J Neurooncol (2011) 103:121–127
DOI 10.1007/s11060-010-0361-z

CLINICAL STUDY – PATIENT STUDY

Supratentorial and cerebellar liponeurocytomas: report of four cases with review of literature

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Received: 2 April 2010 / Accepted: 9 August 2010 / Published online: 29 August 2010

Abstract Liponeurocytoma is not exclusive to the cerebellar or fourth ventricular location. Since its inclusion in the central nervous system tumor classification in 2000, six cases with similar radiological, histomorphological and immunohistochemical features have also been described in the lateral ventricles. In the present study, we report clinical, radiological and pathological findings of three supratentorial and one cerebellar liponeurocytoma from our records, evaluated with an extensive panel of immunohistochemistry, and review published cases in the literature. The immunohistochemical pattern of supratentorial and infratentorial liponeurocytomas are almost identical, which indicates that these tumors are homologous.

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Keywords Lateral ventricle · Fourth ventricle · Supratentorial · Infratentorial · Cerebellar · Immunohistochemistry

Introduction

Cerebellar liponeurocytoma as a distinct entity was first introduced in the WHO 2000 classification of tumors of central nervous system [1]. This low grade tumor with consistent neuronal, variable astrocytic and focal lipomatous differentiation was described predominantly in the cerebellar hemispheres and the vermis and rarely in the fourth ventricle [2]. Subsequently, occurrence of similar tumors was described in the supratentorial compartment within lateral ventricles. To date, 33 published cases of cerebellar [2–6] and 6 cases of supratentorial intraventricular liponeurocytoma [7–12] are on record. In this study, we present the clinical, radiological and immunohistochemical features of 4 cases of liponeurocytomas, one arising in the cerebellum and 3 others located supratentoreally, and review the similarities and differences between the tumors in two different intracranial locations.

Materials and methods

We reviewed our records at the Department of Neuropathology, National Institute of Mental Health and Neurosciences, Bangalore, South India, a tertiary care center for neurological disorders, over a period of 10 years (1999–2008). Among 66 cases of central neurocytomas (constituting 0.6% of neurosurgical biopsies) reported between 1999 and 2008, 3 cases of supratentorial intraventricular liponeurocytomas and a single case of cerebellar liponeurocytoma

were found. The clinical presentation, neuroimaging features, and treatment details are summarized in Table 1 (Fig. 1a–c).

The excised tumor tissue received was processed for paraffin embedding and sectioning. Sections were stained with hematoxylin and eosin stain for routine histological evaluation. Immunohistochemistry was performed on representative sections using indirect immunoperoxidase technique with antibodies to Glial fibrillary acid protein (GFAP; monoclonal, 1:50 dilution, BioGenex, USA), Synaptophysin (polyclonal, 1:50 dilution; Dako USA), Neuronal nuclear antigen (NeuN; monoclonal, 1:50 dilution; BioGenex), Neurofilament (NF; monoclonal, 1:1,000 dilution; BioGenex), Chromogranin A (ChgA; monoclonal, prediluted; BioGenex), MIB-1 (monoclonal, 1:30 dilution; BioGenex;), MAP-2 (monoclonal, 1:1,000; Sternberger Monoclonals, USA), p53 (monoclonal, 1:100 dilution; BioGenex) and S-100 (polyclonal, 1:100 dilution; BioGenex). The immunohistochemical features are tabulated in Table 2.

Results

Histopathological examination of the tumor from all the three cases in the supratentorial compartment showed sheets and lobules of monomorphic round cells interspersed by arborizing network of capillaries and multiple large punched out empty spaces (Fig. 2a). The tumor cell nuclei were round with speckled chromatin and micronucleoli, and pale eosinophilic to clear cytoplasm. The empty spaces in the tumor represented large fat vacuoles surrounded by a thin rim of cytoplasm (Fig. 2a). Dispersed foci of cell-free, finely fibrillary neuropil was seen punctuating the tumor. Diligent search did not reveal mitotic activity in any of the cases. Interstitial foci of hemorrhage

Table 1 Clinical and neuroimaging features

Case no. Age/sex	Clinical features	CT/MRI	Treatment	Follow-up
Case 1 36 years/ M	Bifrontal headache: 4 months; blurring of vision: 15 days. Examination: normal memory, speech and attention span, no neurological deficits, bilateral papilloedema	CT scan: mixed density, irregular mass lesion, bilateral lateral ventricles causing hydrocephalus. Third and fourth ventricles normal	Radical excision	Recurrence 9 years and 4 months later. MRI: large, non-enhancing, mixed iso to hyperintense on T1WI and isointense on T2WI lesion, involving both lateral ventricles, causing hydrocephalus, displacing the shunt tube. Re-exploration and radical excision of the tumor performed with postoperative radiotherapy. No recurrence at 2-year follow-up
Case 2 30 years/ M	Intermittent, holocranial headache and vomiting: 2 years. Examination: bilateral papilloedema	CT: irregular, hypodense lesion occupying both the lateral and third ventricles. MRI: lesion was hyperintense on T1WI and T2WI images with specks of hypointensity (Fig. 1a–c)	Gross total resection. Intraoperatively the lesion was pale yellow, avascular, soft and suckable. Possibilities of lipoma or intraventricular dermoid cyst were considered	Post-operative period was uneventful and the patient was lost for follow-up
Case 3 32 years/ M	Intermittent, holocranial headache, left lower limb weakness: 6 months. Examination: bilateral papilloedema. HIV positive	CT: hyperdense, irregular, lobulated, minimally enhancing mass lesion, in the septal area extending into both lateral ventricles, asymmetrically causing hydrocephalus	Gross total excision	Post-operative period was uneventful and the patient was lost for follow-up
Case 4 45 years/ F	Diminishing vision, headache, swaying while walking: 2 months. Examination: cerebellar ataxia, bilateral papilloedema and decreased vision (6/24) both eyes	CT: hypo- to isodense, midline posterior fossa space occupying lesion, involving the cerebellum and compressing the fourth ventricle, mildly enhancing on contrast	Gross total excision. Intraoperatively the tumor was grey-white, fleshy, and mildly vascular	Post-operative period was uneventful and the patient was lost for follow-up

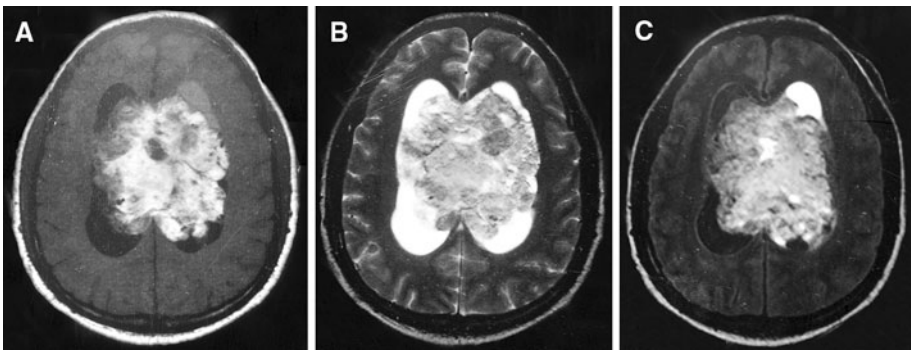

Fig. 1 Case 2. MRI shows a large irregular lesion filling up both lateral ventricles that is patchily hyperintense on T1 (**a**) and T2WI (**b**) with areas of hypointensity, not inverting on FLAIR (**c**)

Table 2 Summary of immunohistochemical profile of present series of liponeurocytomas

	Case 1	Case 2	Case 3	Case 4
Syn	Strong and uniform positive in tumor cells. Rim positivity around vacuoles in lipidized cells	Focally tumor cells and fibrillary matrix positive	Strong and uniform positive in tumor cells. Rim positivity around vacuoles in lipidized cells	Tumor cells and fibrillary matrix uniformly positive
NF	Tumor cells and fibrillary matrix positive	Strong and Uniform positive in tumor cells and fibrillary matrix	Negative	–[a]
NeuN	Positive in nuclei of tumor cells	Positive in nuclei of tumor cells	Positive in nuclei of tumor cells	–[a]
MAP-2	Uniform positive in tumor cells and matrix	Uniform positive in tumor cells and matrix	Uniform positive in tumor cells and matrix	Uniform positive in tumor cells and matrix
GFAP	Focal tumor cells and fibrillary tumor matrix positive, with accentuation around vacuoles in lipidized tumor cells	Focal tumor cells and fibrillary tumor matrix positive	Focal tumor cells and fibrillary tumor matrix positive, with accentuation around vacuoles in lipidized tumor cells	Focal tumor cells and fibrillary tumor matrix positive
Chg A	Focally positive in tumor cells	Focally positive in tumor cells	Focally positive in tumor cells	–[a]
S-100	Focally rim positivity around vacuoles in lipidized tumor cells	Negative	Focally rim positivity around vacuoles in lipidized tumor cells	–[a]
MIB-1	<1%, 3–4% (recurrence)	<1%	<1%	<1%
p53	Negative	Negative	Negative	–[a]

Syn Synaptophysin, *NF* Neurofilament, *ChgA* Chromogranin A, *NSE* Neuron-Specific Enolase, *CK* Cytokeratin, *EMA* Epithelial Membrane Antigen, *GFAP* Glial Fibrillary Acidic Protein, *NeuN* Neuronal Nuclear antigen

[a] Immunohistochemical stain not done

and fibrin was prominent in initial tumor sample of Case 1, while in the recurrent tumor, lipidization of the tumor cells was prominent. Numerous hyalinized blood vessels and predominantly lipidized zones were seen in Case 2. In all the three supratentorial tumors, the cytoplasm of the cells and small areas of cell free fibrillary matrix were diffusely immunopositive for Synaptophysin (Fig. 2b) and MAP-2 (Fig. 2c) while focally positive for Neurofilament (Fig. 2d) and Chromogranin A (Fig. 2e). In Case 3, no NF labeling was demonstrable, though diffuse positivity was present for MAP-2. Tumor cell nuclei demonstrated immunopositivity for NeuN (Fig. 2f). GFAP immunostaining highlighted glial cells in the midst of neurocytoma cells and surrounding the blood vessels (Fig. 2g). The lipid-laden tumor cells showed thin rim of cytoplasm immunolabeled by synaptophysin in Cases 1 and 3, while GFAP and S100 positive glial fibres were found encircling some of the lipid spaces (Fig. 2g, h). The MIB-1 labeling index in all the cases was uniformly less than 1% (Fig. 2i) except for the recurrent tumor in Case 1, which was focally 3–4%. p53 immunolabeling was negative in all. The posterior fossa tumor in Case 4, had identical histology and immunoprofile as the supratentorial group, with MIB-1 labeling index of less than 1%.

Discussion

To date, only six cases of supratentorial intraventricular central liponeurocytomas have been reported in the medical literature, all involving lateral ventricles (Table 3). The

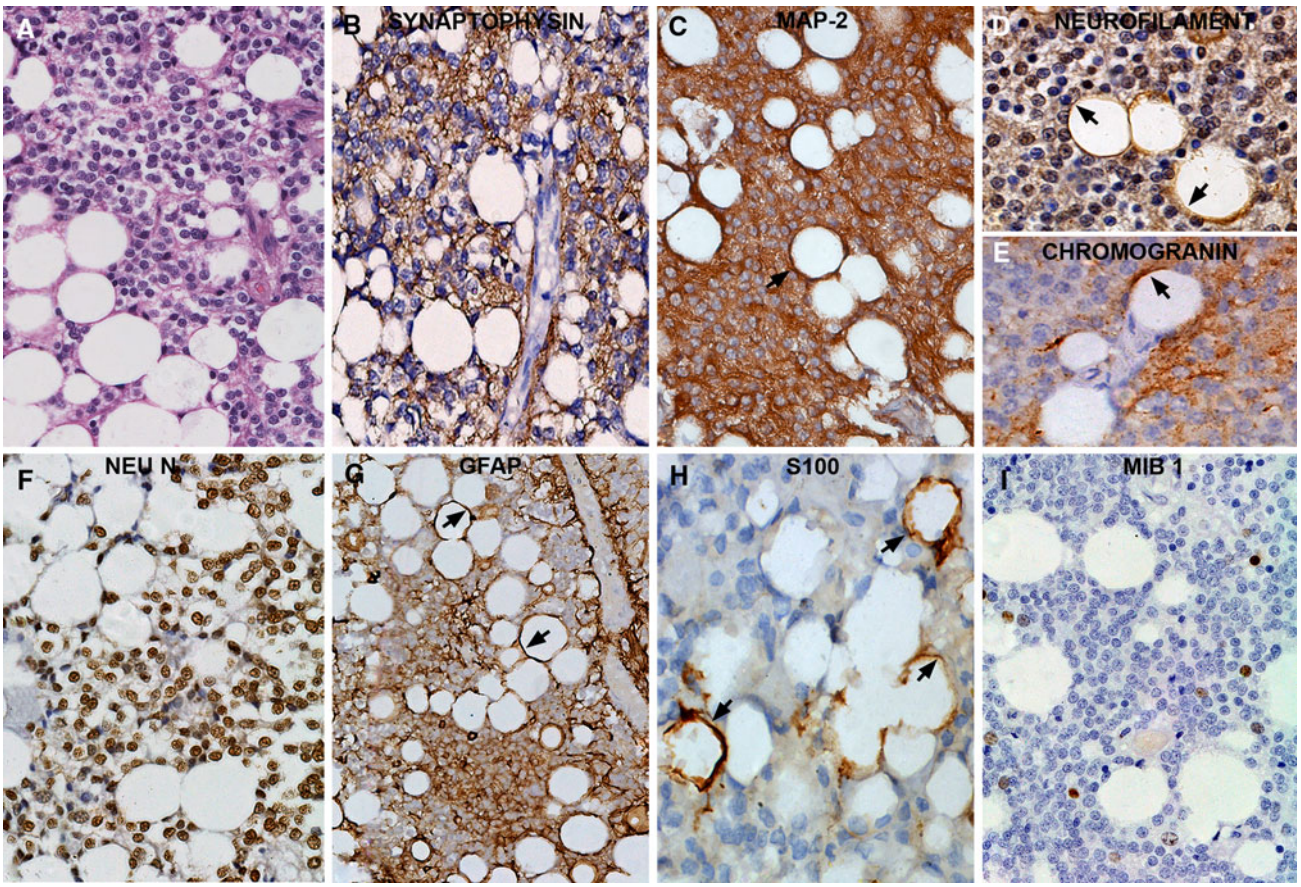

Fig. 2 Sheets of tumor cells with small, monomorphic nuclei and clear cytoplasm seen intersected by large lipid vacuoles (**a**) dispersed in a fine, neuropil stroma that shows diffuse positivity for synaptophysin (**b**). Tumor cells and matrix show strong labeling with MAP-2 (**c**) but focal labeling with NF (**d**) and ChgA (**e**) particularly surrounding vacuoles. Uniform nuclear labeling seen with NeuN in tumor cells (**f**). Scattered cells and perivascular glial cells highlighted by GFAP (**g**) with vacuoles encircled by GFAP (**g**) and S100 (**h**). Proliferative activity as seen by MIB-1 is low (**i**) (**a** HE ×180, immunoperoxidase; **b–d** ×180, **e**,**g** ×280, **f** ×120, **h** ×180)

mean age at presentation was 38.5 years (age range 30–59 years), with male preponderance (male:female = 3:1). In the present study, all the three supratentorial tumors involved bilateral lateral ventricles, with extension into third ventricle in one case (Case 2). Mean age at presentation was 32 years (range 30–36 years), and all three were males. The age distribution and anatomical localization was essentially similar to central neurocytomas that are typically located supratentorially in the lateral ventricle and/or the third ventricle, clinically manifesting in the third and fourth decades of life [13]. In contrast, cerebellar liponeurocytomas involve the cerebellar hemispheres, vermis and occasionally cerebellopontine angle, and occurs in older age group (fourth to sixth decades) [2] similar to Case 4 in the present study, that occurred in a 50-year-old lady.

Cerebellar liponeurocytomas on CT scans appear as hypo- to isodense tumors. The hypodensity has the attenuation values of fatty tissue, with variable enhancement on contrast. On MRI, the tumors appear hypointense on T1WI with scattered foci of hyperintensity in the form of mottling, laminated or serpiginous streaks, corresponding to fat, with minimal, heterogenous contrast enhancement. On T2W imaging, the tumors appear mildly hyperintense with absent or minimal edema. The presence of fat as determined on CT scan and T1W MRI are very characteristic and help distinguish this rare neoplasm from medulloblastomas or ependymomas of the fourth ventricle [14]. In published cases of supratentorial ventricular liponeurocytomas, the tumors are non-homogenous lesions with mixed signals indicating presence of fat and possibly calcium [7] with enhancing heterogenously on CT [9, 11] and MRI [7, 11]. Cranial CT scan in the present series showed hypodensity (Case 2) corresponding to microscopically predominant fat component and hyperdensity (Case 3) reflecting high cellularity. On MRI, hyperintensity on T1WI was noted in the recurrent tumor (Case 1), corresponded to increased fat component in the tumor.

Microscopically, cerebellar liponeurocytoma has a biphasic appearance, composed of sheets of isomorphic round tumor cells, having neurocytic and focal lipomatous

Table 3 Clinical summary of six reported cases of supratentorial liponeurocytomas

Authors	Age	Sex	Site	Clinical features	Treatment	Follow-up
Horoupian et al. [7]	30	M	Left lateral ventricle, roof of third ventricle, encroaching periventricular white matter	Headache, vomiting, decreased vision	Subtotal resection	Lost for follow-up
Mena et al. [8]	Clinical details of the case not specified in the original article					
George et al. [9]	59	F	Anterior horn left lateral ventricle	Difficulty in walking, decreased memory, cold sensation right arm and leg	Partial excision	Recurrence after 5 years 7 months followed by gross total resection
Rajesh et al. [10]	30	M	Body and frontal horn of the lateral ventricles	Headache, instability of gait for 3 months, right-sided cerebellar signs and sixth cranial nerve paresis	Near total excision	Not available
Kuchelmeister et al. [11]	35	M	Left lateral ventricle	Headache, dizziness, fatigue and paroxysmal paraesthesias in arms and legs	Near total excision	Not available
Wang et al. [12]	No details/abstract available					

Table 4 Summary of immunohistochemical profile of published cases

	Horoupian et al. [7], $n = 1$	George et al. [9], $n = 1$	Rajesh et al. [10], $n = 1$	Kuchelmeister et al. [11], $n = 1$
Syn	Tumor cells and fibrillary matrix positive	Strong and uniform positive in tumor cells; rim positivity around vacuoles in lipidized cells	Tumor cells positive	Tumor cells and fibrillary matrix positive
NF	–[a]	Negative	Negative	Negative
Chg A	–[a]	–[a]	–[a]	Negative
NSE	–[a]	–[a]	Tumor cells positive	Tumor cells and fibrillary matrix positive
GFAP	–[a]	–[a]	Reactive astrocytes positive	Reactive astrocytes, few tumor cells and tumor matrix positive
S-100	–[a]	–[a]	Negative	Nuclear and cytoplasmic positivity in reactive astrocytes, and GFAP positive tumor cells; focal diffuse positivity in tumor matrix
CK	–[a]	–[a]	–[a]	Negative
EMA	–[a]	Negative	–[a]	–[a]
Ki67	–[a]	5%, 5.8% (recurrence)	–[a]	4%
p53	–[a]	–[a]	–[a]	10–15% of tumor cells positive

n Number of cases, *Syn* Synaptophysin, *NF* Neurofilament, *ChgA* Chromogranin A, *NSE* Neuron-Specific Enolase, *CK* Cytokeratin, *EMA* Epithelial Membrane Antigen, *GFAP* Glial Fibrillary Acidic Protein

[a] Immunohistochemical stain not done

differentiation. The tumor cells and lipidized cells express the neuronal markers synaptophysin, neuron specific enolase, MAP-2 and focally GFAP [2]. The histological features of all the cases in the present study were comparable to cerebellar liponeurocytoma. Central neurocytomas have similar histology and immunoprofile except for the absence of fat component.

The immunostaining profile of previously published cases of supratentorial central liponeurocytomas (Table 4) document uniform positivity for neuronal markers like synaptophysin [7, 9–11], and neuron-specific enolase (NSE) [10, 11]. Expression of glial markers GFAP and S-100 is limited to scattered reactive astrocytes [10, 11] and a few tumor cells along with tumor matrix [11]. The tumor cells are negative for NF [9–11], epithelial membrane antigen (EMA) [9], CK and Chg A [11]. MIB-1 labeling index was 4% [11] and 5% [9] and increased to 5.8% in the recurrent tumor [9].

George et al. suggest that lipomatous differentiation occurs in synaptophysin positive neurocytic cells with

progressive accumulation of lipid and coalescence of neurocytic cells [9]. Rim positivity for GFAP has also been demonstrated in lipidized tumor cells suggesting pluripotent immunophenotypic expression [11]. By electron microscopy, the demonstration of neurosecretory granules, and clear and dense core vesicles in some of the cells with osmiophilic lipid spaces confirms lipidization of the neurocytic cells or metaplastic transformation of neuroectodermal cells into adipocytes [10, 11].

Heterologous elements like fat, melanocytes [15] and skeletal muscle [16] have been described within central neurocytomas. Horoupian et al. suggested the origin of central neurocytoma from pluripotent progenitor cells in the germinal layers, and therefore the presence of fat cells within them is not surprising [7]. Apart from 29 reported cases of cerebellar liponeurocytoma [2], the presence of fat cells or lipidization of neuroepithelial tumor cells of the central nervous system have also been described in sporadic cases of cerebellar astrocytomas [17], multiple intraspinal low-grade astrocytoma mixed with lipoma (astrolipoma) [18], a low-grade astrocytoma variant in pediatric age (lipoastrocytoma) [19], frequently in pleomorphic xanthoastrocytoma [20], occasionally in glioblastoma [21], 88 cases of ependymomas [22], 1 case of mixed cerebral glio-neuronal tumor [23], 22 cases of supratentorial PNET [24, 25], and 1 case of extracerebellar but partly infratentorial PNET with a glioblastoma component [26].

Central neurocytomas have a benign clinical course but local recurrence follows incomplete resection. Radiotherapy is advocated for residual tumors [27, 28]. The 5-year survival rate of cerebellar liponeurocytomas is 48%, but this should be interpreted with caution because of rarity of this tumor and lack of systematic follow-up [2]. Recurrence rate in cerebellar liponeurocytomas is 31% [5], but as it occurs following long disease-free interval, repeat surgery is preferable to radiotherapy, as objective evidence of usefulness of this therapeutic modality is lacking [29]. Radiotherapy may be considered in cases with incomplete resection or in tumors with high proliferation index documented on histology [6]. Of the six reported cases, one recurred following partial excision [9] and the rest were lost to follow-up. Similarly in the present study, one case recurred after 9 years and 4 months of initial surgery, and rest of the cases were lost to follow-up. An increase in lipomatous component was demonstrable both radiologically and histologically in supratentorial tumors, similar to cerebellar liponeurocytomas [30], reflecting slow evolution of lipomatous metaplasia. Following molecular and genetic studies, an International Consortium evaluating 20 cases of cerebellar liponeurocytomas, suggested a relationship to central neurocytomas, with high frequency of TP53 missense mutations [31].

Conclusion

We suggest a change in nomenclature of these tumors to 'liponeurocytoma' rather than the restrictive 'cerebellar liponeurocytomas', to encompass all the sites of occurrence. Defining the exact prognosis of these rare tumors and determine management protocols awaits documentation of more number of cases with long-term follow-up.

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