Published in final edited form as:

Histopathology. 2020 January ; 76(2): 275–282. doi:10.1111/his.13967.

Intracranial Cellular Schwannomas: A Clinicopathologic Study of 20 Cases

Felipe D’Almeida Costa¹, Tiago Miranda Dias¹, Kara A. Lombardo², Aditya Raghunathan⁴, Caterina Giannini⁴, Lawrence Kenyon⁵, Ali G. Saad⁶, Murat Gokden⁷, Peter C. Burger², Elizabeth A. Montgomery², Fausto J. Rodriguez^{2,3}

¹Department of Anatomic Pathology, A.C. Camargo Cancer Center, Sao Paulo, Brazil

²Department of Pathology, Johns Hopkins University, Baltimore, MD

³The Sidney Kimmel Comprehensive Cancer Center, Johns Hopkins University, Baltimore, MD

⁴Department of Pathology, Mayo Clinic, Rochester, MN

⁵Department of Pathology, Thomas Jefferson University Hospital, Philadelphia, PA

⁶Department of Pathology, University of Mississippi, Jackson, MS

⁷Department of Pathology, University of Arkansas, Little Rock, AR

Abstract

Aims: Cellular schwannoma is a specific subtype of schwannoma, prone to misinterpretation as a malignant neoplasm. Involvement of the intracranial compartment by these tumors is very rare. We aim to characterize this clinicopathologic subgroup.

Methods and Results: We identified a total of 20 cellular schwannomas with predominant intracranial involvement. The mean age of the patients at the time of surgery was 37 years (range 16–81), with a slight female predominance (1.5:1 ratio). The most common sites were the 8th (n=8) and 5th (n=6) cranial nerves. Three tumors involved the anterior cranial fossa/olfactory groove, and a single case involved the glossopharyngeal nerve. All tumors met established criteria for cellular schwannoma, and were composed of interlacing fascicles of spindle cells lacking Verocay bodies with minimal Antoni B pattern, and variable chronic inflammation and foamy histiocytes. Rare findings included hemosiderin deposition (n=6), necrosis (n=4), brisk mitotic

Correspondence: Fausto J. Rodriguez, MD, Department of Pathology, Division of Neuropathology, Johns Hopkins University School of Medicine, Sheikh Zayed Tower, Room M2101, 1800 Orleans Street, Baltimore, MD 21231, frodrig4@jhmi.edu.

Author Contributions

Felipe D’Almeida Costa: collected and analyzed data, study planning and conception, wrote the manuscript.

Tiago Miranda Dias: collected and analyzed data, participated in writing the manuscript.

Kara A. Lombardo: analyzed data, reviewed and corrected manuscript.

Aditya Raghunathan: collected data, reviewed and corrected manuscript.

Caterina Giannini: collected data, reviewed and corrected manuscript.

Lawrence Kenyon: collected data, reviewed and corrected manuscript.

Ali G. Saad: collected data, reviewed and corrected manuscript.

Murat Gokden: collected data, reviewed and corrected manuscript.

Peter C. Burger: collected data, reviewed and corrected manuscript.

Elizabeth A. Montgomery: collected data, reviewed and corrected manuscript.

Fausto J. Rodriguez: analyzed and collected data, study planning and conception, participated in writing the manuscript.

Conflicts of interest: none.

activity (>10 mitoses per 10 high power fields)(n=2), focal epithelioid morphology (n=2), myxoid areas (n=2), neuroblastoma-like pattern (n=1) and granular cells (n=1). Immunohistochemical stains demonstrated expression of Schwann cell markers (S100 protein, SOX10, Collagen IV) and preserved H3 K27 trimethylation in all cases tested. Fourteen patients had postoperative follow-up ranging from 2 months to 21 years (mean 66 months). In patients with follow-up, local recurrence/persistence developed in 6 cases; 5 tumors were initially incompletely resected. No metastatic disease or deaths were reported.

Conclusions: Intracranial cellular schwannomas share morphologic and immunophenotypic features with cellular schwannomas at others sites, may demonstrate locally aggressive growth but appear to lack metastatic potential.

Keywords

Cellular Schwannoma; NF2; Intracranial; H3 K27me3; MPNST

Introduction

Cellular schwannomas (CS) are benign nerve sheath tumors that demonstrate a striking predominance of hypercellular (Antoni A) areas, scant or absent hypocellular (Antoni B) areas and no recognizable well formed Verocay bodies¹. In addition, they can show increased mitotic activity, small necrotic foci and signs of destructive growth, such as bone erosion². The presence of all these features in a spindle cell neoplasm commonly causes diagnostic difficulty and can easily lead the pathologist to mistake the tumor for malignancy. These tumors occur more often in females and the most common sites of involvement are the posterior mediastinum and retroperitoneum. Intracranial examples are relatively rare, with reported frequencies ranging from 0 to 11% of all cellular schwannomas in published series²⁻⁷. We reviewed our experience with cellular schwannomas involving the intracranial compartment, and performed a critical review of the literature to better define the clinical features of these patients and to describe the pathological findings of this uncommon tumor, which may constitute a diagnostic challenge.

Materials and methods

Patients

Clinical charts and pathology records were reviewed. All available slides were reviewed by at least 3 observers. The inclusion criteria included: (1) tumor location intra-axial or extra-axial, supratentorial or infratentorial; (2) tumor histology consistent with cellular schwannoma, as defined by the fourth edition of the World Health Organization (WHO) Classification of Tumours of Soft Tissue and Bone¹. Twenty cases diagnosed in a twenty year period were included in this study. Appropriate institutional review board approval was obtained for the study and informed consent or waiver of informed consent per IRB guidelines was followed.

Histopathology and Immunohistochemistry

Hematoxylin and eosin-stained slides and immunohistochemical slides were reviewed in all cases. In cases with unstained slides or paraffin blocks available, additional 4- μ m-thick sections were obtained for immunohistochemistry. The following antibodies were performed systematically in a subset of cases using automated instruments SOX10 (clone N-20; Santa Cruz Biotechnology, Dallas, TX; 1:100 dilution), CD34 (clone QBEnd/10; Ventana, Tucson, AZ; prediluted) and H3 K27 trimethylation (clone C36B11, Cell Signaling Technology, Danvers, MA; 1:100 dilution).

Statistical analysis

Frequencies, mean and median values were calculated as appropriate. Descriptive statistics and frequencies were generated for nominal and ordinal variables, respectively using Microsoft Excel.

Results

Clinical features

A total of 18 patients satisfied criteria for inclusion, identified among approximately 198 (9%) cellular schwannoma diagnoses in the Johns Hopkins pathology database in the study period (1998–2018), with two additional cases obtained at the Mayo Clinic.

Clinicopathologic features are outlined in Table 1. The tumors developed in 12 women and 8 men (1.5:1 ratio). The mean age of the patients at the time of surgery was 37 years (range 16–81). One patient had neurofibromatosis type 2 (NF 2), and a second case had a coexisting meningioma but no other clinical criteria for NF2. One patient had POTS (postural orthostatic tachycardia syndrome), one had Asperger syndrome and one was diagnosed with mental retardation.

The most common sites were the 8th (n=8) and 5th (n=6) cranial nerves. Three tumors involved the anterior cranial fossa/olfactory groove. One tumor involved cranial nerve IX (glossopharyngeal nerve). One lesion was completely intraparenchymal within the brain and one tumor presented as a dominant mass in the falx cerebri, with smaller satellite lesions. Information about preoperative tumor size was present in 12 cases. The maximum dimension of the tumors ranged from 1.0 cm to 6.8 cm (mean 3.2 cm).

Pathologic and immunohistochemical findings

All tumors included in this study were hypercellular spindle cell neoplasms. The cells were arranged predominantly in short interlacing fascicles (Figure 1A–C), in some areas demonstrating a storiform pattern. Most of the nuclei were elongated, with a mixture of plump and wavy nuclear morphologies. Diffuse and uniform hyperchromasia was not observed; only scattered enlarged and hyperchromatic tumor cells with a smudgy chromatin pattern were focally seen in 3 cases (21%), reminiscent of the degenerative changes found in “ancient” schwannomas. Hyalinized vessels were also a focal feature detected in 15 cases (75%) (Figure 1B). Foamy macrophages were present in 12 cases, usually as a focal finding or present as a subtle interstitial infiltrate (60%). Ten cases (50%) exhibited patchy lymphoid aggregates around vessels. Other findings included hemosiderin deposition (n=6), necrosis

(n=4), focal epithelioid morphology (n=2), myxoid areas (n=2), neuroblastoma-like pattern (n=1) and granular cell change (n=1) (Figures 2 and 3). Necrosis was coagulative and encompassed approximately 10–15% of the areas examined in 3 cases, and formed a small focus in one case (<1%). The neuroblastoma-like pattern was characterized by small cells with high nuclear:cytoplasmic ratios and low proliferation. The granular cell change was composed of PAS and CD68 positive granules, consistent with lysosomal accumulation (Figure 3). The mitotic count ranged from <1 to 12 mitoses per 10 high power fields (HPF), with a median of 1 per 10 HPF. Dense pericellular reticulin deposition was identified in 7 (of 7) cases tested.

Immunohistochemically, all cases showed consistent expression of at least one Schwann cell marker (Figures 1 and 3), including strong and diffuse nuclear and cytoplasmic staining for S100 protein (20/20), SOX10 (9/10) and pericellular collagen IV deposition (9/10). Three (of 7) cases tested were positive for CD34 while EMA was negative in 9/9 cases. The proliferative index measured by Ki-67 staining (tested in 12) ranged from 5 to 20% (mean 10%). H3 K27me3 immunostain, performed in 10 cases, was completely retained in 8 (Figure 1F) and partially lost in 2. No cases showed complete H3 K27me3 loss as classically described in most MPNST. INI1 expression was preserved in 1/1 cases with epithelioid morphology tested.

Clinical Outcome

Twelve patients had postoperative follow-up ranging from 2 months to 21 years (mean 66 months) (Table 1). Two were treated with radiation therapy and 1 case with radiation therapy and temozolomide for progression. Local recurrence/tumor regrowth developed in 6 patients, 5 of whom had a subtotal resection. Two cases with brisk mitotic activity exceeding 10 mitoses per 10 high power fields were present in our cohort. One manifested a recurrence 21 years after a subtotal resection. The second patient is asymptomatic with no evidence of recurrence 9 months after a gross total resection. No metastatic disease or deaths were reported in any case during the follow-up period.

Discussion

Intracranial schwannomas are rare benign neoplasms and can show different histologic patterns and variants. The cellular variant is probably the subtype most commonly mistaken for a malignant tumor, because it usually displays concerning histological features that include hypercellularity, increased mitotic activity and necrotic foci⁸.

Cellular schwannomas of the intracranial space are rarer than those developing in peripheral sites, with only rare cases reported in the literature to date, some of them as part of general cellular schwannomas series^{2–7} and others as single case reports convincingly illustrated^{9–15}. Patients in this current series presented with intracranial tumors at a slightly younger age (mean 38 years) compared to cellular schwannoma in general reported in the literature, age ranging from 40 to 55 years in 4 studies^{2, 3, 5, 6}, with an average age of 40.9 years in another study⁴. There was a slight female predilection in our series, which is in accordance with series including tumors inside and outside the intracranial space reporting a

predominance in females^{3, 5, 6}. Two studies showed no difference between males and females (M:F 1:1)^{2, 4}.

The most prevalent parent nerves that were identifiable in the current series were the 8th and 5th cranial nerves, with almost equal occurrence (8 and 6 cases respectively). This is of interest, since the vestibular (8th) nerve is greatly favored in conventional intracranial schwannoma. Three cases developed in the anterior cranial fossa underneath the frontal lobes. One possible parent nerve for these tumors is the nervus terminalis or “cranial nerve 0”, a peripheral intracranial nerve branch composed of unmyelinated fibers, and coursing in the subarachnoid space overlying the gyrus rectus¹⁶.

All 20 cases described here demonstrated consistent histological features, according to the WHO definition of cellular schwannoma, as described above, and their characteristics did not differ from those in other reported cases. The hypercellularity, interlacing fascicles arrangement and increased mitotic activity of cellular schwannomas can lead the pathologist to misdiagnose this tumor as a malignant neoplasm. Regardless of the worrisome overall features of cellular schwannomas, there are some findings that can aid in the correct diagnosis, some of them also present in conventional schwannomas. These microscopic findings are the presence of foamy histiocytes, perivascular lymphocytes, hyalinized vessels and hemosiderin depositions. A careful histological examination is usually needed, since most of these features are only focally present. Unusual features can also be found as part of the morphological spectrum of this tumor, such as epithelioid cytomorphology and aggregates of granular cells, both of these characteristics already described in a previous report¹⁷. Epithelioid morphology may be of concern in schwannomas given that it is one of the forms that malignant transformation takes¹⁸. However, this finding was unassociated with significant atypia or proliferation, and epithelioid change may still be a feature of benign schwannomas¹⁹. In some sites, especially the sinonasal tract, cellular schwannomas can have an infiltrative growth pattern, adding to their mimicry of malignant neoplasms²⁰.

Another useful tool for the correct diagnosis is immunohistochemistry. The hallmark of all schwannomas, including cellular schwannoma, is diffuse S100 positivity, present in all cases in this series. Pericellular collagen IV immunoreactivity was present in almost all cases (9 of 10), which is particularly useful as a marker of Schwann cells, since S100 may be positive in a variety of spindle cell mimics. Another helpful marker is SOX10²¹, expression of the latter being present in all but one of our cases tested.

The main differential diagnosis for the tumors discussed here is MPNST, which can also arise in intracranial sites and in association with cranial nerves²². None of the patients in our cohort died of disease during the follow-up period, although recurrence/persistence developed in a subset, primarily tumors that had been incompletely resected. We have previously reported a patient who died of leptomeningeal dissemination of a low grade Schwann cell tumor with morphologic and immunophenotypic features of cellular schwannoma²³, and therefore it appears to have behaved in a malignant fashion. Leptomeningeal dissemination or metastases were not present in any of the cases of the current series. One patient (#12) presented with a dominant mass that was resected and smaller presumed satellite lesions in the vicinity that were not sampled. The satellite lesions

are growing slowly at last follow-up, but the residual dominant mass decreased in size after postoperative chemoradiation.

A recently recognized relatively specific marker for MPNST is loss of H3 K27 trimethylation, which occurs in 70–90% of the cases but is not a feature of MPNST mimics, including cellular schwannoma²⁴. This is a consequence of mutations in the polycomb repressive complex (PRC2) components *SUZ12* or *EED*, present in the majority of MPNST^{25, 26}. None of the nine cases tested in the current series demonstrated the complete loss of H3 K27 trimethylation in neoplastic cells typical of MPNST, although two cases demonstrated partial loss, which overlaps with the findings in MPNST²⁴. Melanocytic neoplasms represent another entity in the differential diagnosis. However, the morphologic features, the frequent nerve association and the immunophenotype argued against this possibility.

In conclusion, the cellular variant is an important subtype of schwannoma to be recognized. In the intracranial compartment it may be associated with persistence/local recurrence, particularly after subtotal resections, but does not seem to have metastatic potential in our experience. Cellular schwannomas at other sites are also known for their potential for local recurrence, although complete excision may be more of a challenge in intracranial locations. A careful histologic examination and immunohistochemical characterization usually reveal key features that aid in the correct diagnosis and appropriate management.

Acknowledgments

This work was supported in part by the King Khaled Eye Specialist Hospital, Riyadh, Saudi Arabia (to FJR) and NIH grant P30 CA006973 to the Sidney Kimmel Comprehensive Cancer Center (PI: W. Nelson).

References

1. Fletcher CD, Unni KK, Mertens F. Who classification of tumours. Pathology and genetics of tumours of soft tissue and bone. . 4th ed. Lyon: IARC press, 2013.
2. Woodruff JM, Godwin TA, Erlandson RA, Susin M, Martini N. Cellular schwannoma: A variety of schwannoma sometimes mistaken for a malignant tumor. *Am J Surg Pathol* 1981;5;733–744. [PubMed: 7337161]
3. Casadei GP, Scheithauer BW, Hirose T, Manfrini M, Van Houton C, Wood MB. Cellular schwannoma. A clinicopathologic, DNA flow cytometric, and proliferation marker study of 70 patients. *Cancer* 1995;75;1109–1119. [PubMed: 7850709]
4. Fletcher CD, Davies SE, McKee PH. Cellular schwannoma: A distinct pseudosarcomatous entity. *Histopathology* 1987;11;21–35. [PubMed: 3557324]
5. Lodding P, Kindblom LG, Angervall L, Stenman G. Cellular schwannoma. A clinicopathologic study of 29 cases. *Virchows Arch A Pathol Anat Histopathol* 1990;416;237–248. [PubMed: 2105560]
6. White W, Shiu MH, Rosenblum MK, Erlandson RA, Woodruff JM. Cellular schwannoma. A clinicopathologic study of 57 patients and 58 tumors. *Cancer* 1990;66;1266–1275. [PubMed: 2400975]
7. Deruaz JP, Janzer RC, Costa J. Cellular schwannomas of the intracranial and intraspinal compartment: Morphological and immunological characteristics compared with classical benign schwannomas. *J Neuropathol Exp Neurol* 1993;52;114–118. [PubMed: 8095064]
8. Rodriguez FJ, Folpe AL, Giannini C, Perry A. Pathology of peripheral nerve sheath tumors: Diagnostic overview and update on selected diagnostic problems. *Acta Neuropathol* 2012;123;295–319. [PubMed: 22327363]

9. Andrade GC, Paiva Neto MA, Braga FM. [thalamic intracerebral schwannoma: Case report]. *Arq Neuropsiquiatr* 2002;60;308–313. [PubMed: 12068368]
10. Erlich SA, Tymianski M, Kiehl TR. Cellular schwannoma of the abducens nerve: Case report and review of the literature. *Clin Neurol Neurosurg* 2009;111;467–471. [PubMed: 19200646]
11. Esposito F, Cappabianca P, Del Basso De Caro M, Cavallo LM, Rinaldi C, De Divitiis E. Endoscopic endonasal transsphenoidal removal of an intra-suprasellar schwannoma mimicking a pituitary adenoma. *Minim Invasive Neurosurg* 2004;47;230–234. [PubMed: 15346320]
12. Koyye RT, Mahadevan A, Santosh V et al. A rare case of cellular schwannoma involving the trigeminal ganglion. *Brain Tumor Pathol* 2003;20;79–83. [PubMed: 14756445]
13. Mariniello G, Horvat A, Popovic M, Dolenc VV. Cellular dumbbell schwannoma of the hypoglossal nerve presenting without hypoglossal nerve palsy. *Clin Neurol Neurosurg* 2000;102;40–43. [PubMed: 10717403]
14. Rallis G, Stathopoulos P, Mezitis M, Sakellaris N, Machera H, Zachariades N. Combined craniofacial approach for the removal of a large trigeminal schwannoma invading the infratemporal fossa. *Oral Maxillofac Surg* 2012;16;211–216. [PubMed: 21842149]
15. Schlieter M, Zoubaa S, Kress B, Unterberg A, Jacobi C, Hahnel S. Hemorrhagic acoustic schwannoma: Radiological and histopathological findings. *J Neuroradiol* 2005;32;210–212. [PubMed: 16134303]
16. Fuller GN, Burger PC. Nervus terminalis (cranial nerve zero) in the adult human. *Clin Neuropathol* 1990;9;279–283. [PubMed: 2286018]
17. Carpenter PM, Grafe MR, Varki NM. Granular cells in a cellular neurilemmoma. *Arch Pathol Lab Med* 1992;116;1083–1085. [PubMed: 1417449]
18. McMenamin ME, Fletcher CD. Expanding the spectrum of malignant change in schwannomas: Epithelioid malignant change, epithelioid malignant peripheral nerve sheath tumor, and epithelioid angiosarcoma: A study of 17 cases. *Am J Surg Pathol* 2001;25;13–25. [PubMed: 11145248]
19. Hart J, Gardner JM, Edgar M, Weiss SW. Epithelioid schwannomas: An analysis of 58 cases including atypical variants. *Am J Surg Pathol* 2016;40;704–713. [PubMed: 26752543]
20. Hasegawa SL, Mentzel T, Fletcher CD. Schwannomas of the sinonasal tract and nasopharynx. *Mod Pathol* 1997;10;777–784. [PubMed: 9267819]
21. Karamchandani JR, Nielsen TO, van de Rijn M, West RB. Sox10 and s100 in the diagnosis of soft-tissue neoplasms. *Appl Immunohistochem Mol Morphol* 2012;20;445–450. [PubMed: 22495377]
22. Scheithauer BW, Erdogan S, Rodriguez FJ et al. Malignant peripheral nerve sheath tumors of cranial nerves and intracranial contents: A clinicopathologic study of 17 cases. *Am J Surg Pathol* 2009;33;325–338. [PubMed: 19065105]
23. Rodriguez EF, Blakeley J, Langmead S et al. Low-grade schwann cell neoplasms with leptomeningeal dissemination: Clinicopathologic and autopsy findings. *Hum Pathol* 2017;60;121–128. [PubMed: 27666764]
24. Schaefer IM, Fletcher CD, Hornick JL. Loss of h3k27 trimethylation distinguishes malignant peripheral nerve sheath tumors from histologic mimics. *Mod Pathol* 2016;29;4–13.
25. Lee W, Teckie S, Wiesner T et al. Prc2 is recurrently inactivated through eed or suz12 loss in malignant peripheral nerve sheath tumors. *Nat Genet* 2014;46;1227–1232. [PubMed: 25240281]
26. Zhang M, Wang Y, Jones S et al. Somatic mutations of suz12 in malignant peripheral nerve sheath tumors. *Nat Genet* 2014;46;1170–1172. [PubMed: 25305755]

Figure 1. Histologic and immunohistochemical features of intracranial cellular schwannoma. All tumors demonstrated increased cellularity with a lack of Verocay bodies (A-C). Hyalinized vessels were present in a subset of cases (B). Strong expression of S100 protein was present in all cases (D) and pericellular collagen IV in most (E). H3 K27 trimethylation showed preserved immunoreactivity, at least in part, in all cases (F).

Figure 2. Unusual findings in intracranial cellular schwannoma.

Morphologic features of note present in rare cases included myxoid change (A), focal epithelioid morphology (B), cellular component with cells with high nuclear:cytoplasmic ratios but low proliferation ('neuroblastoma-like')(C) and brisk mitotic activity. This example showed greater than 10 mitoses in 10 high power fields (arrows)(D).

Figure 3. Intracranial cellular schwannoma with granular cell change.

This neoplasm developed in a 51-year old woman, and presented as a large contrast enhancing mass in the anterior cranial fossa (A). The neoplasm had a conventional cellular schwannoma component, including spindle cells (B) and aggregates of histiocytes (C). Granular cell change was conspicuous in areas (D) and composed of bright PAS granules (E). SOX10 was strongly expressed (F).

Table 1.

Clinicopathologic features of intracranial cellular schwannomas

Case	Age	Sex	Location	Size*	Extent of Resection	Other disorders	Additional Treatment	Follow-up (months)	Follow-up
1	34	F	CN VIII	2.8	STR	NF2 history		>250	Recurrence [‡]
2	31	M	CN VIII	3.5	GTR				LFU
3	49	M	CN VIII	2	NOS				LFU
4	16	M	CN VIII	1.4	STR	Asperger syndrome		11	Stable disease (1.4 cm), unchanged from postop MRI
5	20	F	CN VIII	5.5	GTR	mental retardation		11	NED
6	40	F	CN VIII	NA	STR		Surgery	7	Additional surgery for tumor regrowth
7	44	M	CN VIII	2.3	GTR			2	NED
8	38	F	CN VIII	NA	GTR	Coexisting L petroclival meningioma, no evidence of NF2		84	NED
9	16	F	CN V - Parasellar	6.8	GTR			28	NED
10	46	M	CN V -cavernous sinus	4	STR				LFU
11	16	M	CN V -Meckel's cave	1	NOS	POTS syndrome		10	NED
12	28	F	CN V-Posterior fossa	NA	STR		Radiation therapy	8	Tumor growth despite treatment
13	39	F	CN V	NA	GTR		Radical surgery	>220	Recurrences at 7 and 14 years. Currently NED after radiosurgery
14	42	F	Skull base/ infratemporal fossa, compressing CN V,VII	NA	STR				LFU
15	51	F	Anterior skull base	4.3	GTR			24	NED
16	49	F	Anterior skull base	NA	GTR			120	NED
17	81	F	Anterior skull base/Olfactory groove	NA	NOS				LFU
18	20	F	CN IX	NA	STR			148	Surgeries for progression at 1 and 4 years, currently NED
19	23	M	Occipital lobe(Intraparenchymal)	1.5	GTR				LFU
20	68	M	Falk Cerebri	3.3	STR		TMZ +irradiation (50Gy) on progression	5	Progression [‡] after 6 weeks on MR. Treated with chemoradiation. MRI at 5 months:progression of 2 right frontal

Case	Age	Sex	Location	Size [*]	Extent of Resection	Other disorders	Additional Treatment	Follow-up (months)	Follow-up parafoalpine lesions, reduction in the size of the residual callosal component

^{*} Maximum dimension in cm, GTR gross total resection, STR subtotal resection, NOS not otherwise specified, LFU lost to follow-up, NED no evidence of disease, recurrence refers to overt tumor growth after GTR or generous resection, while progression refers to progressive growth of evident residual tumor on clinical follow-up.