A 46-year-old never-smoker man presented to the emergency room with a 4-day history of progressive cough and right-side chest wall pain in October 2011. A computed tomography scan of his chest showed large anterior mediastinal and right pleural-based masses associated with pleural effusion. The tumor was confirmed to be squamous cell carcinoma by percutaneous gun biopsy of the right pleural-based mass (Fig. 1A). The tumor cells expressed KIT (Fig. 1B) and p63 proteins, and the patient was diagnosed with thymic cancer. Even after two cycles of paclitaxel/cisplatin chemotherapy and a subsequent line of cyclophosphamide/doxorubicin/cisplatin chemotherapy, his pleural effusion increased and the primary anterior mediastinal mass progressed. He required frequent thoracentesis to relieve dyspnea. At that time, we evaluated whether the initially biopsied tumor specimen harbored KIT mutation, and identified a mutation in exon 11 (loss of aspartic acid at position 579: D579del).

After administration of imatinib (400 mg daily) since December 2011, the tumorous lesion has decreased to the range of partial response by the Response Evaluation Criteria in Solid Tumors. Positron emission tomography–CT scans at the same time points also showed decreased FDG uptake in the corresponding lesions of the anterior mediastinum (C) and pleura (D). CT, computed tomography; FDG, fluorodeoxyglucose.
in Solid Tumors (Fig. 2). The patient is still on imatinib (total 15 months) and is in good general condition.

DISCUSSION

Although KIT protein expression is prevalent in thymic neoplasms, KIT mutation has rarely been observed.\textsuperscript{1,2} Since the first report of KIT mutation and tumoral response to imatinib in thymic cancer in 2004, a few similar cases have been reported.\textsuperscript{3,4}

In this case, we demonstrated the presence of a novel somatic deletion mutation involving the proto-oncogene KIT in thymic cancer. The patient currently shows a prolonged partial response to imatinib with no significant toxicity. Among the many KIT exon 11 mutations, a deletion in codon 579(Asp) in the juxtamembrane region of the KIT gene was reported in a gastrointestinal stromal tumor,\textsuperscript{5} but has never been reported in thymic cancer. As in our case, that tumor was reported to show a good response to imatinib therapy.

To date, there have been few treatment options for metastatic thymic carcinoma, and its outcome is generally disappointing. In agreement with previous reports, our study suggests that the KIT pathway can be an important driving oncogenic pathway and can be a useful target of molecular therapy such as imatinib.

REFERENCES