A previously healthy 24-year-old woman presented with shortness of breath. Chest radiograph (Fig. 1A) showed complete right upper and lower (RUL) lobe collapse with leftward mediastinal shift and abrupt cutoff of the right mainstem bronchus suggestive of an obstructing lesion. Chest computed tomography (CT) (Fig. 1B, C) revealed a 9-cm RUL mass (white arrowheads) with endobronchial invasion and suspected mediastinal involvement (black arrows). Pathology from bronchoscopic cytology (Fig. 2) yielded translocation-positive Ewing sarcoma. Positron emission tomography-CT showed hypermetabolic osseous foci compatible with metastases (Fig. 1D, E) and a hypermetabolic RUL mass extending toward the left atrium (LA) (Fig. 1F, G). An 18-month combination chemotherapy of cyclophosphamide–doxorubicin–vincristine/ifosfamide–etoposide was started to downsize the tumor before a possible surgical resection. To clarify possible tumor invasion into the heart, echocardiography was performed, identifying a 2.8 × 2.2 cm mass (Fig. 1H) entering the LA through the pulmonary vein. The differential diagnosis was thrombus versus invading tumor. For further clarification, cardiovascular magnetic resonance (CMR) imaging was performed, revealing a well-circumscribed heterogeneous RUL mass with areas of necrosis and hemorrhage (Fig. 1I). The mass invaded the right hilum, obstructed the right mainstem bronchus, and grew into the LA through the right superior pulmonary vein (Fig. 1I, black arrow). In addition, comparison between coronal positron emission tomography-CT (Fig. 1F) and CMR (Fig. 1I) revealed additional tissue apposed to the hypermetabolic tumor thrombus. Early post-contrast CMR confirmed the presence of a bland LA thrombus (Fig. 1J, black arrow), adherent to the enhancing tumor thrombus (white arrowhead). On the basis of the findings obtained by CMR, the patient was immediately placed on anticoagulation therapy.

The Ewing sarcoma family of tumors has a neural crest cell origin. Most of these tumors exhibit a translocation between chromosomes 11 and 22.1 The majority of tumors typically affect the skeleton. Rare manifestations, however, have been reported in several extraosseal tissues.1 Primary pulmonary Ewing tumor is an extremely rare manifestation of the extraosseous Ewing sarcomas (EES). As of today, the number of primary pulmonary EES cases reported in literature is less than 15, including only one case of primary pulmonary EES invading the heart.2 Our case report highlights the importance of multimodality imaging for the diagnostic assessment of the primary tumor, tumor direct invasion, and metastasis and emphasizes the role of CMR in the differential diagnosis of intracardiac tumor mass and thrombus.

REFERENCES
FIGURE 1. Representative chest radiograph (A, anteroposterior view), CT (B and C, coronal and four-chamber views), PET-CT (D–G, axial and coronal views), echocardiography (H, four-chamber view), and cardiac MRI (I and J, coronal and four-chamber views) images are shown. CT, computed tomography; PET, positron emission tomography; MRI, magnetic resonance imaging.
FIGURE 2. Photomicrographs of the H&E-stained cell-block sample (upper panel, ×40 magnification) and Papanicolaou-stained bronchoscopic cytology sample (bottom panel, ×40 magnification) are shown. The H&E slide demonstrates a “small round blue cell” tumor with dark nuclei and minimal cytoplasm consistent with Ewing sarcoma cells. The Papanicolaou-stained slide shows better morphology and nuclear characteristics with central nucleoli and a rare mitosis. The characteristic cytologic finding in Ewing sarcomas is a dimorphic cell population; one being larger with a more pale nucleus (black arrow, pink/clear vacuoles in the pale cytoplasm) and the second with smaller size and darker nucleus (surrounding the pale cells).1 H&E, hematoxilyn–eosin.