



Editorial

Editorial: High-output state due to an arteriovenous fistula for hemodialysis access as a cause of the aggravation of congestive heart failure



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High-output heart failure is an uncommon type of acute heart failure, and is caused by various pathological conditions, such as anemia, sepsis, beriberi, congenital or acquired arteriovenous fistula, hyperthyroidism, psoriasis, and Paget disease of bone [1,2]. The underlying pathophysiological condition in high-output heart failure is reduced systemic vascular resistance, which is elicited by either vasodilation of systemic resistant vessels or systemic arteriovenous shunt. The decreased systemic vascular resistance activates hemodynamic and neurohormonal responses that maintain blood supply to the organs. The responses include an increase in cardiac output [3] and the activation of the sympathomimetic and renin–angiotensin–aldosterone systems [4,5]. These neurohormonal alterations result in the retention of sodium and water and an expansion of both extracellular fluid and plasma volume, which predispose the patients to the development of congestive heart failure (Fig. 1).

Symptoms, physical findings, and laboratory tests in high-output heart failure

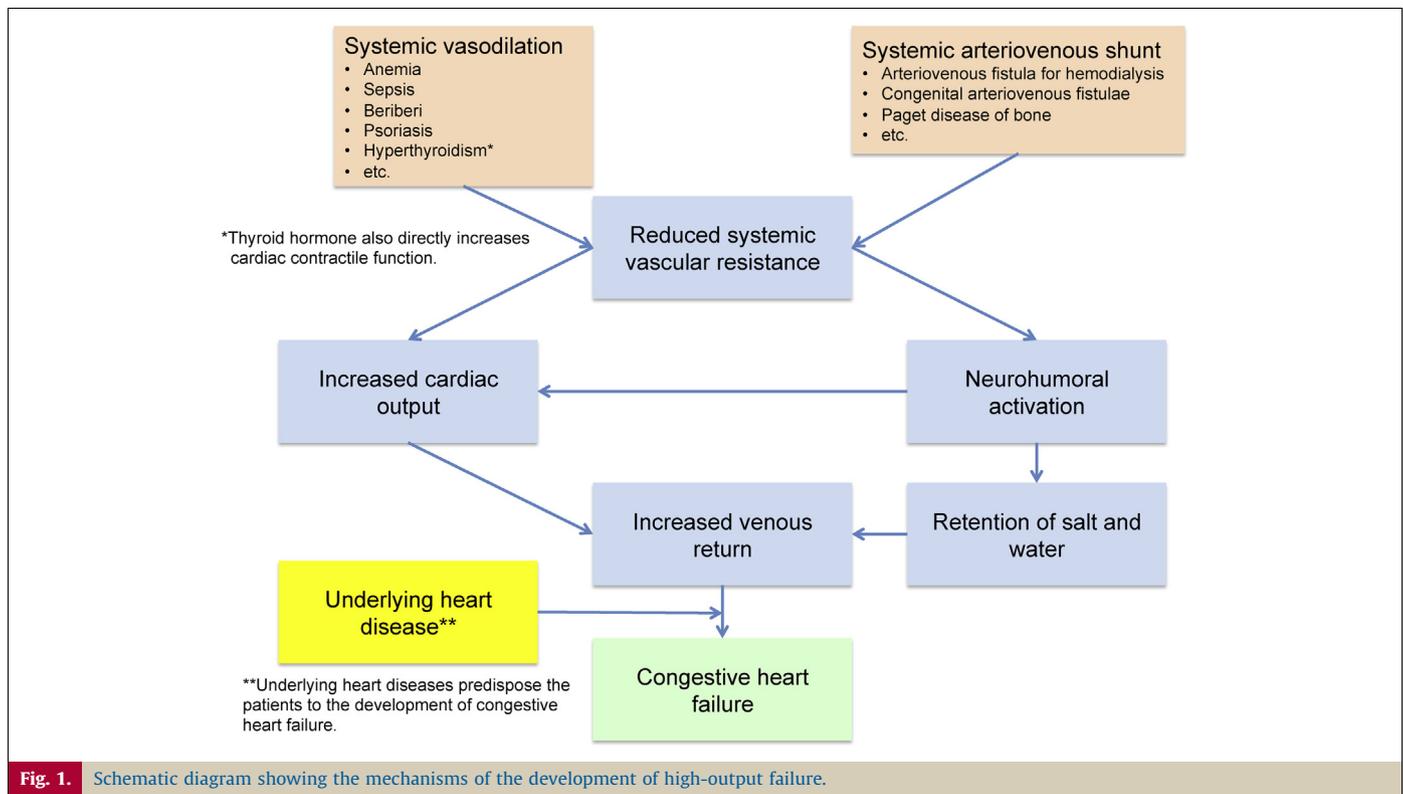
Patients with high-output heart failure may complain of typical symptoms of congestive heart failure including exertional dyspnea, orthopnea, and peripheral edema. Physical examination may reveal the dilatation of jugular veins, coarse crackles on lung auscultation, a third sound on cardiac auscultation, and leg edema, all of which are commonly found not only in patients with high-output heart failure but also in those with much more common low-output heart failure. However, the patients usually show warm extremities suggesting that the extremities are well perfused, which is sometimes a useful finding to notice the existence of high-output state in patients with acute heart failure. Patients with high-output heart failure may also have bounding pulses and wide pulse pressure although these findings can be

seen in other cardiac conditions, such as aortic regurgitation and patent ductus arteriosus [6]. Echocardiography is essential to assess chamber sizes, valvular function, and both systolic and diastolic ventricular performances. Right heart catheterization may be required in some cases to confirm the existence of high-output state in which cardiac output is high (e.g. more than 8 L/min) [1].

Causes of high-output heart failure and its treatment

Identifying the cause of symptomatic high-output heart failure is vital to manage acute heart failure due to this condition, and feasibility of the treatment depends on the underlying disease and its severity. For example, in patients with refractory sepsis and high-output heart failure, it may not be always easy to manage this underlying disease of acute heart failure. It is also important to be aware that underlying heart diseases play a pivotal role in the development of symptomatic high-output heart failure (Fig. 1). In other words, we have to search for an underlying heart disease as soon as we notice the existence of high-output heart failure [6]. According to the guidelines for the diagnosis and management of heart failure in adults published by the American Heart Association and American College of Cardiology, anemia must be severe (e.g. hemoglobin levels less than 5 g/dl) to be the sole cause of high-output heart failure [7].

In addition to the treatment of underlying diseases, such as anemia, sepsis, beriberi, arteriovenous fistulae, and hyperthyroidism, correction of excessive fluid retention may also be useful to manage symptomatic high-output heart failure. Dietary restriction of sodium intake and judicious use of diuretics may be helpful in some patients [1]. In the case of septic shock, intravenous infusion of norepinephrine should be considered to reverse the reduced systemic vascular resistance induced by peripheral vasodilation [8]. There is a huge amount of evidence supporting the use of angiotensin-converting enzyme inhibitors, angiotensin receptor blockers, and beta-blockers in patients with chronic heart failure and a low left ventricular ejection fraction [7]. However, the administration of angiotensin-converting enzyme inhibitors or angiotensin receptor blockers further decreases systemic vascular resistance, and aggravates the high-output state. Cautious use of a beta-blocker in combination with an antithyroid drug may be beneficial for some patients with high-output heart failure due to hyperthyroidism when they have sinus tachycardia or atrial fibrillation with rapid ventricular response [6].



A case of high-output heart failure

Imran et al. reported an interesting case of high-output heart failure that was caused by a radiocephalic arteriovenous fistula for hemodialysis access with abnormally high shunt flow [9]. The patient presented with typical symptoms and signs of congestive heart failure such as dyspnea, coarse crackles on auscultation, and pitting edema of the lower extremities. Laboratory tests revealed a high level of brain natriuretic peptide (5386 pg/ml) without an increase in cardiac enzymes. Chest X-ray showed cardiomegaly and pulmonary vascular congestion. It may not have been difficult to diagnose the patient as having acute exacerbation of congestive heart failure, and the patient was successfully treated with intravenous furosemide, bi-level positive airway pressure, and the intravenous infusion of nitroglycerin. Since echocardiography demonstrated severe aortic stenosis with a peak velocity of greater than 4 m/s across the aortic valve, the authors scheduled aortic valve replacement. The authors noticed the possibility of excessively high shunt flow at the arteriovenous fistula for hemodialysis access during physical examination, and ultrasound revealed an abnormally high shunt-flow volume of greater than 5 L/min, which strongly suggested the existence of high-output state in the patient. Finally, the patient underwent right heart catheterization, which demonstrated an excessive cardiac output of 10.6 L/min and a cardiac index of 5.2 L/min/m². Before aortic valve replacement, the patient was subjected to banding of the arteriovenous fistula, which reduced the shunt flow to 1.8 L/min.

The importance of considering both an underlying heart disease and shunt-flow volume in patients with symptomatic heart failure with an arteriovenous fistula

Abnormally high shunt flow at an arteriovenous fistula for hemodialysis access can cause high-output heart failure in patients

with end-stage renal disease, and several case reports have already been published [10,11]. The excessively high shunt-flow volume can be reduced by banding, which mitigates the adverse effects of systemic arteriovenous shunt on the hemodynamic condition [12]. Even though the shunt flow is appropriate, creation of an arteriovenous fistula elicits persistently high cardiac-output state and increases myocardial oxygen demand, which may predispose the patients with end-stage renal disease to a risk of myocardial ischemia [13]. Therefore, patients with an underlying heart disease, such as severe aortic stenosis as described in the case report by Imran et al. [9] may be more likely to develop acute exacerbation of heart failure after the creation of an arteriovenous fistula than those without a concomitant heart disease. In patients with an underlying heart disease and abnormally high shunt-flow volume at the arteriovenous fistula, the hemodynamic condition may be easily aggravated.

In conclusion, the case reported by Imran et al. [9] highlighted the importance of considering both an underlying heart disease and shunt-flow volume at the arteriovenous fistula when we treat patients with symptomatic heart failure who are on hemodialysis.

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Yutaka Kagaya (MD, PhD, FJCC)*

Office of Medical Education, Tohoku University Graduate School of Medicine, Sendai, Japan

*Correspondence to: Office of Medical Education, Tohoku University Graduate School of Medicine, 2-1 Seiryomachi, Aoba-ku, Sendai 980-8575, Japan. Tel.: +81 22 717 8510; fax: +81 22 717 8223
E-mail address: kagaya@med.tohoku.ac.jp (Y. Kagaya).

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