hyperthyroidism is described by the presence of symptoms found in thyrotoxicosis are as follows: nervousness and anxious; excessive sweating, warm skin and heat intolerance; irritability; palpitations; excessive bowel movements; fatigability; weight loss with increased appetite \((\text{Von Muller paradox})\) and
menstrual disturbances. The presence of a thyroid crisis is also described. The clinical findings are a picture of excessive hyperthyroidism accompanied by manifestations of multi-organ dysfunction in the presence of precipitating factors. As for this patient, the clinical signs observed were as follows: thyroid gland hyper trophy or struma; Soft bruits are found on palpation of the patient’s neck; warmth and increased sweating of the skin; tremor; muscle fatigue & exophthalmos.

Cardiovascular manifestations in these patients include palpitations, thrill condition & systolic hypertension, tachycardia, exercise intolerance, dyspnea on exertion, dilated pulse pressure, and atrial fibrillation. The central nervous system (CNS) manifestations include agitation, delirium, confusion, stupor, and coma. CNS involvement is a poor prognostic factor for mortality. In this reported patient, the patient appeared to be agitated and confused, this was shown during follow-up observation of the patient where the patient did not understand well the instructions given by the physician, on the contrary the patient tended to be restless and held his hand repeatedly. Gastrointestinal (GI) symptoms include nausea, vomiting, diarrhea, abdominal pain, intestinal obstruction, and acute hepatic failure.

Meanwhile, in this patient, the following gastrointestinal disorders were experienced: nausea and diarrhea, abdominal palpation found tenderness, hepato-splenomegaly and acute hepatic failure. This condition can occur due to hepatic congestion or hypoperfusion, it is also accompanied by jaundice in the skin and eyes of the patient which indicates liver dysfunction.

Liver dysfunction in thyroid disease can range from asymptomatic enzyme elevations to acute liver failure (ALF). In patients with hyperthyroidism, there can be several explanations for liver dysfunction. In vitro studies in animals have shown that excess triiodothyronine (T3) activity can induce hepatocyte apoptosis through a mitochondrial-dependent pathway. In addition, T3 is responsible for regulating enzymes involved in bilirubin metabolism, and excess levels can result in the accumulation of bilirubin precursors.

To establish the diagnosis of hyperthyroidism, it is obtained from the patient’s history, physical examination which includes blood pressure, pulse, palpation and auscultation of the thyroid gland, eye and heart examination and diagnostic support. Laboratory tests required are free T4 (FT4) and thyroid stimulating hormone (TSH) levels for hyperthyroidism. Other investigations needed are chest X-ray, electrocardiography, and echocardiography to see heart problems.

Heart failure as a complication of hyperthyroidism can be established using the Framingham criteria. The ECG examination showed rapid atrial fibrillation at a heart rate of 124 beats/min (Fig. 1). She was diagnosed with congestive heart failure and rapid atrial fibrillation caused by hyperthyroidism. The ECG finding were an irregular rhythm, with a pulse rate of 124x/minute, normal axis, normal P waves and a PR interval of 0.20 seconds, QRS duration of 0.08 seconds and signs of left ventricular enlargement.

Management of cardiovascular disease in hyperthyroidism is to immediately reduce the hypermetabolic state by administering antithyroid drugs to reduce thyroid hormone levels and treating other cardiovascular manifestations such as decreasing heart rhythm and administering antihypertensive drugs.
The use of the Wayne Index can assist in establishing the diagnosis of hyperthyroidism, consisting of 9 symptoms and 10 signs each with a score of 85% with an accuracy rate of 85%. In this case, the patient was diagnosed with hyperthyroidism based on the Wayne index, a score of 35 was obtained. Based on laboratory tests, a decrease in TSH levels was found, namely TSH <0.05 Ul/ml (N : 0.250-5.00), FT4 >7.77 Pg/ml (N : 0.930-1.700). In addition to serum levels of TSH and FT4, on other laboratory investigations, an increase in serum SGOT of 64.00 mg/dl was also found which indicated changes/disorders in the patient's liver and heart. Serum albumin value of 2.9 indicates that the patient has hypoalbuminemia which results in a decrease in intravascular oncotic pressure and causes fluid to seep into the interstitial space and results in limb edema, ascites, and pleural effusion. Total bilirubin >25.00 and direct >15.00, these values indicate impaired liver function so that excessive bilirubin secretion occurs. On electrocardiographic examination that was performed on the patient, atrial fibrillation was found with normal axis and hypertrophy of the left ventricle. Left ventricular hypertrophy can be suspected as an enlargement of the heart and this is also supported by the findings from the physical examination which revealed a lateral shift of the icterus cordis from the left midclavicular ICS V. Anatomically, thyroid hormone can cause cardiac hypertrophy as a result of increased protein synthesis. The increase in minute volume is due to an increase in heart rate and stroke volume, a decrease in peripheral resistance, and the presence of heating-induced peripheral vasodilation due to increased tissue metabolism. The cause of liver failure in hyperthyroid patients is caused by several factors. The cholestatic type of liver disorder of the intrahepatic lobule is more prominent in the above cases. Cholestasis is a condition in which the flow of bile into the duodenum is slowed or blocked so that it fails to meet the normal amount. Clinically, cholestasis can be defined as the accumulation of substances excreted into the bile such as bilirubin, bile acids and cholesterol in the blood and body tissues. The patient is said to be cholestatic if the direct bilirubin level is more than 1 mg/dl and the total bilirubin is less than 5 mg/dl. In Grave's disease, cholestasis is associated with an autoimmune disease that can attack liver cells and cause primary biliary cirrhosis (PBC) or autoimmune hepatitis. It can occur in 10% of patients with hyperthyroidism. Liver damage caused by thyrotoxicosis or hyperthyroidism can also be caused by ischemic hepatitis, which occurs due to reduced blood flow to the liver despite increased metabolism. Other mechanisms of liver damage can also be caused by congestive heart failure as in the case above. Antithyroid drugs can also cause hyperbilirubinemia such as propylthiouracil (PTU). The treatment of thyroid crisis includes the administration of beta-blockers, anti-thyroid drugs, iodine, and corticosteroids (Table 2). Treatment is supportive by maintaining the patient’s body temperature by placing a cooling blanket and administering acetaminophen. Patients with thyroid storm deserve fluid resuscitation, supportive breathing, and monitoring in the intensive care unit.
Table 2. Thyroid crisis medication and dosage

<table>
<thead>
<tr>
<th>Drug</th>
<th>Dosage</th>
<th>Mechanism of Action</th>
</tr>
</thead>
<tbody>
<tr>
<td>Propylthiouracil (PTU)</td>
<td>Initial dose 500–1000 mg, then 250 mg every 4 hours</td>
<td>Inhibits hormone synthesis Inhibits conversion of T4 to T3</td>
</tr>
<tr>
<td>Methimazol</td>
<td>60–80 mg/day</td>
<td>Inhibits hormone synthesis</td>
</tr>
<tr>
<td>Propanolol</td>
<td>60–80 mg every 4 hours</td>
<td>Administered to patients with congestive heart failure Inhibits conversion of T4 to T3</td>
</tr>
<tr>
<td>Lugol’s Iodine (potassium iodine)</td>
<td>5 drops (0.25 mL or 250 mg) orally every 6 hours</td>
<td>Inhibits hormone synthesis Inhibits hormone release into the bloodstream</td>
</tr>
<tr>
<td>Hydrocortisone</td>
<td>Initial dose of 300 mg intravenously, then 100 mg every 8 hours</td>
<td>May inhibit conversion of T4 to T3 For prophylaxis of adrenal insufficiency</td>
</tr>
</tbody>
</table>

In this case, the patient received therapy with Propylthiouracil 4x300 mg, Propranolol 4x20mg, PZ Infusion 1000ml/24 hours, Pantoprazole Injection 2x40 mg, Furosemide 2x10 mg, Spironolactone 1x100mg as treatment for congestive heart failure. The patient is in the High care unit room and monitors the Electrocardiogram every day. After giving propranolol for 3 days, the patient’s heart rhythm showed a slight improvement. After 12 days of treatment, the patient’s condition had not improved clinically and in the laboratory, the patient was planned to be referred to RS Soetomo Surabaya for further treatment of Percutaneous Transhepatic Biliary Drainage (PTBD).

CONCLUSION

Reported a case of Thyroid heart disease in which the patient experienced multi-organ failure in the heart, liver and blood. In patients with multi-organ failure, thyroid crisis should be considered as the cause. Rapid diagnosis and treatment of hyperthyroid crisis in areas with limited access to services for prevention and treatment can reduce mortality rates significantly.

References


