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Recurrent Cardiac Arrest as the Initial Manifestation of Thyroid Storm

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ABSTRACT/SUMMARY

A 53-year-old man with no known medical history presented with new-onset lower-extremity edema, acute shortness of breath, and atrial fibrillation. He rapidly deteriorated, suffering three episodes of cardiac arrest in the emergency department, requiring intubation and advanced cardiac life support. Laboratory evaluation revealed severe thyrotoxicosis, and the clinical presentation fulfilled diagnostic criteria for thyroid storm. This case underscores the profound cardiovascular consequences of extreme thyroid hormone excess and highlights the importance of early recognition and immediate multimodal therapy.

Keywords: Cardiac Arrest; Thyroid; Cardiovascular

CASE PRESENTATION

Initial Presentation

A 53-year-old man with no significant past medical history presented to the emergency department, with progressive shortness of breath and bilateral lower-extremity swelling, accompanied by palpitations. Symptoms had developed over several days prior to presentation. He denied prior thyroid disease, medication use, stimulant exposure, or recent iodinated contrast. There was no known history of cardiac disease.

On arrival, he appeared acutely ill and dyspneic. Vital signs were notable for:

- Heart rate: 200 beats per minute
- Respiratory rate: 24 breaths per minute
- Blood pressure: 120/90 mm Hg
- Temperature: 98°F (36.7°C)

Physical examination revealed tachycardia with an irregularly irregular rhythm, bilateral lower-extremity edema, and signs of volume overload. He was alert but visibly distressed.

Cardiovascular Collapse and Cardiac Arrest

An electrocardiogram demonstrated atrial fibrillation with rapid ventricular response (Figure 1). Shortly after arrival, the patient developed hemodynamic instability, followed by cardiac arrest. Advanced cardiac life support was initiated, resulting in return of spontaneous circulation. He experienced two additional episodes of cardiac arrest in the emergency department, each requiring full resuscitative efforts.

Following the final arrest, the patient was endotracheally intubated for airway protection and transferred to the intensive care unit.

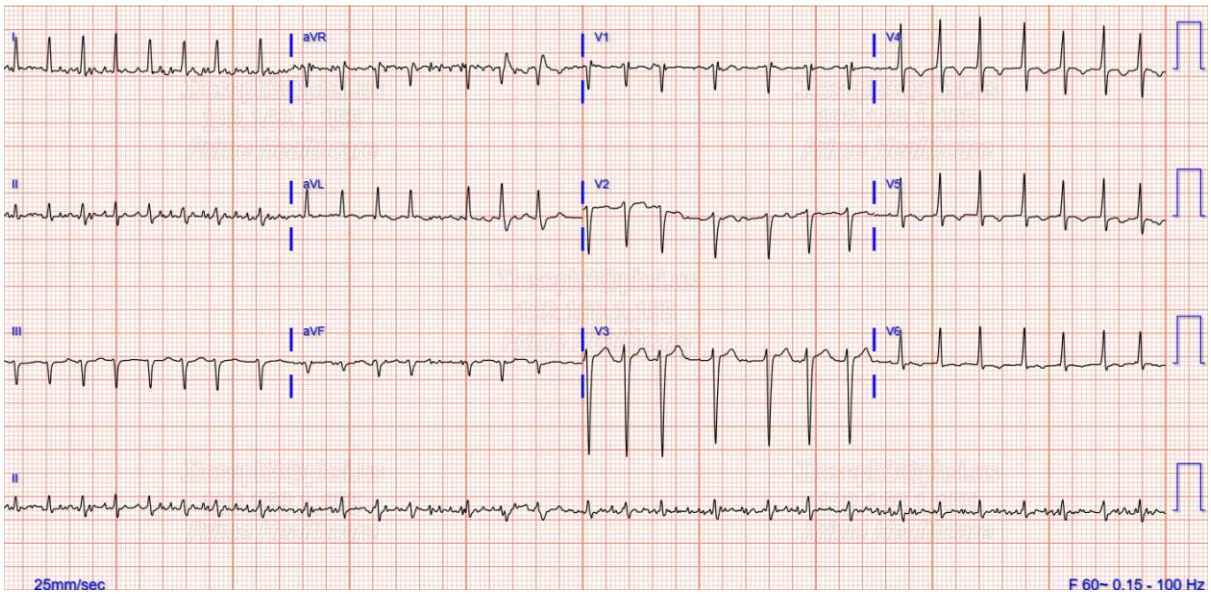

Figure 1

Diagnostic Studies

Initial laboratory evaluation showed:

- Sodium: 138 mmol/L
- Potassium: 5.0 mmol/L
- Chloride: 100 mmol/L
- Bicarbonate: 17.2 mmol/L
- Anion gap: 20.8
- Lactic acid: 4.0 mmol/L
- TSH: <0.005 µIU/mL
- Free T4: 3.8 ng/dL

These findings were consistent with high–anion gap metabolic acidosis and severe thyrotoxicosis.

A bedside transthoracic echocardiogram demonstrated hyperdynamic cardiac function, without evidence of regional wall-motion abnormalities or pericardial effusion.

Taken together—extreme tachycardia, atrial fibrillation, cardiovascular collapse with recurrent cardiac arrest, metabolic acidosis, and markedly abnormal thyroid function tests—the findings supported a diagnosis of thyroid storm.

Physiology of Thyroid Hormone and Cardiovascular Effects

Thyroid hormones exert potent effects on the cardiovascular system by increasing myocardial contractility, heart rate, and cardiac output while reducing systemic vascular resistance. These effects are mediated through both genomic mechanisms—such as upregulation of β-adrenergic receptors—and non-genomic actions on ion channels and vascular smooth muscle.

In hyperthyroidism, these mechanisms predispose patients to atrial fibrillation, high-output heart failure, and myocardial ischemia due to increased oxygen demand. A classic review in *The New England Journal of Medicine* described atrial fibrillation as one of the most common and clinically significant cardiac manifestations of thyrotoxicosis.

Pathophysiology of Thyroid Storm

Thyroid storm represents the most severe manifestation of thyrotoxicosis and reflects decompensated systemic thyroid hormone excess, often precipitated by physiologic stress. Importantly, thyroid storm is not defined by absolute hormone concentrations; free T4 and T3 levels often overlap with those seen in uncomplicated hyperthyroidism.

Instead, diagnosis depends on clinical evidence of multisystem involvement, particularly cardiovascular and central nervous system dysfunction. The presence of atrial fibrillation with extreme tachycardia, metabolic acidosis, heart failure manifestations (peripheral edema), and cardiovascular collapse in this patient reflects the hallmark features of thyroid storm described in major clinical reviews and guidelines.

Clinical Discussion

Diagnostic Reasoning

Using the Burch–Wartofsky Point Scale, this patient accumulated a score well above the diagnostic threshold for thyroid storm:

- Heart rate >140 bpm: major cardiovascular points
- Atrial fibrillation: additional cardiovascular points
- Congestive heart failure (peripheral edema): additional points
- Metabolic derangement and cardiovascular collapse: supportive features

This framework reinforces that thyroid storm is a clinical diagnosis, and treatment should not be delayed while awaiting confirmatory testing.

Mechanisms of Cardiac Arrest

In severe thyrotoxicosis, atrial fibrillation with rapid ventricular response can result in acute myocardial ischemia, hypotension, and cardiogenic shock. Hyperadrenergic states may also precipitate malignant ventricular arrhythmias or pulseless electrical activity. The hyperdynamic circulation observed on bedside echocardiography in this patient reflects profound thyroid hormone–mediated cardiac stimulation preceding circulatory collapse.

Management

Management of thyroid storm requires simultaneous, multi-targeted therapy, as emphasized in the American Thyroid Association guidelines and major reviews in *The New England Journal of Medicine (NEJM)* and *Journal of the American Medical Association (JAMA)*.

Initial Stabilization

The patient was intubated following cardiac arrest and managed with mechanical ventilation, circulatory support, and aggressive treatment of metabolic derangements.

Targeted Thyroid Storm Therapy

Standard therapy includes:

1. Beta-adrenergic blockade (often with a titratable agent such as esmolol in unstable patients) to control adrenergic excess and ventricular rate.
2. Thionamide therapy (methimazole or propylthiouracil) to inhibit new thyroid hormone synthesis.
3. Inorganic iodine, administered after thionamide therapy, to inhibit release of preformed hormone.
4. Glucocorticoids to reduce peripheral conversion of T4 to T3 and address potential adrenal insufficiency.
5. Supportive ICU care, including temperature control, volume management, and treatment of potential precipitating factors.

Recent data from large observational cohorts published in *JAMA Network Open* suggest no significant difference in mortality between propylthiouracil and methimazole in thyroid storm, supporting individualized drug selection.

CONCLUSION

This case illustrates that thyroid storm can present as recurrent cardiac arrest in patients without known thyroid disease. Extreme tachycardia, atrial fibrillation, heart failure, and metabolic acidosis should prompt urgent consideration of thyroid storm, even in the absence of fever. Early recognition and prompt initiation of multimodal therapy are critical to reducing mortality in this life-threatening endocrine emergency.

Key Teaching Points

- Thyroid storm is a clinical diagnosis, not defined by hormone levels alone.

- Atrial fibrillation with extreme tachycardia can precipitate cardiovascular collapse and cardiac arrest in severe thyrotoxicosis.
- Immediate empiric treatment is essential when thyroid storm is suspected.
- Hyperdynamic cardiac function may precede sudden decompensation.
- Patients may lack a prior history of thyroid disease.

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