A 64-year-old man was diagnosed with a large tumor of the anterior mediastinum (Fig. 1), which, by ultrasound-guided needle aspiration, was initially characterized as an atypical carcinoid (neuroendocrine tumor [NET]; mitotic count 2-20 per 10 high power fields and/or 3-20% Ki67 index [G2 (NET G2)]; MIB1-Proliferations Index 3%-8%, positive chromogranin A). The patient was referred to our department for further investigation and surgical resection. The resection of the tumor was performed in January 2011. The left phrenic nerve, both mammary arteries, the brachiocephalic vein, and a part of the pericardium were engulfed by the tumor and were removed. A complete dissection of the tumor from the superior vena cava was not possible, so a very small amount of tumor tissue was inevitably left at the junction of the excised brachiocephalic vein with the superior vena cava. Because of the R2 resection and the chronic renal insufficiency only additional radiotherapy was suggested. The patient having completed the suggested radiotherapy (40Gy) remains without recurrence of the tumor or metastases at 15 months after surgery. Macroscopically, the tumor was a white-yellowish, partly encapsulated, soft, elastic nodular mass and weighed 1770 g. The cut surface showed lobules with hemorrhage and myxoid as well as cystic areas. Microscopic core features are characterized in Figure 2.

Case reports of adult neuroblastoma (ANB) occurring in nonpediatric patients more than 18 years of age are rare, with less than 50 published cases. To our knowledge only 21 cases of mediastinal ANB, including three thymic neuroblastomas, have been reported. ANB does not show a sex predilection. The site of the primary tumor purely reflects the distribution of the sympathetic nervous system, with the distribution being similar among adults and children. In our case the tumor might originate somewhere along the course of the sympathetic aortic plexus or the phrenic nerve. A thymic origin of the neuroblastoma could not be demonstrated. Alternatively, mediastinal ANB can originate from a preexisting mediastinal teratoma, which also could not be demonstrated. Figure 3 gives a synopsis over the immunohistochemical differential diagnosis. Because of the positive staining for Chromogranin A, these tumors can be mistaken for carcinoid tumors. However, the different cellular structure, the necrosis, and the mitotic/proliferation index, as well as immunohistochemical panel usually helps avoiding a misdiagnosis. Tumors of this size are in general inoperable especially without prior treatment. The false initial diagnosis as atypical carcinoid lead us to an attempt of operative resection. In spite of the R2 resection and after adjuvant radiotherapy the patient remains tumor free 15 months after surgery. There is no consensus regarding the treatment of ANB. Some centers apply similar protocols for the treatment of the tumor in children and adults. Treatment options include surgical resection, radiotherapy, or chemotherapy and the most active therapeutic agents used alone or in combination include cyclophosphamide, cisplatin, doxorubicin, carboplatin, and ifosfamide. Regarding prognosis and survival, there is a trend of improvement in survival in recent decades, which may reflect advances in treatment options. Patients with localized disease or regionally advanced disease had higher survival rates than those with advanced metastatic disease.

REFERENCES
FIGURE 1. Magnetic resonance imaging (A, B) and posteroanterior chest radiograph (C) demonstrating an extensive tumor mass occupying the anterior mediastinum. The tumor extends from the upper thoracic aperture cranially almost to the diaphragm caudally (A), with shifting of the major vessels posteriorly (B). Postoperative posteroanterior chest radiograph after successful tumor resection (D).

FIGURE 2. A, Tumor overview (hematoxylin and eosin staining; red bar = 100 μm): extended areas of necrosis in the center of the tumor. Parts of the connective tissue capsule and some fat tissue in the periphery. B, Tumor (higher magnification; hematoxylin and eosin staining): relatively small tumor cells with hyperchromatic nuclei are partially arranged in single files. A neuropil-like matrix can be noted in the background embedding the tumor cells. C, Immunohistochemistry (synaptophysin): positive reaction of the neuropil-like matrix, but not of the tumor cells, vessels, the fibrous capsule, and adipose tissue. D, Immunohistochemistry (neuron-specific enolase): faint reactivity of the neuropil-like matrix and a weak positivity of the tumor-cell cytoplasmics. E, Immunohistochemistry (chromogranin A): strong reactivity of the neuropil-like Matrix, but not of the tumor cells, vessels, the fibrous capsule, and adipose tissue. Focally, tumor invasion of the surrounding adipose tissue is observed (arrow). F Immunohistochemistry (CD 56): strong reactivity of the neuropil-like matrix and single tumor cells.
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FIGURE 3. Immunohistochemical profile of neuroblastoma and possible differential diagnoses. (Annotation: “−” negative, “+” positive, “±” variable positive or negative [“−” to “++++” describes the relative quantity of cases featured in the literature but not the strength of staining]). NSE, neuron specific enolase; PNET, primitive neuroectodermal tumor.