May-Thurner Syndrome Resulting in Paradoxical Embolism

Frank Amico, MD; Harit Desai, DO; Vincent Varghese, DO; Jon C. George, MD
From the Deborah Heart and Lung Center, Browns Mills, New Jersey.

ABSTRACT: A 53-year-old female with a past medical history of hypertension, hyperlipidemia, asthma, and prior transient ischemic attack presented after an embolic cerebrovascular accident, and a tranesophageal echocardiogram detected a patent foramen ovale (PFO). We present herein a rare case of MTS resulting in paradoxical embolism through a PFO.

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A 53-year-old female with a past medical history of hypertension, hyperlipidemia, asthma, and prior transient ischemic attack (TIA) presented after an embolic cerebrovascular accident (CVA) requiring the use of thrombolytic therapy. The patient underwent a transesophageal echocardiogram that detected a 12-mm patent foramen ovale (PFO). Given her dramatic clinical presentation and prior TIA, a decision was made to treat the patient’s PFO with percutaneous closure for secondary stroke prevention.

She was brought to the cardiac catheterization lab and prepped in a sterile fashion. Right common femoral vein (CFV) access was obtained and a 12 Fr sheath was placed. Next, access was obtained in the left CFV and an 8 Fr sheath was placed. An Accunav intracardiac echocardiography (ICE) catheter (Siemens) was advanced through the left CFV but was met with significant resistance in the left common iliac vein (CIV) (Figure 1). Selective venogram of the left CIV confirmed a subtotal thrombotic occlusion of the CIV at the inferior vena cava (IVC) junction (Figure 2) consistent with May-Thurner syndrome. A glidewire

Figure 1. Fluoroscopy demonstrating inability to pass the intracardiac echocardiography catheter (arrow) through an obstructed left common iliac vein.
(Terumo) with a support catheter was advanced to cross the subtotal occlusion into the IVC and a caval venogram was performed to confirm intraluminal position (Figure 3).

The ICE catheter was then advanced via the left CFV into the right atrium and positioned to visualize the interatrial septum as well as the PFO. After crossing the PFO with a glidewire and exchanging for an Amplatzer stiff wire, a 25 mm HELEX occluder (Gore) was advanced through the sheath and deployed across the PFO with minimal residual flow through the PFO.

A 5 Fr Omni Flush catheter (Angiodynamics) was advanced through the right CFV and positioned at the left CIV-IVC junction. A 12 mm x 20 mm Mustang balloon (Boston Scientific) was advanced through the left CFV and inflated to predilate the left CIV. A 14 mm x 40 mm Protégé stent (Covidien) was deployed in the IVC, extending across the lesion in the left CIV and postdilated using the same balloon with excellent venographic result (Figure 4). The patient tolerated the procedure well and was discharged home the following day with dual antiplatelet therapy for at least 3 months.

DISCUSSION

May-Thurner syndrome (MTS) is caused by compression of the left CIV between the overlying right common iliac artery (CIA) and the underlying vertebral body. It is also known as iliac vein compression syndrome and “Cockett syndrome.” This anatomic pattern can result in unprovoked left iliofemoral deep venous thrombosis (DVT) and has been reported to have a prevalence of 49% to 62% in patients with DVT. Generally, compression of CIV alone rarely causes
thrombus to form, but when combined with another exacerbating factor (e.g. tobacco use, hypercoagulable state, contraceptive pills, etc.), thrombus formation is more likely. The clinical stages of MTS have been described as follows: Stage 1, asymptomatic iliac vein compression; Stage 2, development of a venous spur; and Stage 3, development of iliac vein DVT.

Visualization of thrombus secondary to MTS can be difficult using conventional screening ultrasound. Physical examination may reveal a swollen thigh, pain, and venous claudication. If clinical suspicion is high, contrast venography, magnetic resonance imaging, or intravascular ultrasound imaging must be performed. May–Thurner syndrome is most common in females and primarily affects them between the second and fifth decade. Furthermore, it is more common in patients with reduced CIV diameters or severe iliac vein compression and occurs more commonly in the left CIV, which is attributed to compression of the left CIV by the right CIA. In a study of 2,576 patients with unilateral DVT, left-sided MTS was found in 55.9%.

Treatment of DVT in patients with MTS is two-fold: firstly, management of thrombosis with anticoagulation and thrombectomy; and secondly, treatment of the mechanical obstruction with angioplasty and stenting. However, patients with MTS can be poorly responsive to treatment with anticoagulation alone due to persistent mechanical obstruction, resulting in recurrent DVTs, thereby necessitating catheter-directed thrombolysis, venous angioplasty, and/or intravascular stenting. In a study by Jeon et al, patients who underwent catheter directed thrombolysis and stent placement had a 1-year primary patency rate of 83.3%. However, this study had limited power due to its small population of only 30 patients. A larger study of 982 patients showed an excellent stent patency rate of 79% after 5 years.

Long-term complications of MTS include post-phlebitic syndrome, especially in patients treated solely with anticoagulation. On the other hand, pulmonary emboli rarely occur in patients with this syndrome because the compression by the overlying artery acts as a natural mechanical barrier.

We present herein a rare case of MTS resulting in paradoxical embolism through a PFO.

Figure 4. Final venogram confirming excellent result (*) post stenting in the left common iliac vein.

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Address for correspondence: Jon C. George, MD, Director of Clinical Research, Division of Cardiovascular Medicine, Deborah Heart and Lung Center, 200 Trenton Road, Browns Mills, NJ, 08015, USA. Email: jcgeorgemd@gmail.com.

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