

Peculiarities in the Treatment of Cardiomyopathy Associated with High-Output Conditions

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Abstract

High-output heart failure (HOHF) is a less common form of heart failure and presents as the inability of the heart to supply enough blood for the body's needs.

HOHF can be divided into two categories: 1. increased demand for increased metabolism or 2. arteriovenous shunts increasing venous circulation flow and decreasing resistance, leading to increased oxygen consumption and low systemic vascular resistance.

The most common causes are obesity, liver disease, shunts, lung disease, myeloproliferative diseases, hyperthyroidism, and thiamine deficiency.

Introduction

This article aims to carry out a systematic review of high-output heart failure (HOHF) and its peculiarities.

HOHF is a less common form of heart failure, and although it may seem contradictory at first glance, it presents itself as the inability of the heart to supply enough blood for the body's needs.¹

Patients with HOHF have normal cardiac function and decreased systemic vascular resistance, either secondary to diffuse arteriolar dilation or possible shunts of arterioles and capillary beds, leading to neurohormonal activation.^{2,3}

High cardiac output is a state of elevated cardiac output at rest greater than 8 L/min or a cardiac index greater than 4.0 L/min/m²,¹ and heart failure occurs when this cardiac output is insufficient to meet the demand hemodynamics.

Methods

Systematic review on high-impact publishing platforms.

Development

Regarding the causes of HOHF, they can be divided into two categories: 1. increased demand for greater metabolism or 2. arteriovenous shunts that increase the flow of venous

circulation and decrease systemic vascular resistance, leading to increased oxygen consumption and low systemic vascular resistance, respectively.^{1,4}

According to a study by Anand of over 16,000 patients, the main causes of high-output HF are obesity 31%, liver disease (cirrhosis) 23%, arteriovenous shunts (systemic arteriovenous fistulas (AVF), hemodialysis fistulas and hereditary hemorrhagic telangiectasia) 22%, lung disease (chronic obstructive pulmonary disease, bronchiectasis bronchiolitis obliterans, interstitial disease) 16% and myeloproliferative disease 8%. Other causes that can lead to high-output HF include thiamine deficiency, causing a disease known as Beriberi, and finally, thyrotoxicosis.⁵

The treatment can be divided into two parts. First, it starts with acute heart failure intervention. Depending on the severity, treatment should be aimed at acute respiratory failure due to volume overload and, if present, hypotension. Initial management may range from intermittent diuretic therapy and oxygen supplementation to continuous diuretic infusion, non-invasive positive pressure ventilation, or intubation. If hypotension and decreased organ perfusion are evident, inotropic medications are required. Once patients are stabilized and are no longer in a decompensated state, treatment can be directed to the underlying etiology.^{6,7}

The treatment of hyperthyroidism is focused on symptomatic therapy with the aim of reducing circulating thyroid hormones, either through medication and/or the use of radiotherapy or surgery if necessary. Myeloproliferative diseases are treated according to the specific underlying disease and may involve hematopoietic cell transplantation, depending on the severity. Treatment is widely variable depending on the disease and severity of symptoms. The Surviving Sepsis Campaign guidelines guide the treatment of sepsis and involve early recognition, immediate and aggressive resuscitation with intravenous fluids, and antibiotic therapy with investigation to determine the source of infection. Thiamine deficiency that causes Beriberi is treated with thiamine replacement for at least two weeks. Chronic lung disease is a progressive disease with treatment targeting the underlying lung disease. In general, hypoxia and hypercapnia are addressed, and management is symptomatic therapy that slows the progression of the underlying disease. Acquired AVFs are treated by closing or reducing blood flow. It is more pertinent in patients on hemodialysis who require access; however, if it presents with HOHF, closure may be necessary, and alternative access sites must be obtained. Treatment of congenital AVFs may involve medical therapy, invasive embolization, or surgical excision, depending on the exact cause. Liver cirrhosis advanced enough to cause high-output heart failure is end-stage, and treatment involves liver transplantation. Medical therapy has

Keywords

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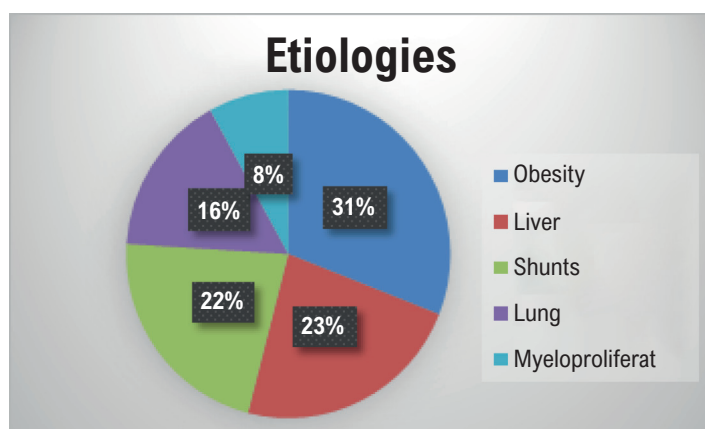


Figure 1 – Etiologies of high-output HF.

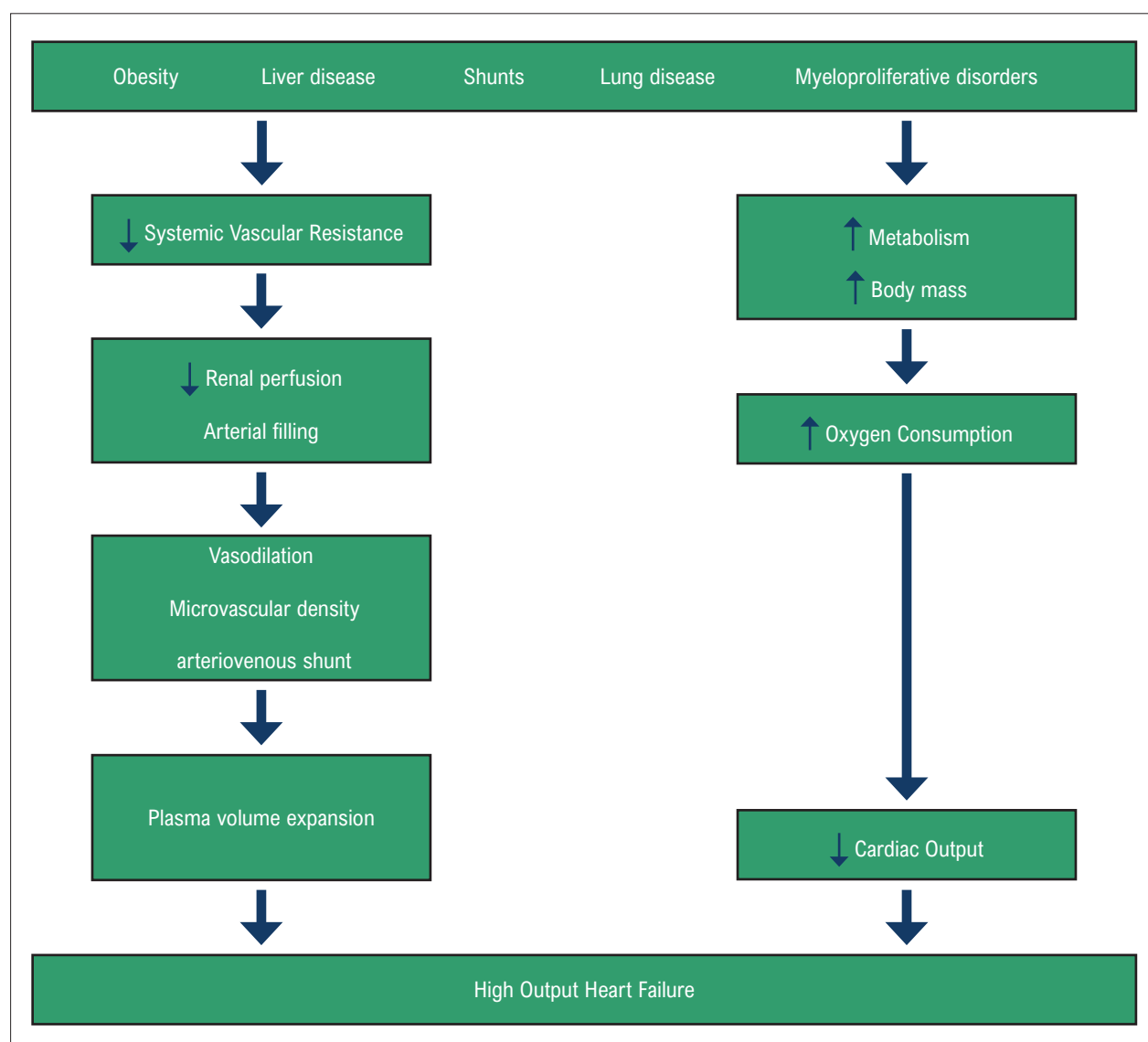


Figure 2 – Pathophysiology of the disease. Adapted Reddy et al.¹

a role in fluid management for hypervolemia, involving the combined use of loop diuretics and antimineralocorticoids, which can limit flow through shunts.

Differential diagnosis

Clinical heart failure is a diagnosis based on the patient's history and physical examination at initial evaluation. Although the type may not be clear, most patients experience very similar, if not the same, symptoms. Hypervolemia, dyspnea at rest or exertion, orthopnea, and fatigue are the general symptoms of heart failure. Different types of heart failure include heart failure with reduced ejection fraction and heart failure with preserved ejection fraction, which can be differentiated from HOHF by measuring cardiac output and/or cardiac index and low systemic vascular resistance.

Prognosis

The prognosis for HOHF depends on the cause of the disease. Reddy published a retrospective analysis of patients with HOHF, reporting an increased mortality rate of three patients compared to the control group (individuals free from heart failure). The study reported a hazard ratio of 3.4 (1.6 to 7.6). This study further stated that among patients with HOHF, obesity-related causes had the lowest 5-year mortality, 19%, compared to liver disease (58%) and heart failure associated with shunt formation (59%), which presented the highest mortality in 5 years.¹

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HOHF is a rarely reported cardiovascular complication associated with non-cardiovascular diseases and is found in 0.07% of all HF cases. Although comparable in-hospital mortality was observed among the cohorts studied, there is a need to increase awareness and timely recognition of this entity.

Author Contributions

Conception and design of the research, Writing of the manuscript and Critical revision of the manuscript for content: Santos CC, Rossi Neto JM, Finger MA; Acquisition of data: Santos CC; Analysis and interpretation of the data: Santos CC, Rossi Neto JM.

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Study association

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Ethics approval and consent to participate

This article does not contain any studies with human participants or animals performed by any of the authors.



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