Neoplasm

Lipoglioblastoma: a lipidized glioma radiologically and histologically mimicking adipose tissue

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Abstract

\textbf{Background:} We report the case of a man with glioblastoma containing a component radiologically and histologically mimicking adipose tissue.

\textbf{Case Description:} A 48-year-old man recently complaining of headaches and difficulty with speech presented with a cystic peripherally enhancing left temporoparietal mass with focal intrinsically (precontrast) bright nodules in fluid attenuated inversion recovery and T1-weighted images similar to adipose tissue. Histologically, the enhancing component was classic glioblastoma, whereas the bright nodules comprised tumor cells that in aggregate closely resembled adipose tissue.

\textbf{Conclusions:} The case illustrates the extent to which lipidized central nervous system tumors of glial origin, or components thereof, can radiologically and histologically resemble adipose tissue. However, immunohistochemical staining and electron microscopy can eliminate diagnostic confusion.

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\textbf{Keywords:} Glioblastoma; Lipidization; Lipoglioblastoma; Lipoastrocytoma; Adipose tissue

1. Introduction

The CNS tumors are sometimes lipidized—for example, pleomorphic xanthoastrocytoma (PXA)—but only rarely to the extent that the affected areas resemble adipose tissue histologically. Even less common are tumors wherein the adipose tissue is extensive enough to become apparent grossly on MRI. We report a glioblastoma, with lipidization that resembled adipose tissue both radiologically and histologically.

2. Case history

A 48-year-old man presented with a 2-week headache and speech difficulties. Magnetic resonance imaging demonstrated a $5 \times 4 \times 3 \text{ cm}$ solid and cystic/necrotic ring-enhancing mass in the left temporoparietal region (Fig. 1A). Within the solid enhancing component were several distinct well-circumscribed nodules that were hyperintense on both precontrast T1-weighted images and T2 FLAIR-weighted images. A gross total excision was achieved. The patient died 15 months after the surgery despite radiotherapy and chemotherapy.

3. Results

Most of the solid tumor consisted of solid sheets of mitotically active pleomorphic cells with angular nuclei with coarse chromatin typical of high-grade astrocytomas (Fig. 2A). There was extensive vascular proliferation (Fig. 2B) and focal necrosis (Fig. 2C). Less cellular, but mitotically active, infiltrating areas were also present.
Corresponding to the T1-precontrast, bright nodules was rarefied tissue indistinguishable at low and high magnification from adipose tissue, with its small dark nuclei flattened against the margins of the cells. The nodules were all well circumscribed, with sharp borders between lipidized and nonlipidized zones. Only the narrowest transition zone separated these 2 components (Fig. 3A and B). There was no spindled component to suggest gliosarcoma. The tumor in general and “adipocytes,” in particular, were strongly positive for GFAP, the latter in a rim pattern (Fig. 3D). The tumor, including the lipidized component, was negative for synaptophysin.

4. Discussion

Fat accumulation in CNS neoplasms usually takes the form of small cytoplasmic droplets that create a bubbly or

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**Fig. 1.** Axial MRI (FLAIR, T1-weighted, and postcontrast T1-weighted) shows a large ring-enhancing mass in the superior left temporoparietal lobe, with thickened soft tissue in the posterior-lateral margin demonstrating irregular and nodular enhancement (T1 + gadolinium). Note also small nodular areas, isointense to adipose tissue, within the solid portion of the mass that are hyperintense on FLAIR and T1-weighted images.

**Fig. 2.** Glioblastoma. Much of the tumor, corresponding to the enhancing nodule on MRI, is classically glioblastoma with mitotically active pleomorphic cells (A), vascular proliferation (B), and tumor necrosis (C). D: The tumor cells are GFAP-positive.
xanthomatous appearance as in the iconic lipidized CNS tumor, PXA [8]. Large clear vacuoles suggesting fat droplets are not infrequently encountered in other CNS tumors as well, but confirmatory fat stains on frozen sections are rarely done. Nevertheless, one suspects that the vacuities must have been lipid and that the incidence of lipidization of CNS tumors is greater than is realized.

Cytoplasmic vacuoles known to represent lipid enliven the lipid-rich glioblastomas [7,9] including epithelioid [14] and giant-cell variants [10]. More impressive are tumor cells so lipid-laden that they resemble adipocytes individually or to adipose tissue in aggregate, sometimes even macroscopically [12]. Such tumors include rare, otherwise typical, glioblastomas [12], ependymomas (“lipomatous”) [15,16], astrocytomas (“lipoastrocytoma,” “astrolipoma,” “astrocytoma with extensive lipidization”) [4,18], neurocytomas (“liponeurocytoma”) [2,3,5], primitive neuroectodermal tumors (“lipomatous”) [6], and meningiomas (“lipomatous”) [1,11,13]. Gliosarcomas rarely have a liposarcomatous component [17].

Lipidization in CNS tumors is usually an unexpected histologic finding but may be so prominent, as in the present case, as to be apparent preoperatively on MRI [3,4,6,13,15,18]. The same tissue may be present at the microscopic level but not abundant and concentrated enough to be recognized radiologically [12]. The present case therefore is one of the former, wherein a lipidized component is evident, both histologically and radiologically.

The nature of lipidized CNS tumor cells may be apparent by the context and transition forms between obvious tumor cells and the lipid-laden variants, electron microscopy, or immunohistochemistry. Electron microscopy identifies telltale structures such as desmosomes in meningiomas or reveals a unit membrane that encircles the lipid in adipocytes but not in lipidized tumor cells [13]. Immunohistochemistry is helpful if lipidized cells are immunoreactive for GFAP, EMA, or synaptophysin, for gliomas, meningiomas, and neurocytomas, respectively.

Although there is little doubt about the glioblastomatous nature of the present case as well as others with classic features of “GBM,” molecular studies in a series of 3 cases found changes consistent with high-grade astrocytoma, for example, gain of chromosome 7 and losses of chromosomes 9 and 10 [12].

References