

High-Output Heart Failure Caused by Thyrotoxicosis and Beriberi



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KEYWORDS

- High-output heart failure • Thyrotoxicosis • Thionamides • Beriberi • Wet beriberi
- Shoshin beriberi • Thiamine deficiency

KEY POINTS

- High-output heart failure is less common than low-output heart failure and treatment options generally recommended for low-output heart failure may not be beneficial.
- Thyrotoxicosis causes many cardiovascular signs and symptoms, and can lead to high-output heart failure. Symptoms may not abate until the thyroid function has returned to near normal.
- Acute thiamine deficiency, or wet beriberi, can lead to high-output heart failure. Signs and symptoms include shortness of breath and marked peripheral edema. Patients at most risk for this include those with chronic alcoholism and malnutrition. Once thiamine administration is started, symptoms can rapidly improve.

INTRODUCTION

Heart failure is a complex clinical syndrome resulting from structural or functional impairment of ventricular filling or ejection of blood. This leads to symptoms of fatigue, dyspnea, and the retention of fluid. Disease of the pericardium, myocardium, endocardium, or heart valves can cause heart failure. Heart failure can also be caused by metabolic and nutritional abnormalities, such as severe hyperthyroidism and vitamin deficiency.¹ Heart failure is most commonly associated with a low-output state (cardiac index <2.5 L/min/m²),² but high-output heart failure may be seen in some patients. A high-output state has been defined as greater than 8 L/min or cardiac index greater than 3.9 L/min/m². In high-output heart failure, systemic vascular resistance is reduced because of vasodilation or systemic arteriovenous shunting.³

Current guideline recommendations for low-output heart failure treatment, such as vasodilators or positive inotropic agents, may not be appropriate for patients with

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high-output heart failure because they have low systemic vascular resistance and normal or near-normal ventricular contractility. Several disease states can cause high-output states, including sepsis, anemia, systemic arteriovenous fistulas, Paget disease, and multiple myeloma. Two additional causes of high-output heart failure include thyrotoxicosis and beriberi.² With prompt and complete treatment, both of these can be reversible. Thyrotoxicosis and beriberi leading to high-output heart failure are further described in this article.

THYROTOXICOSIS

Thyrotoxicosis is a syndrome caused by the inappropriate overproduction of thyroid hormone, leading to a hypermetabolic, hyperdynamic state. The excess production of thyroid hormones has a detrimental effect on the cardiovascular system. Such symptoms as resting tachycardia, breathlessness, and palpitations are some of the most prevalent. It is an acute disorder requiring prompt recognition and treatment. The association between thyrotoxicosis and heart failure has been well-established and long described in the literature.⁴

The most common cause of thyrotoxicosis is Graves disease, an autoimmune disorder responsible for up to 90% of cases of thyrotoxicosis.⁵ Graves disease is more common in women than in men.⁶ Thyrotoxicosis can also be caused by toxic multinodular goiter, toxic adenoma, thyroiditis, and excessive thyroid hormone replacement.^{5,7} Thyrotoxic heart failure is more common in patients older than age 60.^{8,9} It is more likely to occur when there is underlying cardiac disease; however, it can also occur when there is no cardiac disease. It has also been reported during pregnancy.¹⁰

Thyrotoxicosis can lead to thyrotoxicosis crisis, referred to as thyroid storm. It is not common and the important associated symptom includes hyperpyrexia. Body temperature can become elevated to 40.5°C to 41.1°C (105°F–106°F). Rapid treatment of thyroid storm in the hospital setting is essential.^{6,11} Thyroid storm is beyond the scope of this article.

The Thyroid

The thyroid gland is located in the anterior neck behind the thyroid cartilage and its two lobes lie on either side of the trachea. The thyroid secretes two major hormones: tetraiodothyronine (thyroxine, or T₄) and triiodothyronine (T₃). Production of thyroid hormones is controlled by the hypothalamic-pituitary-thyroid axis, a complex feedback system (Box 1). T₄ is the predominant thyroid hormone accounting for about 80%

Box 1

Hypothalamic-pituitary-thyroid axis

Hypothalamus secretes thyrotropin-releasing hormone (TRH)

↓

TRH stimulates the anterior pituitary to secrete thyroid-stimulating hormone (TSH)

↓

TSH increases T₃ and T₄

Data from Elston MS, Conaglen JV. Thyrotoxicosis: pathophysiology, assessment, and management. J Prim Health Care 2005;32(6):407–13; and Dahlen R. Managing patients with acute thyrotoxicosis. Crit Care Nurse 2002;22(1):62–9.

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