Primary cervical melanoma with brain metastases

Case report and review of the literature

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Primary intramedullary melanoma is a very rare tumor that occurs most frequently in the middle or lower thoracic spinal cord. The authors present a case of primary cervical cord melanoma that developed in a 62-year-old man who was surgically treated and subsequently underwent radiation therapy. Clinical and histogenetic features of this neoplasm and results of chemo-, radio-, and immunotherapy are reported. Both “dysembryogenetic” and “mesodermal” hypotheses on the origin of primary spinal melanoma are discussed.

KEY WORDS • melanoma • spinal cord tumor • intramedullary lesion

Primary malignant melanoma in the central nervous system (CNS) accounts for approximately 1% of all cases of melanoma. Primary spinal melanoma is a rare tumor that was first reported by Hirschberg in 1906. Since then, only 35 cases have been reported, with the tumor occurring most frequently in the middle and lower thoracic spinal cord. A review of the 37 cases, including the present case of primary spinal melanoma, is detailed in Table 1.

To diagnose a primary intramedullary melanoma, Hayward’s criteria should be fulfilled: 1) no malignant melanoma, either primary or metastatic, should be present elsewhere outside the CNS; 2) nerve roots and the dura should not be involved by the tumor; and 3) the lesion should be confirmed on pathological studies. Primary spinal melanoma must be differentiated from primary melanotic tumor of the leptomeninges, usually known as meningeal melanocytoma, which is a benign lesion with a prolonged clinical course and which carries a better prognosis.

We present a case of primary intramedullary cervical melanoma. An angiogram of the spine obtained in our patient disclosed the presence of high vascularity and a remarkable circular blush of the lesion, which was unique because no such pathological circle has so far been described in the literature. The patient underwent surgical treatment followed by radiation therapy and survived for 15 months after the onset of symptoms, 1 month after brain metastases that arose from cerebrospinal fluid (CSF) dissemination were observed.

Case Report

History. This 62-year-old man was admitted to the Department of Neurosurgery in October 1996 complaining of mild neck pain, gradual onset of weakness in the left arm and leg, and “pins and needles” sensation in the left hand. The pain had progressively worsened 1 month before admission.

Examination. A neurological examination revealed spastic paresis with hyperreflexia of the left arm and sensory disturbances in the neck and proximally in both arms. The results of laboratory tests indicated no abnormalities. Magnetic resonance (MR) imaging of the cervical spine...
revealed an intramedullary lesion at the C-3 level, with a hyperintense signal relative to the spinal cord on both T1- and T2-weighted MR images. Intravenous injection of gadolinium diethylenetriaminepentaacetic acid clearly showed an enhanced higher signal intensity lesion on T1-weighted images (Fig. 1). The patient underwent angiography of the spine, which revealed a pathological circular blush with feeding vessels from the anterior spinal artery, visible through the left radicular artery positioned between C-4 and C-5 (Fig. 2).

**Operation and Pathological Examination.** On October 17, 1996 the patient underwent spinal surgery and a laminectomy was performed at C-3 via a posterior approach. At operation, the dura appeared to be intact. Once exposed, the spinal cord appeared normal on the surface, although it was swollen and enlarged pial arteries were visualized. No nerve root involvement or extramedullary extension were observed. After a midline cordotomy was performed, a soft vascularized blue-black mass was revealed within the spinal cord on the left side at the C-3 level.

### TABLE 1

<table>
<thead>
<tr>
<th>Case No.</th>
<th>Authors &amp; Year</th>
<th>Age (yrs), Sex</th>
<th>Duration of Symptoms</th>
<th>Site</th>
<th>Diagnosis Made</th>
<th>Metastases</th>
</tr>
</thead>
<tbody>
<tr>
<td>1</td>
<td>Hirschberg, 1906</td>
<td>67, F</td>
<td>3 mos</td>
<td>thoracic</td>
<td>no at autopsy</td>
<td>NR</td>
</tr>
<tr>
<td>2</td>
<td>Boit, 1907</td>
<td>51, M</td>
<td>11 mos</td>
<td>thoracic</td>
<td>no at autopsy</td>
<td>liver, spleen</td>
</tr>
<tr>
<td>3</td>
<td>Esser, 1907</td>
<td>32, M</td>
<td>15 days</td>
<td>thoracic</td>
<td>yes during life</td>
<td>&amp; at autopsy</td>
</tr>
<tr>
<td>4</td>
<td>Kawashima, 1910</td>
<td>26, F</td>
<td>7 mos</td>
<td>thoracic</td>
<td>no at autopsy</td>
<td>diffuse leptomeningeal dissemination</td>
</tr>
<tr>
<td>5</td>
<td>Lindbom, 1912</td>
<td>45, F</td>
<td>2 mos</td>
<td>cervical</td>
<td>no at autopsy</td>
<td>NR</td>
</tr>
<tr>
<td>6</td>
<td>Koelichen, 1916</td>
<td>25, M</td>
<td>18 mos</td>
<td>cervical</td>
<td>yes at autopsy</td>
<td>NR</td>
</tr>
<tr>
<td>7</td>
<td>Ringertz, 1926</td>
<td>61, F</td>
<td>6 mos</td>
<td>thoracic</td>
<td>yes at autopsy</td>
<td>brain not examined</td>
</tr>
<tr>
<td>8</td>
<td>Schmid, 1926</td>
<td>71, M</td>
<td>14 mos</td>
<td>thoracic</td>
<td>no at autopsy</td>
<td>small pigmented foci over cerebral dura mater</td>
</tr>
<tr>
<td>9</td>
<td>Bau-Prussak &amp; Mackiewicz, 1929</td>
<td>29, M</td>
<td>21 days</td>
<td>thoracolumbar</td>
<td>yes during life</td>
<td>NR</td>
</tr>
<tr>
<td>10</td>
<td>Bell, 1930</td>
<td>48, F</td>
<td>3 mos</td>
<td>cervicothoracic</td>
<td>yes during life</td>
<td>NR</td>
</tr>
<tr>
<td>11</td>
<td>de Blasi, 1930</td>
<td>71, F</td>
<td>6 mos</td>
<td>thoracic</td>
<td>yes at autopsy</td>
<td>brain not examined</td>
</tr>
<tr>
<td>12</td>
<td>Van Bogaert &amp; Verbrugge, 1933</td>
<td>38, M</td>
<td>6 mos</td>
<td>thoracic</td>
<td>yes during life</td>
<td>brain not examined</td>
</tr>
<tr>
<td>13</td>
<td>Schnitker &amp; Ayer, 1938</td>
<td>49, F</td>
<td>?</td>
<td>thoracic</td>
<td>yes at autopsy</td>
<td>lung, liver, uterus, diffuse leptomeningeal dissemination</td>
</tr>
<tr>
<td>14</td>
<td>Da Costa &amp; Love, 1939</td>
<td>55, F</td>
<td>24 mos</td>
<td>thoracic</td>
<td>yes during life</td>
<td>NR</td>
</tr>
<tr>
<td>15</td>
<td>Garcin, et al., 1941</td>
<td>52, M</td>
<td>21 days</td>
<td>lumbosacral</td>
<td>yes during life</td>
<td>NR</td>
</tr>
<tr>
<td>16</td>
<td>Mackay &amp; Hureau, 1942</td>
<td>32, F</td>
<td>10 mos</td>
<td>cervical</td>
<td>no at autopsy</td>
<td>lesion extending to lower medulla &amp; cervical spinal cord</td>
</tr>
<tr>
<td>17</td>
<td>Castaner Vendrell, et al., 1950</td>
<td>52, F</td>
<td>12 mos</td>
<td>lumbar</td>
<td>yes during life</td>
<td>NR</td>
</tr>
<tr>
<td>18</td>
<td>Forbes &amp; Maloney, 1950</td>
<td>57, M</td>
<td>?</td>
<td>thoracic</td>
<td>yes at autopsy</td>
<td>diffuse leptomeningeal dissemination</td>
</tr>
<tr>
<td>19</td>
<td>Kissel, et al., 1950</td>
<td>25, F</td>
<td>2 mos</td>
<td>cervical</td>
<td>yes during life</td>
<td>NR</td>
</tr>
<tr>
<td>20</td>
<td>De Assis &amp; De Luccia, 1951</td>
<td>26, M</td>
<td>7 mos</td>
<td>lumbar</td>
<td>yes during life</td>
<td>NR</td>
</tr>
<tr>
<td>21</td>
<td>King &amp; Probst, 1951</td>
<td>47, M</td>
<td>2 mos</td>
<td>lumbar</td>
<td>yes during life</td>
<td>nodules at base of brain &amp; ventricles ependyma, diffuse leptomeningeal dissemination</td>
</tr>
<tr>
<td>22</td>
<td>King, et al., 1952</td>
<td>53, M</td>
<td>12 mos</td>
<td>lumbar</td>
<td>yes during life</td>
<td>pigmented foci dura mater base brain</td>
</tr>
<tr>
<td>23</td>
<td>Perino, 1953</td>
<td>40, M</td>
<td>?</td>
<td>thoracic</td>
<td>yes during life</td>
<td>NR</td>
</tr>
<tr>
<td>24</td>
<td>Roca de Vinãls, et al., 1954</td>
<td>50, F</td>
<td>6 mos</td>
<td>thoracolumbar</td>
<td>yes during life</td>
<td>NR</td>
</tr>
<tr>
<td>25</td>
<td>Gibson, et al., 1957</td>
<td>51, F</td>
<td>?</td>
<td>thoracic</td>
<td>no at autopsy</td>
<td>diffuse leptomeningeal dissemination</td>
</tr>
<tr>
<td>26</td>
<td>Hirano &amp; Carton, 1960</td>
<td>42, M</td>
<td>1 mo</td>
<td>thoracic</td>
<td>yes during life</td>
<td>NR</td>
</tr>
<tr>
<td>27</td>
<td>Kiel, et al., 1961</td>
<td>33, F</td>
<td>5 mos</td>
<td>cervical</td>
<td>yes during life</td>
<td>cerebral leptomeningeal pigmentation</td>
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<tr>
<td>28</td>
<td>Özden, et al., 1984</td>
<td>15, F</td>
<td>4 mos</td>
<td>cervical</td>
<td>yes during life</td>
<td>NR</td>
</tr>
<tr>
<td>29</td>
<td>Larson, et al., 1987</td>
<td>30, F</td>
<td>5 mos</td>
<td>thoracic</td>
<td>yes during life</td>
<td>NR</td>
</tr>
<tr>
<td>30</td>
<td>Larson, et al., 1987</td>
<td>73, M</td>
<td>6 mos</td>
<td>thoracic</td>
<td>yes during life</td>
<td>NR</td>
</tr>
<tr>
<td>31</td>
<td>63, M</td>
<td>96 mos</td>
<td>thoracic</td>
<td>yes during life</td>
<td>NR</td>
<td></td>
</tr>
<tr>
<td>32</td>
<td>67, F</td>
<td>18 mos</td>
<td>thoracic</td>
<td>yes during life</td>
<td>NR</td>
<td></td>
</tr>
<tr>
<td>33</td>
<td>57, F</td>
<td>3 mos</td>
<td>cervical</td>
<td>yes during life</td>
<td>NR</td>
<td></td>
</tr>
<tr>
<td>34</td>
<td>69, F</td>
<td>24 mos</td>
<td>thoracic</td>
<td>yes during life</td>
<td>NR</td>
<td></td>
</tr>
<tr>
<td>35</td>
<td>Yamasaki, et al., 1989</td>
<td>31, M</td>
<td>6 mos</td>
<td>thoracic</td>
<td>yes during life</td>
<td>NR</td>
</tr>
<tr>
<td>36</td>
<td>Magni, et al., 1996</td>
<td>64, M</td>
<td>24 mos</td>
<td>thoracic</td>
<td>yes during life</td>
<td>NR</td>
</tr>
<tr>
<td>37</td>
<td>present study</td>
<td>62, M</td>
<td>1 mo</td>
<td>cervical</td>
<td>yes during life</td>
<td>nodules diffused to entire brain; 3 big lesions in cerebellum, rt frontal hemisphere, &amp; lt ponsmidbrain; diffuse leptomeningeal dissemination</td>
</tr>
</tbody>
</table>

* NR = not reported; ? = unknown.
level. This intramedullary tumor was only partially re-
moved because of vascularization, adherence in the ante-
rior region, and poorly defined margins of the lesion itself.
Intraoperative histological examination of a frozen section
demonstrated a malignant melanoma. At ultrastructural
examination, the permanent specimens of the lesion dem-
strated proliferation of spindle cells that were organized
in clusters and fascicles, characterized by nuclear ana-
plasia, long intertwining processes, complex membrane
interdigitations, scattered intermediate-type junctions, and
lysosomal dense bodies. The neoplasm exhibited a high
mitotic rate; the presence of extensive deposition of
melanin granules and pleomorphic melanosomes was also
observed (Fig. 3). The tumor was studied using immu-
nocytocchemical methods in which monoclonal antibody
anti-HBM-45, a useful marker for melanocytic differenti-
ation in a neoplasm, was used. This marker is indicative of
active melanosome production and is localized in Stage 1
and 2 melanosomes. Tumor cells stained positive for
HBM-45+.

Postoperative Course. The patient’s postoperative clin-
cal examination revealed mild neurological improvement.
The patient underwent dermatological and ophthalmolog-
ical examinations, which did not show other evidence of a
primary origin of his melanoma. He was discharged on
October 31, 1996 and underwent postoperative radiother-
apy of the entire spine with two opposed, evenly
weighted, laterolateral fields. Radiation therapy was per-
formed using cobalt-60 to a dose of 44 Gy administered in
22 fractions of 2 Gy per fraction 5 days per week, with
both fields treated daily over a period of 36 days. The
superior and inferior borders of the fields included at least
two vertebral bodies in either direction from the target
volume as determined by MR imaging. The field width
encompassed the whole vertebra, and the dosage was cal-
culated at midplane. The patient tolerated the treatment
well and showed good improvement of sensory and motor
symptoms. The toxicity of treatment was negligible (sec-
ond degree dermatitis responsive to local steroid–antibiotic
drug therapy). Follow-up examination at 14 months
showed good improvement of the left hemiparesis.
However, in February 1998 the patient started compla-
ning of headache, vomiting, and weight loss. Magnetic re-
onance images of the brain were obtained, and we ob-
served metastases arising from CSF dissemination in the
right frontal hemisphere, cerebellum, left pons, and mid-
brain. Small nodules diffused to the entire brain were also
detected (Fig. 4). Death occurred 1 month later, 15 months
after the onset of symptoms.

Discussion

We report on a patient with a rare primary malignant
melanoma of the cervical spinal cord. In previous cases
reported in the literature various melanomas are described
throughout the neuraxis. Primary spinal melanoma most
frequently occurs in the middle and lower thoracic
cord. To date, only 37 cases of primary spinal melanomas have been reported in the literature, including
intramedullary, intradural, and extradural lesions. An
extensive review of 27 cases of malignant melanoma
involving the CNS was reported by Hayward, in which
four of these lesions were located in the spinal cord. One
lesion was situated in the cervical cord, two were dorsal, and one involved the conus medullaris. In records of patients with primary melanoma of the spinal cord obtained from the Mayo Clinic and reviewed by Larson et al., only one patient presented with a cervical spinal cord lesion; in the other patients, the melanoma was found in the thoracic cord at the T-6 level or more distally. Magni et al., in their recent review of the literature reported that...
Primary intramedullary melanoma

the tumor is more often located in the middle or lower thoracic cord.

Among the 37 cases so far described in the literature, only eight, including that detailed in our report, were located in the cervical spinal cord.30,32,33,36,41,46,51,73 One case of a cervicothoracic melanoma was reported by Bell5 in a 48-year-old woman. Regarding metastases, our case is the first cervical melanoma so far described in which diffuse metastases arising through CSF dissemination were demonstrated in the brain. The patient’s MR images obtained 14 months postsurgery documented the presence of nodules diffused to the entire brain and three large lesions in the right frontal hemisphere, cerebellum, and left pons and midbrain were observed. However, no secondary lesions were detected at 9 months in a follow-up brain study.

In our case, in accordance with Hayward’s30 classification criteria, melanoma was not detected outside the CNS, the nerve root and the dura were not involved by the tumor, and the lesion was confirmed on pathological studies. As Hayward points out, a malignant melanoma can occur in any organ in which melanin-containing cells are present. These cells are a normal constituent of many sites in addition to the skin, for example, the iris, ciliary body, choroid, and retina of the eye, chromaffin tissue, and leptomeninges. Even though melanocytic cells have been found in the leptomeninges, primary spinal cord melanomas are quite rare. A thorough physical examination to search for a primary cutaneous, mucosal, or ocular melanoma is recommended and is usually sufficient to exclude evidence of systemic disease.

In our patient, the angiogram of the spine was very striking because it revealed high vascularity and a remarkable circular blush. This is a unique characteristic of our report: no pathological circle has so far been described in the literature, in contrast to the recent case of thoracic melanoma reported by Yamasaki, et al.,75 in which no feeding vessels or pathological blush were observed. The appearance of the lesion on MR imaging was found to be compatible with the reports in the literature: preoperative MR imaging revealed a pattern similar to that of intracerebral melanoma, which is thought to be caused by the paramagnetic products of subacute and chronic hemorrhage, as well as the paramagnetic stable free radicals known to exist within melanin.41,72 The peripheral intramedullary location of the tumor in the cervical spinal cord explained the asymmetric myelopathy observed at neurological examination.

Primary intramedullary melanoma exhibits either slow progression or rapid decline, and this lesion is different from either meningeal melanocytoma or the more common type of melanoma of the skin with metastases to the CNS. Meningeal melanocytoma (or primary melanotic tumor of the leptomeninges) is a rare benign melanotic tumor deriving from the melanocytes of the leptomeninges and occurring anywhere in the cranial and spinal meninges. Complete surgical removal is the treatment of choice, and radiotherapy is usually unnecessary. In spite of the tumor’s benign biological behavior, the prognosis remains uncertain because of possible local recurrences. Magnetic resonance imaging reveals a variable appearance because of different degrees of melanization and cannot be used to distinguish reliably between meningeal melanocytomas and other pigmented tumors such as primary malignant melanoma.

Meningeal melanocytoma usually shows increased signal intensity on T₁-weighted images, iso- or low intensity on T₂-weighted images, and mild but homogeneous enhancement after administration of contrast material.15,69 Surgical, ultrastructural, and immunohistochemical criteria are more relevant in differentiating meningeal melanocytoma from primary malignant melanoma. Surgical findings of a lesion with well-defined margins and the absence of nerve roots and dura involvement are suggestive of meningeal melanocytoma. Ultrastructurally, melanocytomas lack anaplastic features such as necrosis, significant mitotic activity, and pleomorphism. Recent or old hemorrhage is not present, in contrast with primary melanomas. Magnetic resonance imaging cannot be used to distinguish reliably between the presence of melanin and hemosiderin, both of which produce decreased T₂-weighted relaxation on images. Immunohistochemically, meningeal melanocytoma cells are immunoreactive for S-100 protein and vimentin but not for epithelial membrane antigen. The low proliferative activity in bromodeoxyuridine labeling associated with the absence of anaplastic features means a better prognosis in patients affected by melanocytomas who can, according to these findings, be cured by complete surgical resection of the lesion.95

Melanoma of the skin with metastases to the CNS progresses rapidly and is often fatal within 6 months.10,72,33,41,42,59,61,73 Primary spinal melanoma is a more indolent malignancy than melanoma that metastasizes to the CNS.30,32,41,64,67 The biology of these tumors is not well known and reviewing the accumulated experience may help to delineate further the behavior of such unusual spinal cord tumors. The surgical excision alone of the lesion is generally incomplete and is not usually curative.2,31,65 For this reason, adjuvant radiotherapy is frequently recommended after surgery,13,26,31,34,41,42,46,50,55,68 although malignant melanoma does not seem to be particularly radiosensitive. Local external radiation treatment, in fact, is aimed at sterilizing the neoplastic bed to decrease the risk of local regrowth. The average survival of patients with primary spinal melanoma after combined surgery and radiotherapy is 5 to 6 years, clearly longer.
compared with the more common melanoma of the skin, which demonstrates a fast progression with systemic spread and fatal outcome. Among 36 previous reported cases of patients with spinal melanomas, 11 received radiation therapy and had different survival times. Bau-Prussak and Mackiewicz, Bell, García, and Hirano and Carter reported a patient survival time of 6 months; Kissel, et al., reported an 11-month survival time. However, Da Costa and Love and later, Moersch, et al., reported a survival time of 10 years. The Mayo Clinic experience from 1974 to 1985 offers data regarding survival: among five patients affected by primary thoric melanoma who received postoperative radiation therapy, one patient is reported to be alive after a short follow-up period, another at 7 years after onset of symptoms, and three patients died, 2.5, 3.5, and 13 years, respectively, after the diagnosis was made. The findings in this series show an average survival time of 6 years 7 months after surgery and postoperative radiation therapy, suggesting that primary spinal melanoma may have a better prognosis when compared with cases previously reported.

Chemotherapy is another complementary form of treatment believed to be useful in the therapy of malignant melanoma and it may be administered either after surgery alone or following postoperative radiation therapy. Only two cases treated thus are reported in the literature: in one case reported by Özden, et al., 1,3-bis(2-chloroethyl)-1-nitrosourea and dimethyltriazenomidazole carboxamide (dacarbazine) were intravenously administered in a 30-year-old woman affected by a primary dorsal thoracic melanoma: at 16-month follow up, the patient was free of symptoms and no signs of tumor recurrence were observed. Yamasaki, et al., reported a case in which intrathecally administered dacarbazine, effective in the treatment of CSF metastases, was given after radiation and systemic interferon-β therapies in a 31-year-old man affected by primary intramedullary thoracic melanoma. In this patient, follow-up examination in which MR images obtained 1 year after surgery were used did not show a significant increase in the size of the residual tumor and no recurrence or metastasis was observed.

Regarding the prognosis for primary melanoma, worldwide experience has shown survival periods varying from 2 months to 13 years, even though partial resection of the tumor is often reported to improve mean survival times. Among the 36 cases reported in the literature, seven nonsurgically treated patients had a mean survival time of 2 months; only one of these patients survived 1.5 years. The other 29 patients, who underwent surgical excision alone or associated with additional treatments, showed survival periods ranging from 6 months to 13 years. Active specific immunotherapy has also been used, often with encouraging results, and the best results have been obtained in patients with minimal residual disease after resection of the tumor. Prolonged survival of several years has been achieved, with little toxicity attributable to the treatment.

It is revealing to discuss the histogenesis of primary intramedullary melanoma. Melanocytic cells arise in the neural crest by the 6th week of gestation and migrate to their destination, reaching the skin by the 10th week and the meninges by the 20th week of embryological development. An interesting pathogenetic hypothesis for primary intramedullary melanoma suggests that, during embryogenesis, a few neuroectodermal rest cells migrate to and reside within the neural tube and its coverings. These cells are, therefore, not “committed” and are unable to establish correct connections with other cells and to interact with chemical markers, which may mediate outward migration and cellular maturation. The development of neoplasms could be dependent on the “staminal” character of these cells. During embryonal development, differentiation, namely as cellular maturation and synthesis of specific products, is preceded by a stage in which the cell is orientated, namely committed, toward a precise cellular stock and a specific function, which were already planned before the migration. After the migration, the definitive differentiation is achieved in consequence of ambiental factors or specific inductors. If migration does not occur, the cells regain the original character of staminal cells, which are the totipotent cells from which they are derived before being committed: in situations promoting underdifferentiation and cellular proliferation, the cell lacks a specific phenotype and reverts to totipotency at a genetic level. These properties of the staminal cell cause an abnormal growth of undifferentiated cells, which then will give rise to the tumor.

Cellular migration-related anomalies occur early or late. Early migration disorders are usually genetically determined, but they may be secondary to environmental causes. Primary intramedullary melanoma, consequently, could be dependent on defective genomic expression. Another theory on the origin of primary spinal melanoma, on the other hand, holds that melanoma may occur in any organ in which melanoblasts can normally be found. This includes the pia mater, which not only covers the brain but also sheathes the blood vessels passing into it and the spinal cord. These pial pigmented cells, although present everywhere, are most numerous around the spinal cord and on the ventral aspect of the brain. According to this theory, the neoplasm arises from melanoblasts of the pia mater, which will undergo an anaplastic transformation. Therefore, spinal melanoma would be a mesodermal neoplasm, because it arises from the mesenchyma located between the neural tube and the ectoderm from which leptomeninges and, therefore, the pia mater take origin. This is the so-called “mesodermal” theory. The former hypothesis is most interesting, because it suggests a genetic origin of this tumor, thus raising the possibility of gene therapy treatment of this lesion in the future.

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References

3. Antanitus DS, Choi BH, Lapham LW: The demonstration of glial fibrillary acidic protein in the cerebrum of the human fetus

F. M. Salpietro, et al.
Primary intramedullary melanoma

by indirect immunofluorescence. *Brain Res* **103**:613–616, 1976


20. Esler P: Über eine seltene Rückenmarkshautgeschwulst (Chromatophoroma durae matris spinalis). *Dtsch Nervenheilk** **32**:118–123, 1907


44. Lindbom O: Ett fall af chromatophoroma durae matris spinalis. *Hygiea* **74**:198–218, 1912


49. Moersch FP, Love JG, Kernohan JW: Melanoma of the central nervous system. Report of thirty-four cases, in nineteen of which the diagnosis was verified by operation or necropsy. *JAMA* **115**:2148–2154, 1940


55. Rate WR, Solin LJ, Turrisi AT: Palliative radiotherapy for


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