Collision Tumor of Meningioma and Non Hodgkin Malignant Lymphoma of Cerebellum

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Abstract

Primary central nervous system lymphoma (PCNSL) constitutes a rare group of extranodal non-Hodgkin’s lymphomas (NHLs), primarily of B cell origin, the incidence of which, has markedly increased in the last three decades. Immunodeficiency is the main risk factor, but the large majority of patients are immunocompetent. This report presents the case of a 71-year-old woman with a collision tumor of primary malignant lymphoma and meningioma in the cerebellum. Collision tumor of primary malignant lymphoma and meningioma have not been described in the literature. The morphological aspect is interesting with regard to the problem of collision tumors.

Keywords: Primary central nervous system lymphoma; Meningioma; Collision tumor

Introduction

Primary central nervous system lymphoma (PCNSL) is a rare central nervous system tumor, accounting for approximately 4% of all primary central nervous system tumors [1]. Collision tumors of histologically different primary brain tumors are rare, and these generally occur as multicentric tumors. Coexistence of histologically different tumors in a single lesion is extremely rare. We report the coexistence of primary malignant lymphoma and meningioma with collision tumors in a single mass. The discussed case is a report with histological findings and neuroimaging.

Clinical Summary

A 71-year-old woman developed dizziness, imbalance in the body, and headache one months before admission. Thereafter, she suffered increasing complaints. Physical examination showed no pathological finding as well (excluding tandem walk). Computed tomography (CT) revealed a mass lesion with a size of 3.2 x 2.5 cm and in the cerebellar region (Figure 1).

CT scan revealed a mass lesion predominantly located in the right cerebellar region. Intratumoral calcification was depicted by the CT scan. She underwent subtotal resection of the tumor. Macroscopically, the tumor consisted of purple violet tissue.

Pathological Findings

Macroscopically, the resected specimen consisted of two distinct components, purple violet soft tissue and yellowish hard tissue, separated by a relatively clear border. Microscopically, the tumor tissue from the cerebellar tissue consisted of cells with spindle cells and calcification (Figure 2). Because the cells mimic fibroblasts in appearance and also form true reticulin and collagen fibrils. The cells of the fibroblastic meningioma are usually arranged in fascicles crossing other fascicles. The pink collagen bundles seen in hematoxylin-eosin stained sections vary from place to place. Moreover, histopathological examination revealed that the tissue was composed of large having vesicular nucleus, 1-3 conspicuous nucleoli and moderate amount of pale to eosinophilic cytoplasm with brisk mitoses lymphoid malignant cells infiltrating cerebellar parenchyma diffusely (Figure 3). The cells were invasive within meningioma. Differential diagnoses of PCNSL revealed primitive neuroectodermal tumors, undifferentiated carcinomas, small cell astrocytomas, metastatic amelanotic melanomas, or anaplastic oligodendrogliomas and reactive changes. The edges of the infiltrating masses often contained mixtures of tumor cells, small reactive lymphocytes, and reactive astrocytes. It was observed that the tumor cells stained with LCA, CD20 (strongly), ki-67(LI: 90%) diffusely positive. Staining for GFAP, CD68, NSE, chromogranine, synaptophysin was negative (Figures 4-6). The patient was considered

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to have B-cell diffuse non-Hodgkin primary central nervous system lymphoma and meningiomas (Fibroblastic, WHO GRADE I) (collision and coexistence).

**Discussion**

The simultaneous occurrence of collision tumors in the cerebellum and in the same patient, particularly in patients without a history of phacomatosis or prior radiation therapy, is rare [2-5]. A significant number of collision tumors happen in cases with phacomatosis [2].

The term ‘collision’ has been used for the cases in which two tumors are being intermixed or appear close to each other without brain tissue in between [4] gliomas and meningiomas are the most frequently reported combination of histologically different brain tumors [6].

However, primary malignant lymphoma and meningioma are not reported in the literature.

Several hypotheses have been offered to link the occurrence of multiple primary brain tumors of different histogenesis in the same patient. The observation that a significant number of the reported cases had their tumoral localization in juxtaposition raises the possibility that one tumor may influence as an irritating agent for the local proliferation and growth of the other [6-8]. A purely coincidental event has been suggested by others [6-8]. Surgical trauma, genetic factors and ionizing radiation may act as factors for tumor development [4].

In collision tumors, the possibility that one tumor may be responsible for inducing the formation of the other tumor has been suggested.

Though patients with AIDS have approximately 20% more lifetime risk of developing PCNSL, incidence in patients without AIDS is also increasing rapidly. This rise seems to be unassociated with AIDS or immunosuppression and is being seen more commonly in HIV-negative individuals [8,9]. This case in our study was HIV-negative.

PCNSLs in the present study accounted for 12% of total extranodal lymphomas and 4.3% of total non-Hodgkin’s lymphomas (NHLs). The incidence reported in the literature is approximately 4.2% of extranodal lymphomas and 0.7-1.7% of total NHLs [7-9].

Misdiagnosis may be one of the reasons for not receiving appropriate treatment. PCNSL may occasionally pose a problem while differentiating from other round cell tumors including neurocytoma, primitive neuroectodermal tumors (PNET’S), small cell glioblastoma,
and rarely viral encephalitis with perivascular reactive lymphoid cuffing.

Central neurocytoma is a rare intraventricular brain tumor that affects young adults and presents with increased intracranial pressure secondary to obstructive hydrocephalus and NSE (strongly) positive [10].

PNET’S have diffuse proliferation of small round cells having round nuclei, fine chromatin, scantly clear or eosinophilic cytoplasmic and indistinct cytoplasmic borders. Immunohistochemically, tumor cells were negative for epithelial markers (Cytokeratin, EMA), leucocyte common antigen (LCA) and neuroendocrine markers (synaptophysin, chromogranin, CD56) [11]. Viral encephalitis with perivascular reactive lymphoid cuffing have reactive (normally) lymphocyte.

High index of suspicion for lymphoma and a panel of antibodies should be used in differentiating lymphomas from other round cell tumors. In the present study, this case was differentially diagnosed as primitive neuroectodermal tumor with reactive changes (CD68, synaptophysin, NSE, chromogranine negative). In conclusion, the case has been diagnosed as PCNSL in histological examination. It is possible that meningiomas develop as secondary malignant neoplasm due to transformation of the arachnoid cells in response to the growth of a subjacent glioma or after radiation therapy [2,4,6,10-13].

Collision tumors composed of glioma and meningioma are rare occurrences and only few cases in the brain have been described in the literature. Prayson et al. [3] reported a 87-year-old woman with collision tumors that consisted of syncytial meningioma (WHO grade I) and malignant astrocytoma (WHO grade III) [3]. Vasquero et al. [5] reported a 75-year-old woman who had a collision tumor composed of psammomatous meningioma and glioblastoma multiforme. Similarly Drlicek et al. [14] reported a 51-year-old male with glioblastoma (WHO grade IV) and a meningothelial meningioma (WHO-grade I). Here, we report the first collision tumor composed of PCNSL and meningioma in a woman.

Our patient’s disease status was not associated with phacomatosis, and no trauma or surgery has been recorded in the history of her disease. Although a purely coincidental event is possible for an intracerebellar meningioma and adjacent PCNSL to occur in a woman, the collision of two tumors suggested that local causes were probably responsible for our patient’s disease status.

In addition, the differential diagnosis of PCNSL is even more difficult in collision tumors. As a result, although these tumors are rare, many studies are needed to elucidate the structures of heterogeneous tumors which would be useful for diagnosis and treatment.

References