Exertional Dyspnea Due to Iliac Vein Occlusion, Treated by Recanalization

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ABSTRACT: Exertional dyspnea has many causes. We report a case of exertional dyspnea resulting from preload insufficiency due to inadequate venous return. This occurred after a deep vein thrombosis resulted in chronic bilateral iliac vein occlusions. It was managed with endovascular iliac vein recanalization resulting in symptom improvement.

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A 41-year-old woman with a history of rotator cuff repair and cholecystectomy underwent neck fusion surgery for degenerative joint disease. She developed a left leg deep vein thrombosis (DVT) 3 days later. Anticoagulation was relatively contraindicated relatively early in the postoperative period. An Option Elite nitinol temporary inferior vena cava (IVC) filter (Argon) was placed. She unfortunately developed a pulmonary embolism (PE) within another 2 days without hemodynamic compromise. Anticoagulation was commenced 2 weeks later. A computed tomography (CT) study revealed bilateral iliac vein thrombosis.

Eight months after the index event, she was referred to our institution for removal of her IVC filter as well as evaluation of exertional dyspnea. In addition, she reported left leg aching with standing and mild bilateral lower-extremity edema. She wore compression stockings, which helped reduce the edema. CT venography revealed bilateral common iliac vein occlusion extending into the iliac confluence.

The patient reported that since the DVT and PE she experienced marked exertional dyspnea and exercise intolerance. Before the event, she could use the treadmill three times a week. Since the event, she could no longer do so. The mere act of walking up the stairs made her feel as if "she was going to die." In addition, she reported resting heart rates between 90-100 beats/min, which would rise to 190 beats/min within 30 seconds of walking.

Her physical examination was mostly unremarkable: resting heart rate, 94 beats/min; blood pressure, 124/81 mm Hg; respiratory rate, 14 breaths/min; temperature, 36.9 °C; saturations, 98% on room air; and body mass index, 27 kg/m^2. On auscultation there were no murmurs. The lungs were clear. There was trace edema around both ankles without pigmentation. Pedal pulses were 2+ bilaterally with brisk capillary refill.

To rule out chronic thromboembolic pulmonary hypertension (CTEPH), a ventilation/perfusion V/Q scan was obtained that showed no perfusion defects or mismatch. A CT angiogram revealed no residual pulmonary artery thrombus. Transthoracic echocardiography revealed normal systolic and diastolic function, no valvulopathy, and normal right ventricular systolic pressure of 25 mm Hg. Right heart catheterization revealed normal right-sided and left-sided filling pressures (3 mm Hg and 10 mm Hg, respectively) and normal mean pulmonary artery pressure (18 mm Hg). Exercise treadmill testing revealed no ischemia.

Pulmonary function testing (PFT) was performed to evaluate other potential pulmonary causes of exertional dyspnea; the findings were unremarkable.

A 6-minute walk test was carried out. She was able to walk 360 meters with a Borg Dyspnea scale of 5 (severe dyspnea). Based on her gender, height, weight, and age, she walked 61% of the expected distance. Her symptoms did not improve despite attempts to exercise and increase her cardiovascular endurance.

As there was no history of pulmonary disease or CTEPH and since the echocardiogram, right heart catheterization, and PFT did not shed light on the etiology of the dyspnea, we considered the possibility of preload insufficiency. The patient had bilateral iliac vein occlusions and we postulated that adequate venous return to the right ventricle was being compromised.

We decided to intervene on the iliac venous occlusive disease. During the first part of the procedure, using a transjugular approach, the IVC filter was removed with a retrieval device (Cook Medical).

Subsequently, using bilateral common femoral vein access, we infused tissue plasminogen activator at a total rate of 1 mg/hr for 24 hours through the sheaths. The following day, the right common iliac vein was recanalized. Intravascular ultrasound was used to guide balloon angioplasty and to deploy a 14 x 90 mm self-expandable Wallstent (Boston Scientific) from the right common iliac vein extending slightly into the distal IVC (Figures 1 and 2). Due to the prolonged duration of the procedure, the left iliac vein was not intervened upon. The patient was commenced on a direct-acting oral anticoagulant and discharged the following day.

At 2-week follow-up, she reported dramatic improvement of her exertional dyspnea as well as reduced bilateral leg edema. She began to go to the gym again. During a repeat 6-minute walk test performed 3 weeks post procedure, she completed 449 meters

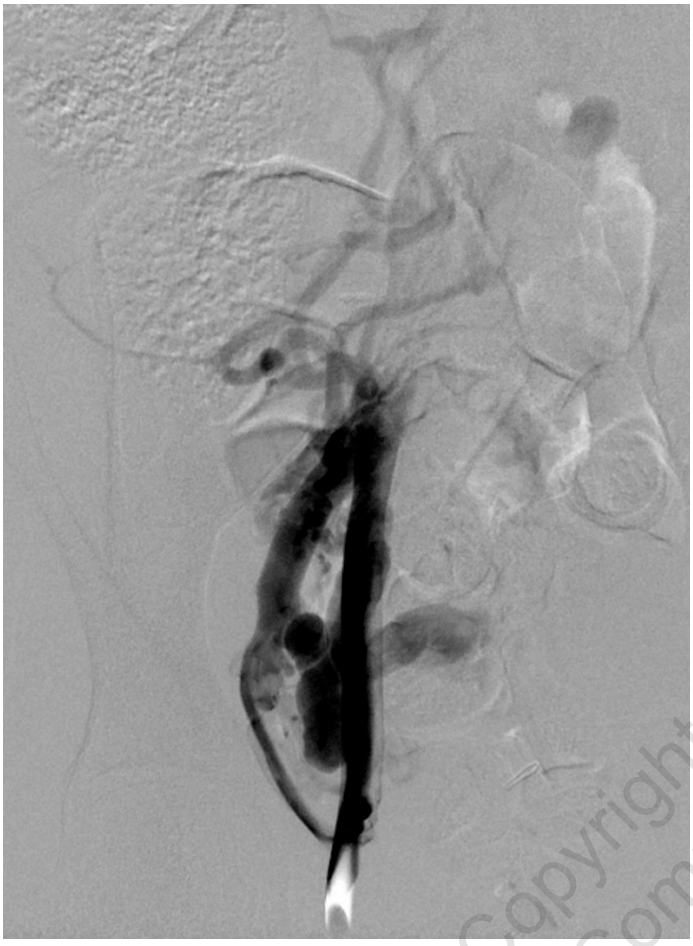

Figure 1. Venogram demonstrating right common iliac vein occlusion.

(76% of expected), which was an improvement from 360 meters before the procedure. Her Borg Dyspnea scale improved from 5 (severe) to 2 (slight). The patient remained asymptomatic at 6-month follow-up exam.

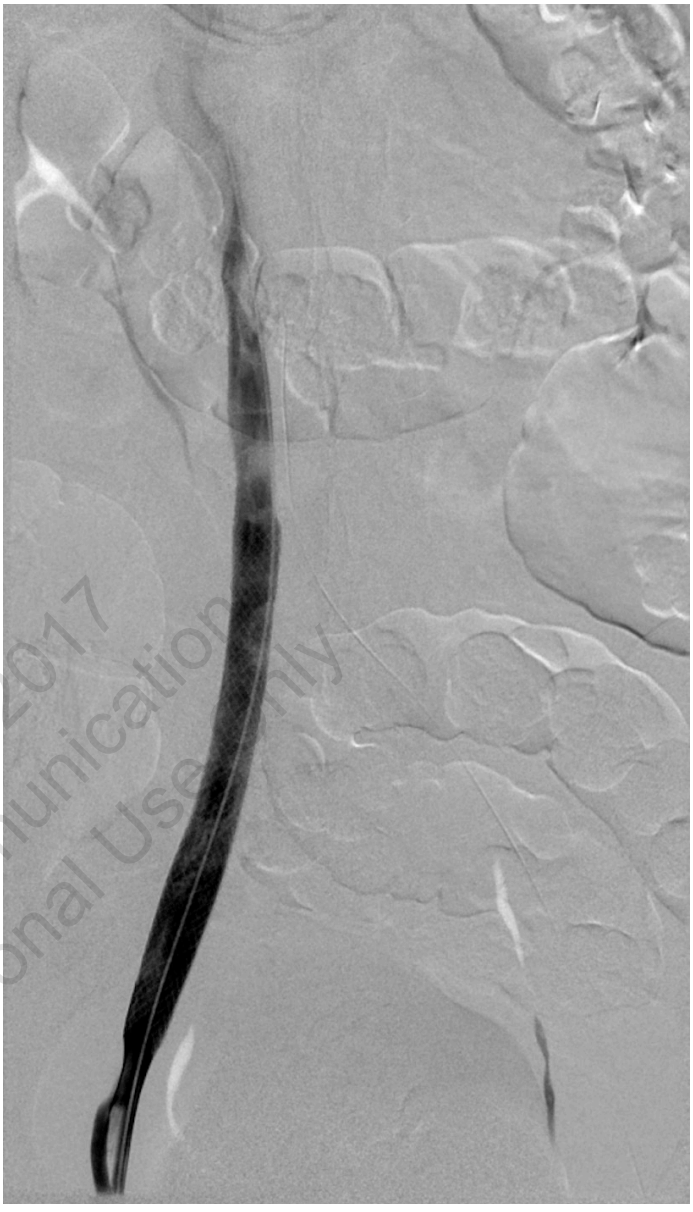

Figure 2. Venogram demonstrating restored flow through the right common iliac vein after recanalization and stenting.

DISCUSSION

Exertional dyspnea is a common complaint with many causes. In the case of our patient, it began after DVT/PE in the setting of neck surgery. The workup did not reveal evidence of CTEPH or cardiopulmonary disease.

In a database of patients referred for clinically indicated invasive cardiopulmonary exercise stress testing at Massachusetts General Hospital, a group of 49 patients with unexplained exercise tolerance was identified.[1] This group was found to have low maximum aerobic capacity due to inadequate peak cardiac output, with normal biventricular ejection fractions and without pulmonary hypertension. The group was compared to patients with a normal exercise response. The impaired group demonstrated significantly lower right-sided and left-sided filling pressures at peak exercise, despite initial intravenous fluid challenge. The group with unexplained exercise response also demonstrated decreased stroke volume augmentation with exercise (13 ± 10 mL/m^2 vs 18 ± 10 mL/m^2; P=.01). The authors suggested that low venous pressure leading to inadequate ventricular filling can be a cause for exercise intolerance. The mechanism, they postulated, may be inadequate venoconstriction of capacitance vessels. In the case of our patient, we postulated that bilateral iliac vein occlusion resulted in inadequate venous return to maintain adequate right ventricular filling during exertion. The patient did not note dyspnea at rest because the venous return to the right ventricle was adequate, through collaterals. However, the increased demands presented by exercise could not be met via the collaterals and the right ventricle was underfilled, leading to inadequate stroke volume. Tachycardia was likely a compensatory response.

After the DVT/PE, our patient was noted to be relatively tachycardic and her heart rate would rise briskly with minimal exercise. There was no record of this occurring previously. Patients with similar symptomatology may be labeled as having postural orthostatic tachycardia syndrome (POTS). Even though we are not suggesting POTS as a diagnosis for our patient, it merits mention here due to some characteristics that apply to this case.

Previous work has suggested that inadequate peripheral venoconstriction,[2] sympathetic dysautonomia,[3] or autonomic neuropathy[4] may contribute to POTS. There is also evidence, however, that low stroke volume and low venous return can be involved. In a study of 27 patients with the diagnosis of POTS, they had lower stroke volume and cardiac output compared with a control group.[5] Baroreflex and autonomic function were intact. Another study of patients with exaggerated changes in heart rate and intact sympathetic function used red blood cell tagging to suggest venous pooling in the legs as a possible mechanism.[6]

Similarly, Masuki et al[7] reported lower stroke volume among patients labelled as having POTS. This was noted at rest and with exercise. Heart rate, however, rose greater with the POTS group during exercise. In fact, the increase in stroke volume was inversely proportional to the increase in heart rate. This may suggest that the increase in heart rate can serve as a compensatory mechanism for lower stroke volume.

Deconditioning alone was unlikely to explain our patient's symptoms. She made several attempts to exercise, but persistently found the slightest exertion to be challenging. Oldham et al[1] found that deconditioning was in fact likely to lead to elevated filling pressures, an observation echoed by Stickland et al,[8] where subjects with greater fitness demonstrated lower pulmonary artery wedge pressure and superior stroke volume and end-diastolic volume at peak exercise, suggesting improved diastolic function.

CONCLUSION

Adequate venous return is an integral component of the cardiovascular system. When compromised, it can have dramatic effects on exercise tolerance. To our knowledge, this is the first published case of exertional dyspnea from preload insufficiency associated with obstruction of (iliac) venous return treated successfully with endovascular revascularization. ■

Disclosure: The authors have completed and returned the ICMJE Form for Disclosure of Potential Conflicts of Interest. The authors report no financial relationships or conflicts of interest regarding the content herein.

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