The Thrill Is Gone
A Case of Progressive Dyspnea

Natalia C. Berry, MD, MBA; Piotr S. Sobieszczzyk, MD; Joshua A. Beckman, MD, MSc

Foreword

Information about a real patient is presented in stages (boldface type) to expert clinicians (Drs Beckman and Sobieszczzyk), who respond to the information, sharing their reasoning with the reader (regular type). A discussion by the authors follows.

An 85-year-old white woman with longstanding atrial fibrillation and hypertension presented to our emergency department with a 2-day history of progressive dyspnea. Earlier that week she had been seen by an outpatient provider and was found to be hypotensive. Her home bumetanide was discontinued, and she developed dyspnea that progressed after missing 2 doses. She reported that she was sleeping with more pillows and that she had a limited ability to walk and a new cough.

Her recent history was notable for progressive fatigue and dyspnea over the past year. In the past 3 months she had been treated as an outpatient with loop diuretics for presumed congestive heart failure by a cardiologist. Her recent history was also notable for recurrent hematochezia requiring 5 admissions, including an intensive care unit admission for hemorrhagic shock, over the preceding 6 months. An extensive evaluation, including esophagogastroduodenoscopy, several colonoscopies, and a small-intestine capsule study were unrevealing; she was noted only to have severe diverticulosis. During this period, her warfarin and, after additional bleeding episodes, her aspirin were stopped in light of her recurrent bleeding.

Additional medical history included a diagnosis of cataracts, macular degeneration, and osteoarthritis. Medications included bumetanide 1 mg daily, metoprolol succinate 50 mg daily, omeprazole 40 mg daily, and calcium supplements. She had no known drug allergies.

Dr Beckman: Dyspnea in an older patient is a common concern and is predominantly caused by a range of cardiopulmonary diseases. In this case, the patient’s presentation is notable for progressive dyspnea over 1 year that has acutely worsened over several days. Her report of orthopnea, cough, and exertional intolerance suggest that an exacerbation of congestive heart failure is likely. Moreover, her diagnosis of congestive heart failure is only presumed, and further investigation is needed to understand this acute presentation. Other etiologies of progressive dyspnea include anemia related to her recent gastrointestinal bleeding, or a primary pulmonary process such as interstitial lung disease, chronic obstructive pulmonary disease, malignancy, pulmonary hypertension, or an infectious process such as a viral respiratory infection or pneumonia.

Patient presentation (continued): Initial vital signs on presentation included an oral temperature of 95°F, a heart rate of 81 beats per minute, blood pressure of 110/56 mm Hg, respiratory rate of 20 breaths per minute, oxygen saturation of 85% on room air with improvement to 95% on 3 L nasal cannula oxygen supplementation. Physical examination was notable for a cachectic-appearing woman in no acute distress. Her neck examination revealed marked internal jugular venous engorgement with v waves to 20 cm H2O. Inspiratory rales were noted at the bases of her lungs bilaterally. Cardiac examination was notable for an irregularly irregular rhythm, a diffuse and laterally displaced point of maximal impulse, a normal s1 and s2, and a III/VI holosystolic murmur at the left lower sternal border that increased with inspiration and decreased with exhalation and Valsalva maneuver. A right ventricular heave was present. Her abdomen was soft and nontender, and bowel sounds were present with a fluid shift noted. Over the right flank area, there was a 2 × 2 cm palpable and prominent thrill. The bruit was continuous throughout the cardiac cycle. Her extremities were cool below the knees, without peripheral edema. Pulses were 2+ at brachial, radial, popliteal, dorsalis pedis, and posterior tibial sites. Her neurological and skin examinations were unremarkable.

Chest radiograph revealed marked cardiomegaly, pulmonary vascular congestion, and no parenchymal lung abnormalities (Figure 1). Laboratory studies showed normal electrolytes and a creatinine level of 1.33 mg/dL which was equivalent to the patient’s baseline. The hematocrit was 33.4%, and N-terminal probrain natriuretic peptide was 4497 pg/mL (normal 0–1799). ECG showed atrial fibrillation at 72 beats per minute, right axis deviation, and an Ashman beat (Figure 2). Transthoracic echocardiogram obtained to evaluate her systolic murmur revealed a normal-sized left ventricle with preserved systolic function, mild-to-moderate mitral regurgitation, moderate-to-severe...
right ventricular enlargement with decreased function, biatrial enlargement, and severe tricuspid regurgitation (Figure 3A and 3B). Right ventricular systolic pressure was estimated at 73 mm Hg. The inferior vena cava was dilated and did not collapse with respiration.

Dr Beckman: Physical examination findings of elevated jugular venous pressure, ascites, and pulmonary rales are consistent with biventricular failure and both pulmonary and systemic volume overload. Her right ventricle (RV) is proportionally more affected than her left ventricle, as evidenced by her RV heave on physical examination, and by her echocardiographic findings of RV enlargement with decreased function, severe tricuspid regurgitation, and a dilated inferior vena cava. In contrast, her left ventricle is of normal size with preserved systolic function. The differential diagnosis for her cardiopulmonary findings alone includes, among other diagnoses, primary pulmonary disease causing pulmonary hypertension and RV failure, central shunting causing RV dilation and failure, primary valvular heart disease involving the tricuspid valve with resultant RV failure, and restrictive cardiomyopathy affecting predominantly the RV. However, given the prominent abdominal thrill and continuous bruit on physical examination, a vascular malformation – more specifically an arteriovenous fistula – must be considered as a possible root cause of her congestive heart failure. The patient’s wide pulse pressure would support this diagnosis, because the reduced afterload from a systemic arteriovenous fistula would be expected to lower diastolic blood pressure. Moreover, her RV predominant congestive heart failure would well be explained by chronic RV volume overload from the excess venous return.

Patient presentation (continued): A computed tomography angiogram, ordered to better define the vascular abnormality present on physical examination, demonstrated a large arteriovenous fistula at the level of the right kidney. There was aneurysmal dilatation of the right renal artery, draining into a markedly dilated right renal vein and into a large inferior vena cava (Figure 4). The contrast density in the renal vein is identical in appearance to that within the aorta and the aneurysm, confirming the presence of an arteriovenous fistula. Noncontrast imaging demonstrated calcification in the wall of the aneurysm. A 3-dimensional reconstruction of the arteriovenous fistula demonstrates the fistula anatomy (Figure 5).

Dr Beckman: The suspected diagnosis of an arteriovenous fistula is confirmed. The size of the fistula on imaging is striking, and the calcification in the aneurysm wall suggests a chronicity of years, if not decades. It seems likely that the large arteriovenous fistula may be the root cause of the patient’s presenting symptoms. The arteriovenous fistula caused a chronic high-output state, which in turn was likely the cause of the patient’s longstanding atrial fibrillation, pulmonary hypertension, recent-onset congestive heart failure, and unexplained dyspnea and fatigue in the presence of preserved left ventricular ejection fraction and normal left ventricle size. Moreover, her multiple episodes of hematochezia may also be attributed to chronically elevated visceral venous pressures; indeed, gastrointestinal bleeding has been associated with high-output cardiac failure from arteriovenous malformations.1,2 In considering the

---

**Figure 1.** Chest x-ray demonstrated revealed marked cardiomegaly, pulmonary vascular congestion, and no parenchymal lung abnormalities.

**Figure 2.** ECG showed atrial fibrillation at 72 beats per minute, right axis deviation, and an Ashman beat.
patient’s recent course, it is possible that her history of gastrointestinal bleeding and recurrent episodes of anemia may have exacerbated the high-output state already present, contributing to the recent development of congestive heart failure. Given the diagnosis of a large arteriovenous fistula in a patient with symptomatic high-output congestive heart failure, further diagnostic steps should be pursued to better characterize the fistula and provide therapeutic options.

**Patient presentation (continued):** Right heart catheterization was performed to quantify hemodynamic status, and arteriography was performed for fistula characterization. Right heart catheterization revealed right atrial pressure of 23 mm Hg with elevated cV waves, right ventricular pressure 61/17 mm Hg, pulmonary arterial pressure 58/26 mm Hg, pulmonary capillary wedge pressure 22 mm Hg, and pulmonary vascular resistance 295 dynes-s^-1-cm^-5. Cardiac output was 8.5 L/min by Fick calculation. Arteriography confirmed a single right renal artery supplying a 4.5-cm aneurysm, draining into a markedly dilated right renal vein and inferior vena cava (Figure 6). Angiography of the left renal artery was normal. With temporary fistula occlusion, cardiac output decreased to 4.6 L/min, but pulmonary hypertension remained fixed. The fistula was deemed amenable to safe percutaneous closure because the arterial supply of the kidney originated proximal to the aneurysm; the proximal renal artery gave 3 small branches that supplied flow to the actual kidney.

**Dr Sobieszczyk:** Right heart catheterization revealed elevated filling pressures bilaterally with pulmonary hypertension. Cardiac output was supranormal consistent with the suspected high-output state. With fistula occlusion, cardiac output decreased nearly 50% confirming that the fistula accounted for a significant portion of her cardiac output. Her pulmonary hypertension remained fixed with fistula occlusion, suggesting that longstanding right-sided volume overload had led to vascular remodeling in the pulmonary bed, with fixed pulmonary hypertension as a result.

**Patient presentation (continued):** Treatment options available to the patient included surgical repair, endovascular repair, and continued medical management of her congestive heart failure. Endovascular repair would consist of attempted embolization of the aneurysm and the interruption of blood flow to the aneurysm with the use of vascular occlusion devices. This was possible because the arterial supply of the kidney could be preserved despite the embolization of the aneurysm and fistula. Compromise or injury to the kidney is an important consideration, for further reduction in function would not be well tolerated in an older patient with a baseline impaired glomerular
filtration rate. Surgical repair of this fistula would require an open repair, with ligation and removal of the aneurysm and fistula. Open surgical repair would entail a greater risk for morbidity and mortality than endovascular repair.

Several extensive discussions were held with patient and her daughter about the risks and benefits of treatment. Potential benefits of a procedure included improvement of her congestive heart failure, improvement gastrointestinal bleeding, and abrogation of the risk of aneurysm rupture; risks included procedure risk, her age and frailty, and unclear benefit given the chronicity of the fistula and the fixed pulmonary hypertension on catheterization. This was neither a simple nor straightforward decision for the patient and her family. Ultimately, given the hemodynamic significance of the fistula, it was elected to pursue closure of the fistula. Endovascular repair was chosen over surgical repair in light of the patient’s advanced age, frailty, and correspondingly high surgical risk.

The patient underwent endovascular repair, which was performed 6 weeks after discharge from her original admission. The procedure was performed successfully with the use of 22 coils and 2 vascular occluder devices (Figure 7). Pre- and postprocedure hemodynamics are displayed in Table 1.

The patient has been seen for 3 outpatient visits since the procedure. She feels she has gained a new life, with improved energy, appetite, and resolution of her dyspnea. Three months postprocedure, she no longer required diuretic therapy, and 6 months postprocedure, her anticoagulation was restarted without any recurrent gastrointestinal bleeding. It remains to be seen whether her RV dysfunction and pulmonary hypertension may improve over time with correction of this overload.

Dr Beckman: In this patient who presented with an acute on chronic exacerbation of congestive heart failure, careful physical examination and the discovery of a prominent abdominal thrill were paramount to arriving at the correct diagnosis. Fistula characterization revealed that nearly half of the patient’s cardiac output was being diverted to the fistula, causing a high-output state and resultant congestive heart failure. With successful closure of the fistula, the patient reported marked improvement of her symptoms of exertional dyspnea and fatigue, and her functional status has increased significantly.

Discussion

We present a patient with new-onset congestive heart failure related to high output from a large, chronic renal arteriovenous fistula. This case highlights the pathophysiology of high-output cardiac failure, the importance of the physical examination in the diagnosis, and the decision process behind performing invasive procedures in the elderly population.

High output is defined as a resting cardiac output in adults exceeding 8 L/min or 3.9 L·min⁻¹·m⁻². When accompanied by pulmonary or systemic congestion, this state is referred to as high-output cardiac failure. Conditions associated with high cardiac output include anemia, systemic arteriovenous fistulae, pregnancy, sepsis, hepatic disease, renal disease, hyperthyroidism, nutritional deficiencies, and a variety of other conditions (Table 2).

The pathophysiology of congestion arises as a result of decreased systemic vascular resistance secondary to the aforementioned conditions. With a fall in systemic vascular resistance and arterial blood pressure, baroreceptor activation of the renin-angiotensin-aldosterone system, sympathetic nervous system, and arginine vasopressin occurs. This in turn leads
The mechanism of salt retention is similar to that which occurs in low-output cardiac failure as a response to arterial hypotension secondary to decreased cardiac output.

The signs and symptoms of high-output cardiac failure overlap with those of low-output cardiac failure. Patients may present with symptomatic dyspnea, exertional intolerance, fatigue, and fluid retention manifested by abdominal or peripheral swelling. Physical examination may reveal tachycardia, tachypnea, elevated jugular venous pulsations, pulmonary rales, pleural effusions, ascites, and peripheral edema. A wide pulse pressure is often present. In contrast to low-output cardiac failure, patients with high-output cardiac failure are more likely to have warm extremities in the context of low peripheral vascular resistance and relative peripheral vasodilation.

Treatment of high-output cardiac failure requires treating the underlying etiology of the failure. The heart is usually structurally normal in high-output failure, and therefore correcting the high-output state and ameliorating fluid retention, are mainstays of management.

Arteriovenous fistulae are an uncommon cause of high-output cardiac failure. Renal arteriovenous fistulae have an estimated prevalence of <0.04%, and represent between 14% and 53% of all arteriovenous abnormalities. Fistulae can be acquired or congenital. Approximately 70% of renal arteriovenous fistulae are acquired, with the remaining 30% thought to be congenital. Acquired fistulae occur usually in the case of trauma, or as a result of a surgical or percutaneous procedure. Uncommonly, acquired fistulae are classified as idiopathic, presumed to be caused by a preexisting renal artery aneurysm that erodes into an adjacent vein. Frequently, high-output failure can result from a known arteriovenous fistula used for hemodialysis in patients end-stage renal disease. A case of renal cell carcinoma causing systemic arteriovenous shunting and high-output cardiac failure has also been described.

Signs and symptoms of renal arteriovenous fistulae include an abdominal bruit (100%), hypertension and cardiomegaly (50%), congestive heart failure (32%), and gross hematuria (21%). Ultrasonography, renal computed tomography, and magnetic resonance angiography are the diagnostic procedures of choice. Treatment options for symptomatic renal arteriovenous fistulae include surgical ligation with or without partial or total nephrectomy, or percutaneous transcatheter embolization. Transcatheter embolization of a renal arteriovenous fistula was first described in 1973. Important considerations for percutaneous approach include access route (arterial or venous), materials required to close the fistula, and the avoidance of normal perfusing vessels.

In this case, the life-saving procedure was only possible because careful physical examination uncovered a key finding in diagnosing the patient’s condition. The patient had been examined by >25 physicians in the 6 months preceding the current admission without appreciation of the thrill. We believe that the history and examination remain the paramount diagnostic tool for clinicians. In a study looking at >400 consecutive patients admitted to an emergency department, it was the history and physical examination together that proved to be the most accurate modality in making the correct diagnosis; a diagnosis was accurately made by history and physical examination in ~60% of the cases. Another study observed that a pivotal physical examination finding by an experienced clinician led to a substantial change in a patient’s treatment course.

### Table 1. Hemodynamics Before and After Endovascular Repair Procedure

<table>
<thead>
<tr>
<th></th>
<th>Preprocedure</th>
<th>Postprocedure</th>
</tr>
</thead>
<tbody>
<tr>
<td>Right atrium</td>
<td>22 mm Hg</td>
<td>20 mm Hg</td>
</tr>
<tr>
<td>Right ventricle</td>
<td>61/17 mm Hg</td>
<td>56/15 mm Hg</td>
</tr>
<tr>
<td>Pulmonary artery</td>
<td>58/26 mm Hg</td>
<td>56/23 mm Hg</td>
</tr>
<tr>
<td>Pulmonary capillary wedge pressure</td>
<td>22 mm Hg</td>
<td>20 mm Hg</td>
</tr>
<tr>
<td>Cardiac output (Fick)</td>
<td>8.5 L/min</td>
<td>4.7 L/min</td>
</tr>
<tr>
<td>Aorta</td>
<td>106/49 mm Hg</td>
<td>120/67 mm Hg</td>
</tr>
<tr>
<td>Inferior vena cava saturation</td>
<td>83%</td>
<td>63%</td>
</tr>
</tbody>
</table>

### Table 2. Conditions Causing High-Output Cardiac Failure

- Anemia
- Pregnancy
- Sepsis
- Beriberi disease
- Paget disease
- Chronic obstructive pulmonary disease
- Renal disease
- Hyperthyroidism
- Hepatic disease (cirrhosis, hepatitis)
- Systemic arteriovenous fistula
in 25% of cases. Indeed, it is not the testing that makes a diagnosis, it is the physician who uses these tools as guided by his/her evaluation of the patient. The history and physical examination are unique tools available at the patient bedside, whereas all other testing can be ordered from afar. If we deemphasize our roles at the bedside, we risk obsolescence in the care of patients. Although more diagnostic modalities will be made available to us in the years to come, it is clear that the physical examination must remain – as it has for centuries – a foundation of clinical care.

Performing invasive procedures in elderly patients can be controversial for the narrower window of risk and benefit. The advanced age, frailty, and a relatively higher number of comorbid conditions in such patients are a cause for concern among clinicians. It is clear that the potential benefits of any procedure must be carefully weighed against procedural risk on an individual case-by-case basis. When discussing the possibility of an invasive procedure with a frail, elderly patient, appropriate expectation setting is necessary. The physician must explain in detail the ramifications of the procedure so the patient understands the expected level of disruption to their daily lives, the possible loss of autonomy during recovery, the length of the recovery period, and the expected change in duration and quality of life as a result of a safe procedure. Despite a narrower window of benefit, the correct use of interventions may provide a significant improvement in an elderly patient’s quality of life. Indeed, a recent systematic review of elderly patients undergoing percutaneous coronary intervention demonstrated that patients >80 years of age can derive as much benefit in terms of gain in quality of life as their younger counterparts.

Conclusion

This case underscores the importance of considering a wide differential even in a seemingly routine presentation of acute congestive heart failure in an elderly patient with progressive dyspnea and fatigue. Physical examination was paramount to the discovery of a large arteriovenous fistula as the cause of the patient’s presenting symptoms and high-output congestive heart failure. With the diagnosis confirmed, the fistula was closed only after judicious consideration of the risks and the benefits of such a procedure in an elderly patient. The patient has reclaimed a level of energy, well-being, and functional status that she has not had in years.

Disclosures

None.

References


**Key Words:** arteriovenous fistula ■ cardiac output, high ■ heart failure
The Thrill Is Gone: A Case of Progressive Dyspnea
Natalia C. Berry, Piotr S. Sobieszczyk and Joshua A. Beckman

Circulation. 2015;132:953-959
doi: 10.1161/CIRCULATIONAHA.115.017267

Circulation is published by the American Heart Association, 7272 Greenville Avenue, Dallas, TX 75231
Copyright © 2015 American Heart Association, Inc. All rights reserved.
Print ISSN: 0009-7322. Online ISSN: 1524-4539

The online version of this article, along with updated information and services, is located on the World Wide Web at:
http://circ.ahajournals.org/content/132/10/953

Permissions: Requests for permissions to reproduce figures, tables, or portions of articles originally published in Circulation can be obtained via RightsLink, a service of the Copyright Clearance Center, not the Editorial Office. Once the online version of the published article for which permission is being requested is located, click Request Permissions in the middle column of the Web page under Services. Further information about this process is available in the Permissions and Rights Question and Answer document.

Reprints: Information about reprints can be found online at:
http://www.lww.com/reprints

Subscriptions: Information about subscribing to Circulation is online at:
http://circ.ahajournals.org//subscriptions/