Indonesian Journal of Cardiology
Indonesian J Cardiol 2025:46:89-95
pISSN; 2830-3105 / eISSN: 2964-7304
doi: 10.3070/ijc.1701

Case Report

Hemodynamic Conundrum of Thyroid Storm-Induced Acute Heart Failure: Challenging Case in a Remote Area

Dya Pratama Andryan[1], Susandy Oetama[2], Oktavia Lilyasari[3]

Abstract

Background: Thyroid storm (TS) is an acute and critical presentation of hyperthyroidism. It can lead to multiple organ dysfunction and has a high rate of mortality. Heart failure is one of the grave complications of hyperthyroidism and thyroid storm. Rapid progression of TS can lead to hypoperfusion and shock even with normotensive blood pressure and normal hemodynamic parameters. Unfortunately, the prevalence of hyperthyroidism is high in developing areas that lack of advanced medical facilities. This case presentation aims to present the rare condition of acute high-output failure secondary to thyroid storm with hypoperfusion and normotensive shock.

Case Illustration: A 28-year-old man came to the emergency department of a private hospital in East Borneo with worsening dyspnea on effort for three days before admission. His blood pressure was 169/103 mmHg with an irregular heart rate at 135-148 bpm. His axillary temperature was 37.9°C. ECG showed rapid Atrial Fibrillation (AF) with Ashman phenomenon. Chest x-ray revealed cardiomegaly with flattened cardiac waist and lung infiltrate. His echocardiogram has a hyperdynamic LV with LVEF 70%, normal RV function, concentric LV hypertrophy, and increased LAVi (51.19 mL/m^2). From the initial echocardiogram hemodynamic assessment, eRAP was 15 mmHg, CO was 6.5 to 7.4 L/min, and SVR was 1167 to 1329 dyne/sec/cm^{-5}. His peak E wave velocity was 92-95 cm/s. His fT4 was increased (100 ng/dL) while TSH was reduced (0.007 mU/L). H2FPEF score estimated 38.7% probability of heart failure with preserved ejection fraction (HFpEF). Burch-Wartofsky score was 60, suggesting thyroid storm. He was diagnosed with acute high-output heart failure secondary to thyroid storm due to uncontrolled Graves' Disease, and AF with rapid ventricular response. During follow-up in the Intensive Care Unit (ICU), patients underwent hypoperfusion with normotensive blood pressure (normotensive shock). Norepinephrine was initiated. The patient kept deteriorating and then passed away in our critical care unit at day of the seventh day.

Conclusion: Thyroid storm-induced acute heart failure might have a conundrum presentation due to normotensive and good cardiac output, giving a false impression of hemodynamic condition. Clinical presentation was very important to identify hypoperfusion, and aggressive treatment was needed to stabilize the patient's condition.

(Indonesian J Cardiol, 2025;46;89-95)

Keywords: *Acute heart failure, high output, thyroid storm, hyperthyroidism, hypoperfusion*

[1]Umar Wirahadikusumah General Hospital, West Java, Indonesia.

[2]Hadji Boejasin General Hospital, Pelaihari, South Kalimantan, Indonesia.

[3]Division of Pediatric Cardiology and Grown-Up Congenital Heart Disease, Department of Cardiology and Vascular Medicine, National Cardiovascular Centre Harapan Kita, Jakarta, Indonesia.

Correspondence:
Dya Pratama Andryan,

Umar Wirahadikusumah General Hospital, West Java, Indonesia.

Email: dyandryanmd@gmail.com

Introduction

Thyroid storm (TS) is an acute and critical manifestation of hyperthyroidism.[1-2] It can lead to multiple organ dysfunction and has a high mortality rate. Heart failure is one of the grave complications associated with hyperthyroidism and TS. Thyroid-related heart failure is characterized by high-output heart failure, with uncompromised (and often increased) cardiac output; however, congestion occurs due to diastolic dysfunction and poor Left Ventricular (LV) filling secondary to tachyarrhythmia.[3-4] Rapid progression of TS can lead to hypoperfusion and shock, even with normotensive blood pressure and normal hemodynamic parameters. Unfortunately, hyperthyroidism has become increasingly prevalent in developing regions that lack advanced medical facilities.[1] Hence, this case presentation aims to illustrate the rare condition of acute high-output heart failure secondary to thyroid storm, accompanied by hypoperfusion and normotensive shock.

Case Illustration

A 28-year-old man was presented to the emergency department of a private hospital in East Borneo with worsening dyspnea three days before admission. He also reported palpitations, constant fever, and "air hunger" when lying flat. Additionally, he noticed swelling in both legs. His medical history indicated stage II hypertension, for which he was not taking his medication regularly; nevertheless, there were no other remarkable findings in his medical records. However, upon examination, his blood pressure was 169/103 mmHg, with an irregular heart rate of 135 bpm to 148 bpm. His axillary temperature was 37.9°C. Notable physical findings included alopecia areata, exophthalmos, icteric sclera (Figure 2B-C), an enlarged non-nodular thyroid gland measuring 2x1.5 cm^2, minimal rales at the bilateral lung bases, a faint systolic murmur at the lower sternal border and apex graded 3/6, and bilateral pitting oedema. Capillary refill time was over 2 seconds, with warm but clammy extremities. His ECG result revealed rapid atrial fibrillation with Ashman phenomenon (Figure 2A). A chest X-ray test showed cardiomegaly with a flattened cardiac waist and lung infiltrates. His echocardiogram indicated hyperdynamic LV function with an LVEF of 70%, normal RV function, concentric LV hypertrophy (Figure 2D-E), and increased Left Atrial Volume Index (LAVI) of 51.19 mL/m^2. The initial echocardiographic hemodynamic assessment demonstrated an estimated Right Atrial Pressure (eRAP) of 15 mmHg, Cardiac Output (CO) of 6.5 to 7.4 L/min, and Systemic Vascular Resistance (SVR) of 1167 to 1329 dyne/sec/cm^5. His peak E wave velocity was 92 - 95 cm/s (Figure 2F); however, due to limitations, we cannot measure Tissue Doppler Index (TDI) or calculate Pulmonary Arterial Wedge Pressure (PAWP). Additionally, abdominal ultrasound findings were consistent with a "starry sky" appearance, indicative of ischemic acute hepatitis. His free T4 (fT4) was increased (100 ng/dL), while TSH was suppressed (0.007 mU/L). The H2FPEF score estimated a 38.7% probability of Heart Failure with Preserved Ejection Fraction (HFpEF). The Burch-Wartofsky score was 60, suggesting a thyroid storm.

He was diagnosed with acute high-output heart failure secondary to a thyroid storm due to uncontrolled Graves' disease and rapid ventricular response, atrial fibrillation. For acute heart failure, he was treated with intravenous furosemide at 5 mg/hr, ramipril 5 mg once daily, hydrochlorothiazide (HCT) 25 mg once daily, and spironolactone 25 mg once daily. For his atrial fibrillation, he received warfarin 2 mg once daily, propranolol starting at 10 mg thrice daily, and digoxin IV 0.25 mg as needed. For his thyroid storm, he was treated with methimazole 20 mg four times daily and dexamethasone 2x5 mg IV. Unfortunately, Lugol's solution was unavailable. During his follow-up in the Intensive Care Unit (ICU), the patient showed improvement over the first two days, with reduced dyspnea, improved congestion profile, and stabilized blood pressure and heart rate. His urine output was 2 L to 3 L per day. However, on day three, the patient began to deteriorate. His blood pressure dropped to 116/45 mmHg without support, and his heart rate was 78 bpm, but his diuresis fell below <1 cc/kg/min. On day four, the patient developed metabolic acidosis, with blood gas analysis revealing a pH of 7.26, pCO_2 of 45.2 mmHg, pO_2 of 102 mmHg with nasal cannula at 3 L/min, and HCO_3^- of 21.7 with an actual base excess of -5.1. The patient's awareness and renal function deteriorated, prompting the discontinuation of medications affecting blood pressure and renal condition, such as ramipril, HCT, and spironolactone. Serial hemodynamic echocardiograms revealed an eRAP of 8 mmHg, CO of 3.9 – 4 L/min, and SVR of 1100–1128 dyne/sec/cm^5. We suspected this condition was due to hypoperfusion with normotensive blood pressure (normotensive shock). Subsequently, we initiated norepinephrine at 0.05 mcg/kg/min, titrated upwards, and corrected acidosis with IV bicarbonate. The patient continued to deteriorate and passed away in the critical care unit on the seventh day.

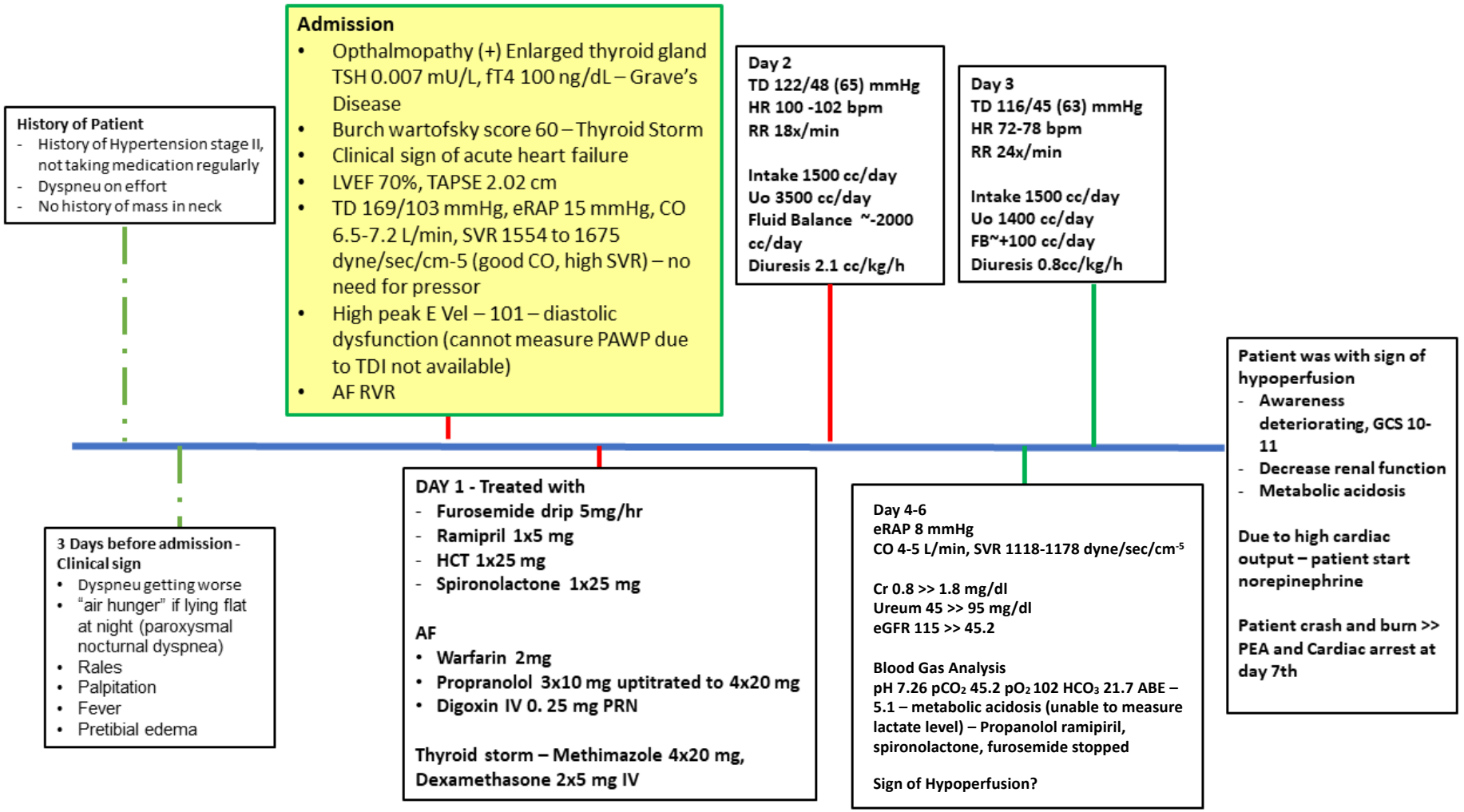

Figure 1. Timeline of the case.

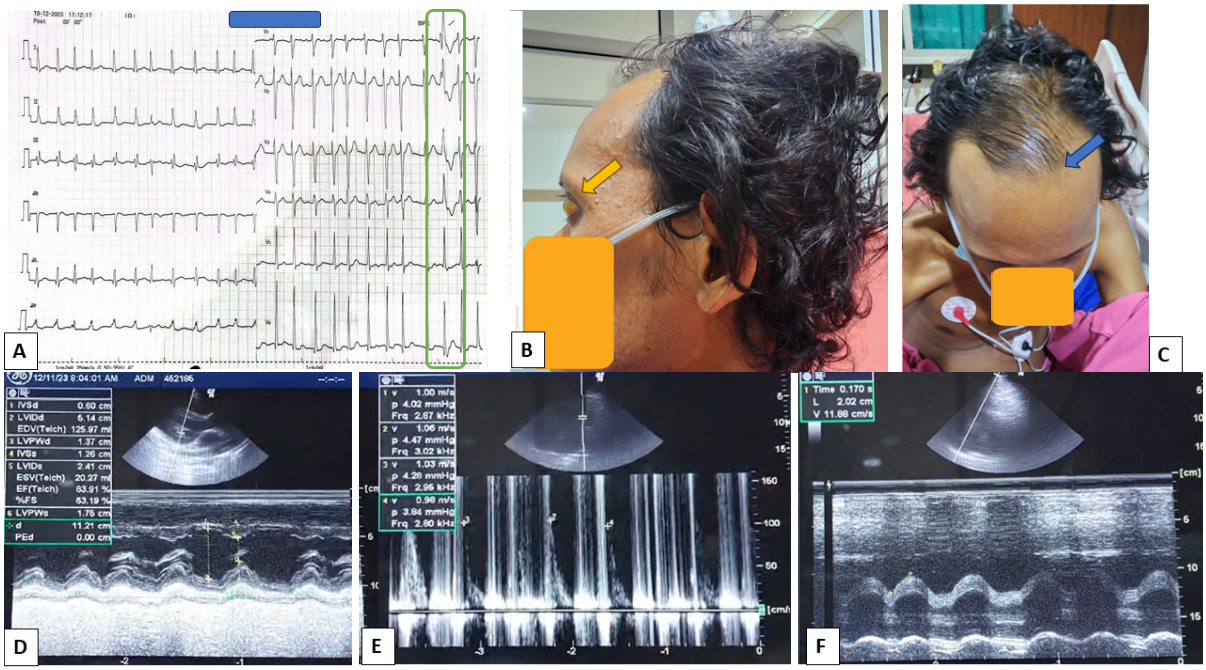

Figure 2. Clinical and hemodynamic echocardiogram profile of the patient.

Discussion

A thyroid storm is a rare and life-threatening exacerbation of hyperthyroidism, characterized by fever, delirium, seizures, coma, jaundice, arrhythmia, and high-output heart failure. The mortality rate due to cardiac failure, arrhythmia, or hyperthermia can be as high as 30%, even with appropriate treatment. TS is typically precipitated by acute illness (e.g., stroke, infection, trauma, diabetic ketoacidosis), surgery—especially thyroid surgery—or radioiodine treatment in patients with partially treated or untreated hyperthyroidism. Patients with TS and hyperthyroidism exhibit different normal ranges of hemodynamic parameters due to the effects of thyroid hormones on the cardiovascular system; therefore, monitoring these parameters should be conducted with heightened vigilance.[3,5]

Thyroid hormones, particularly T3, exert positive inotropic and chronotropic effects, increasing heart rate and cardiac output. This ultimately results in decreased systemic vascular resistance as a hemodynamic response. However, in chronic states, this can lead to pathological shifts in hemodynamic and neurohumoral conditions. In the early stages, heart failure manifests as HFpEF, with diastolic dysfunction as the primary mechanism. In later stages, biventricular failure with Heart Failure with Reduced Ejection Fraction (HFrEF) may occur. Arrhythmias, primarily in the form of rapid atrial fibrillation, can lead to rate-related reductions in both left ventricular systolic and diastolic functions due to decreased ventricular filling. Right ventricular strain may also increase due to elevated pulmonary vascular resistance.[6]

From a metabolic perspective, hyperthyroidism also has a crucial role in the acceleration of atherosclerosis, thrombosis formation, and secondary hypertension, leading to hypertensive heart disease. Pre-existing ischemic or hypertensive heart disease may further impair the ability to compensate for hemodynamic and neurohumoral changes, particularly in TS.[3,6-7]

Hence, effective management requires intensive monitoring and supportive care, identifying and treating precipitating causes as well as adopting measures to reduce thyroid hormone synthesis. Fluid resuscitation with crystalloid solutions must be administered. Large doses of Propylthiouracil (500–1000 mg loading dose, maintenance 250 mg QID), or methimazole as an alternative at 20 mg QID, should be given. Stable iodide should be administered at 5 drops QID to block thyroid hormone synthesis. Glucocorticoids (hydrocortisone 300 mg IV bolus, then 100 mg TID, or methylprednisolone 50 mg IV) should be administered to reduce inflammation and inhibit the conversion of T4 to T3.[2,4,8] Propranolol of 20 mg to 80 mg every 6 to 4 hours can help control adrenergic symptoms, increase ventricular filling, and control the arrhythmia rate. However, caution is warranted due to the negative inotropic effects, especially in patients who are native beta-blocker users.[5–7]

We managed our case of TS and acute heart failure in a remote setting. The patient presented

with acute heart failure, atrial fibrillation with rapid ventricular response, and thyroid storm due to untreated Graves' disease. The patient exhibited signs of congestion and hyperdynamic function based on physical examination and echocardiography hemodynamic assessment (Table 1). There were no signs of hypoperfusion initially. Our first strategy was to alleviate congestion and manage heart failure with IV furosemide, ramipril, spironolactone, hydrochlorothiazide, and propranolol at an initial dose of 10 mg TID, which was titrated in the ICU to control tachycardia and TS. Digoxin IV 0.25 mg and warfarin 2 mg were administered for atrial fibrillation. The treatment for TS included methimazole and dexamethasone, given the unavailability of hydrocortisone and iodine. Unfortunately, we could not monitor our patients with invasive techniques. We could not recognize early signs of hypoperfusion due to the hemodynamic complexities in hyperthyroid patients. We identified that the patient was deteriorating and hypoperfusion state on the third day, following a decrease in urine output and the onset of delirium (Figure 1). We also initiated hemodynamic support on the fifth day, despite the patient having normal cardiac output and vascular resistance, due to persistent hypoperfusion and metabolic acidosis. There is ongoing debate regarding the aggressiveness of propranolol use in high-output states. Pulse-dose steroids are a potential rescue strategy; however, we did not implement this due to pneumonia infection, and we opted for high-dose dexamethasone.[9-10]

Table 1. Hemodynamic and lab profile.

Day 1: Admission	Day 3: Start deteriorating	Day 4	Day 6
Hemodynamic TD 169/103 (123) mmHg HR 135-148 bpm eRAP 15 mmHg SV 45-50 ml CO 6.5-7.4 L/min SVR 1167-1329 dyne/sec/cm^{-5} Peak E Vel 100 cm/s LVH Concentric Mild functional MR Mild-Trivial TR PvAcct 200 ms Diuresis 2.0 cc/kg/hr	TD 116/45 (63) mmHg HR 75-78 bpm eRAP 8 mmHg SV 52 ml CO 3.9-4 L/min SVR 1100-1128 dyne/sec/cm^{-5}	Norepinephrine 0.05 mcg/kg/min TD 100/45 (63) mmHg HR 70-78 bpm eRAP 8 mmHg SV 39 ml CO 3.0-3.2 L/min SVR 1375-1466 dyne/sec/cm^{-5}	Norepinephrine 0.05 mcg/kg/min Dobutamine 5 mcg/kg/min TD 92/45 (61) mmHg HR 80-85 eRAP 8 mmHg SV 40-45 ml CO 3.2-3.8 L/min SVR 1115-1325 dyne/sec/cm^{-5}
Lab Hb 15,4 g/dl Ht 47,6% Leucocyte 8430/mm^3 Tro 222.000/mm^3	Diuresis 0.8 cc/kg/hr	Diuresis 0.5 cc/kg/hr	Diuresis 0.3 cc/kg/hr
Renal Ur 45.1 mg/dl Cr 0.8 mg/dl eGFR (MDRD) 115	**Ureum 45 >> 95 mg/dl** **Cr 1.8 mg/dl** **eGFR 45.2**		
Other GDS 211 mg/dl Na 133 mEq/L K 4.1 mmol/L Cl 107 mEq/L SGOT 250 IU/L SGPT 300 IU/L	**Blood Gas Analysis** pH 7.26 pCO_2 45.2 pO_2 102 HCO_3 21.7 ABE -5.1 – metabolic acidosis (unable to measure lactate level)	**Abdominal Ultrasound** – Starry sky appearances – suggestive to acute (ischemic – hypoperfusion) Hepatitis	

Remote area settings present certain limitations. Blood Gas Analysis (BGA) had to wait several hours due to limited laboratory facilities; BGA examination was conducted at another hospital. We also faced challenges in obtaining invasive hemodynamic monitoring modalities. This modality is recommended by guidelines such as the Japanese Thyroid Storm Guidelines (2016). In this case, we relied heavily on echocardiographic hemodynamic examination. Additionally, we lacked advanced treatment modalities such as hemodialysis, plasmapheresis, mechanical support, or urgent total thyroidectomy in refractory critical settings.

Conclusion

Thyroid storm-induced acute heart failure may present a conundrum due to normotensive status and good cardiac output, potentially giving a false impression of the hemodynamic condition. Clinical presentation is crucial for identifying hypoperfusion, and aggressive treatment is necessary to stabilize the patient's condition.

List of Abbreviations

AF	Atrial Fibrillation
BGA	Blood Gas Analysis
CO	Cardiac Output
ECG	Electrocardiogram
eRAP	Estimated Right Atrium Pressure
fT4	Free Thyroxine Hormone (T4)
HFpEF	Heart Failure with Preserved Ejection Fraction
IV	Intravenous
LAVi	Left Atrial Volume Index
LV	Left Ventricle
LVEF	Left Ventricular Ejection Fraction
PAWP	Pulmonary Arterial Wedge Pressure
RV	Right Ventricle
SVR	Systemic Ventricular Resistance
TS	Thyroid Storm
TSH	Thyroid-Stimulating Hormone

Ethical Clearance

Not applicable. Written and informed consent was obtained from the parents of the patient.

Publication Approval

All authors consent to the publication of this manuscript.

Authors' Contributions

All authors contributed to the literature searching and review. All authors read and approved the final manuscript.

Acknowledgments

The authors would like to express their sincere gratitude to PKT Bontang General Hospital, Universitas Indonesia, and the National Cardiovascular Center Harapan Kita for their valuable support and collaboration in facilitating this study.

Conflict of Interest

The authors declared that they have no competing interests.

Availability of Data and Materials

All data supporting this case are contained within the manuscript. No additional datasets were generated or analyzed during the current study.

Funding

Nothing to disclose. The study received no external funding.

Copyright/Permissions for Figures

All figures in this manuscript are original and created by the authors. No copyrighted material has been reproduced. The authors confirm that all figures are their own and have not been previously published elsewhere.

References

1. Taylor PN, Albrecht D, Scholz A, Gutierrez-Buey G, Lazarus JH, Dayan CM, et al. Global epidemiology of hyperthyroidism and hypothyroidism [Internet]. Vol. 14, Nature Reviews Endocrinology. Nature Publishing Group; 2018. 301–16.
2. J. Larry Jameson, Susan J. Mandel, Anthony P. Weetman. Chapter 377 Hyperthyroidism. In: Jameson JL, Fauci AS, Kasper DL, Longo DL, Hauser SL, Loscalzo J, editors. Harrison's Principles of Internal Medicine. 20th Edition. United States: McGraw-Hill; 2018. 2703–10.

3. Razvi S, Jabbar A, Pingitore A, Danzi S, Biondi B, Klein I, et al. Thyroid Hormones and Cardiovascular Function and Diseases. J Am Coll Cardiol. 2018 Apr 24;71(16):1781–96.
4. Kahaly GJ, Bartalena L, Hegedüs L, Leenhardt L, Poppe K, Pearce SH. 2018 European thyroid association guideline for the management of Graves' hyperthyroidism. Eur Thyroid J. 2018 Aug 1;7(4):167–86.
5. Firdaus I. *Fibrilasi Atrium Pada Penyakit Hipertiroidisme: Patogenesis dan Tatalaksana*. Indonesian Journal of Cardiology. 2007 Sep;28(5):375–85.
6. Klein I, Danzi S. Thyroid disease and the heart. Circulation. 2007 Oct;116(15):1725–35.
7. Bachman ES, Hampton TG, Dhillon H, Amende I, Wang J, Morgan JP, et al. The metabolic and cardiovascular effects of hyperthyroidism are largely independent of β-adrenergic stimulation. Endocrinology. 2004 Jun;145(6):2767–74.
8. Satoh T, Suzuki A, Wakino S, Iburi T, Tsuboi K, Kanamoto N, et al. 2016 Guidelines for the management of thyroid storm from The Japan Thyroid Association and Japan Endocrine Society (First edition). The Japan Thyroid Association and Japan Endocrine Society Taskforce Committee for the establishment of diagnostic criteria and nationwide surveys for thyroid storm. 2016;63(12):1025–64.
9. Kiriyama H, Amiya E, Hatano M, Hosoya Y, Maki H, Nitta D, et al. Rapid Improvement of thyroid storm-related hemodynamic collapse by aggressive anti-thyroid therapy including steroid pulse. Medicine (United States). 2017 Jun 1;96(22).
10. Andrade Luz I, Pereira T, Catorze N. Thyroid storm: A case of haemodynamic failure promptly reversed by aggressive medical therapy with anti-thyroid agents and steroid pulse. BMJ Case Rep. 2018 Dec 1;11(1).