Case Reports

Exertional Dyspnea as a Symptom of Infrarenal Aortic Occlusive Disease

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Advanced atherosclerosis of the aorta can cause severe ischemia in the kidneys, refractory hypertension, and claudication. However, no previous reports have clearly associated infrarenal aortic stenosis with shortness of breath. A 77-year-old woman with hypertension and hyperlipidemia presented with exertional dyspnea. Despite extensive testing and observation, no apparent cause for this patient's dyspnea was found. Images revealed severe infrarenal aortic stenosis. After the patient underwent stenting of the aortic occlusion, she had immediate symptomatic improvement and complete resolution of her dyspnea within one month. Twelve months after vascular intervention, the patient remained asymptomatic.

In view of the distinct and lasting elimination of dyspnea after angioplasty and stenting of a nearly occluded infrarenal aortic lesion, we hypothesize that infrarenal aortic stenosis might be a treatable cause of exertional dyspnea. Clinicians should consider infrarenal aortic stenosis as a possible cause of dyspnea. Treatment of the stenosis might relieve symptoms. ***(Tex Heart Inst J 2014;41(3):316-8)***

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Advanced atherosclerosis in the aorta can cause critical ischemia in the kidneys and lower extremities. Severe stenosis and occlusion of the aorta is often associated with refractory hypertension and leg claudication[1]; however, dyspnea on exertion has not been established as a correlated symptom. We describe the case of a 77-year-old woman with chronic, severe atherosclerotic disease of the aorta who presented with worsening exertional dyspnea and underwent stenting of a nearly occluded calcified lesion in her infrarenal abdominal aorta.

Case Report

In January 2012, a 77-year-old woman with hypertension, hyperlipidemia, and diabetes mellitus presented with a 2-month history of progressive exertional dyspnea that had begun to interfere with her performing light housework and walking farther than the length of her home. Other than dyspnea, she reported only bilateral calf and buttocks pain that reproducibly occurred with ambulation. She had initially sought medical care at an emergency department after having been awakened by acute-onset shortness of breath and left-sided chest pain. At that presentation, she had a blood pressure of 171/71 mmHg; heart rate, 69 beats/min; respiratory rate, 17 breaths/min; and oxygen saturation, 100% on room air. At that time, results of physical examination and laboratory studies were unrevealing. A ventilation-perfusion scan showed a low probability of pulmonary embolism. A computed tomographic (CT) angiogram revealed diffuse coronary and aortic atherosclerosis, without evidence of pulmonary embolism or parenchymal lung disease.

The CT angiographic findings and ongoing symptoms led to the patient's transfer to our institution for further evaluation. Upon arrival, she appeared to be comfortable and was asymptomatic at rest. She had a blood pressure of 156/82 mmHg despite reported compliance with antihypertensive medications, a resting pulse rate of 71 beats/min, and an oxygen saturation of 99% on room air. Her physical examination was notable for mild sternal chest discomfort upon palpation, occasional ectopic beats during cardiac auscultation, and faint pulses in her bilateral femoral, dorsalis pedis, and posterior tibial arteries. Results of laboratory studies, including D-dimer, pro-brain natriuretic peptide, hemoglobin, thyroid-stimulating hormone, creatinine, and

sequential troponin measurements, were within normal limits. However, the patient had an elevated total cholesterol level of 242 mg/dL and her low-density-lipoprotein cholesterol level was 152 mg/dL. An initial electrocardiogram (ECG) showed sinus rhythm with occasional premature ventricular beats and no evidence of left ventricular hypertrophy or myocardial ischemia.

During the patient's hospitalization, her chest pain rapidly subsided after she took anti-inflammatory medication; however, the episodes of dyspnea and fatigue with minimal ambulation persisted. No changes were made to her previously prescribed antihypertensive regimen, because blood pressure recordings throughout her stay ranged from 110 to 140 mmHg systolic and 40 to 60 mmHg diastolic. She continued to take amlodipine (5 mg) and losartan (50 mg) daily, and carvedilol (12.5 mg) twice daily. Exercise on a modified Bruce protocol treadmill stress test was limited to 7 minutes (4 metabolic equivalents) because of her progressive dyspnea. Otherwise, there was no ECG or echocardiographic evidence of ischemia, she had no chest pain or claudication, and her peak blood pressure response during stress was normal at 144/72 mmHg. An echocardiogram revealed normal left ventricular function with no diastolic dysfunction or significant valvular disease. A coronary CT angiogram showed mild luminal irregularities without evidence of obstructive coronary artery disease. Pulmonary function testing yielded no obstruction or restriction.

Magnetic resonance angiograms of the chest, abdomen, and lower extremities revealed a high-grade stenosis of the abdominal aorta 3 cm distal to the renal arteries (Fig. 1). An abdominal CT angiogram suggested moderate stenosis of the left renal ostium, and it confirmed infrarenal stenosis secondary to densely calcified plaque and atherosclerotic changes within the infrarenal abdominal aorta, which reduced the luminal diameter to 2 mm. Consequently, a vascular surgeon performed a diagnostic angiogram, which revealed more than 80% stenosis of the infrarenal aorta with no significant pressure gradient across the renal arteries. The aortic lesion was treated with angioplasty and placement of a balloon-expandable PALMAZ® stent (Cordis, a Johnson & Johnson company; Bridgewater, NJ), which increased the aortic diameter to 8 mm. To reduce the risk of aortic rupture secondary to severe calcification, the treating surgeon allowed a residual 40% non-flow-limiting stenosis (Fig. 2).

One day after the intervention on hospital day 6, the patient's claudication symptoms completely resolved—she was able to walk around the ward with noted improvement in her exertional dyspnea, and she was therefore discharged from the hospital. Within one month, her exertional dyspnea had fully resolved, and she was able to resume daily exercise, including 30-minute bicycle rides, weight-lifting, and frequent bowling sessions. A modified Bruce protocol stress test 4 months after the procedure yielded a 4-minute improvement in walking time, from 7 to 11 minutes (7 metabolic equivalents), without recurrence of her previous symptoms. During clinical visits 6, 9, and 12 months after intervention, the patient was completely asymptomatic, and her hypertension remained controlled by her unchanged antihypertensive regimen.

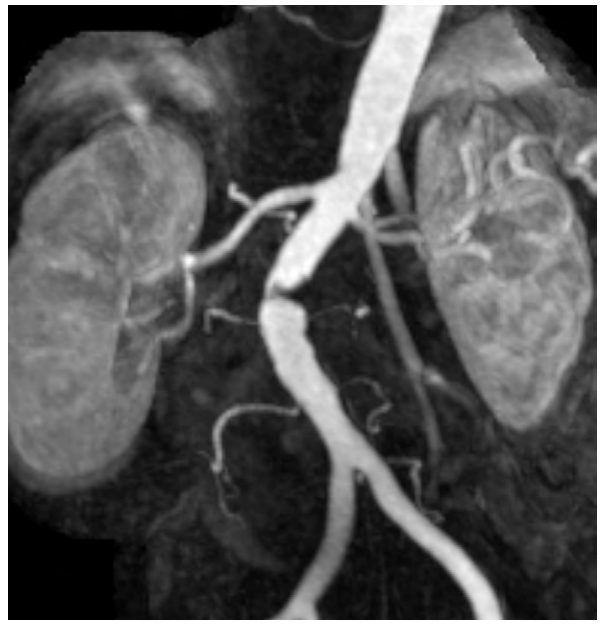

Fig. 1 *Magnetic resonance angiogram reveals high-grade stenosis of the abdominal aorta 3 cm distal to the renal arteries.*

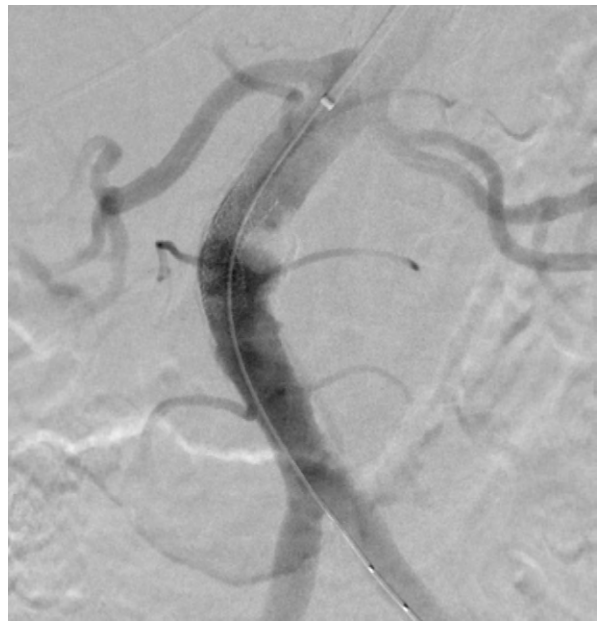

Fig. 2 *Angiogram after angioplasty shows stent placement and residual 40% non-flow-limiting stenosis.*

Discussion

We recognized a causative relationship between the patient's infrarenal abdominal aortic stenosis and her claudication symptoms and diminished peripheral pulses; however, we did not expect to find an association between the abdominal aortic stenosis and the shortness of breath. We investigated typical causes of exertional dyspnea, including hypertensive heart disease with diastolic dysfunction, ischemic heart disease, chronotropic incompetence, pulmonary embolism, and primary pulmonary disease. Our suspicion of an intrinsic cardiac problem was low, because the nature of the patient's chest pain seemed most consistent with musculoskeletal inflammation and did not correlate with exertion or shortness of breath. In addition, the admission echocardiogram and the results of laboratory and stress tests did not suggest ischemic heart disease or congestive heart failure. Nothing from the chest images, ventilation-perfusion scan, or pulmonary function test results suggested pulmonary embolism or primary pulmonary disease as a cause of the dyspnea. In hindsight, we hypothesized that the patient's infrarenal aortic disease was responsible for her entire presentation, because her dyspnea resolved completely after angioplasty and stenting of her aortic lesion.

Near-complete aortic occlusion, which can be seen in middle aortic syndrome or advanced atherosclerotic disease with focal infrarenal aortic stenosis, is apparently relatively rare.[2,3] Associated risk factors include heavy smoking (in 98% of reported cases), hypertension (58%), and hypercholesterolemia (8%).[3] The clinical presentation of aortic occlusion includes claudication as the most prevalent symptom (in 81% of reported cases), followed by pain at rest (25%) and tissue loss (15%).[3] In patients with atherosclerosis-associated chronic total occlusion of the infrarenal aorta and claudication, approximately half present with buttocks pain as a primary symptom, and one quarter have calf pain.[4] Several investigators have reported claudication in relation to severe infrarenal aortic stenosis or occlusion[2-4]; however, only one study mentions a possible correlation with dyspnea.[5] To our knowledge, our patient is the first to display a relatively clear symptomatic relationship between infrarenal aortic disease and shortness of breath.

A few theories support this conclusion. Severe hypertension caused by aortic occlusion and renal artery stenosis could have produced flash pulmonary edema and shortness of breath in our patient. However, the angiographic findings and pressure gradients failed to confirm renal artery stenosis. Regardless, middle aortic syndromes without concomitant renal artery stenosis have been known to create hypertension proximal to the aortic stenosis and can manifest themselves with early fatigue on exertion.[6] Another, less likely, explanation is dyspnea caused by metabolic acidosis. This patient routinely attempted exercise, which in the presence of her infrarenal abdominal aortic stenosis could have induced transient muscle ischemia and a buildup of lactic acid, especially in the absence of adequate collateral circulation.[7] Subsequent metabolic acidosis could have resulted in subjective dyspnea because of an increased compensatory ventilation rate—a situation previously noted in a 73-year-old woman with chronic subtotal occlusion of the infrarenal abdominal aorta after it became occluded.[5] Finally, aortic atherosclerosis is known to reduce circulatory compliance and increase peripheral resistance.[8] Perhaps our patient's focal aortic occlusion impeded blood flow, further elevated systemic vascular resistance, and limited the augmentation of cardiac output in response to dynamic exercise—causing symptoms of poor exercise tolerance, including shortness of breath.

Perhaps the finding of exertional dyspnea has not been previously well established because patients tend to be so physically limited by claudication that their dyspnea is not recognized. Patients with symptomatic infrarenal atherosclerotic aortic lesions have classically been treated surgically with either endarterectomy for focal disease or bypass grafting for more extensive disease. Endovascular intervention and primary stenting have more recently been recommended as first-line treatment in selected patients.[2] Increased awareness of the possible connection between infrarenal aortic stenosis and dyspnea might reveal a more prevalent phenomenon and guide management when the clinician is unsure whether revascularization is indicated. Although our hypothesis merits further investigation, we think that this case report supports interventional treatment. This therapy enabled our patient to recover fully and to resume a previously active lifestyle.

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