Malignant Brain Tumor With Rhabdoid Features in an Adult
—Case Report—

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Abstract

Rhabdoid tumor (RT) of the central nervous system is an uncommon and aggressive neoplasm that usually affects pediatric patients. Currently, these tumors are classified as malignant RT or atypical teratoid/RT. Another entity of intraparenchymal brain tumor with a rhabdoid component is the extremely rare rhabdoid glioblastoma. A 23-year-old woman presented with a malignant RT in the right thalamus. The tumor was adjacent to the right lateral ventricle and was partially resected. Histological examination revealed prominent proliferation of rhabdoid cells, which is consistent with a diagnosis of malignant RT; the typical features of glioblastoma were not observed. The tumor cells stained positively for integrase interactor–1 and glial fibrillary acidic protein. Therefore, the tumor may have originated from glial components. Genetic analysis using comparative genomic hybridization showed a deoxyribonucleic acid copy-number gain on chromosome 7 but not on chromosome 22. The tumor did not respond to chemotherapy or radiotherapy, and the patient survived for only 4 months after surgery. The present case of malignant RTs shows certain similarities with those of rhabdoid glioblastoma. Further accumulation and analysis of data, including data from genetic analyses, may lead to the identification of a new type of malignant RT.

Key words: intracerebral malignant rhabdoid tumor, atypical teratoid/rhabdoid tumor, adult, comparative genomic hybridization, rhabdoid glioblastoma

Introduction

Rhabdoid tumors (RTs) are aggressively malignant neoplasms of unknown histogenesis that occur in children and infants.45) RT was first described in the kidney, and later RTs arose from various organs and tissues, including soft tissue, the liver, and the central nervous system (CNS).29) A subtype of RT originates in the CNS and occurs in infancy and childhood.29) This subtype consists of classical RT tissue mixed with primitive neuroectodermal tumor (PNET) tissue, epithelium, and neoplastic mesenchyme.29) These tumors are named atypical teratoid/rhabdoid tumors (AT/RTs) based on the disparate combination of rhabdoid, primitive neuroepithelial, epithelial, and mesenchymal components.30,42) Nearly 200 cases of CNS AT/RTs have been reported, and most patients were less than 3 years old at the time of diagnosis.5,25) Only 25 adult cases of cerebral RTs, including AT/RTs and malignant RTs, have been reported.1,2,8,10,13–15,19,23,24,27,31,32,38,39,41,42,45) Only 6 cases of epithelioid glioblastoma with a rhabdoid component have been reported.16,26,28,47) However, the World Health Organization (WHO) Classification of Tumours of the Central Nervous System classifies AT/RTs as embryonal tumors, but does not list malignant RT and rhabdoid glioblastoma as distinct categories.30)

We describe a case of malignant RT in the brain in an adult which fulfilled the diagnostic criteria for a rhabdoid glioblastoma.

Case Report

A 23-year-old woman presented with a 1-month history of headache and nausea. Neurological examination revealed sensory disturbance on the left side of the body. Computed tomography and magnetic resonance (MR) imaging showed that the tumor was located in the right thalamus adjacent to the right lateral ventricle wall, contained calcification, and was heterogeneously enhanced after the administration of gadolinium (Fig. 1A). Partial resection of the tumor was performed via a right parietal corticotomy under neuronavigation guidance. The tumor was soft, reddish, hemorrhagic, and exposed to the lateral ven-
The well-defined nodular mass contained moderately vascular and soft-solid areas.

Histological examination of the tumor specimens revealed that the neoplasm mainly consisted of sheets or nests of ovoid cells with abundant pale eosinophilic cytoplasm and ovoid or convex eccentrically located nuclei with prominent nucleoli, the so-called rhabdoid cells, with no additional primitive neuroectodermal, epithelial, or mesenchymal components. The tumor cells adhered to the walls of the blood vessels and formed a pseudopapillary structure. The connections between the cells were fragile. The amount of chromatin in the nuclei was increased. Apoptotic cells, cells undergoing karyokinesis, and giant cells were found (Fig. 1B, C). The tumor cells were positive for epithelial membrane antigen (EMA) (DAKO Denmark A/S, Glostrup, Denmark), vimentin (DAKO Denmark A/S), glial fibrillary acidic protein (GFAP) (DAKO Denmark A/S), α-smooth muscle actin (DAKO Denmark A/S), S-100 protein (DAKO Denmark A/S), and neurofilament (DAKO Denmark A/S) (Fig. 2A–F). The labeling index for Ki-67 (DAKO Denmark A/S) was 30% (Fig. 2G). The tumor tissue was also positive for integrase interactor-1 (INI-1) protein (BD Transduction Laboratories, Franklin Lakes, New Jersey, USA) (Fig. 2H). Genetic analysis by comparative genomic hybridization (CGH) was performed as described elsewhere. The tumor showed various deoxyribonucleic acid (DNA) copy-number aberrations, including gains on chromosomes 3, 7, 9, 12, 17q, and 21q and a loss on 14q; no detectable loss of genetic material on chromosomal arms 3, 7, 9, 12, 17q, and 21q; no detectable loss of genetic material on chromosomes 3, 7, 9, 12, 17q, and 21q; no detectable loss of genetic material on chromosome 17q. The tumor showed various deoxyribonucleic acid (DNA) copy-number aberrations, including gains on chromosomes 3, 7, 9, 12, 17q, and 21q and a loss on 14q; no detectable loss of genetic material on chromosomes 3, 7, 9, 12, 17q, and 21q; no detectable loss of genetic material on chromosome 17q. 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Fig. 3 Graphical profiles of comparative genomic hybridization. The chromosome number is shown under the appropriate idiogram. The mean (red line) and standard deviation (yellow lines) of the fluorescence intensity ratios for the indicated chromosomes are shown; $n$ is the number of metaphase spreads from which data were collected. The thick green lines to the right of each chromosome idiogram show regions with increased deoxyribonucleic acid (DNA), and the thick red lines to the left show regions of relative loss and copy-number aberrations. The tumor showed various DNA copy-number aberrations (gains on chromosomes 3, 7, 9, 12, 17q, and 21q and a loss on 14q), but no loss on chromosome 22. The study did not reveal any abnormality in chromosome 22q, which contains the integrase interactor-1 gene.

After the histological diagnosis was established, the patient was treated with a chemotherapeutic regimen that included ifosfamide (2 g/m², 1 day), etoposide (100 mg/m², 3 days), and carboplatin (450 mg/m², 3 days). Only 1 course of chemotherapy was used, because the lesion progressed rapidly, and the patient’s condition deteriorated. Corticosteroid treatment did not improve the symptoms. External beam radiation therapy of 40 Gy was planned in 20 fractions, but was suspended after 50% of the scheduled dose was administered because the patient’s general condition deteriorated. The patient developed hydrocephalus 1 month after the first surgery and underwent 4 shunt operations because of repeated tube occlusion by tumor cells. MR imaging 1 month after the first surgery showed that the tumor had regrown to its original size at the time of the surgery and had disseminated to the posterior fossa (not shown). The patient died approximately 4 months after the diagnosis. Autopsy was not performed.

Discussion

The WHO Classification of Tumours of the Central Nervous System (2007) describes AT/RT as consisting of rhabdoid cells and often PNET tissue, epithelium, and neoplastic mesenchyme, and is almost always associated with INI1/hSNF5 inactivation. Mutation or loss of the INI-1 locus at 22q11.2 is the genetic hallmark of AT/RT. This tumor can occur sporadically or as part of RT predisposition syndrome (RTPS). Moreover, according to the WHO, a germline INI-1 mutation is diagnostic of RTPS. RTPS is characterized by a markedly increased risk of developing malignant RTs due to the constitutional loss or inactivation of the 1 allele of the INI-1 gene.30

RTs in adults, including malignant RTs and AT/RTs in the CNS, are characterized by diffuse growth of rhabdoid cells with areas of alveolar or trabecular arrangement of cells and an angiocentric pattern, with infiltration along blood vessels.13 These tumors generally contain a mixture of undifferentiated round polygonal or spindle-shaped cells, and cells with typical paracellular, glassy eosinophilic inclusions. The nuclei are eccentric and vesicular, with occasional prominent nucleoli, and mitosis is common.15,31,45 Rhabdoid cells are almost always found to express vimentin, EMA, and cytokeratin by immunohistochemical analysis.15,31 The presence of vimentin-positive cytoplasmic globular inclusions and membranous EMA reactivity are characteristic features of RTs at any site. Adult RTs may also be positive for S-100 protein, GFAP, cytokeratin,11,22 and α-smooth muscle antigen, and negative for desmin, neurofilament, and myoglobin.11 In our patient, the tumor specimen was positive for vimentin, EMA, α-smooth muscle actin, and negative for desmin, neurofilament, and myoglobin.11 In our patient, the tumor specimen was positive for vimentin, EMA, α-smooth muscle actin, and negative for desmin, neurofilament, and myoglobin.11 In our patient, the tumor specimen was positive for vimentin, EMA, α-smooth muscle actin, and negative for desmin, neurofilament, and myoglobin.11 In our patient, the tumor specimen was positive for vimentin, EMA, α-smooth muscle actin, and negative for desmin, neurofilament, and myoglobin.11 In our patient, the tumor specimen was positive for vimentin, EMA, α-smooth muscle actin, and negative for desmin, neurofilament, and myoglobin.11 In our patient, the tumor specimen was positive for vimentin, EMA, α-smooth muscle actin, and negative 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doid features. Rhabdoid meningioma corresponds to WHO grade III and shows only focal rhabdoid features. We considered that the tumor in the present patient was distinct from rhabdoid meningioma because of the intramedullary location.

Primary brain tumors that transform to a rhabdoid phenotype exhibit the immunohistochemical characteristics specific to the primary tumor. Since the present tumor was diffusely positive for GFAP, the origin could have been glial cells. Rhabdoid glioblastoma cells have not only rounded cytoplasmic contours but also prominent cytoplasmic filaments. The cell nuclei are large, eccentric, and contain prominent nucleoli. These tumors express neuronal markers, vimentin, α-smooth muscle actin, and EMA. In the present case, the diagnosis of RT was established because the typical features of glioblastoma were not observed and because the definition of rhabdoid glioblastoma is unclear. The tumor cells were positively stained for GFAP in 5 of 25 reported cases of RT in adults, suggesting rhabdoid glioblastoma.

Table 1 Summary of cases of rhabdoid glioblastoma

<table>
<thead>
<tr>
<th>Author (Year)</th>
<th>Age (yrs)/Sex</th>
<th>Location</th>
<th>Surgery</th>
<th>Chemo-therapy</th>
<th>Radio-therapy</th>
<th>IHC</th>
<th>Genetic analysis</th>
<th>Survival</th>
</tr>
</thead>
<tbody>
<tr>
<td>Wyatt-Ashmead et al. (2001)</td>
<td>18/male</td>
<td>rt F</td>
<td>3 times, PR</td>
<td>yes (NA)</td>
<td>yes (NA)</td>
<td>vimentin, EMA, cytokeratin (AE1/AE3)</td>
<td>monosomy ch 22 (NA)</td>
<td>5 mos</td>
</tr>
<tr>
<td>Lath et al. (2003)</td>
<td>16/female</td>
<td>rt F-T</td>
<td>TR</td>
<td>yes (NA)</td>
<td>yes (NA)</td>
<td>vimentin, EMA, keratin</td>
<td>–</td>
<td>3 mos</td>
</tr>
<tr>
<td>Fung et al. (2004)</td>
<td>66/male</td>
<td>rt T</td>
<td>done (NA)</td>
<td>no</td>
<td>no</td>
<td>vimentin, EMA, GFAP, SMA</td>
<td>polysomy ch 22 (FISH)</td>
<td>short (NA)</td>
</tr>
<tr>
<td>Perry et al. (2005)</td>
<td>36/male</td>
<td>NA</td>
<td>NA</td>
<td>NA</td>
<td>NA</td>
<td>NA</td>
<td>–</td>
<td>NA</td>
</tr>
<tr>
<td>Kleinschmidt-DeMasters et al. (2006)</td>
<td>20/female</td>
<td>NA</td>
<td>NA</td>
<td>NA</td>
<td>NA</td>
<td>NA</td>
<td>polysomy ch 7 (FISH)</td>
<td>NA</td>
</tr>
<tr>
<td>18/male</td>
<td>NA</td>
<td>NA</td>
<td>NA</td>
<td>NA</td>
<td>NA</td>
<td>NA</td>
<td>–</td>
<td>22 wks</td>
</tr>
<tr>
<td>Present case</td>
<td>23/female</td>
<td>rt Thal</td>
<td>PR</td>
<td>IFO, CDDP, VP-16</td>
<td>22 Gy</td>
<td>vimentin, GFAP, EMA, S-100 protein, NFP, INI-1 (+)</td>
<td>normal ch 22 (CGH) gains on ch 3, 7, 9, 12, 17q, 21q; loss on 14q</td>
<td>4 mos</td>
</tr>
</tbody>
</table>

immediately recognizable, and a specific diagnosis, beyond that of a high-grade malignant neoplasm, is not attainable. Therefore, such tumors could have been diagnosed as malignant RTs. We compared previously reported cases of typical AT/RTs in adults with respect to the patients’ age, tumor location, incidence, presence of mutations and deletions in 22q chromosome, and patient survival (Table 2). Again, the definition of adult RT, including rhabdoid glioblastoma, is unclear; however, these rare tumors show features that are extremely distinct from pediatric AT/RTs.

In conclusion, cumulative data including genetic aberrations other than INI-1 status or chromosome 22 alterations are required to better characterize tumors diagnosed as malignant RTs or rhabdoid glioblastomas; this information could lead to the identification of a new tumor entity.

**Disclosure**

The authors report no conflict of interest concerning the materials or methods used in this study or the findings specified in this paper.

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