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Rosette-forming glioneuronal tumour of the fourth ventricle: case report and review of the literature

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Abstract

Rosette-forming glioneuronal tumour (RGNT) of the fourth ventricle is one of the newly described primary tumours of the central nervous system. These tumours have two components of both neurocytic and glial areas but usually the glial component of the tumour predominates. They have biphasic cytoarchitecture with two elements; neurocytic rosettes resembling Homer-Wright rosettes, and astrocytic component resembling a pilocytic astrocytoma. They are low-grade tumours with lack of histopathological signs of malignancy. Here, clinical, magnetic resonance, computed tomography (CT) and pathological features of rosette-forming glioneuronal tumour of posterior fossa are presented. A 29-year-man was admitted with an acute neurological deterioration. A three ventricular hydrocephalus and a hypo-density around vermis in the posterior fossa were seen in his CT scans. He did well after an emergency external ventricular drainage. He had an elective operation and a mass that was reported to be a rosette-forming glioneuronal tumour of the fourth ventricle was excised.

Key words: *cerebellum, fourth ventricle, glioneuronal tumour, neuropathology, rosette-forming glioneuronal tumour.*

Introduction

Rosette-forming glioneuronal tumour (RGNT) of the fourth ventricle is one of the newly described primary tumours of the central nervous system [12]. Komori *et al*. [18] were the first who described this entity. They explained the characteristics of these tumours in terms of location, distinctive histological appearance with formation of neurocytic component and indolent biologic behaviour in a series of 11 cases report in 2002 [19]. Rosette-forming glioneuronal tumour was confirmed as a new type in the newly updated World Health Organization (WHO) classification of tumours of the central nervous system in 2007 [12]. Previously, these tumours assumed to be only infratentorial and located around fourth ventricle. Different locations of RGNT as chiasma [31], suprasellar region [30] and pineal region [10,13,19,32], septum pellucidum [36], intraventricular dissemination [35] and two spinal cord [5,28] were published later.

Although the histopathological features of these tumours are regarded as distinct and typical, they can show wide spectral clinical symptoms (Table I). We aimed to make a case addition to the limited clinical, radiological and pathological experience of this kind of rare and "poorly" described tumours.

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Table I. Review of the literature of cases of rosette-forming glioneuronal tumour

Author	No.	Sex, age	Presenting symptom	Location	Treatment	Postoperative morbidity
Komori *et al.*	1	25, M	Headache, 4th nerve palsy	4. V, aquaduct, pineal region	Biopsy	–
	2	59, M	Ataxia	4. V, aquaduct	ST + RT	+
	3	24, F	Headache, ataxia, dysartria	4. V, aquaduct, vermis, pons	PR	–
	4	18, M	Seizure	4. V, aquaduct	GTR	–
	5	40, F	Headache	4. V, vermis	GTR	+
	6	38, F	Headache	4. V	GTR	–
	7	39, F	Headache, neck pain, blurred vision	4. V, aquaduct	GTR	+
	8	27, M	Headache, confusion, ataxia	4. V	PR	+
	9	18, F	Incidental, headache, ataxia	4. V, aquaduct	PR	+
	10	46, M	Headache, blurred vision, ataxia	4. V, vermis, cerebellum	PR	+
	11	12, F	Headache, ataxia	Tectum, aquaduct, pineal r.	PR	–
Preusser *et al.**	12	35, M	Incidental	4. V, vermis	Surgery!	Not known
Adachi *et al.*	13	18, F	Incidental, slight ataxia	4. V	PR	+
Albanese *et al.*	14	32, F	Headache, cervical pain, neck rigidity	4. V	GTR	+
Jacques *et al.*	15	39, M	Vertigo, headache, diplopia, nystagmus	4. V	GTR	–
	16	33, F	Headache, diplopia, ataxia, dysarthria, lethargy	4. V	GTR	Not known
	17	42, M	Headache	4. V	GTR	Not known
Johnson *et al.*	18	29, F	Headache, vertigo	4. V	NGTR	Not known
Rickert *et al.*	19	16, F	Therapy resistant back pain	Spinal cord (C7-T2)	GTR	–
Vajtai *et al.*	20	16, F	Vertigo, nausea, tinnitus, ataxia	Roof of 4. V	GTR	+
	21	30, F	Vertigo, vomiting, headache, clumsy walking	Roof of 4. V	GTR	–
Pimentel *et al.*	22	38, F	Headache	4. V	NGTR	+
	23	51, F	Dizziness	Cerebellum	GTR	–
Marhold *et al.*	24	20, M	Somnolence, anisocoria, ataxia	Pineal region, vermis, 4. V	GTR	+
	25	47, F	Headache, ataxia, hemiparesthesia	Inferior vermis	GTR	+
	26	39, F	Headache, vertigo, nausea, tinnitus	Foluculus, lateral (CPA)	GTR	–
Tan *et al.*	27	42, M	Headache, feeling "odd"	Upper cerebellar aquaduct	Biopsy	+
	28	38, F	Lightheadedness	Cerebellar vermis	Biopsy	–

Table I. Cont.

Author	No.	Sex, age	Presenting symptom	Location	Treatment	Postoperative morbidity
Joseph *et al.*	29	38, F	Headache, vomiting	Cereballar vermis	GTR	–
	30	24, F	Worsening of gait, anisocoria	4. V	PR	–
Scheithauer *et al.*	31	23, M	Eye pain, blurred vision, headache	Chiasma	PR	–
Anan *et al.*	32	44, F	Tetraparesis, dysesthesia, neurogenic bladder	Spinal cord, cervicothoracic	GTR	+ (slight aggravation)
Wang *et al.*	33	16, F	Seizure, loss of consciousness	ventricle	Biopsy	–
Kinno *et al.***	34	18, M	Gait disturbance, ataxia	Cerebellar vermis	PR	–
Li *et al.*	35	27, M	Headache, vomiting, clumsy walking	4. V	GTR	–
Arai *et al.*	36	15, F	Headache	4. V, ventricle, vermis	GTR	+
Luan *et al.*	37	30, F	Headache	Right cerebellar hemisphere	GTR	–
Ghosal *et al.*	38	22, M	Headache, diplopia (3rd nerve palsy)	Pineal gland & tectum	Decompression?	–
Frydenberg *et al.*	39	29, M	Headache, vomiting, decrease in the level of consciousness	Pineal gland	GTR	–
Matyja *et al.*	40	20, F	Headache, nausea, balance disturbance	4. V	PR	?
Sharma *et al.*	41	16, F	Headache, diplopia	Midbrain	Biopsy + RT	–
	42	17, M	Loss of consciousness	Suprasellar, 3rd ventricle	Biopsy	–
Gessi *et al.*	43	18, M	Not known	Vermis	GTR	–
Fushimi *et al.*	44	28, F	Intermittent headache	Cerebellar midline	PR	–
Ellezam *et al.*	45	29, F	Not known	Inf vermis/4. V	Not known	Not known
	46	23, F	Not known	Inf vermis/4. V	Not known	Not known
	47	12, M	Not known	Inf vermis/4. V	Not known	Not known
	48	50, M	Not known	Inf vermis/4. V	Not known	Not known
	49	45, M	Not known	Midbrain/tectal	Not known	Not known
	50	18, F	Not known	Inf vermis/4. V	Not known	Not known
	51	30, F	Not known	3rd V	Not known	Not known
	52	15, M	Not known	Inf vermis/4th V	Not known	Not known
Karafin *et al.*	53	18, ?	Developmental delay	Posterior fossa	GTR	–
Solis *et al.*	55	16, F	Headache, vomiting	Pineal region	PR	–
Xiong *et al.*	56	38, M	Visual disturbance	Septum pellicidum	PR	–
Podlesek *et al.*	57	70, M	Persistant vertigo	Vermis, 4. V	GTR	–
Present study	58	29, M	Neurological deterioration with somnolence	Vermis, 4. V	NGTR	–

PR – partial removal, GTR – gross total removal, RT – radiotherapy, NGTR – nearly gross total removal
**This case reported again in Marhold serious*
***One of the cases presented in this report had been published previously by Adachi et al.*

Case report

A 29-year-man was admitted with an acute neurological deterioration and somnolence. His neurological examination was normal except a positive Babinski sign on his right side. In his medical history, there was a diagnosis of an incidental and asymptomatic hydrocephalus that was seen in his cranial computed tomography (CT) following a mild head injury three years before. A cranial CT showed a three ventricular hydrocephalus and a hypodensity around vermis in the posterior fossa (Fig. 1A). A cranial magnetic resonance imaging (MRI) revealed a partly cystic lesion located in vermis. It was hypointense on T1 and hyperintense on T2 weighted images and had no contrast enhancement (Fig. 1B-D). The lesion was hyperintense on flair sequences. The neurological status of the patient improved promptly following an emergent external ventricular drainage insertion. On the following day, the patient underwent an operation and a nearly gross total tumour removal was performed.

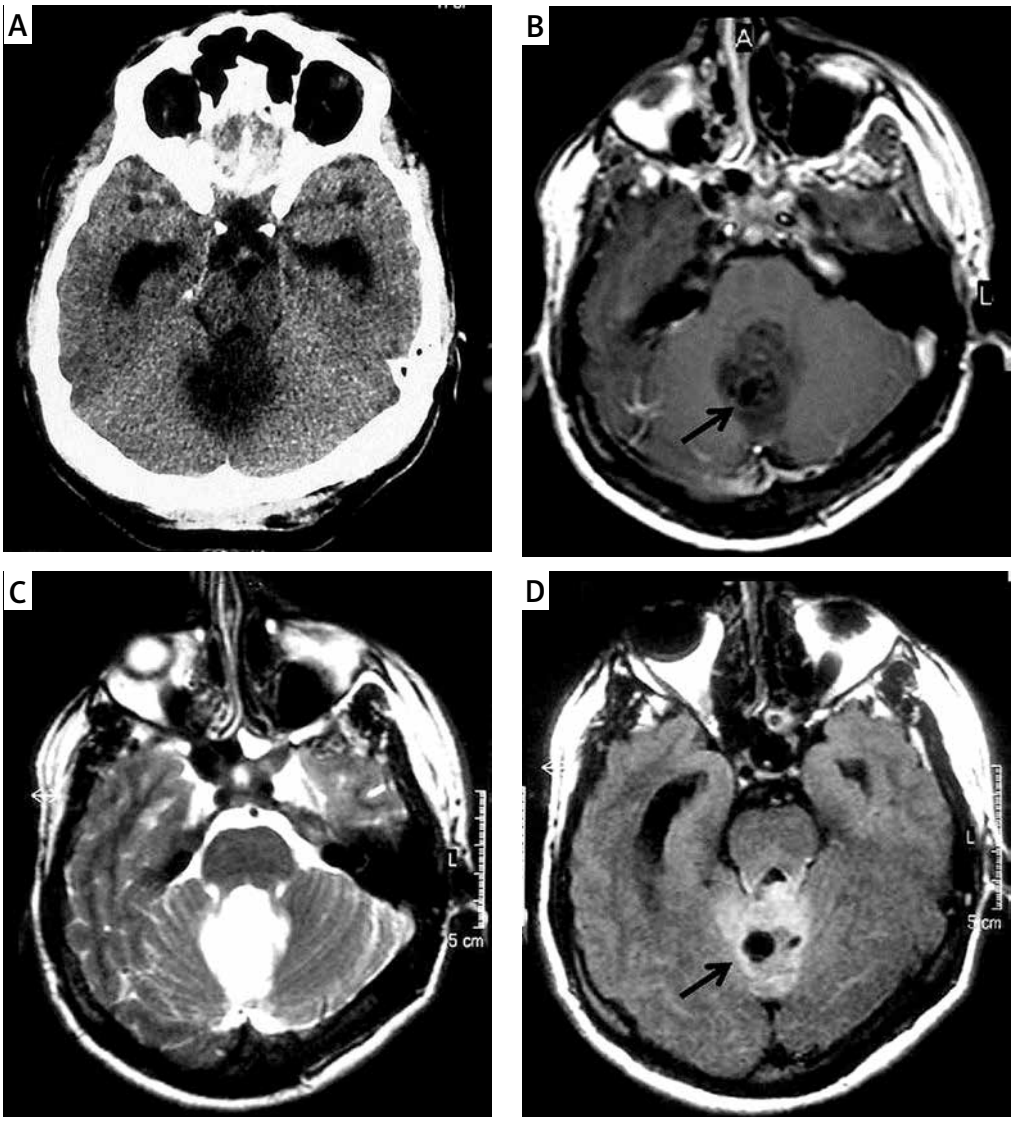

Fig. 1. A) Non-contrast computed tomography scan shows a hypodense lesion at vermis. **B**) On T1 weighted magnetic resonance scans this lesion shows heterogeneous contrast enhancement and a cystic component also is found (arrow) in the lesion. **C**) The lesion seems hyperintense on T2 weighted magnetic resonance imaging (MRI). **D**) On flair weighted MRI images the lesion seems hyperintense but the cystic component is hypointense (arrow).

The resected tumour specimen was transferred to the pathology department after fixing in a phosphate buffered 4% solution of formalin. Serial sections of 5 µm thick paraffin-embedded tumour tissues were stained with hematoxylin and eosin (HE). Additionally, an immunohistochemical (IHC) staining with monoclonal antibodies against glial fibrillary acidic protein (GFAP) (1 : 75; Neomarkers, Fremont, CA, USA), synaptophysin (1 : 100; Neomarkers) and Ki 67 (1 : 50; Neomarkers) was performed. Immunohistochemical staining was performed using an enhancement method based on a repetitive microwave heating technique using sodium citrate buffer. In microscopic examination, the tumour consisted of two components and it was also well demarcated from cerebellar tissue. The tumour was characterized by glial and neuronal components (Fig. 2A). In the area of the glial component, the cytoplasmic processes formed a compact textured fibrillary background. Some areas resembled pilocytic astrocytoma and oligodendroglioma (Fig. 2D). There were a few Rosenthal fibres and hemosiderin deposits. The neurocytic component consisted of uniform neurocytes which are forming rosettes and pseudorosettes (Fig. 2B-C). Neurocytic tumour cells had ovoid or round nuclei with fine chromatin pattern, inconspicuous nucleoli and delicate cytoplasmic processes. Overall, cellularity was low. Mitosis, atypia, necrosis or vascular proliferations were not seen. In immunohistochemical examination, synaptophysin immunoreactivity was only restricted in the peripapillary area of perivascular pseudorosette and rosette

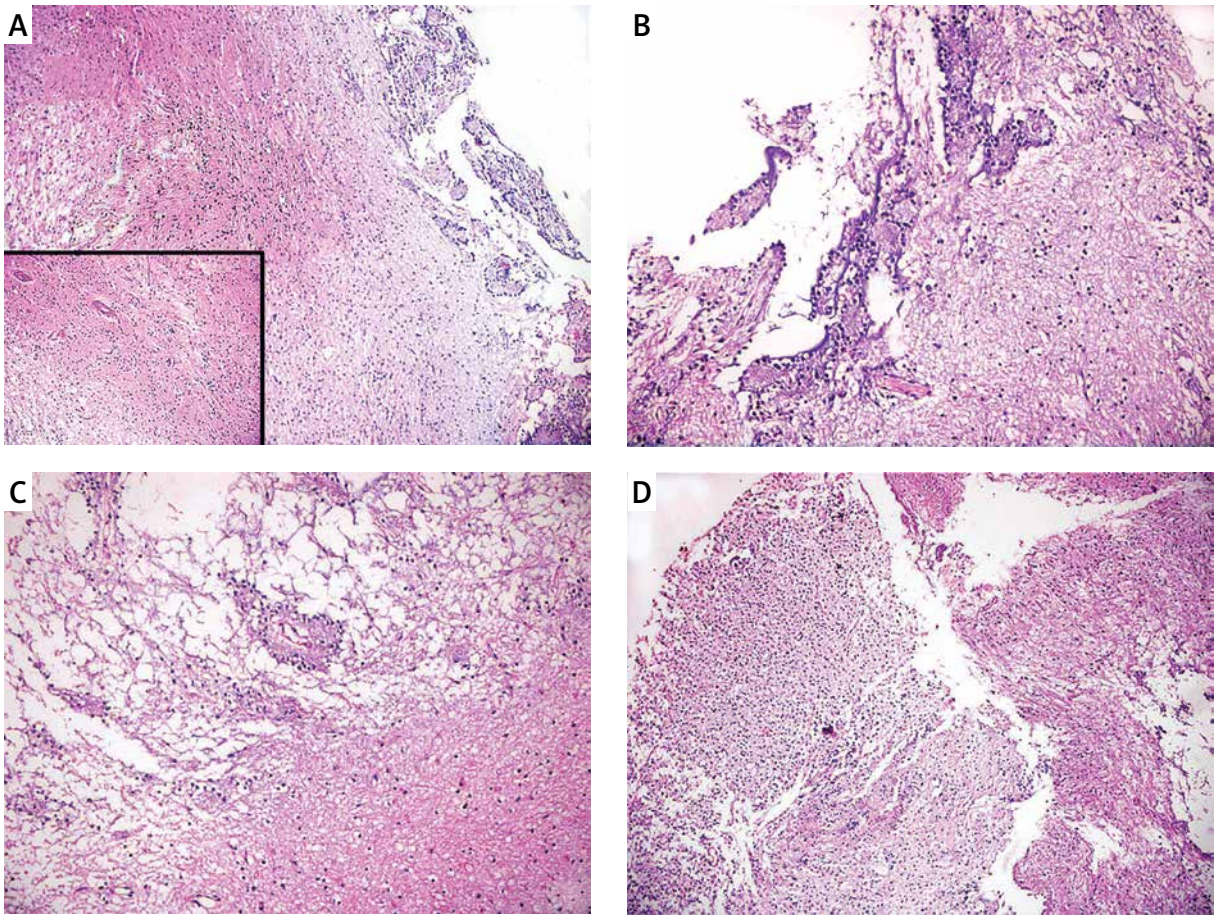

Fig. 2. Two components: neurocytic and astrocytic (H&E x40), pilocytic astrocytoma like component on the left side (H&E x200). **A**) Neurocytic pseudorosettes and rosettes around the astrocytic component. **B**) Neurocytic pseudorosette: delicate cell processes radiating toward a capillary (H&E ×100). **C**) The other glial area resembling oligodendroglioma intermingle with pilocytic astrocytoma like area (H&E ×100). **D**) There is no mitosis and atypical histopathological findings.

(Fig. 3A). Staining with GFAP antibody was positive in the astrocytic component of the tumour (Fig. 3B). Ki 67 labelling index was very low (1% and below). The tumour was reported to be a rosette-forming glioneuronal tumour of the fourth ventricle.

The postoperative period was unremarkable. He was followed for 32 months without evidence of progressive clinical deterioration.

Discussion

Kuchelmeister *et al*. [20] reported a dysembryoplastic neuroepithelial tumour (DNT) in cerebellum which morphologically resembled RGNT in 1995. The term rosette-forming glioneuronal tumour was used to define the low-grade tumour of the fourth ventricle by Komori *et al*. [18] for the first time in 1998. The characteristic features of these tumours were explained as 1) its unique location, 2) neurocytic pseudo-rosette formation and 3) the presence of a pilocytic astrocytoma component. Until the publishing of two cases that were located in chiasma [31] and spinal cord [5], these tumours were believed to be found only in posterior fossa. So, the "unique location" of these tumours seems to need some change. Anan *et al*. [5] suggested that the lesion might derive from the gray matter or the middle motor nuclei in the spinal cord, so RGNT may not be limited in distribution [5]. Scheithauer *et al*. [31] reported that periventricular germinal matrix is the likely origin of RGNTs and so, they may be occurring in "ectopic" sites. Rosette-forming glioneuronal tumours may be located in different areas such as the pineal region [9,10] and lateral ventricles [35] and some satellite tumours may also be seen in the patients [6,18,27,33].

Rosette-forming glioneuronal tumour is encountered more frequently in females. Thirty-one of the 56 reported cases in the English literature, including the present case, up to date were seen in women. Patient age was 12-70 years with a mean of 29 years (Table I).

Headache and cerebellar signs as ataxia and nystagmus are the most common initial clinical symptoms. Diplopia, ptosis [13], dysarthria [13,19], blurred vision [19], seizure [19,35], dizziness [25], vertigo [14,34], vomiting [21,34), clumsy walking [21,23] and neck pain and rigidity [2,19] are the other signs and symptoms of this tumour. Lethargy [13], somnolence, loss of conscious [35] and anisocoria [23] due to increased intracranial pressure may also be seen in these patients as in the presented case. However, there are some cases which are diagnosed incidentally [1,19,27], too.

A three ventricular hydrocephalus may be seen in RGNT patients. It can be so serious that a ventriculo-peritoneal shunt [13] or insertion of an external ventricular drainage (EVD) may be required [10,23]. We also performed an EVD in the emergency room for the presented case, because the patient was admitted with an acute neurological deterioration with somnolence. Fortunately, most of these tumours are slowly growing lesions. The development of ventriculomegaly can be chronic and may be clinically compensated [23].

The radiological findings of RGNTs may show some variations. These lesions are relatively circumscribed and heterogeneous, they may have some cal-

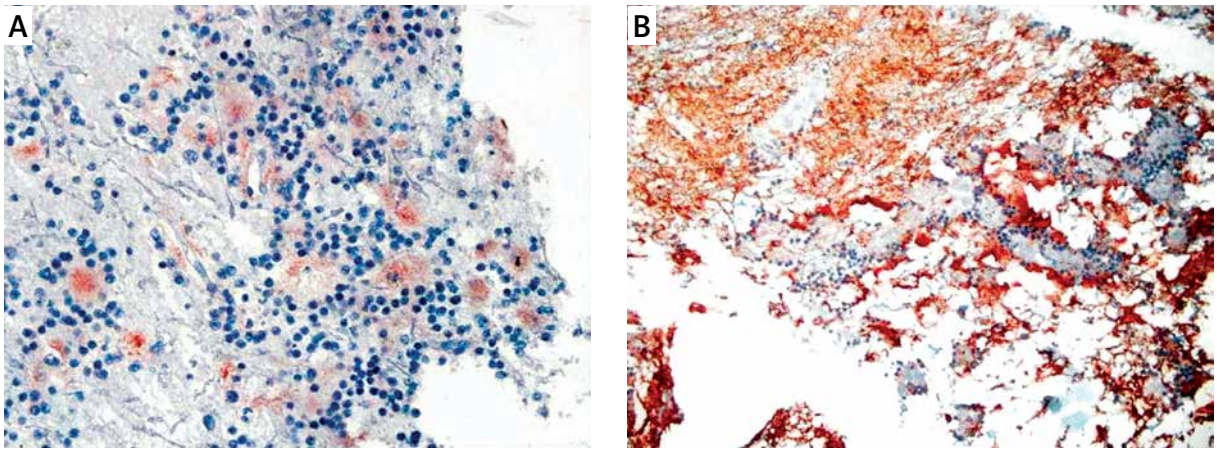

Fig. 3. In immunohistochemical study synaptophysin is present at the centre of neurocytic rosettes (**A**) and glial fibrillary acidic protein (GFAP) immunoreactivity is present in the glial component (**B**).

cifications and cystic components [4,11,13,21,23,27]. Some smaller tumour nodules may accompany these tumours [19,23,27]. These tumours are usually hypointense on T1 and hyperintense or isointens on T2 weighted MRI sequences [10]. They may have no contrast enhancement as in our case or may have a ring shaped contrast enhancement that led to consideration of malignancy [24,27]. Low density on non-enhanced CT as in the presented case is also typical of these lesions [4]. These tumours may show evidence of previous intratumoral haemorrhage on MRI [21,30].

Rosette-forming glioneuronal tumours have two components of both neurocytic and glial areas, but usually the glial component of the tumour predominates [3]. Rosette-forming glioneuronal tumours are low-grade tumours with no histopathological signs of malignancy [1,27]. They have a low labelling index of Ki-67 antigen, minimal or no cellular atypia and there is no evidence of recurrence, increase in tumour size or metastasis until now [19]. Solis *et al*. [32] found mutations of isocitrate dehydrogenase 1 (IDH1) and IDH2 that are relatively specific of diffuse gliomas; but Xiong *et al*. [36] did not detect somatic mutations of IDH1 and IDH2 in their cases. PIK3CA mutations were other mutations reported in RGNT [7]. PIK3CA mutants are seen with high frequencies in glioblastomas and anaplastic oligodendrogliomas.

The recommended treatment modality of these tumours is surgery. As these tumours are accepted low-grade in nature, an aggressive approach can increase surgical morbidity [15]; so, subtotal removal or gross total removal may be chosen. Until now, 4 cases of recurrence have been reported. The recurrence time of the tumours was 10 years in 2 cases [13,19] and 9 and 4 years in the others [7].

The main differential diagnosis of RGNT includes pilocytic astrocytoma (PA), dysembryoplastic neuroepithelial tumour, central neurocytoma, plexus papilloma, oligodendroglioma, ependymoma, primitive neuroectodermal tumour (PNET), glioneuronal tumour with neuropil-like islands (rosette) glioneuronal tumour, papillary glioneuronal tumour and metastasis [3,15,19,29]. Pilocytic astrocytoma is the most difficult tumour type for differential diagnosis with RGNT. Pilocytic astrocytoma has small round cells as well as astrocytic cells but rosette structures like those of RGNT are not found in PA [17,19]. There is also no evidence of neural differentiation in the rounded cells of pilocytic astrocytoma [3]. Dysembryoplastic neuroepithelial tumour has mature neurons in mucinous pools that help for differentiating from RGNT. Rosette-forming glioneuronal tumour does not have "floating neurons" as is the case for the "specific glioneuronal element" of DNT [34]. Central neurocytoma is characterized by uniform round cells and has similar immunohistochemical features of neuronal differentiation as in RGNT, but it does not have biphasic architecture with a distinctly separate glial component and true neurocytic rosette formation [35]. Central neurocytomas have no astrocytic components as RGNTs. Although neurocytes or well-formed rosettes may be found in oligodendrogliomas, oligodendroglial components are not seen in RGNTs [6]. The distribution pattern of synaptophysin and GFAP staining in ependymomas are quite the opposite to that seen in RGNTs [34].

In conclusion, RGNTs are clinically slow-growing tumours and typically occur in the midline. They affect mainly young adults with a slight female predominance. They can be seen outside the posterior fossa as ectopic tumours. They have biphasic cytoarchitecture with two elements; neurocytic rosettes resembling Homer-Wright rosettes and astrocytic component resembling a pilocytic astrocytoma. Glioneuronal tumours are a heterogeneous entity; so, further pathologic and clinical subclassifications may be required for prognostication and treatment. Careful and long-term follow-up monitoring will be wise for these uncommon tumours.

Disclosure

Authors report no conflict of interest.

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