A 47-year-old woman with superior vena cava syndrome (SVCS) presented a metastatic small-cell lung cancer (SCLC). An initial computed tomography revealed a large tumor of the right lung, compressing the superior vena cava (Fig. 1A). A vena cava stent was inserted at the time of diagnosis and the patient was sent for chemotherapy. After the patient was administered four courses of chemotherapy by cisplatin and etoposide, the computed tomography revealed a partial tumor response (Fig. 1B) and migration of the stent into the left pulmonary artery (Fig. 2). Because of the poor oncological prognosis and the asymptomatic character of the migration, surgery was not indicated. Anticoagulation treatment was challenged because of the risk of hemorrhage. The patient died 3 months later because of a hepatic encephalopathy.

Migration of an endoprosthesis can lead to potentially serious complications and is estimated at 3%1,2 Some authors suggest that the vena cava stent should be avoided in patients with SCLC because of the chemo- and radiosensitive nature of this histologic type of cancer, which increases the risk of stent migration because of rapid tumor shrinkage under treatment.1 In some cases of acute SVCS spectacular clinical results could be observed during the 24 to 48 hours after insertion of the stent, whereas the efficacy of chemotherapy or radiotherapy

FIGURE 1. A, Initial chest CT scan showing vena cava stenosis caused by a mediastinal mass (arrow). B, Chest CT scan showing a normal diameter of vena cava after four courses of chemotherapy (arrow). CT, computed tomography.
was not observed until 3 to 4 weeks after.\textsuperscript{2,3} The insertion of a vena cava stent must be avoided in SVCS caused by SCLC.\textsuperscript{2}

**REFERENCES**


**Erratum**

**Overexpression of EPH Receptor B2 in Malignant Mesothelioma Correlates with Oncogenic Behavior: Erratum**

We would like to point out a methodologic error regarding peptide sc-1763P from Santa Cruz which we used for part of our EphB2 functional assays. SC-1763P is, in reality, not an EphB2 blocking peptide, and in fact is sold as a control to block the anti-EphB2 antibody sc-1763P. Hence, based on the information available on the sc-1763P peptide the effects we observed in the experiments shown in Fig. 5 are nonspecific effects and should not be attributed to inhibiting EphB2. Our shRNA data in the manuscript, however, will stand as proof of principle for the demonstration of the functional events associated with EphB2 in mesothelioma. Our gratitude to Dr. Elaine Pasquale, Professor at the Sanford-Burnham Medical Research Institute in La Jolla and an internationally renowned ephrin expert, for pointing out this error. Harvey I. Pass, MD Chandra Goparaju, PhD NYU Langone Medical Center New York, NY

Reference: