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CASE REPORT

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Right ventricular failure due to acute pulmonary embolism associated with Graves' disease: A case report

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ABSTRACT:

Background: Graves' disease is a prevalent endocrine disorder characterized by diverse clinical manifestations affecting multiple organs, exhibiting varying degrees of severity. Cardiovascular system involvement is one of the most common manifestations, which include palpitation, tachycardia, cardiomyopathy, atrial fibrillation, pulmonary hypertension, or heart failure. Additionally, excessive thyroid hormone can lead to a hypercoagulable state, increasing the risk of venous thrombosis. However, thrombotic events, particularly deep vein thrombosis, cerebral venous thrombosis, or pulmonary embolism, are rare complications of Graves' disease.

Case presentation: In this report, we present the case of a 53 years old woman who presented with progressively worsening dyspnea, orthopnea, paroxysmal nocturnal dyspnea, bilateral leg edema, and jaundice for 2 weeks. She had also reported a weight loss of 4 kilograms within 3 months. Upon admission to our hospital, she initially received a diagnosis of congestive heart failure with atrial fibrillation and thyrotoxicosis. Following diuretic therapy, the patient developed hypotension and severe hypoxemia. Subsequent investigation revealed acute right ventricular failure due to an acute sub-segmental pulmonary embolism, which was confirmed by computed tomography of the pulmonary artery. The patient's condition improved after resuscitation involving intravenous fluid administration to increase right ventricular preload, intravenous vasopressor infusion to elevate systemic blood pressure, management of severe thyrotoxicosis, and intravenous administration of heparin.

Conclusion: Graves' disease, accompanied by hemodynamic disturbances due to acute right ventricular failure, necessitates admission to the intensive care unit for resuscitation and close monitoring. Although acute pulmonary embolism is an uncommon condition associated with Graves' disease, it should be considered, particularly in patients who develop acute right ventricular failure.

Keywords: Acute pulmonary embolism, Graves' disease, Right ventricular failure

BACKGROUND

Graves' disease is a prevalent endocrine disorder with reported incidence rates ranging from 0.5 to 2.5% worldwide[1]. The excess production of thyroid hormone affects multiple organ systems. The cardiovascular, neurological, and gastrointestinal systems are commonly involved. Clinical manifestations of Graves' disease have various severity ranges, from asymptomatic cases to thyroid storm[2]. Some patients only had weight loss, and increased appetite, while others developed tachycardia, atrial fibrillation, or pulmonary hypertension too. Cardiovascular involvement in Graves' disease is associated with conditions such as dilated cardiomyopathy, antiphospholipid syndrome, and a high prevalence of pulmonary arterial hypertension[3]. In severe cases of Graves' disease or during a thyroid storm, patients may experience heart failure, altered consciousness, and jaundice[4]. Thyrotoxicosis, a state of excessive thyroid hormone, is also associated with endothelial dysfunction and a hypercoagulable state, leading to an increased risk of venous thrombosis[1]. Although rare, pulmonary embolism has been reported as a rare complication of Graves' disease in a few documented cases.

CASE PRESENTATION

We present the case of a 53 years old woman who had a body weight loss of 4 kilograms over a period of 3 months, along with symptoms of palpitations, progressive dyspnea on exertion, abdominal distension, and bilateral leg swelling. She denied having a fever, chest pain, cough, loss of appetite, abdominal pain, or foamy urine. The patient presented to our hospital with worsening dyspnea, paroxysmal nocturnal dyspnea, edema, and jaundice for 2 weeks.

Upon examination, her vital signs were as follows: blood pressure (BP) of 121/87 mmHg, body temperature of 37.2 C, pulse rate of 140 beats per minute with totally irregular rhythm, respiratory rate of 28 breaths per minute, and oxygen saturation of 92% on room air, which improved to 98% on 3 liters of oxygen via a nasal cannula. Her weight was 48 kilograms, and her height was 152 cm.

The patient appeared conscious with a hyposthenic build, marked icteric sclera, exophthalmos, generalized enlargement of the thyroid gland, and no thyroid bruit. Her jugular venous pressure was elevated to the level of the mandible. Examination of the cardiovascular system revealed apical and parasternal heaving, no thrill, an apical impulse at the 6th intercostal space along the midclavicular line, normal S1, loud P2, and no murmur. Bilateral lower lung crepitations were detected on auscultation. Abdomen examination revealed distension with hepatomegaly, no ascites, and bilateral 4+ pitting edema.

Laboratory evaluation showed a normal complete blood count and renal function test, but an elevated N-terminal pro B-type natriuretic peptide (NT-proBNP) level of 826 pg/ml. Thyroid function tests indicated thyrotoxicosis with TSH < 0.005 μIU/ml (reference range 0.27-4.2), Free T4 of 2.82 ng/dL (reference range 0.93-1.17), Free T3 of 4.1 pg/mL (reference range 2.04–4.40) and Anti-thyroid stimulating hormone receptor (Anti-TSH) > 40 IU/L (reference range <0.8). Liver tests revealed direct hyperbilirubinemia with transaminitis (Total Protein 7.6 g/dL, Albumin 2.5 g/dL, Globulin 5.1 g/dL, Total bilirubin 16.47 mg/dL, Direct bilirubin 8.01 mg/dL, AST(SGOT) 172 U/L, ALT(SGPT) 37 U/L, and Alkaline phosphatase 105 U/L). Antiphospholipid antibodies (anticardiolipin IgG, IgM, antiBeta-2 Glycoprotein1 IgG, IgM) were negative. A chest X-ray showed alveolar infiltrates in the right lower lung with bilateral blunting of the costophrenic angles (Figure 1), and an electrocardiogram (ECG) revealed atrial fibrillation with a rapid ventricular rate (Figure 2).

KEY MESSAGES:

- Graves' disease is an autoimmune disorder affecting the thyroid gland, leading to various systemic complications, including cardiovascular issues.
- Apart from the potential complication of pulmonary hypertension in Graves' disease, there is another uncommon issue to be aware of. Pulmonary embolism can trigger acute right ventricular failure in Graves' disease.
- Management of acute right ventricular failure involves a comprehensive approach, adjustment to right ventricular preload, afterload, and contractility, as well as correction of hypoxemia and acidemia.

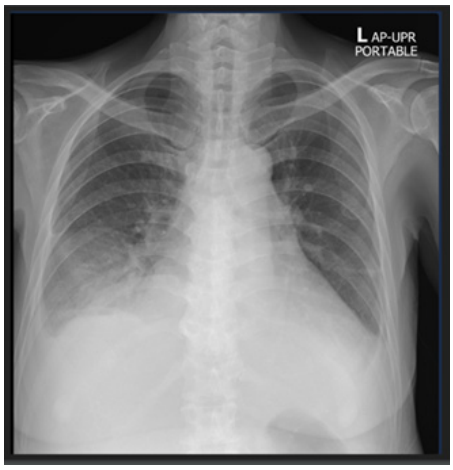

Figure 1. Chest X-ray alveolar infiltrated right lower lung with bilateral blunt costophrenic angle.

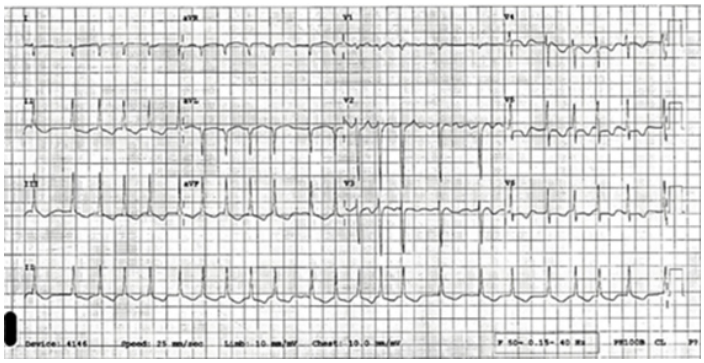

Figure 2. ECG shown atrial fibrillation with rapid ventricular rate, right axis deviation, right ventricular strain.

The patient was initially diagnosed with congestive heart failure and atrial fibrillation with a rapid ventricular rate associated with Graves' disease. Treatment with intravenous furosemide 60 mg and amiodarone was initiated. Over the next 10 hours, the patient's urine output reached 2600 ml. However, she was worsening dyspnea and hypoxemia, requiring intubation due to type 1 respiratory failure. Subsequently, she developed shock with a systemic blood pressure of 88/63 mmHg and an elevated serum lactate level of 10.5 mmol/L. Then norepinephrine was administered and titrated up to 0.25 mcg/kg/min to maintain blood pressure. Given the diagnosis of shock, the patient was transferred to the medical intensive care unit (MICU) for resuscitation.

In the MICU, a bedside echocardiogram revealed findings of inferior vena cava (IVC) plethora, estimated right atrial pressure from the echocardiogram of 15 mmHg, dilatation of the right atrium and right ventricle, left ventricular D-shape, a left ventricular ejection fraction (LVEF) of 48% by modified Simpson's method, medial E/E' of 9, and a tricuspid annular plane systolic excursion (TAPSE) of 8 mm (Figure3-4). A central venous catheter was inserted via the right internal jugular vein, with an initial central venous pressure (CVP) of 10 mmHg. Based on the echocardiographic findings and CVP measurement, the suspected cause of shock was a combination of hypovolemic and obstructive shock. Resuscitation was performed through a fluid challenge test and the initiation of vasopressor therapy. A fluid challenge test involving the intravenous infusion of 200 ml of crystalloid fluid over 15 minutes resulted in an elevation of CVP from 10 to 13 mmHg and an increase in blood pressure to 100/60 mmHg. For vasopressor therapy, norepinephrine infusion was continued and titrated up to a maximum dose of 0.3 mcg/kg/min. Once the patient's blood pressure stabilized at 116/70 mmHg, she was transferred for a computerized tomography of pulmonary artery angiography (CTPA) to confirm the diagnosis of pulmonary embolism as a potential cause of hypoxemia and right ventricular dilatation. The CTPA revealed acute pulmonary embolism in the left lower lobar artery extending to all basal segmental arteries of left lower lung with associated lung infarction and segmental pulmonary embolism of apical segmental branch of right upper lobar artery, anterior, apico-posterior and superior lingular branches of left upper lobar artery (Figure 5). Intracardiac thrombi were also detected within the right and left atrial appendages, and the left ventricular apex.

Following the diagnosis of acute pulmonary embolism, intravenous heparin was administered. The patient's right ventricular preload was adjusted through the fluid challenge test, and right ventricular afterload was managed through adjustments to mechanical ventilation for correct hypoxemia. The patient's heart rate was controlled using digoxin and intravenous amiodarone. With these interventions, the patient's blood pressure and tissue perfusion improved, and norepinephrine infusion was reduced until it was discontinued. For the management of Graves' disease with severe symptoms, the patient was prescribed lithium 900 mg/day, cholestyramine 16 g/day (due to hyperbilirubinemia and transaminitis), and hydrocortisone 200 mg/day.

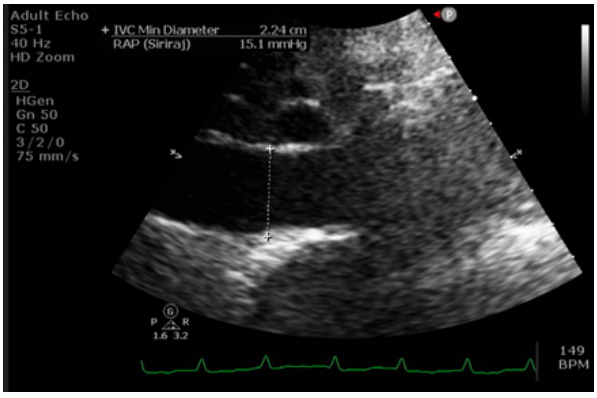

Figure3. Echocardiogram show IVC pethora, estimated RAP from the echocardiogram of 15 mmHg.

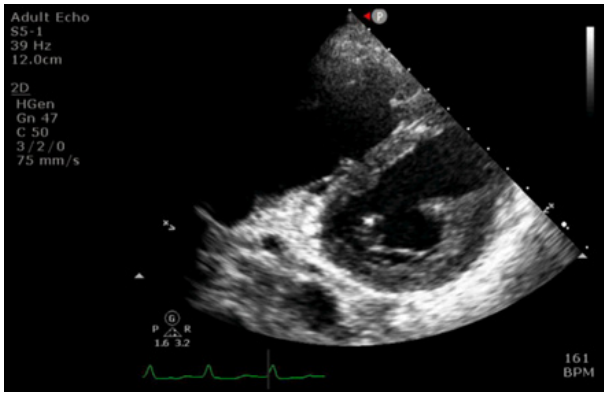

Figure 4. Echocardiogram show Right ventricle dilate, Left ventricular D-shape.

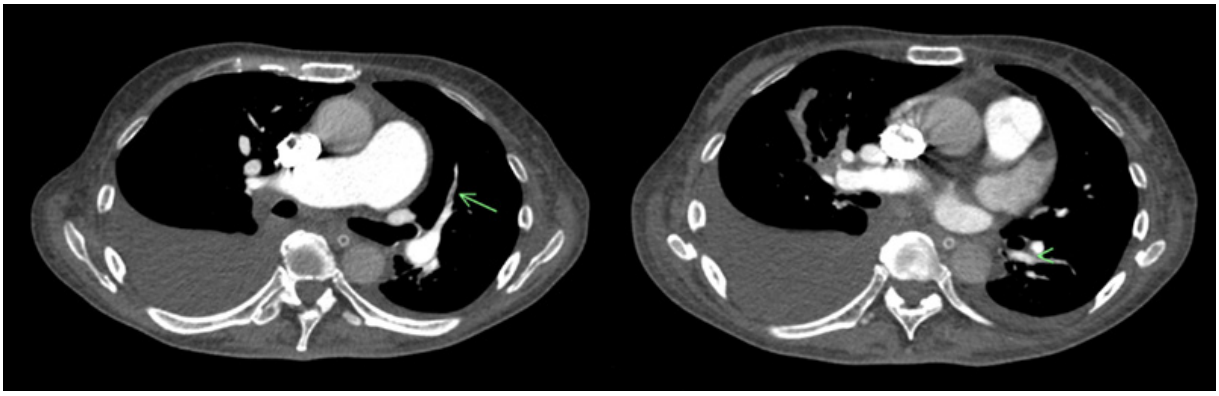

Figure 5. CT pulmonary embolism show acute pulmonary embolism at left lower lobar artery extending to all basal segmental arteries of left lower lobe and superior lingular branches of left upper lobar artery.

Over the next 10 days, the patient underwent follow-up transthoracic echocardiography (Figure 6-7), which revealed mild left ventricular dilatation and concentric hypertrophy. LVEF 63% by modified Simpson's method, uncertain left ventricular diastolic function due to fused E and A waves, mild left atrial dilatation, normal right atrium and right ventricle size, preserved right ventricular systolic function (TAPSE 24 mm, lateral S velocity = 13 cm/s), and the absence of pulmonary hypertension. The mean pulmonary artery pressure (mPAP) was measured as 20 mmHg by the systolic-diastolic method and 20 mmHg by the Abbas method.

The patient was eventually discharged from the hospital and scheduled for follow-up at the thyroid clinic. With improved liver test results, she transitioned to receiving Propylthiouracil (PTU) instead of lithium and cholestyramine for the well-controlled management of Graves' disease. Due to an unprovoked acute pulmonary embolism, she was advised to continue anticoagulant therapy for at least 3 months.

DISCUSSION

This case report describes a patient with an uncommon presentation of Graves' disease. Our patient was compared with previous cases reported of patients with pulmonary embolism with Graves' disease (Table 1). This case initially presented with signs and symptoms resembling congestive heart failure, but her condition worsened after diuresis. Echocardiogram and computed tomography pulmonary artery angiography (CTPA) were performed, confirming the diagnosis of acute pulmonary embolism and right ventricular failure.

The management of this patient involved several steps. First, blood pressure was stabilized through a fluid challenge test and titration of vasopressors to improve ventricular perfusion, which affected biventricular contractility. Second, mechanical ventilation was adjusted to correct hypoxemia that decreased pulmonary vascular resistance and right ventricle afterload. Third, optimization of preload of the right and left ventricles by slowing the heart rate with intravenous digoxin and amiodarone for increased ventricular filling time. In cases of severe thyrotoxicosis accompanied by atrial fibrillation with rapid ventricular response and unstable hemodynamics, cardioversion is recommended when atrial fibrillation rapidly impairs hemodynamics, and after ruling out the presence of left atrial thrombus[4]. This patient had hypovolemic and obstructive shock, that caused atrial fibrillation with a rapid ventricular response. If the patient's hemodynamic stability can't be achieved through adjusted preload, vasopressors, and antiarrhythmic drugs such as digoxin and amiodarone, consider the balance between the risk of thromboembolism and the potential benefits of cardioversion. Notably, amiodarone, despite its iodine content, can still be used as an antiarrhythmic drug in cases of severe thyrotoxicosis or thyroid storms associated with cardiac failure[4]. Fourth, intravenous heparin was administered to prevent ongoing thrombus formation in the pulmonary artery and reduce the risk of stroke. Lastly, excess thyroid hormone, which was the primary cause in this patient, was controlled using lithium and cholestyramine.

When about acute pulmonary embolism presents in the main pulmonary artery branch or obstructive shock persists despite medical intervention, thrombolytic agents are the treatment of choice, and extracorporeal membrane oxygenation can be an adjunct to maintain hemodynamic stability during massive pulmonary embolism. However, in this patient, the pulmonary embolism is not positioned in the main pulmonary artery. Fortunately, the patient's hemodynamic status can be effectively managed through the use of medication.

The authors note that while the patient exhibited signs and symptoms that could be indicative of thyroid storm and followed the diagnostic criteria for thyroid storm of the Japan Thyroid Association (JTA)[4], the exclusion of JTA noted that if signs and symptoms of inclusion criteria had cause other than thyroid storm, the physician should be considered. Acute right ventricular failure in this patient is caused by acute pulmonary embolism and hypoxemia. The cause of hyperbilirubinemia and transaminitis was thyrotoxicosis; there was also congestive hepatopathy that had evidence from CTPA, which showed contrast reflux in the portal vein, heterogenous liver parenchyma, and a negative viral hepatitis profile. In addition, this patient did not present with other signs and symptoms associated with thyroid storm for support in diagnosing thyroid storm such as alteration of consciousness and fever.

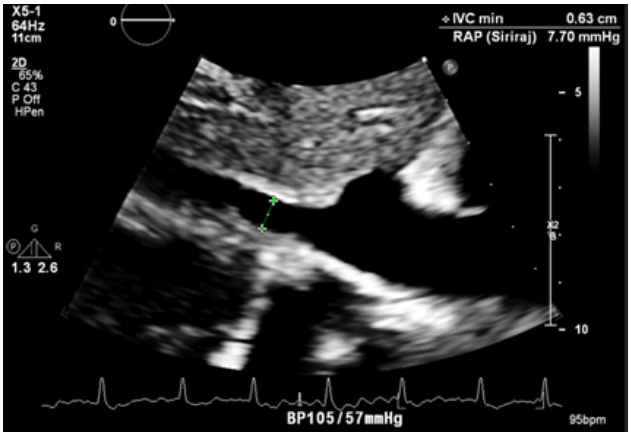

Figure 6. After treatment 10 days, follow up echocardiogram show IVC 0.63 cm.

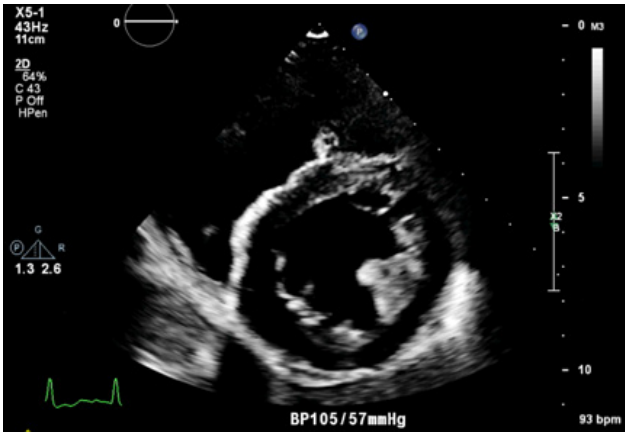

Figure 7. After treatment 10 days, follow up echocardiogram show right ventricle not dilate.

Table 1. Case report of pulmonary embolism in Graves' disease with/without right ventricular failure.

	Case	Cause of thyrotoxicosis	Cause of pulmonary hypertension	Treatment
Grine S, et al.[9]	1. A 32-year-old man presented with palpitation with signs and symptoms of right sided heart failure Echocardiogram: PAP 64 mmHg	Graves' disease -Anti-TSH elevated high titer	Pulmonary embolism with RV failure	- Heparin, followed by oral anticoagulant - Benzylthiouracil (Basdène®) - Radioactive iodine treatment *Repeated echocardiography showed normal pulmonary artery pressure (29 mmHg)
	2. A 23-year-old female presented with a severe chest pain, dyspnea with an exaggeration of tachycardia ECG showed right bundle branch block	Graves' disease -Anti-TSH 43.3 IU/L, (normal range <2 IU/L)	Pulmonary embolism but no clinical RV failure	- Heparin relayed by anticoagulants - Radioactive iodine treatment was administered *then lost to follow up
Bisural R, et al.[10]	A 58-year-old female presented with dyspnea with tachycardia with hypoxemia Echocardiogram: pulmonary systolic arterial pressure of 50-55 mmHg	Graves' disease -TSI 2.51 IU/mL (normal range 0.00-0.55 IU/mL)	Pulmonary embolism in subsegmental artery in the right lower lobe but no clinical RV failure	-Enoxaparin followed by oral anticoagulant -Methimazole
Bilal Lashari, et al.[11]	A 46-year-old man presented with dyspnea with weight loss with tachycardia and desaturation	Hyperthyroidism	Pulmonary embolism But no evidence pulmonary hypertension and RV failure	-Methimazole -Low molecular weight heparin anticoagulation
Abuobeida Ali, et al.[12]	A 38-year-old gentleman presented with shortness of breath, diarrhea, vomiting with tachycardia Echo by FAST shown right ventricular dilation	Graves' disease -TRAb 8.16 IU/L (normal 0–1.74 IU/L) -TPO 593 IU/ml (normal 0–34 IU/ml)	Pulmonary embolism with biventricular heart failure	-Enoxaparin -Cardioversion -PTU, Dexamethasone -Continuous veno-venous renal replacement therapy
Shu-Yu Tang, et al.[13]	A 32-year-old woman presented with dyspnea and desaturation Echocardiogram: elevated right heart systolic pressure (tricuspid regurgitation pressure gradient, TRPG 60.5 mmHg), moderate to severe tricuspid regurgitation, and D-shaped left ventricle	Graves' disease -Anti-TPO Ab 603 IU/mL (normal < 5.6 IU/ mL) -TBII of 93.2% (normal <10%)	Pulmonary embolism Plus left common femoral vein thrombosis	-Propylthiouracil (PTU), hydrocortisone, and proprano-lol, Lugol' s solution -Catheter-directed thrombolysis therapy (CDT) with EKOS® catheter and implantation of IVC filter *Echocardiogram revealed no more D-shaped left ventricle and decreased TRPG (36.2 mmHg), improvement in right heart dysfunction
Tanyalakmara T, Tongyoo S	A 53-year-old woman presented with progressive dyspnea with weight loss with tachycardia and desaturation	Graves' disease Anti-TSH > 40 IU/L (normal <0.8 IU/L)	Pulmonary embolism with acute RV failure	-Heparin -Digoxin, Amiodarone -Lithium, cholestyramine, Hydrocortisone -PTU

Abbreviations: RV, right ventricle; PAP, pulmonary arterial pressure; Anti-TSH, Anti thyroid stimulation hormone; TSI, Thyroid-stimulating immunoglobulin; TRPG, transpulmonary pressure gradient; Anti-TPO Ab, Anti Thyroid peroxidase antibody; TBII, Thyrotropin-binding inhibiting immunoglobulin; PTU, Propylthiouracil; IU/L, international unit/liter; IU/ml, international unit/milliliter

Therefore, the diagnosis of severe Graves' disease rather than thyroid storm was agreed upon with the endocrinologist team. However, due to severe hemodynamic disturbances, we treated severe Graves' disease similar to a thyroid storm.

Pulmonary hypertension is defined by a mean pulmonary arterial pressure (mPAP) >20 mmHg at rest[5]. Thyroid function tests are recommended in all patients with PAH to identify associated conditions[5]. This patient had signs and symptoms of thyrotoxicosis that made us concerned.

The prevalence of pulmonary hypertension in Graves' disease is estimated to be around 30-40%[6,7]. The exact mechanisms underlying this association are not fully understood but may involve the effects of excess thyroid hormone on the cardiovascular system, including hyperdynamic heart function, increased heart rate and contractility, imbalances in vasoconstriction and vasodilation of the pulmonary arterial bed, and increased pulmonary vascular resistance[6-8]. Other than excess thyroid function, which is associated with a hypercoagulable state and endothelial dysfunction, which may contribute to venous thrombosis but pulmonary embolism is a rare presentation in Graves' disease, with a prevalence of 0.16%[1].

In severe cases of thyrotoxicosis or thyroid storm, definitive treatments such as radioactive iodine or thyroidectomy are typically recommended[4]. However, in this particular patient, radioactive iodine was not suitable due to the presence of Graves' ophthalmopathy, and thyroidectomy was not feasible due to the recent acute pulmonary embolism and the need for ongoing anticoagulant therapy. The patient's Graves' disease was managed with Propylthiouracil (PTU) for the time being.

CONCLUSION

Although pulmonary embolism is a rare complication in Graves' disease, it is important for physicians to consider this possibility in patients who present with acute right ventricular failure. The intensive care unit is often necessary for resuscitation and monitoring of severe Graves' disease or thyroid storms.

ETHICS

The patient clinical and demographic data were collected in accordance with the guidelines set forth by the Siriraj Institutional Review Board of the Faculty of Medicine Siriraj Hospital, Mahidol University, Bangkok, Thailand. The patient profiled in this report gave written informed consent to be studied and reported upon in this case report.

CONSENT FOR PUBLICATION

Informed consent to publish identifying data was obtained from the study participant and that this consent was informed.

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AUTHORS' CONTRIBUTIONS

T.T. and S.T. contributed to data acquisition, data interpretation, and drafting and revision of the manuscript. All authors have read and approved the final version of the manuscript to be submitted for journal publication.

ABBREVIATIONS

TSH, thyroid stimulation hormone; μIU/ml, micro international unit/milliliter; ng/Dl, nanograms/deciliter; pg/mL, picogram/milliliter; AST, aspartate transaminase; ALT, alanine transaminase; U/L, unit/liter; TAPSE, Tricuspid annular plane systolic excursion; mPAP, mean pulmonary arterial pressure

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