REVIEW

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High-output heart failure from arteriovenous dialysis access: A structured approach to diagnosis and management

ABSTRACT

High-output heart failure can be a complication of having an arteriovenous fistula (or graft) for hemodialysis access. This review details the pathophysiology, diagnosis, and management of this serious but underdiagnosed condition.

KEY POINTS

The diagnosis of arteriovenous high-output heart failure relies on strong clinical suspicion and should incorporate noninvasive methods such as transthoracic echocardiography and color duplex ultrasonography of the access site before proceeding with right heart catheterization to confirm the diagnosis.

In patients with this condition, right heart catheterization will demonstrate a decrease in intracardiac filling pressures and cardiac indices when the fistula is temporarily occluded.

Definitive treatment includes either ligating or banding the fistula. The decision requires a multidisciplinary approach involving specialists in cardiology, vascular surgery, and nephrology—and the patient.

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Arteriovenous high-output heart failure, a consequence of blood shunting through an arteriovenous fistula created for hemodialysis access, is serious and underrecognized.

Middle-aged adults with moderately or severely reduced kidney function are at high risk of developing heart failure.[1] In 2022, 131,194 Americans with chronic kidney disease progressed to end-stage kidney disease, of whom 82% started hemodialysis,[2] and the prevalence of heart failure in this population was 25%.[3] Some of these patients with heart failure will have heart failure secondary to a high-output fistula. Although this condition was reported as early as the 1960s,[4] its exact incidence and prevalence are hard to estimate, as it lacks a universal definition or criteria.

Arteriovenous high-output heart failure is likely underdiagnosed, as most clinicians are unaware of when to evaluate for it and are unfamiliar with how to evaluate for it. Untreated, arteriovenous high-output heart failure has a high mortality. In a series of 120 patients at Mayo Clinic who had high-output heart failure, the 3-year mortality rate was 38%.[5] Thus, it needs to be recognized and treated promptly.

This narrative review details the pathophysiology, epidemiology, diagnosis, and management of arteriovenous high-output heart failure. Although we mostly talk about patients who have an arteriovenous fistula, the same information applies to those who have a prosthetic arteriovenous graft.

TABLE 1
Common causes of high-output heart failure

Cause	Mechanism
Obesity	Vasoactive adipokines released from visceral adipose tissue lead to peripheral vasodilation, decreased systemic vascular resistance, and increased cardiac output Paracrine release of fatty acids from ectopic adipose tissue can result in direct lipotoxicity-mediated alterations in myocardial metabolism, leading to negative cardiac remodeling[5]
End-stage liver disease (cirrhosis)	Systemic circulation of vasodilators from increased portal pressures results in splanchnic vasodilation and overall decreased systemic vascular resistance and increased cardiac output[8]
Arteriovenous shunting	Connection to the lower-resistance venous system decreases both afterload and systemic vascular resistance while increasing venous return to the right and left ventricle, leading to increased cardiac output[7]
Hypercapnic lung disease (chronic obstructive pulmonary disease, connective tissue disease, bronchiectasis)	Long-standing hypercapnia-induced peripheral vasodilation results in decreased systemic vascular resistance, leading to increased cardiac output[7]
Sepsis (acute and long-standing)	Interleukin 1, interleukin 6, and tumor necrosis factor–induced endocapillary leak and peripheral vasodilation decrease systemic vascular resistance, leading to increased cardiac output[9]
Anemia (severe)	Increased renal nitric oxide production leads to peripheral vasodilation, lower systemic vascular resistance, and increased cardiac output[9]
Hyperthyroidism	Increased thyroid hormone production causes increased cardiac contractility, increased heart rate, and decreased systemic vascular resistance, leading to increased cardiac output[10]
Pregnancy	Peripartum increased stroke volume, chronotropy, and increased endothelial synthesis of vasodilating prostaglandins result in decreased systemic vascular resistance and increased cardiac output[11]
Vitamin B_1 deficiency, beriberi	Vitamin B_1 is a necessary cofactor for aerobic metabolism; severe deficiency results in a switch to anaerobic metabolism, leading to a buildup of pyruvate and lactic acid, causing systemic vasodilation, decreased systemic vascular resistance, and increased cardiac output[9]
Myeloproliferative disease	Poorly understood; proposed mechanisms include myeloproliferative neoplasm causing increased metabolism by malignant cells, extramedullary hematopoiesis, or anemia[12]

DUE TO BLOOD SHUNTING

Heart failure is a clinical syndrome that results from any structural or functional impairment in ventricular filling or ejection of blood at rest or with exertion.[6] It is called *high-output* heart failure if the cardiac index is higher than 3.9 L/min/m^2 or the cardiac output is higher than 8.0 L/min.[7] The classification of heart failure by output is different from the classification system by ejection fraction (reduced, mildly reduced, and preserved). Most patients with arteriovenous high-output heart failure have a preserved ejection fraction.

Common causes of high-output heart failure and their mechanisms are listed in **Table 1**.[5,7,8–12]

In arteriovenous high-output heart failure (**Figure 1**), the fistula that was created for dialysis access allows blood to shunt from the arterial system to the lower-pressure venous system, resulting in increased right ventricular preload with compensatory right ventricular hypertrophy and dilation.[7] This increased right ventricular preload also increases left ventricular preload and stroke volume, which, along with decreased total peripheral resistance, results in increased cardiac output.[7]

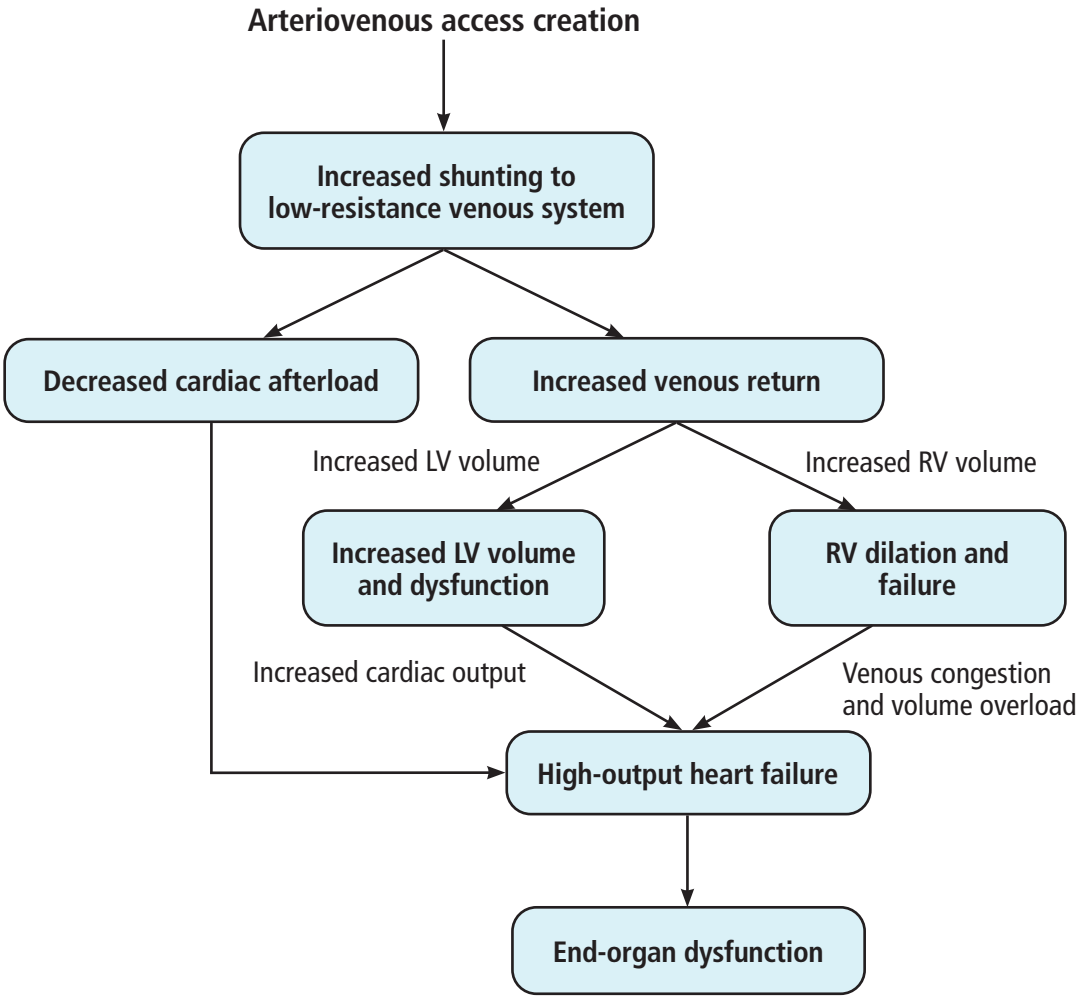

Figure 1. Pathophysiology of arteriovenous high-output heart failure. Creation of arteriovenous access, with mixing of arterial and venous blood, leads to increased shunting into the lower-resistance venous system, resulting in decreased cardiac afterload and increased venous return. These changes impact the right and left ventricles, contributing to the development of high-output heart failure.

LV = left ventricle; RV = right ventricle

Over time, increased preload can lead to left ventricular hypertrophy with impaired diastolic filling, and later progress to left ventricular dilation with impaired systolic function.[7,13] These changes can begin as soon as 3 to 14 days after the fistula is created.[13,14] Factors specific to chronic kidney disease such as hypertension, upregulation of profibrotic cytokines, and impaired iron utilization can also contribute to negative cardiac remodeling.[15]

Although systemic vascular resistance is decreased, renovascular resistance is paradoxically increased, resulting in reduced renal blood flow and subsequent activation of the renin-angiotensin-aldosterone system, thus promoting inappropriate volume retention.[7,13,14] Over time, this volume retention and cardiac remodeling create a state of volume overload with ineffective circulating volume leading to symptomatic heart failure. If left untreated, this ineffective circulating volume can result in end-organ damage.

THE HIGHER THE FLOW, THE HIGHER THE RISK

The higher the rate of blood flow through the fistula, the higher the risk of high-output heart failure.[7,16] Several factors affect the blood flow rate and the risk of heart failure.

Location. Fistulas placed more proximally, where the artery is bigger—4 to 6 mm in diameter or more—have higher flow and are associated with higher risk.[3,7] Begin et al[17] reported that in a series of 45 patients, 24 to 28 weeks after fistula creation the mean flow through brachiocephalic (proximal) fistulas was 1,285 mL/min, which was nearly twice as much as that through distal radiocephalic (distal) fistulas (647 mL/min).

Time. Arteriovenous fistulas may continue to dilate over time, resulting in significantly increased venous return.

Existing structural heart disease, which is common in patients with end-stage kidney disease before they get their fistula, is associated with higher risks of heart failure and death after starting hemodialysis. These changes include a mildly reduced to reduced left ventricular ejection fraction (< 45%) and right ventricular dysfunction, both of which increase the risk of heart failure exacerbations and are associated with a nearly 2-fold higher risk of death following arteriovenous fistula creation.[18,19] These structural changes are believed to further accelerate the pathophysiology of high-output heart failure.[5,19] Importantly though, most patients with arteriovenous high-output heart failure have a preserved left ventricular ejection fraction, ie, 50% or higher.

End-stage kidney disease itself also increases the risk of high-output heart failure through the mechanisms of hypertension, arteriosclerosis, and chronic anemia.[5]

Comorbidities. Additionally, patients who have any of the comorbidities listed in **Table 1** before getting their fistula are at higher risk of multifactorial high-output heart failure.

DIAGNOSIS: A STRUCTURED APPROACH

Although there are no validated risk-stratification tools or algorithms for diagnosing and managing arteriovenous high-output heart failure, we propose an algorithm (**Figure 2**) that starts with noninvasive assessments and progresses to invasive testing only when indicated.

Noninvasive assessments first

History. Patients present with typical symptoms of heart failure such as dyspnea, orthopnea, paroxysmal nocturnal dyspnea, decreased exercise tolerance, peripheral edema, and fatigue.[6]

The sequence of events—fistula creation first, then heart failure onset or worsening—is critical. Arteriovenous high-output heart failure should be strongly suspected if heart failure newly arises or if admissions for decompensated heart failure increase after the fistula is created and no other precipitating factor is evident, especially if the patient's fistula is high up in the arm.[20]

How long after fistula creation do symptoms arise? Information is mostly limited to case reports, but the onset may be dramatic and immediately follow the procedure, or occur more insidiously months to years later as the flow through the fistula increases, concurrent with cardiac remodeling.[3] There is no system for categorizing the time of onset of symptoms, but we propose calling it *early* if symptoms arise less than 6 weeks after the fistula was created, *intermediate* if they arise 6 weeks to 12 months later, and *late* if more than 12 months have passed.

An additional clue could be a paradoxical worsening in heart failure symptoms with the use of guideline-directed medical therapy—specifically, therapy aimed at decreasing cardiac afterload and lowering blood pressure, as these patients already have significantly low systemic vascular resistance.

Physical examination. A knowledgeable and experienced health practitioner should regularly examine the access site to monitor for flow dysfunction, either high or low.[20,21] On palpation, a thrill, pulsatility, and arteriovenous collapsibility are modestly sensitive signs (compared with ultrasonography as the gold standard) for detecting stenosis (ie, low flow) but not high flow through the fistula.[20,21]

Physical findings that should raise suspicion for high-output heart failure include widened pulse pressure, hyperdynamic precordium, a new systolic murmur (secondary to increased flow), and an abnormally large aneurysmal fistula.[20] However, no physical examination techniques have demonstrated reliable reproducibility for arteriovenous high-output heart failure surveillance.

Molecular biomarkers are of uncertain utility

Many biomarkers have been studied for stratifying the risk of high-output heart failure in patients with arteriovenous fistulas.

Brain natriuretic peptide (BNP) and **N-terminal pro–brain natriuretic peptide (NT-proBNP)** levels correlate with the risk of death in patients with end-stage kidney disease,[22] including those on hemodialysis.[23] Both increase after the fistula is created, and they correlate with increased left ventricular diastolic dysfunction.[13,24] NT-proBNP levels decrease after patients with end-stage kidney disease receive a kidney transplant and subsequently have their fistula ligated (see below).[25,26] However, neither BNP nor NT-proBNP have consistently been found to correlate with cardiac output in patients with end-stage kidney disease, and therefore they have not been shown to predict the

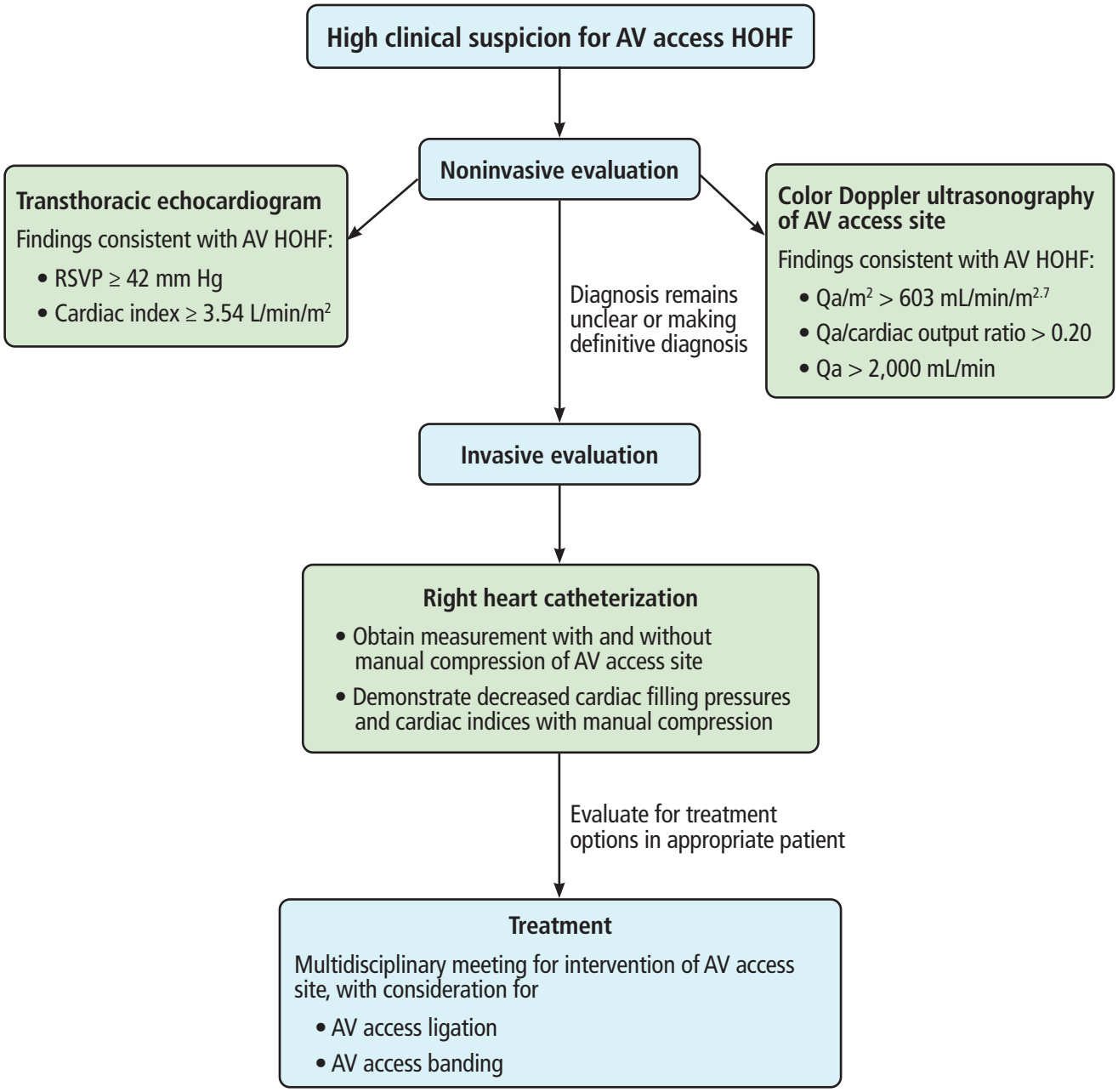

Figure 2. Algorithm for evaluating arteriovenous (AV) access–associated high-output heart failure (HOHF). The evaluation process begins with a high clinical suspicion. Initial assessment is with noninvasive modalities, followed by invasive diagnostic techniques if noninvasive methods are inconclusive or to confirm the diagnosis definitively.

Qa = vascular access blood flow; RSVP = right ventricular systolic pressure

onset of high-output heart failure.[7,13] Additionally, both NT-proBNP and BNP are renally cleared, and their concentrations are directly affected by hemodialysis.[23]

Atrial natriuretic peptide. Iwashima et al[13] found atrial natriuretic peptide levels to be weakly associated with increased cardiac output following arteriovenous fistula creation. However, no other studies have reproduced this finding. Further, the absolute values of both BNP and atrial natriuretic peptide and their percent increase after arteriovenous fistula creation have not

been consistently found to correlate with the blood flow rate through the fistula, further limiting their role in high-output heart failure risk stratification.[7,27]

Cardiac troponins have demonstrated prognostic value in diagnosing myocardial steal syndrome after a fistula is created, but not the onset of high-output heart failure.[3,28]

Newer biomarkers such as suppression of tumorigenicity 2, growth and differentiation factor 15, and galectin 3 have demonstrated associations with left ventricular structural changes, heart failure exacerbations, and cardiovascular mortality in patients on hemodialysis.[5,7] However, testing for these biomarkers is not widely available, and they have not been assessed for correlation with the onset of high-output heart failure.

Transthoracic echocardiography for those with new, suspected, or worsening heart failure

Transthoracic echocardiography is recommended for all patients with new or suspected heart failure as well as those with established heart failure with worsening symptoms.[6,29]

Patients with arteriovenous high-output heart failure can have nonspecific findings corresponding to systolic dysfunction, including increased left ventricular end-diastolic diameter and volume, as well as reduced global longitudinal strain pattern.[27,30] Transthoracic echocardiography can also show characteristics associated with diastolic dysfunction, including an elevated ratio of mitral inflow velocity between diastole and atrial contraction.[5]

Reddy et al,[5] in a retrospective study of 120 patients with known high-output heart failure, found that a cardiac index 3.54 L/min/m^2 or higher on transthoracic echocardiography had a sensitivity of 62% and specificity of 96% for detecting high-output heart failure (area under the receiver operating curve [AUC] 0.85, $P < .0001$). They also found a Doppler-estimated right ventricular systolic pressure of 42 mm Hg or higher had a 92% sensitivity and 100% specificity (AUC 0.97, $P < .0001$). However, these findings can be present in heart failure of multiple etiologies, not just high-output heart failure, and should therefore only be used to further support a suspicion of high-output heart failure in patients with a history and physical examination consistent with this diagnosis, but not by themselves to risk-stratify or diagnose this condition.

Estimated systemic vascular resistance calculated from transthoracic echocardiography could serve as an independent predictive tool for identifying arteriovenous high-output heart failure.[5] However, further studies are needed to validate its reproducibility.

Before performing transthoracic echocardiography, one should try to get the patient down to their dry weight (ie, their weight at the end of dialysis sessions) by removing fluid using intermittent hemodialysis or diuretics. This is to minimize the impact of volume overload on cardiac output measurements, as patients with end-stage kidney disease with significant volume overload may exhibit higher cardiac output attributable to the excess volume.[31]

Color duplex ultrasonography to measure the flow through the fistula

Measuring the rate of flow through the fistula provides critical information about the site's suitability for hemodialysis access as well as the risk of arteriovenous high-output heart failure. The blood flow rate should be greater than 500 or 600 mL/min for an arteriovenous access to be considered mature and adequate for hemodialysis.[20,21] However, high flow rates have been consistently shown to increase the risk of high-output heart failure.[3,16,30,32]

Color duplex ultrasonography is the most common and well-studied technique for measuring the flow.[21,33] It is widely available and relatively inexpensive.[20,21] Its sensitivity is up to 91%, and its specificity is up to 97% compared with fistulography for detecting stenosis.[34] On the negative side, scar tissue, calcification, hematoma, and severe extremity edema can hinder its accuracy. It can also be limited by operator-dependence.[20,21]

Arteriovenous flow can also be measured using magnetic resonance angiography, or indirectly during hemodialysis using ultrasonography dilution or thermodilution.[21] The cost, limited availability, time required, and risk of adverse effects of each limits their practicality.

Unfortunately, after a fistula has matured, there is no consensus on the role of routine surveillance of arteriovenous flow to prevent high-output heart failure.

How much flow is too much? Multiple studies have tried to find the threshold blood flow rate above which cardiac remodeling begins in patients without existing heart failure, or at which surveillance for high-output heart failure should begin.

Saleh et al,[30] in a study of 100 patients on dialysis without existing structural heart disease, found that a flow rate greater than 2,000 mL/min correlated with significantly greater left ventricular dilation as measured by left ventricular end-diastolic diameter, left ventricular end-diastolic volume, and left ventricular mass. Higher flow has also been associated with right ventricular dilation and is an independent risk factor for impaired right ventricular function.[14,35]

The European Society for Vascular Surgery guideline says that a blood flow rate exceeding 1,500 mL/min warrants regular flow measurements and echocardiography to monitor for signs of heart failure, but does not specify time intervals for each.[21] The National Kidney Foundation's guideline does not specify a flow rate warranting further surveillance.[20]

Similarly, there is no universally accepted blood flow rate threshold that results in high-output heart failure. Information about this possible threshold has previously been limited to case reports. Basile et al[16] found a rate greater than 2,000 mL/min had a sensitivity of 89% and specificity of 100% for predicting arteriovenous high-output heart failure (AUC 0.99). This finding was later supported by a study of patients without diabetes on hemodialysis that found significantly greater prevalence of heart failure symptoms in patients with a vascular access flow rate greater than 2,000 mL/min ($P < .05$).[36]

The ratio of arteriovenous access blood flow rate to cardiac output has also been used for risk stratification of high-output heart failure.[16,27,37] Historically, a ratio of 0.20 or 0.30 or greater has been used as a cutoff for high-output heart failure risk; however, this ratio was largely guided by case reports.[19,38–40] Basile et al[16] found a ratio of 0.20 or greater had a 100% sensitivity and 74.7% specificity for identifying high-output heart failure (AUC 0.92).

More recently, the flow rate indexed to the patient's height has been proposed as a better prognosticator for high-output heart failure than the flow rate alone. In a cohort of patients with end-stage kidney disease, all of whom had a vascular access blood flow rate greater than 2,000 mL/min, only 60% of patients had heart failure symptoms.[37] Within this subset, a flow rate indexed to height of 603 mL/min/m$^{2.7}$ or greater demonstrated a sensitivity of 100%, specificity 60%, positive predictive value 83%, and negative predictive value 100% for detecting high-output heart failure (AUC 0.75).

Our recommendations. In view of the high mortality rate associated with arteriovenous high-output heart failure, we believe invasive testing should be considered if the patient has any of the following:

- An arteriovenous access flow rate of 2,000 mL/min or greater,
- A flow rate/cardiac output ratio of 0.20 or greater, or
- A flow rate indexed to height of 603 mL/min/m$^{2.7}$ or greater.

Color duplex ultrasonography of the arteriovenous access should be done as soon as possible after transthoracic echocardiography to prevent confounding interventions, such as volume removal, from affecting the patient's hemodynamics in the interval.

INVASIVE ASSESSMENT: RIGHT HEART CATHETERIZATION

Definitive diagnosis of arteriovenous high-output heart failure requires right heart catheterization, which should be considered only after all the noninvasive studies have been done and the findings have suggested this diagnosis.

Initial measurements should be done without manipulating the arteriovenous fistula. In a patient with arteriovenous high-output heart failure, they will show increased intracardiac pressures including pulmonary capillary wedge pressure, mean pulmonary artery pressure, and mean right atrial pressure; a low to normal systemic vascular resistance; and a high cardiac output and index.[5,40] Then, the fistula should be temporarily occluded and the measurements repeated. Occlusion is commonly performed using an inflated blood pressure cuff.

The essential criterion for diagnosing high-output heart failure is reversibility of both the intracardiac pressures and cardiac indices with temporary occlusion of the arteriovenous fistula. This is particularly important when coexisting ischemia or valvopathy is present that can also be contributing to heart failure.[39] There are no established absolute values or percentage decreases from baseline of either the intracardiac filling pressures or cardiac output or index that establishes the diagnosis of high-output heart failure, however.

Compressing the fistula can also elicit a decrease in heart rate and increase in blood pressure, commonly called the Nicoladoni-Israel-Branham sign. However, this phenomenon is neither sensitive nor specific for high-output heart failure.[3]

A limitation of this procedure is that if the fistula is really big it may be hard to occlude completely, leading to false-negative findings. Another limitation is that some patients cannot tolerate lying flat without shortness of breath or hypoxia. To overcome this, dialysis to remove volume may be necessary; however, this may lead to lower intracardiac filling pressures and cardiac indices, increasing the chance for false-negative diagnosis.

TREATMENT OPTIONS

Ligation

Definitive treatment of arteriovenous high-output heart failure involves removing the shunt pathway through ligation of the fistula site. However, this leaves the patient without ready dialysis access and therefore

is only an acceptable option in those who have received a successful kidney transplant or are suitable candidates for peritoneal dialysis.

Retrospective studies and meta-analyses have shown improvements in cardiac remodeling, ejection fraction, and function after ligation.[26] In a randomized controlled trial in patients with end-stage kidney disease who had received successful kidney transplants, those who had their fistulas ligated had significantly lower NT-proBNP levels and cardiac indices at follow-up compared with patients who did not, whose NT-proBNP levels went up and whose cardiac indices did not change ($P < .001$).[32]

Improvements in heart-failure symptoms and quality of life following arteriovenous fistula ligation have also been observed in case series and retrospective studies.[25,40,41] In a retrospective cohort of 113 patients who successfully received kidney transplants, 29 (26%) had their fistulas closed, mostly because of heart failure symptoms, and their symptoms and exercise capacity improved afterward.[25]

Will prophylactic ligation *prevent* high-output heart failure? In a randomized controlled trial in 28 kidney transplant recipients with a flow rate greater than 1,500 mL/min through their fistulas, no patients in the ligation group developed high-output heart failure, while 5 of 13 (38.5%) of the nonligation group did.[42]

Whether arteriovenous fistula ligation in high-output heart failure decreases the mortality rate remains unclear.[26,42] Studies have found lower 3-year all-cause mortality rates in patients who underwent fistula ligation following kidney transplant than in their counterparts, but this difference was lost after adjustment for confounders.[43] Additionally, the studies showing the benefit of ligation included only patients who had undergone successful kidney transplantation or candidates for peritoneal dialysis. These restrictions limit the generalizability of ligation as a treatment for many patients with end-stage kidney disease who are not current candidates for either peritoneal dialysis or kidney transplantation.

Also, an important consideration is that many transplant recipients experience fistula failure and need to go back on dialysis: as many as 20% by 5 years after transplant, and 50% at 10 years—even as the number of patients on the renal transplant list also continues to grow.[44,45] Many of these patients already have limited vascular access, so the decision to ligate must be multidisciplinary and shared between the cardiologist, nephrologist, vascular surgeon, and patient.

Banding

This procedure offers an alternative to ligation for managing arteriovenous high-output heart failure. It involves surgical dissection down to the fistula and applying bands at various points along its length.[28,46] The bands reduce the radius of the fistula, thereby increasing resistance and decreasing the flow. A study in 50 patients demonstrated more than a 50% reduction in flow following banding, from 3,070 ± 95 mL/min before to 1,490 ± 105 mL/min immediately after ($P < .001$).[47]

Several banding techniques exist,[28,47,48] including precision banding with ultrasonography guidance and the minimally invasive limited ligation endoluminal-assisted revision (MILLER) procedure. In 12 patients with arteriovenous high-output heart failure and average arteriovenous fistula flow of 2,280 mL/min, precision banding resulted in an average reduction of flow of 70% or more, with an improvement in heart failure symptoms in all patients of the cohort.[28] Similarly, in 183 patients with symptomatic heart failure and high arteriovenous fistula flow, the MILLER procedure resulted in complete relief of heart failure symptoms and improved functional capacity in all patients at an average follow-up time of 11 months following band placement.[48]

Despite these improvements, the overall long-term success rates of banding remain low, with high-flow recurrence rates as high as 52% within months of the procedure.[46,47] Other complications reported with banding include stenosis resulting in inadequate flow for hemodialysis access, thrombosis, limb ischemia, distal aneurysms, and infections.[47,48] As with ligation, the decision to perform banding requires a multispecialty meeting with shared consensus between clinicians and patient.

External stenting

Stenting to reduce vascular access flow is a novel method for managing arteriovenous high-output heart failure. However, this technique has shown inconsistent long-term success.[49]

PROMPT DIAGNOSIS NEEDED

High-output heart failure remains an underrecognized but serious complication of arteriovenous access for hemodialysis. Its diagnosis requires a high clinical suspicion and should involve measuring the blood flow through the arteriovenous fistula followed by right heart catheterization to confirm the diagnosis. The high mortality rate and paradoxical worsening with conventional heart failure guideline-directed medical therapy mandate early and prompt diagnosis.

DISCLOSURES

Dr. Ghobrial has disclosed consulting for Edwards Lifesciences, Medtronic, and W.L. Gore & Associates. Dr. Hanna has disclosed serving as an advisor or review panel participant for Akcea Therapeutics, Alexion, Alnylam, Eidos Therapeutics, and Pfizer and consulting for Novo Nordisk. Dr. Finet has disclosed being an advisor or review panel participant for the American Board of Internal Medicine Advanced Heart Failure and Transplant Cardiology Board Examination and Wolters-Kluwer. The other authors report no relevant financial relationships which, in the context of their contributions, could be perceived as a potential conflict of interest.

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