A 72-year-old man presented to the emergency department with a history suggestive of a transient ischemic attack. On physical examination, he had bilateral expiratory rhonchi and no residual neurological deficit. A chest radiograph, obtained on admission, did not reveal any obvious lung mass or nodule. As part of the evaluation protocol, a transesophageal echocardiogram (TEE) was performed to rule out a possible cardioembolic etiology. The TEE showed normal left ventricular systolic function, mildly dilated right ventricle with moderately elevated right ventricular systolic pressure, and a patent foramen ovale with a right-to-left shunt (Figure 1A). With color Doppler interrogation, a high-velocity flow with aliasing was noted in the right upper pulmonary vein (RUPV) (Figure 1B). By continuous wave Doppler, the peak velocity was noted to be 227 cm/s with a mean gradient of 14 mm Hg (Figure 1C), indicating hemodynamically significant stenosis of RUPV. The Doppler waveform showed continuous forward flow in RUPV into the left atrium (LA) with no reversal during atrial systole (Figure 1D), indicating a right-to-left shunt through the patent foramen ovale. The Doppler waveforms in the left lower pulmonary vein (LLPV) (Figure 1E) and left upper pulmonary vein (LUPV) (Figure 1F) showed normal propagated flow without any evidence of stenosis.

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left atrium throughout the cardiac cycle, including atrial systole. The other 3 pulmonary veins (PVs) showed normal velocities (Figure 1D to 1F). Further TEE evaluation of adjoining structures suggested compression of RUPV by an extracardiac mass (Figure 2A). A computed tomographic scan of the chest was subsequently obtained, which showed a right-sided mediastinal tumor compressing the RUPV (Figure 2B). A transbronchial biopsy followed by histological examination confirmed the diagnosis of small cell carcinoma. The patient was referred to the oncology service for further management.

A peak velocity $>100$ cm/s indicates PV stenosis. Hemodynamically significant PV stenosis is often associated with the loss of flow reversal during atrial systole. The common causes of PV stenosis include compression by tumor and narrowing after PV isolation. The increased right ventricular pressure resulting from PV hypertension may lead to a right-to-left shunt through the patent foramen ovale, which predisposes to paradoxical embolism. Our findings underscore the importance of routinely examining PV flow velocities during all clinically indicated TEE procedures.

Disclosures
None.

References

Figure 2. Representative TEE image showing a mass compressing the RUPV (A) and a computed tomographic image at the atrial level showing a mass (arrow) compressing the RUPV.
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