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Technical Notes & Surgical Techniques

Chordoid glioma of the third ventricle: A systematic review and single-center experience

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ABSTRACT

Background: Chordoid gliomas (CG) are neoplasms within the anterior part of the third ventricle.
Objective: To analyze all reported cases of CG with regard to imaging features, neurosurgical approaches, VP-shunt dependency, immunohistochemistry and follow-up. Furthermore, we present three cases.
Methods: Clinical symptoms, MRI features and immunohistochemistry were analyzed in 70 cases. Univariate analyses of extent of resection, surgical approaches, recurrence rates and postoperative hydrocephalus were performed. Three cases are reported with focus on clinical presentation, surgical results, immunohistochemistry and follow-up.
Results: Headache (51.4%), visual deterioration (41.4%) and memory deficits (21.4%) are the main symptoms. MRI shows a homogenously enhancing solid mass in 84.8% with cysts in 20% of all reported cases. 20% of the patients treated by a subtotal resection (STR) and 0% by a gross total resection (GTR) had a recurrence ($p = 0.034$). VP-Shunt dependency was higher in the STR group (19.2%) compared with GTR group (5%; $p = 0.067$). Rate of GTR of the subfrontal trans-lamina terminalis route (83.3%) was superior to the inter-hemispheric transcallosal approach (23.1%; $p = 0.041$). GFAP (100%), Vimentin (100%) and CD 34 (69.6%) are the strongest immunohistochemical markers. Two female and one male patient were admitted to our department. However, in one case recurrence ouccured at 18 months after STR.
Conclusion: Despite the benign histopathology, CGs have high recurrence rates. Morphologically, CGs are solid homogenously enhancing tumors. GTR should be achieved, preferably by the trans-lamina terminalis route. Anatomy and immunohistochemistry seem to show tanycytic origin of this entity.

1. Introduction

Chordoid glioma (CG) is a benign, rare and noninvasive primary neuroepithelial central nervous system tumor. According to the World Health Organization (WHO) it is classified as a grade II tumor. The first classification by the WHO was done in 2000.

Brat et al. [1] was the first one who described the tumor in 1998. The characteristics of this tumor were first described by Wanschitz et al. in 1995 [2]. CGs are characterized by the presence of glial and chordoid components. The histogenesis of this entity is unknown so far [3]. The typical location of this tumor is within the suprasellar region and the anterior part of the third ventricle at the lamina terminalis. Often the tumor is also extending into the hypothalamus [4]. The most common presentation is a uniformly Gd-enhancing lesion on contrast-enhanced T1-weighted MRI, and slightly hyperintensive on T2-weighted MRI. Cystic appearance can also be observed [5]. Histologically, CGs are composed of oval-to-polygonal epitheloid cells with abundant eosinophilic cytoplasm organized in clusters and cords within a mucinous matrix [6]. Immunohistochemically, the tumor is positive for glial fibrillary acidic protein (GFAP), CD34, and Vimentin suggesting an origin from tancycytes which are located in the subcomissural organs [7]. CG is negative for p53 and tumor suppressors or proto-oncogenes

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like TP53, EGFR, CDK4 and MDM2 genes [8]. Molecular losses at the loci 9p21 and 11q13 were found with gains of 11q13 at 11q14 [9]. Due to the typical location of the CG patients present with symptoms of intracranial hypertension because of obstructive hydrocephalus. Hypothalamic dysfunction and visual impairment are also common symptoms.

An elevated morbidity has to be suggested if neurosurgical treatment is considered, as in the treatment of the relevant differential diagnoses within this anterior portion of the 3rd ventricle like ependymomas, pilocytic astrocytoma, central neurocytomas, colloid cysts, craniopharyngiomas and suprasellar meningiomas. Extent of resection (EoR) and the postoperative functional outcome has to be carefully balanced due to the assumption that it is a benign tumor and long-term follow up data of recurrence rates are not available so far. This systematic review analyzed the current data from 70 patients in the literature to identify the initial clinical features, MR imaging features, extent of resection, neurosurgical approaches, postoperative ventriculoperitoneal (VP)-shunt dependency, mortality and immunohistochemical data. Furthermore, we present three cases who were admitted to our neurosurgical department for tumor resection.

2. Methods

2.1. Literature search

To identify the relevant reports on CGs the keywords “chordoid” and “glioma” were used to search on MEDLINE (OVID and Pubmed), Embase, Scopus, and Web of Science. Two reviewers independently extracted data. Full-text versions were obtained from all studies that were considered to be potentially relevant by both reviewers. The references of all relevant studies were searched manually for additional studies until no further publications were found. Any disagreement between the reviewers concerning article inclusion or exclusion was resolved by consensus of a third author. All studies published until September of 2018 were analyzed. Manuscripts which were not written in english were excluded. Following figure (Fig. 1) briefly summarizes the methods and workflow of the literature research. All histopathologically confirmed cases of CGs were included if also appropriate data on initial clinical status of the patient, MRI findings, treatment and results of histopathological examination were given in detail. Follow-up was at least 2 months.

2.2. Data collection

The following variables were analyzed from each study: Age, gender, initial clinical presentation, surgical approach, EoR, adjuvant treatment, VP-Shunt dependency, immunohistochemical reactivity, perioperative mortality, mean and median follow up time, recurrence free survival and time to recurrence. Analyzed studies and main variables are shown in the Table 1. The present sudy was conducted in accordance with the ethical standards of the Declaration of Helsinki. This research did not receive any specific grant from funding agencies in the public, commercial, or not-for profit sectors.

2.3. Statistical design

Statistical analysis was performed with SPSS version 23.0 (Armonk, NY: IBM Corp). The rates of recurrence of gross total (GTR) and subtotal resection (STR) were analyzed with the Kaplan-Meier method and log rank tests. Chi-squared tests (2-sided) were performed to study VP-shunt dependency after gross total or subtotal resection. Fisher's exact tests (2-sided) were also performed to analyze the surgical approaches and EoR. Comparing the means of groups was done by performing the student's *t*-test. $P < 0.05$ was considered as statistically significant.

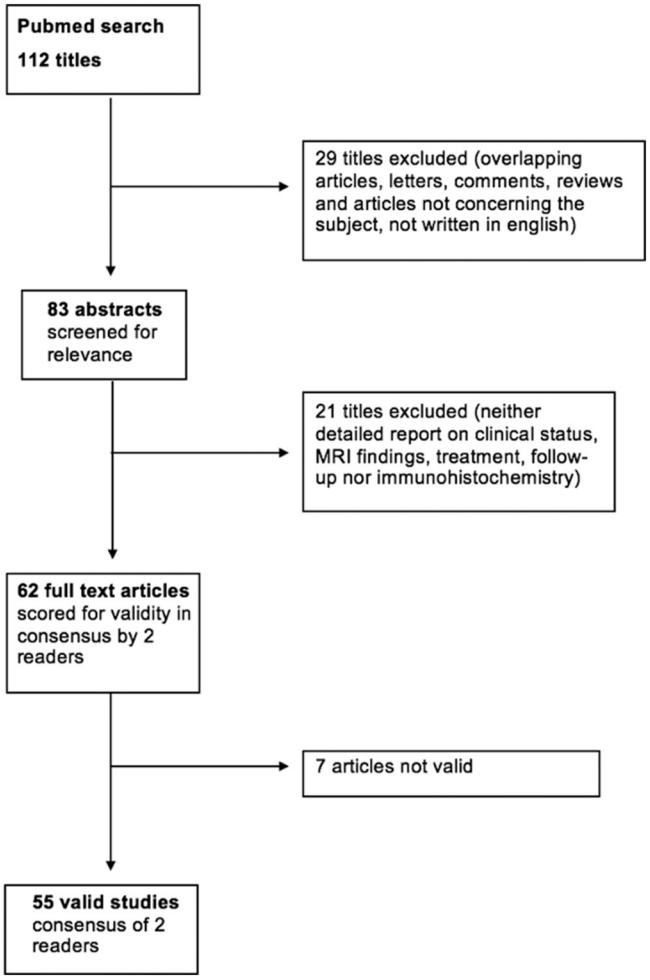

Fig. 1. Flow chart showing search strategy.

3. Results

A total of 70 patients met study criteria and were included in our analysis. There were 45 female (64.3%) and 25 male (35.7%) patients. The male:female ratio was 1:1.8. The patients' age ranged from 5 to 71 years (mean age and Standard Deviation (SD); 44.4 ± 13.171). 50% of patients were between 36 and 53 years old. 4 cases (5.7%) were seen in pediatric patients who were under 18 years old [29,31,40,42].

3.1. Clinical presentation

The most common symptoms, in order, were headache, visual deterioration, memory deficits, nausea and somnolence (for details see Table 2). Less frequent symptoms were urinary incontincence, ataxia, dizziness or polyuria. Papilledema, considered as a sign of increased intracranial pressure was seen in 3 (4.2%) cases. Endocrine dysfunctions like SIADH [11], Diabetes mellitus [18] and amenorrhea [1,6] were also reported.

3.2. MRI features and advanced imaging methods

The CG typically appears as a round solid and ovoid mass within the anterior part of the third ventricle. Generally, the most common MRI finding is the following: A homogenously Gd-enhancing lesion which is hypointense or isointense on T1 weighted native imaging and isointense or hyperintense on T2 weighted imaging. Necrosis is no typical sign of CG. Cysts (20%) and calcifications (8.6%) were also seen. Frequency of MRI characteristics is shown in Table 3. Data concerning

Table 1
Patients' data.

Source	Gender	Age	Presentation	Immunohistochemistry	Surgical approach	EoR	Follow-up	Perioperative mortality	Postoperative VP-Shunt
Bongetta et al. 2015 [10]	f	43	Headache, lethargy, papilledema	GFAP, Vimentin	Pterional-trans-lamina terminalis	GTR	NA	Yes (pneumonia)	Yes
Calanchini et al. 2016 [11]	f	48	SIADH	GFAP, Cam 5.2, TTF-1	Transcortical	STR	12 PF	No	No
Erwood et al. 2017 [12]	f	46	Headache, nausea, memory deficits	GFAP, EMA, CK, D2–40, TTF-1	Transcortical	STR	2 R	No	Yes
Ki et al. 2016 [13]	m	34	Headache	GFAP, CD34, Vimentin	Interhemispheric transcallosal	GTR	45 RF	No	No
Qixing et al. 2015 [14]	f	48	Visual deterioration	GFAP, CD34	Subfrontal trans-lamina terminalis	GTR	12 RF	No	No
Qixing et al. 2015 [14]	m	27	Headache, visual deterioration, abducens paresis	GFAP, CD34	Subfrontal trans-lamina terminalis	GTR	3 RF	No	No
Thavaratnam et al. 2015 [15]	f	30	Bitemporal hemianopsia	GFAP	Subfrontal trans-lamina terminalis	GTR	2 RF	No	No
Yang et al. 2017 [16]	f	50	Memory deficits, visual deterioration	GFAP, CK, Vimentin	Interhemispheric transcallosal	STR	14 PF	No	Yes
Al-Zubidi et al. 2014 [17]	m	37	Visual deterioration, bitemporal hemianopsia	GFAP, S-100, PanCK	NA	STR	6 PF	No	No
Shiramizu et al. 2013 [18]	m	49	Diabetes mellitus, arterial hypertension, memory deficits	NA	Interhemispheric transcallosal	STR	24 R	No	No
Scheurkogel et al. 2012 [19]	m	30	Visual deterioration, bitemporal hemianopsia	GFAP, CD34	Subfrontal trans-lamina terminalis	STR	NA	No	No
Can et al. 2012 [20]	m	37	Hemiparesis, headache, memory deficits	GFAP, CD34, S-100	NA	GTR	NA	No	No
Ghosal et al. 2012 [21]	m	48	Ataxia, urinary incontinence, papilledema, aphasia	GFAP	Pterional trans-lamina terminalis	STR	8 R	No	Yes
Kawasaki et al. 2009 [22]	m	51	Visual deterioration	GFAP, Vimentin	NA	STR	40 PF	No	No
Kawasaki et al. 2009 [22]	f	42	Headache, somnolence, visual deterioration	GFAP, Vimentin	NA	GTR	72 RF	No	No
Nga et al. 2006 [23]	f	49	Somnolence, hemiparesis, visual deterioration	GFAP, CD34	Transcortical	GTR	NA	Yes (pneumonia)	NO
Hsu et al. 2005 [24]	m	50	Headache, memory deficits	GFAP, Vimentin	NA	GTR	NA	No	No
Buccoliero et al. 2004 [25]	f	56	Ataxia	GFAP, NFP, Vimentin, CD34	NA	GTR	8 RF	No	No
Lee et al. 2002 [26]	f	48	Headache, dizziness	GFAP, Vimentin, S-100	Pterional trans-lamina terminalis	GTR	5 RF	No	No
Chung et al. 2007 [27]	m	48	Headache, memory deficits, ataxia	GFAP, S-100	Interhemispheric transcallosal	GTR	18 RF	No	No
Carrasco et al. 2008 [28]	f	53	Visual deterioration, memory deficits, dizziness, bitemporal hemianopsia	GFAP	Pterional trans-lamina terminalis	GTR	2 RF	No	No
Castellano-Sanchez et al. 2000 [29]	m	36	Visual deterioration	GFAP, Vimentin	Pterional trans-lamina terminalis	STR	NA	No	No
Castellano-Sanchez et al. 2000 [29]	m	12	Visual deterioration	GFAP, S-100	Pterional trans-lamina terminalis	GTR	NA	No	No
Sanches et al. 2012 [30]	f	59	Headache	GFAP, Vimentin	Stereotactic Biopsy	–	NA	No	–
Morais et al. 2015 [31]	f	13	Headache	GFAP, EMA	Transcortical	GTR	NA	No	No
Sugita et al. 2010 [32]	m	55	Headache, visual deterioration	Vimentin, CD34	Interhemispheric transcallosal	STR	36 PF	No	No
Molnar et al. 2008 [33]	f	34	Visual deterioration, headache, nausea	GFAP, S-100	NA	STR	NA	Yes (cardiac failure)	No
Al Hinai et al. 2011 [34]	f	50	Headache, memory deficits	GFAP, EGFR, CD45	Pterional trans-lamina terminalis	GTR	18 RF	No	No
Romero-Romero-Rojas et al. 2012 [35]	m	39	Headache, nausea, ataxie	GFAP, CD99	NA	GTR	36 RF	No	Yes
Ni et al. 2013 [6]	f	41	Headache, dizziness	GFAP	NA	GTR	NA	No	No
Ni et al. 2013 [6]	f	25	Headache, nausea	GFAP	NA	GTR	NA	No	No
Ni et al. 2013 [6]	f	35	Amenorrhea, headache	GFAP	NA	GTR	NA	No	No

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Table 1 (*continued*)

Source	Gender	Age	Presentation	Immunohistochemistry	Surgical approach	EoR	Follow-up	Perioperative mortality	Postoperative VP-Shunt
Ni et al. 2013 [6]	f	57	Somnolence, memory deficits, headache	GFAP	NA	GTR	NA	No	No
Desouza et al. 2010 [36]	f	48	Headache, visual deterioration	GFAP, CD34, S-100, Vimentin	Interhemispheric transcallosal	STR	12 PF	No	No
Desouza et al. 2010 [36]	f	36	Headache, polyuria, polydipsia	GFAP, CD34	Interhemispheric transcallosal	GTR	40 RF	No	No
Kim et al. 2010 [37]	f	27	Headache, visual deterioration	GFAP, Vimentin, S-100, NSE, CD56, EMA, CK, PDGFR-alpha, GLUT-1	Transcortical	STR	NA	Yes (respiratory failure)	No
Iwami et al. 2009 [38]	f	61	Syncope	GFAP, Vimentin, EMA, NFP	Interhemispheric transcallosal	STR	12 PF	No	No
Xian et al. 2012 [39]	f	53	Headache, visual deterioration	GFAP, Vimentin, EGFR, Merlin	NA	GTR	18 RF	No	No
Horbinski et al. 2009 [9]	m	41	Obtundation	GFAP, Vimentin, p53	NA	GTR	24 RF	No	Yes
Jain et al. 2008 [40]	f	7	Headache, vomitting	GFAP, CD34, Vimentin	NA	GTR	NA	NO	NO
Jain et al. 2008 [40]	m	55	Memory deficits, urinary incontinenze, seizures	GFAP, CD34, Vimentin, CK, EMA, S-100	NA	GTR	NA	Yes (bacterial meningitis)	No
Gallina et al. 2007 [41]	f	56	Incidental	GFAP, NFP, Vimentin, CD34	Pterional trans-lamina terminalis	GTR	48 RF	No	No
Goyal et al. 2007 [42]	m	5	Headache, vomitting	GFAP, S-100, Vimentin	NA	GTR	NA	Yes (cardiac arrest)	No
Jung et al. 2006 [43]	f	50	Cognitive dysfunction, hyperphagia, memory deficits, weight gain	GFAP, Vimentin, CD34	Interhemispheric transcallosal	STR	3 R	No	Yes
Leeds et al. 2006 [44]	m	57	Headache, malaise, anorexia	GFAP, Vimentin, EMA, CK, CD34	Stereotactic biopsy	–	NA	No	–
Raizer et al. 2003 [45]	f	57	Cognitive dysfunction	GFAP, Vimentin, CD34	Subfrontal trans-lamina terminalis	GTR	13 RF	No	No
Nakajima et al. 2003 [46]	f	49	Headache, memory deficits, urinary incontinence	GFAP	Interhemispheric transcallosal	STR	24 PF	No	No
Sato et al. 2003 [7]	f	65	Headache	GFAP, Vimentin, CD34, *E*-Cadherin	NA	STR	24 PF	No	No
Grand et al. 2002 [47]	f	41	Headache, visual deterioration	GFAP, Vimentin, CD34, EMA, S-100	Interhemispheric transcallosal	STR	NA	No	No
Pasquier et al. 2002 [48]	m	35	Headache, visual deterioration, insomnia, nausea	GFAP, Vimentin, CD34	NA	GTR	68 RF	No	No
Pasquier et al. 2002 [48]	f	39	Headache, visual deterioration	GFAP, Vimentin, CD34	NA	STR	16 PF	No	No
Cenacchi et al. 2001 [49]	m	34	NA	GFAP, Vimentin	NA	GTR	24 RF	No	No
Cenacchi et al. 2001 [49]	f	40	NA	GFAP, Vimentin	NA	GTR	36 RF	No	No
Cenacchi et al. 2001 [49]	f	43	NA	GFAP, Vimentin	NA	GTR	NA	Yes (pulmonary embolism)	No
Ricoy et al. 2000 [50]	f	41	Visual deterioration	GFAP, VImentin	Subfrontal trans-lamina terminalis	GTR	13 RF	No	No
Vajtai et al. 1999 [51]	f	60	Headache, memory deficits, somnolence	GFAP, Vimentin	Interhemispheric transcallosal	STR	NA	Yes (hyponatriemia, tracheobronchitis)	No
Baehring et al. 2006 [52]	f	71	Visual deterioration	GFAP	Stereotactic biopsy	–	NA	No	–
Vij et al. 2011 [53]	m	48	Visual deterioration, focal seizures	GFAP, CD34, CK	Pterional trans-lamina terminalis	STR	10 PF	No	No
Reifenberger et al. 1999 [8]	f	56	Headache, fatigue, scotoma	GFAP, Vimentin, CD34, S-100	Interhemispheric transcallosal	STR	42 PF	No	No
Reifenberger et al. 1999 [8]	f	31	NA	GFAP, Vimentin, CD34	NA	NA	NA	No	No
Reifenberger et al. 1999 [8]	f	53	Weight gain, bitemporal hemianopsia	GFAP, CD34, Vimentin	NA	GTR	NA	No	No
Reifenberger et al. 1999 [8]	m	65	Speech problems, facial weakness	GFAP, Vimentin, CD34, EMA	NA	GTR	NA	Yes (pulmonary embolism)	No
Reifenberger et al. 1999 [8]	m	35	Organic brain syndrome	GFAP, Vimentin, CD34, EMA, S-100	NA	GTR	NA	Yes (pulmonary embolism)	No
Taraszewska et al. 2003 [54]	m	62	Headache, polydipsia, polyuria	GFAP, Vimentin, CD34	Pterional trans-lamina terminalis	GTR	NA	yes (cardiac arrest)	no

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Table 1 (*continued*)

Source	Gender	Age	Presentation	Immunohistochemistry	Surgical approach	EoR	Follow-up	Perioperative mortality	Postoperative VP-Shunt
Taraszewska et al. 2003 [54]	f	51	Hypersomnia, visual deterioration	GFAP, Vimentin, CD34	Pterional trans-lamina terminalis	STR	4 PF	No	No
Liu et al. 2011 [55]	f	45	Headache, memory deficits, lethargy	GFAP, Vimentin, EMA, CK AE 1/3	Transcortical	STR	6 PF	No	No
Vanhauwaert et al. 2008 [56]	f	58	Memory deficits, inattention, hypersomnia	GFAP, Vimentin, CD 34, S-100	Transcortical	GTR	18 RF	No	No
Michotte et al. 2014 [57]	f	48	Memory deficits, depression, urinary incontinence	GFAP, CD34, TTF-1	NA	STR	6 PF	No	No
Suh et al. 2003 [58]	f	48	Headache, dizziness, bitemporal hemianopsia	GFAP, Vimentin, S-100	Transsphenoidal	GTR	17 RF	No	No
Zeinalizadeh et al. 2016 [59]	f	43	Headache, somnolence, visual deterioration	GFAP, Vimentin, CD34	Endoscopic transnasal	GTR	NA	No	No

NA: not available; IVH: intraventricular hemorrhage; RF: Recurrence free; PF: Progression free; R: Recurrence.

TABLE 2
Clinical signs and symptoms.

Symptoms	*n* (%)
Headache	36/70 (51.4%)
Visual deterioration/bitemporal hemianopsia	29/70 (41.4%)
Memory deficits	15/70 (21.4%)
Nausea	7/70 (10.0%)
Somnolence	7/70 (10.0%)
Urinary incontinence	4/70 (5.7%)
Lethargy	4/70 (5.7%)
Dizziness	4/70 (5.7%)
Ataxia	3/70 (4.2%)
Papilledema	3/70 (4.2%)
Hemiparese	3/70 (4.2%)
Polydipsie/Polyuria	2/70 (2.9%)
Epileptic seizures	2/70 (2.9%)
Speech deficits	2/70 (2.9%)

Table 3
MRI and radiological features of Chordoid Glioma.

MRI characteristics	*n* (%)
T1 Gd-enhancement	
Homogenous	56/66 (84.8%)
Heterogenous	10/66 (15.2%)
Native T1- sequence	
Hypointense	15/31 (48.4%)
Isointense	15/31 (48.4%)
Hyperintense	1/31 (3.2%)
T2 – sequence	
Hypointense	1/32 (3.1%)
Isointense	10/32 (31.3%)
Hyperintense	21/32 (65.6%)
Necrosis	3/70 (4.3%)
Cysts	14/70 (20.0%)
Calcification	6/70 (8.6%)

MR-spectroscopy was found in 4 case reports. An elevated choline peak intensity was found in all cases. Decreased *N*-acetylaspartate (NAA) peaks were also seen in all cases. The Choline/Creatine ratio was above 2 in all reported cases [14,24,37].

MR-perfusion data is reported in only 2 cases. Grand et al. [47] showed that the CBV of the tumor is not elevated compared to that of white matter. The relative maximum CBV ratio was 1 in this study. Ki et al. [13] reported a maximum rCBV of 6.95 in a case report of a CG with intraventricular dissemination.

3.3. Surgical techniques and extent of resection

Appropriate data regarding surgical techniques and approaches were given in 40 cases. The most common used neurosurgical approach was the interhemispheric transcallosal route (32.5%). The highest rate of gross total resection (rGTR) were seen in the CGs treated by subfrontal - (83.3%) or pterional trans-lamina terminalis approach (63.6%). Fisher's exact tests (2-sided) analyzed that the extent of resection of the subfrontal craniotomy combined with trans-lamina terminalis approach group was statistically significant superior to the interhemispheric transcallosal route group (23.1%; $p = 0.041$). Tables 4 and 5 summarize the results.

3.4. Extent of resection and VP-shunt dependency

Data concerning EoR and dependency of postoperative permanent cerebrospinal fluid (CSF) diversion via VP-shunt were given in 66 cases. The rate of VP-shunt dependency in the STR group was 19.2% (5/26) and 5.0% (2/40) in the GTR group, respectively ($p = 0.067$).

Table 4
Frequency of reported neurosurgical approaches.

Surgical approach	*n* (%)
Interhemispheric transcallosal	13/41 (31.7%)
Pterional trans-lamina terminalis	11/41 (26.8%)
Temporal transcortical	7/41 (17.1%)
Subfrontal trans-lamina terminalis	6/41 (14.6%)
Stereotactic biopsy	2/41 (4.9%)
Transsphenoidal	1/41 (2.4%)
Endoscopic transnasal	1/41 (2.4%)

3.5. Extent of resection and recurrence-free survival

Data regarding EoR as well as clinical and MRI follow-up was given in 42 cases. Mean and median follow-up was 19.8 and 13.5 months. 4 recurrent CGs were seen in patients treated by a STR. Overall recurrence rate was 9.5%. 1 and 2 year PFS was 85.0% and 80.5% in the STR group. Overall 1 and 2 year PFS was 92.9% and 90.5%. The mean time to progression was 9.25 months. Pearson's chi-squared test revealed that the recurrence rates of gross total resection group (0%) and subtotal resection group (20%) differed statistically significant ($p = 0.034$). Kaplan-Meier analysis is shown in Fig. 2. Perioperative mortality was reported in 11 (15.7%) of 70 cases. Pulmonary embolism (3; 4.3%) and cardiac arrest (3; 4.3%) were the most common causes for perioperative mortality. Bacterial meningitis after revision surgery of postoperative intraventricular hemorrhage was also seen in one (1.4%) case.

3.6. Immunohistochemical features

CG is histopathologically composed of ovoid and polygonal epitheloid cells with vast cytoplasm and abundant with clusters and chords. The cells are localized within a mucinous vacuolated stroma [6,8,32]. Lymphocytic infiltrates are also very common [17,22]. Mitotic activity is typically very low. Mean MIB-I was reported to be 2.5% [60].

The most common strongly expressing immunohistochemical markers were GFAP (100%), Vimentin (100%) and CD 34 (69.6%) (Table 6). Frequency of focal positive immunohistochemical expression is shown in Table 7.

4. Case series of 3 patients

There were three histopathologically diagnosed CGs in our department. Two female and one male patient with a mean age of 48.7 years were treated by neurosurgical interventions.

4.1. Case 1

The first case in our department was in 2002. A 35 years old woman was admitted to us because of chronic headache and central diabetes insipidus. No visual deficits or signs of hydrocephalus were present. MRI showed a homogenously enhancing solid round lesion within the third ventricle (Fig. 3).

GTR was achieved by removal of the tumor via a subfrontal craniotomy and trans-lamina terminalis approach. Postoperatively, the symptoms were regressive. Unfortunately, the patient died in the postoperative period because of pulmonary embolism.

Histopathological examination of the tissue showed cords of epitheloid cells within a chondroid-myxoid stroma. Lymphoplasmacytic infiltrates and Russel bodies were also seen (Fig. 4). Furthermore, Rosenthal fibers which are stereotypical for pilocytic astrocytoma were also detected.

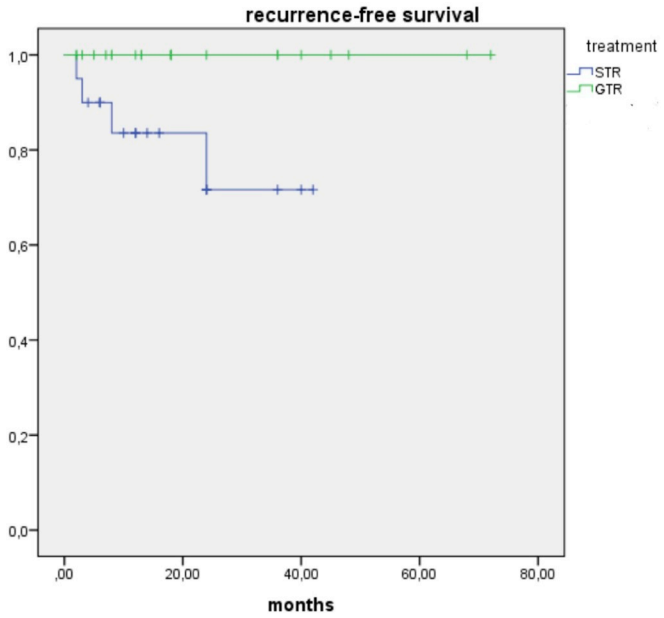

Fig. 2. Kaplan-Meier analysis of the subtotal and gross total resection group.

Table 6
Frequency of strong positive immunohistochemical markers.

Immunohistochemical marker	*n* (%)
GFAP	68/68 (100%)
Vimentin	43/43 (100%)
CD 34	32/46 (69.6%)
S-100	16/39 (41.0%)
EMA	10/52 (19.2%)
CK	7/42 (16.7%)
NFP	2/20 (10.0%)

Table 7
Frequency of focal positive immunohistochemical markers.

Immunohistochemical marker	*n* (%)
D2–40	4/4 (100%)
EGFR	2/2 (100%)
NSE	4/5 (80%)
EMA	34/52 (65.4%)
CK	24/42 (57.1%)
S-100	18/39 (46.2%)
CD 34	12/46 (26.1%)
NFP	1/20 (5.0%)

Table 5
Rates of gross total resection of the different approaches.

Neurosurgical approach	Rate of GTR *n* (%)	*p*-values
Subfrontal trans-lamina terminalis	5/6 (83.3%)	vs. interhemispheric transcallosal: 0.041
Pterional trans-lamina terminalis	7/11 (63.6%)	vs. interhemispheric transcallosal: 0.095
Transcortical temporal	3/7 (42.8%)	vs. subfrontal: 0.266 vs. pterional: 0.630
Interhemispheric transcallosal	3/13 (23.1%)	vs. transcortical: 0.613

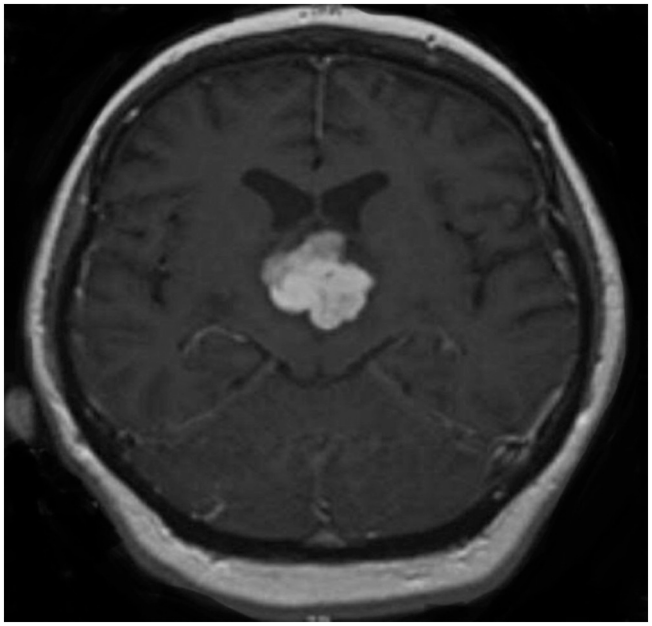

Fig. 3. Axial contrast-enhanced T1-weighted MRI of case 1 with a CG.

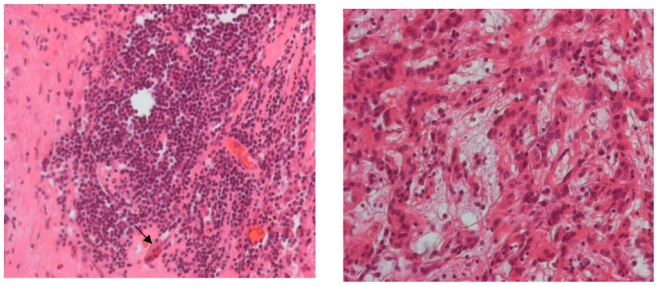

Fig. 4. Hematoxylin and eosin stains of a CG with Russel bodies (arrow) on the left and chondroid-myxoid stroma on the right in case 1.

4.2. Case 2

The second case was a 67 years old woman. The patient presented to our hospital with chronic headache without other symptoms. Formal visual field testing showed no defects. The electrolytes were also in an even balance. Contrast-enhanced T1 weighted MRI of the head confirmed again a vividly enhancing, homogeneous solid mass above the optic chiasm, projecting into the third ventricle (Fig. 5).

Surgical resection was guided by neuronavigation and a subfrontal craniotomy with a trans-lamina terminalis approach was performed. Postoperative MRI revealed a GTR. There was no new neurological deficit in the postoperative period. Histopathologically, there were pseudoepithelial cell groups. Furthermore, there was again a chondroid-myxoid stroma and parts of lymphoplasmacytic infiltrates including Russel bodies. The tumor was separated from surrounding tissue with a desmoid-fibroid pseudocapsule.

4.3. Case 3

The third case of a CG in our department was in 2013. A 44 years old man was admitted to us because of progressive visual deterioration lasting for two months. MRI of the head showed again a solid ovoid-shaped homogeneously enhancing lesion within the third ventricle and growth within the hypothalamus. T2-weighted and noncontrast T1-weighted MR images revealed an isointense third ventricular mass without perilesional edema (Fig. 6). Due to the very typical MR appearance a CG was suspected.

A GTR was not achievable. Consequently, accurate surgery guided by neuronavigation (Brainlab Curve, Brainlab, Feldkirchen, Germany) and an intraoperative 1.5-Tesla MRI (Philips Diamond Select Achieva 1.5 T, Philips, Amsterdam, Netherlands) was performed with a subfrontal craniotomy and trans-lamina terminalis approach (Fig. 7). Postoperative MRI showed a STR. Visual deterioration was persistent after surgery because of strong growth and infiltration of the tumor within the optic nerve and optic chiasm.

The histopathological specimen showed chords of epitheloid tumor cells empted within a chondroid-myxoid stroma. Parts of the tumor showed a trabecular growth pattern. The lymphoplasmocytic infiltrates and organization with lymphoid follicles and Russel bodies was remarkable.

At 18 months follow-up, the patient presented again with progressive visual deterioration and progressive tumor in MRI. Consequently, mass reduction and a STR was indicated again.

Immunohistochemical expression of the three case reports

Immunohistochemical analysis showed a low MIB-I index of 1% in two cases. Only the third case with the recurrent tumor after 18 months showed a MIB-I index of 3%. Strong immunohistochemical expression for GFAP, Vimentin, EMA and S-100 was observed in all cases. Results of the analyzed markers are summarized in the Table 8.

5. Discussion

CG typically occurs at a mean age of 44.4 years and 50% percent of patients are between 36 and 53 years old. 4 cases (5.7%) were in the pediatric age group [29,31,40,42]. There is a female predominance of this entity (male/female ratio: 1:1.8). Nevertheless, the tumor is negative for estrogen and progesterone receptors [10,45,48,58,61]. Syndromic associations were not found.

5.1. Clinical presentation

Overall, CG presents with multiple non-specific symptoms and they are often caused by obstructive hydrocephalus. Visual deterioration is the most common specific abnormality on examination. Ophthalmic examinations and follow-up should be done for clinical monitoring. Memory deficits have to be considered if there is a site-specific tendency of the lesion in MRI. Despite the suprasellar location and possible extension into the hypothalamus, endocrine dysfunctions are usually not common at presentation.

5.2. MRI features

CGs are hypo- (48.4%) or isointense (48.4%) on native T1 weighted imaging. 84.8% of all reported CGs are homogenously enhancing after gadolinium administration, whereas 65.6% are hyperintense on T2 weighted imaging. Most tumors are solid or ovoid in shape. Cystic areas are present in 20%. Calcification was observed in 8.6% [9,13,14,34,38,43]. This is almost congruent with data published by Liu et al. [55] who described a homogenously enhancing mass in 70% and cystic appearance in 28% of 64 analyzed cases. Craniopharyngioma is one relevant differential diagnosis and shows calcification in approximately 90% of pediatric and 70% of adult cases. Cysts are also more common in this entity [62]. Intraventricular Ependymomas show cystic appearance very often and are heterogeneously enhancing after gadolinium administration [63]. Subependymomas [64] show no or minimal enhancement and the central neurocytoma has calcifications in up to 50% [65]. Intraventricular meningiomas are also homogenously enhancing but at perfusion weighted imaging they demonstrate higher blood volume [66].

5.3. Surgical techniques and extent of resection

Subfrontal craniotomy with the trans-lamina terminalis route was the best reported approach concerning achievement of a GTR. This result can be explained by the location of the CG within the anterior

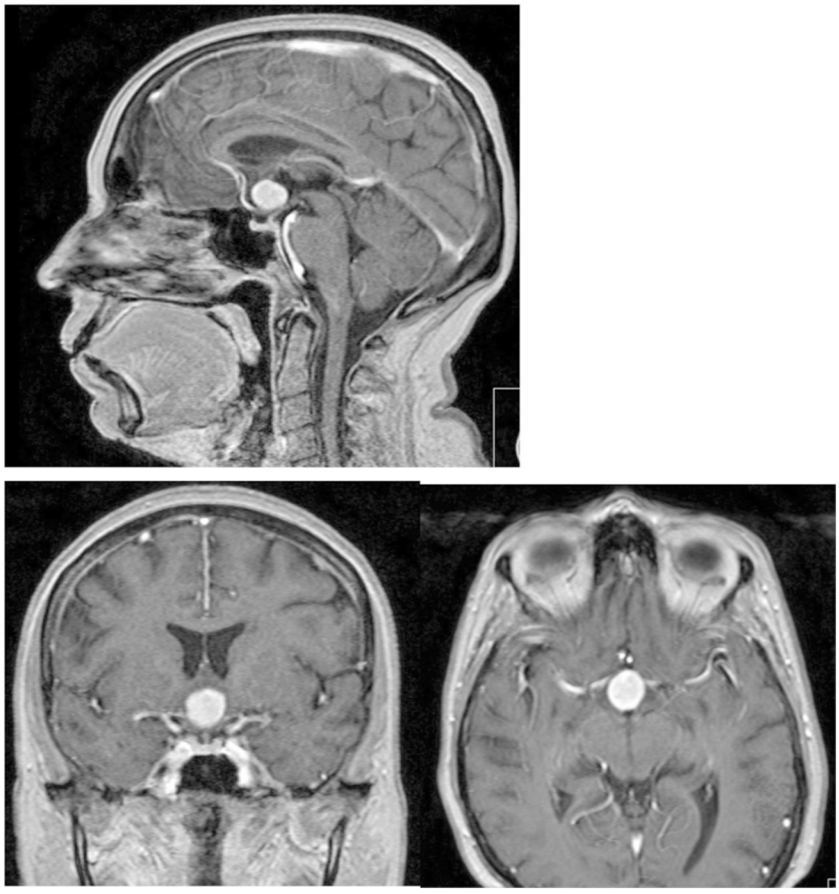

Fig. 5. Sagittal, coronar and axial contrast-enhanced T1-weighted MRI demonstrating homogenous enhancement.

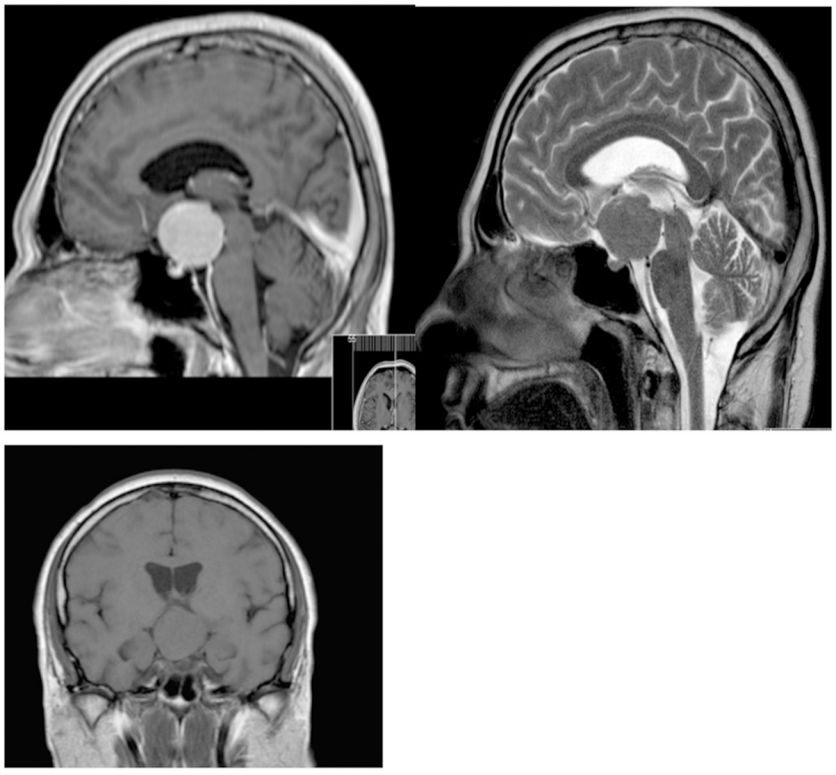

Fig. 6. Sagittal contrast-enhanced T1-weighted and T2-weighted MRI and coronar noncontrast T1-weighted MRI of case 3 with a CG.

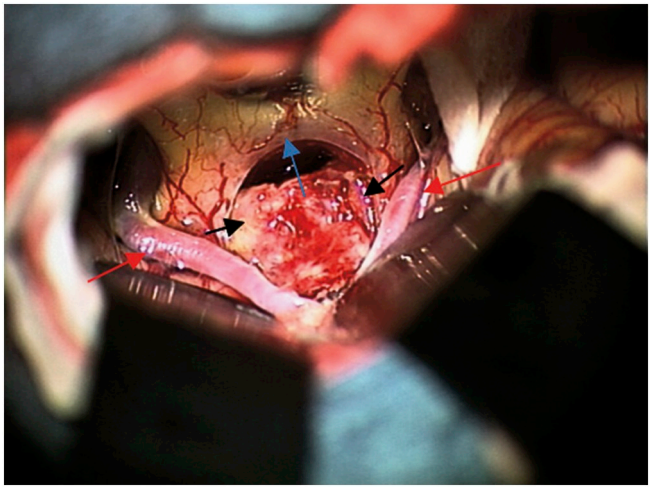

Fig. 7. Intraoperative microscopic visualization via trans-lamina terminalis approach showing a CG (black arrows), A1 segment of the anterior cerebral artery (red arrows) and the adjacent optic chiasm (blue arrow). (For interpretation of the references to colour in this figure legend, the reader is referred to the web version of this article.)

Table 8
Immunohistochemical profile of the case reports.

Immunohisto-chemical markers	Case 1	Case 2	Case 3
MIB-1	1%	1%	3.00%
CK 7	+	+	−
CK 5/6	−	−	−
CK 18	−	+	−
CK 20	−	+	−
EMA	+	+	+
GFAP	+	+	+
VIMENTIN	+	+	+
S-100	+	+	+
KL-1	−	u.	u.
CD 31	+	u.	u.
CD 68	u.	u.	+
NFP	u.	u.	−
IDH-1	u.	u.	−

+ = positive; − = negative; u. = untried.

part of the third ventricle near by the optic chiasm. The major advantage of subfrontal or pterional trans-lamina terminalis routes compared to the interhemispheric transcallosal route is the superior exposure of the chiasmal region. Therefore, subfrontral ($p = 0.041$) and pterional ($p = 0.095$) approaches improve the EoR compared to the inthemispheric transcallosal route. Furthermore, there are less intervening neurovascular structures and the perioperative morbidity is lower in trans-lamina terminalis routes compared to interhemispheric transcallosal approaches.

Permanent postoperative CSF diversion via VP-Shunt placement was necessary in 19.2% of the subtotal resected patients compared to 5% in the gross total resected ones. This result tended to be statistically significant ($p = 0.067$). Overall VP-shunt dependency in 66 operated cases was 10.6% which is nearly congruent with the reported rate of 9.6% in a review of 52 patients suffering from CG [36]. Furthermore, the VP-shunt dependency after colloid cyst removal which are also located within the anterior part of the third ventricle is smiliar to the data in CG and reported as 6.2% in 583 patients [67].

5.4. Extent of resection and recurrence-free survival

Data with regard to EoR in the postoperative MRI and MRI follow-up was given in 42 patients. All recurrences occured in the patients treated by a STR. Rate of recurrence was 20% in this group. Other studies reported recurrence rates of patients treated by STR as 22.7% [36] and 14.7% [60]. Overall rate of recurrence in our analysis was 9.52%. Pearson's chi-squared test (2-sided) showed a statistically significant result to perform a GTR ($p = 0.034$). This result confirms the analysis of other studies that recurrences only occurred after STR [36,60]. The only recurrence in our three case reports was also seen in the subtotal resected patient and occured after 18 months. The use of adjuvant radiotherapy or radiosurgery is so far restricted to cases treated by a STR and seems to be necessary [6,25,68]. Because of the location of the tumor nearby the hypothalamus and the optic nerves Gamma Knife is also an option [69]. All in all, the data regarding recurrence and follow-up of different treatment strategies is poor so far. Consequently, it is not possible to construct a reliable treatment workflow to choose between two pathways including either “high-risk” gross total resection or subtotal resection and adjuvant radiotherapeutic options.

5.5. Immunohistochemical profile

GFAP and Vimentin are strongly positive immunohistochemical markers of CG in all reported cases. These findings favor a glial origin. CD 34 is also an important marker for diagnosis. Focal positivity for EMA, CK and S-100 are also seen [4,8,48,50]. Tanycytes are a subtype of ependymal cells which are located in the ventral part of the third ventricle. Tanycytes are strongly positive for GFAP [70,71]whereas ependymal cells are uniformly negative for GFAP [72]. The anatomic location of the CG is in congruence with the location of tanycytes, namely the infundibular recess and the processes into the hypothalamus. CG seem to be of tanycytic origin.

6. Conclusion

Chordoid gliomas are homogenously enhancing solid tumors at the suprasellar space. The surgical aim should be the achievement of a GTR whenever possible. However, CG has a high rate of recurrence in spite of it's benign nature as a WHO grade II tumor. The trans-lamina terminalis route performed by a subfrontal or pterional craniotomy seems to be the approach of choice. Signs of postoperative hydrocephalus after STR have to be noted. Because of the anatomic location and the immunohistochemical marker profile CG seems to be of tanycytic origin.

Declaration of Competing Interest

The authors declare no conflict of interest.

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