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Infiltrative cerebellar ganglioglioma: conventional and advanced MRI, proton MR spectroscopic, and FDG PET findings in an 18-month-old child

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Introduction

Advanced magnetic resonance imaging (MRI) techniques, such as diffusion tensor imaging (DTI), dynamic susceptibility-weighted contrast-enhanced (DSC) perfusion, and proton magnetic resonance spectroscopy (MRS) have proven to be useful in predicting tumour grade and outcome in glial brain tumours.^{1–5} However, discrepancies do occur when advanced techniques suggest a biologically more malignant behaviour in low-grade lesions such as gangliogliomas.^{6–10} Recently, we had the opportunity to diagnose and follow a child with infiltrative cerebellar ganglioglioma. The purpose of this paper is to contribute to the existing body of knowledge about this rare entity by reporting and discussing the results of conventional and advanced MRI, including DTI, DSC, MRS, susceptibility-weighted imaging (SWI) and 2-[¹⁸F]-fluoro-2-deoxy-D-glucose positron emission tomography (FDG PET) with respect to the histopathological features of the tumour, and reviewing currently available literature.

Case report

An 18-month-old White male (weight 10 kg; height 79.4 cm) with a 5-month history of mild left ptosis and weakness of the ipsilateral lower limb was referred to our institution (March 2008). On admission, his neurological examination also revealed nystagmus and a subtle asymmetric smile. He could pull himself to stand since the age of 12 months, but fell over when attempting to stand freely. He could walk with support but had a broad-based ataxic gait with circumduction of the left leg; Babinski sign was positive on the left side. Neurocognitive development appeared appropriate for age, but he spoke only approximately 5 words. At birth he was diagnosed with a cleft lip and had corrective surgery when 4 months old. This study was approved by the institutional review board and written informed consent was obtained from the patient's family. As part of the initial diagnostic work-up at our institution, computed tomography (CT), FDG PET (Discovery LS, GE Healthcare, Waukesha, WI, USA) and MRI (Trio, Siemens Medical Solutions, Erlangen, Germany) were performed on the patient under general anaesthesia with propofol. MRI included DTI [twice-refocused spin-echo echo-planar imaging (TRSE-EPI) sequence,¹¹ with subsequent calculation of apparent diffusion coefficient (ADC) and fractional anisotropy (FA) maps], SWI,¹² DSC perfusion imaging [two-dimensional (2D) echo-planar sequence, with

subsequent calculation of cerebral blood flow (CBF), cerebral blood volume (CBV), and mean transit time (MTT) maps] and MRS [performed as 2D chemical shift imaging (2D-CSI); Table 1]. Although single voxel spectroscopy (SVS) is known to provide a higher signal-to-noise ratio, 2D-CSI was chosen over SVS as it enables evaluation of variations of the metabolic profile within the tumour field, and hence identification of possible lower and higher grade lesion components. For PET imaging, the patient was intravenously injected with 2.2 mCi FDG. Transmission CT and emission FDG PET images of the brain were obtained approximately 1 h later.

CT revealed an ill-defined slightly hyperdense cerebellar mass lesion without signs of calcification (Fig. 1, row 6). On T2-weighted (T2W) and post-contrast T1-weighted (T1W) MR images, the lesion was slightly hyperintense and hypointense, respectively, with only faint late enhancement after intravenous contrast agent (gadopentetate dimeglumine) injection, conspicuous only on subtraction images (Fig. 1, row 1). The lesion involved the left superior and middle cerebellar peduncles, pons, vermis, and parts of the contralateral cerebellar hemisphere exerting some mass effect on the fourth ventricle.

Using an region of interest (ROI)-based approach (the mean of four ROIs each, placed on adjacent section, was calculated for the tumour, the contralateral cerebellar hemisphere and the middle cerebellar peduncles), ADC values within the lesion were higher and FA values lower than those of normal-appearing cerebellar white matter (WM; Fig. 1, row 3). CBF and CBV values were low for most parts of the tumour, with one focal area of increase (red focus in Fig. 1, row 4) corresponding to a cluster of vessels, presumably veins, on SWI (Fig. 1, row 2).

In contrast, choline/N-acetylaspartate (Cho/NAA) ratio (Fig. 1, row 5, Fig. 3, Table 2) and FDG uptake within the tumour were markedly increased (Fig. 1, row 6). Despite MRS and PET findings indicating a high-grade lesion, applying the diagnostic algorithm proposed by Al-Okaili et al.¹³ to the MR-based data suggested a low-grade neoplasm.

An open biopsy was performed 48 days after admission. Histopathological evaluation of the specimen revealed normal cerebellar cortex. Underlying WM contained small atypical glial cells (showing immunoreactivities for S-100 and GFAP) and dysmorphic ganglion cells, the latter being immunoreactive to synaptophysin and neurofilament proteins. The growth fraction of neoplastic glial cells was low (Fig. 2). No high-grade neoplastic feature was identified; glioneuronal tumour and developmental abnormality, including Lhermitte–Duclos disease (dysplastic gangliocytoma of the cerebellum), were therefore considered differential diagnostic options, but the presence of atypical glial cells among dysmorphic ganglion cells supported the diagnosis of infiltrative ganglioglioma (WHO grade I).

Conventional MRI, DTI, SWI, and PET findings were stable during follow-up. In contrast, the Cho/NAA ratio increased steadily during the same period [3.13 (baseline), 3.98 7 (68 days), 3.89 (238 days), 5.10 (291 days), 2.95 (349 days), 4.77 (404 days), 6.22 (467 days); Table 2].

Discussion

Gangliogliomas, which are mixed neuronal–glial neoplasms, accounting for 2.5–3% of paediatric brain tumours,^{14, 15} most commonly develop within temporal lobes,^{16, 17} but may also arise in brainstem, cerebellum, spinal cord, anterior visual system, or ventricles.^{18–23} Gangliogliomas are usually well-demarcated, intra-axial lesions, both histologically and by imaging. They classically present on MRI as cystic lesions, frequently associated with a T2W hyperintense mural nodule.^{24–32} Focal supratentorial gangliogliomas, in particular, often harbour a cystic component and show obvious contrast enhancement.²⁴ Contrast

enhancement is common in children (up to 66%).^{16,27} Tumour calcification is relatively infrequent (44%).^{24,31,33} CT and T1W imaging findings are often non-specific.^{25–27,29,31,34} In the present patient, ill-defined, faint signal enhancement was seen only after subtraction of unenhanced and contrast-enhanced images. This finding was interpreted as consistent with the histopathology, which showed no signs of angiogenesis. No previous reports have compared the imaging semiology of paediatric and adult infratentorial gangliogliomas. However, in the supratentorial location, tumours are generally larger in children than adults.³⁵ To the best of the authors' knowledge, none of the 46 reported cases of paediatric cerebellar ganglioglioma showed infiltrative behaviour (Table 3) making this entity extremely rare. Conventional and advanced MRI features of infiltrative infratentorial gangliogliomas have not been described previously.

Advanced MRI techniques (DTI, DSC, MRS) are useful in characterizing cerebral neoplasms in vivo, sometimes predicting tumour grade and outcome in glial brain tumours more reliably than histopathology.^{1–5} The present case illustrates possible discrepancies between the histological classification of the tumour, conventional MRI, DTI, and DSC (consistent with low-grade neoplasm) on the one hand, and MRS and FDG data suggestive of a more malignant behaviour on the other hand.^{6–10}

Most parts of the tumour had higher ADC and lower FA than normal-appearing cerebellar WM, as expected for a low-grade lesion. Although minimal ADC values were lower than those previously reported for adults,³⁶ the present findings did not show actual diffusion restriction in any area of the lesion to suggest high tumour cellularity/grade. These data corresponded to the low cell density by histopathology. Overall, the diffusion parameters indicate a loss of integrity of cerebellar tissue matrix due to tumour infiltration. Higher FA values in some tumour areas, notably within the left middle cerebellar peduncle (green on FA colour map [Fig. 1, row 3]) may result from compaction of fibre tracts secondary to displacement or the infiltrative nature of the tumour with cells growing along fibre tracts and preserving diffusion anisotropy to some extent.

Although gangliogliomas have been reported to have significantly higher relative CBV values than low-grade gliomas,³⁷ CBV and CBF values were low in the present patient, except in one area of focal hyperperfusion. However, SWI suggested that this area harboured prominent vessels. Theoretically, this may have corresponded to a small developmental venous anomaly (DVA) or to tumour vessels (draining veins). The diagnosis of DVA was formally ruled out by an MR angiography (using a gadolinium-enhanced three-dimensional time-of-flight technique) during a later follow-up examination. A repeat SWI examination 2 months after the initial work-up continued to show these vessels, even with nearly diminished venous contrast in all other parts of the brain (Fig. 1, row 3). At this time, the low venous contrast throughout the brain was believed to be due to the high end-tidal carbon dioxide during anaesthesia and resulting vasodilatation with increased CBF.³⁸ The paradoxical conspicuity of veins near the tumour could be due to lack of reactivity to anaesthesia-induced hypercarbia, which is common in tumour vessels.³⁹

MRS is valuable in differentiating high-grade neoplasms (e.g., medulloblastoma) from other common low-grade posterior fossa tumours (e.g., juvenile pilocytic astrocytoma, ependymoma)¹⁰ and tumour recurrence from radiation necrosis⁴⁰, but its utility has not been validated in rare tumours. To distinguish healthy from tumour tissue, and good from poor outcome, the most widely accepted cut-off values range from 1.7 to 2.5.^{1,41} The specificity of these values is 10–86%, and therefore outliers clearly exist. For example, juvenile pilocytic astrocytomas, which are also WHO grade I tumours and are accordingly characterized by low Cho, myoinositol, and creatinine,⁴² have also been found to show Cho/NAA ratios up to 3.4.⁴³ MRS data in the present patient are consistent with previous

findings of higher Cho/NAA ratios in gangliogliomas compared to normal brain tissue and other low-grade gliomas.²⁷ Although increase in choline in a patient with supratentorial paediatric ganglioglioma has been identified as a potential indicator of malignancy,⁸ in the present case the stable anatomical MRI findings over a considerable follow-up period suggested overall benign tumour biology.

Although low-grade gangliogliomas typically show hypo- or ametabolism,^{6,27} the tumour of the present patient showed striking hypermetabolism on FDG PET.^{9,27,44} Similar discrepancies among imaging techniques, suggesting a high-grade neoplastic process in low-grade lesions, have been reported. For example, both low- and high-grade anaplastic gangliogliomas show high uptake on ²⁰¹Tl-SPECT and elevated choline levels by MRS.^{7,8} Choline is a marker of membrane turnover, which includes myelin build-up and maintenance. The increased choline concentration within the tumour in the present patient may indicate high glial and myelin content, which is a hallmark of the lesion by histopathology. High-energy metabolism shown by FDG PET may also be related to the large neuronal cell population of gangliogliomas, which have higher metabolism than normal cells of WM.⁴⁵

Tumours in children younger than 2 years often have atypical histopathological features, and because they are rare and few data exist on advanced MRI findings, their potential imaging correlates remain poorly understood. As it is unclear whether the discrepancies observed in the present patient are characteristic of infiltrative cerebellar gangliogliomas, it is unclear whether they can be used for differential diagnosis or represent incidental findings. Despite this, the features and findings were relatively stable over a long follow-up period and are a reliable contribution to existing anatomical and “functional” imaging data. Although the present report illustrates the challenges of interpreting advanced MRI findings, it also underlines the value of systematic use of multi-parametric lesion assessment and suggests remarkable robustness of the diagnostic algorithm of Al-Okaili et al.¹³ Further studies will help determine the importance of advanced MRI to accurately diagnose and describe complex, but specific imaging phenotypes of uncommon paediatric tumours of the central nervous system.

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Figure 1. MRI, CT, and FDG PET results obtained for the present patient (see main text for further detail).

Figure 2.

Histopathological evaluation of the tumour. Small atypical glial cells were identified in the WM underlying normal cerebellar cortex (top row). These cells showed immunoreactivities for S-100 and GFAP. The second population of cells, dysmorphic ganglion cells, is immuno-reactive to synaptophysin (bottom left) and neurofilament proteins. Ki-67 immunoreactivity detecting cells in cycle was confined to a few of the atypical glial cells (bottom right).

Figure 3.
Representative spectrum for 2D CSI spectroscopy acquired at the first follow-up.

Table 1

Magnetic resonance imaging (MRI) performed for our patient

Parameters (3 T)	DTI	3D SWI	MRS	DSC
TR/TE (ms)	6600/120	56/25	1700/135	1800/45
Matrix	128 × 128	384 × 190		128 × 128
FOV	192 × 192	210 × 104		105 × 105
Voxel (mm ³)	1.5 × 1.5 × 3	0.55 × 0.55 × 2	10 × 10 × 15	0.82 × 0.82 × 4
Gap (mm)	3	n/a	n/a	None
Comment	12 Diffusion encoding directions, 4 averages	Fully velocity-compensated, high-resolution 3D gradient-echo sequence	Weak water suppression, bandwidth: 1200 Hz/pixel	15 Contiguous sections, 50 measurements, 0.2 ml/kg Magnevist through a 22 G intravenous line at a rate of 1 ml/s delivered by a power injector

DTI, diffusion tensor imaging; 3D, three-dimensional; SWI, susceptibility-weighted imaging; MRS, multivoxel proton magnetic resonance spectroscopy; DSC, dynamic susceptibility-weighted contrast-enhanced perfusion MRI; TR/TE, relaxation time/echo time; FOV, field of view; n/a: not applicable.

Table 2

Results of two-dimensional chemical shift imaging magnetic resonance spectroscopy

Follow-up	Cho	Cr	NAA	Cho:NAA
Baseline	0.64	0.49	0.21	3.13
68	4.78	2.95	1.20	3.98
238	61.10	35.90	15.70	3.89
291	37.10	13.70	7.27	5.10
349	46.90	65.20	15.90	2.95
404	1.36	1.17	0.29	4.77
467	4.85	2.36	0.78	6.22

The table shows data obtained from voxels exhibiting the highest Cho/NAA ratios of the dataset for each study Cho, choline; Cr, creatinine; NAA, *N*-acetylaspartate.

Table 3

Previously reported patients with paediatric cerebellar ganglioglioma in literature

Study	Patients (No. of children/ No. of adults)	Number and location of tumour	Paediatric cerebellar ganglioglioma (No. of cases)	Comments
Castillo et al., 1990 ²⁶	8/10	4 cerebellum, (14 cerebrum)	2	Clinical presentation, CT and conventional MRI findings, histopathology and disease course of intracranial gangliogliomas.
Diepholder et al., 1991 ⁴⁶	6/7	3 cerebellum, (1 BS/PF, 10 cerebrum)	2	Clinical presentation, CT findings, histopathology and disease course of intracranial gangliogliomas.
Silver et al., 1991 ⁴⁷	7/13	1 cerebellum, (12 cerebrum)	1	Clinical presentation, CT and conventional MRI findings, histopathology and long-term follow-up of gangliogliomas.
Mickle, 1992 ³⁰	32/0	2 cerebellum, (24 cerebrum)	2	Clinical presentation, surgery, histopathology and outcome of paediatric gangliogliomas.
Zimmerman et al., 1992 ³²	115/0	1 cerebellum	1	MRI characteristics of paediatric posterior fossa tumours.
Chang et al., 1993 ³⁴	133/0	1 cerebellum	1	CT appearance of posterior cranial fossa tumours in a Chinese patient cohort.
Wang et al., 1995 ¹⁰	30/0	30 cerebellum	1	Proton MR spectroscopy of paediatric cerebellar tumours.
Johnson et al., 1997 ¹⁶	99/0	(9 BS/PF, 84 cerebrum)	n/a	Clinical presentation, CT and conventional MRI findings, surgery, and outcome.
Im et al., 2002 ²⁷	24/10	6 cerebellum, 28 cerebrum)	6	Clinical presentation, CT, MRI (including MR spectroscopy, PET and SPECT), pathological features, treatment, and outcomes of intracranial gangliogliomas.
Baussard et al., 2007 ²⁴	10/0	9 cerebellum, (1 BS/PF)	9	Clinical presentation, conventional MRI, surgery and outcome
Scalley, 1976 ⁴⁸	1 child	Cerebellum	1	Angiographic findings of a cerebellar ganglioglioma in an 11-year-old male patient.
Probst et al., 1979 ⁴⁹	1 child	Cerebellum	1	Light and electron microscopic study of a cerebellar ganglioglioma in a 2-year-old male patient.
Fukuoka et al., 1985 ⁵⁰	1 child	Cerebellum	1	Histopathological and immunohistochemical studies of a cerebellar ganglioglioma in an infant.
Dhillon, 1987 ⁵¹	1 child	Cerebellum	1	Clinical presentation, CT and pathological features of an 8-year-old female patient with hearing deficit, dizziness, and ataxia diagnosed with a cerebellar ganglioglioma.
Kimura and Suzuki, 1990 ⁵²	1 child	Cerebellum	1	CT, MRI and histopathology of a cerebellar ganglioglioma in a 5-year-old female patient.
Turgut and Ozcan, 1990 ³³	1 child	Cerebellum	1	Clinical presentation and histopathology of paediatric cerebellar ganglioglioma in a 16-year-old male patient.
Nishizawa et al., 1991 ⁵³	1 child	Cerebellum	1	Histopathological evaluation of a cerebellar ganglioglioma in a 14-year-old male patient.
Geyer et al., 1992 ⁵⁴	1 child	Cerebellum	1	Differentiation of a primitive neuroectodermal tumour into a benign ganglioglioma in a 16-month-old female patient, clinical and histopathological findings.
Al-Shahwan et al., 1994 ⁵⁵	1 child	Cerebellum	2	MRI in two patients with unilateral spasms of the face and limbs with ganglioglioma of the cerebellopontine angle.
Blatt et al., 1995 ²⁵	1 child	Cerebellum	1	CT and conventional MRI findings in a 4-year-old male patient with cerebellomedullary ganglioglioma.

Study	Patients (No. of children/ No. of adults)	Number and location of tumour	Paediatric cerebellar ganglioglioma (No. of cases)	Comments
Harvey et al., 1996 ⁵⁶	1 child	Cerebellum	1	Clinical course, MRI and SPECT findings in a 6-month-old female patient with cerebellar ganglioglioma and episodes of hemifacial contraction that were epileptic seizures of cerebellar origin.
Jay and Greenberg, 1997 ⁵⁷	1 child	Cerebellum	1	Histopathological findings in a 16-year-old male patient with an unusual cerebellar ganglioglioma with marked cytological atypia.
Vinchon et al., 2000 ²³	1 child	Cerebellum	1	Cerebellar gliomas in children with NF1 compared to sporadic cerebellar gangliogliomas, histopathological findings, and surgery.
Chae et al., 2001 ⁵⁸	1 child	Cerebellum	1	Hemifacial seizure of cerebellar ganglioglioma origin in a 4-month-old male patient.
Kwon et al., 2001 ²⁸	1 child	Cerebellum	1	Conventional MRI findings in a 2-year-old male patient with cerebellopontine angle ganglioglioma.
Mesiwala et al., 2002 ²⁹	1 child	Cerebellum	1	An infant with focal motor seizures with secondary generalization arising from the cerebellar lesion.
Mink et al., 2003 ⁵⁹	1 child	Cerebellum	1	Clinical and conventional MRI findings in a 22-month-old male patient with progressive myoclonus caused by a cerebellar ganglioglioma.
Milligan et al., 2007 ³¹	1 child	Cerebellum	1	Clinical presentation, conventional MRI and therapy in a 12-year-old male patient with a ganglioglioma of the cerebellopontine angle.
Park et al., 2008 ⁶⁰	1 child	Cerebellum	1	MRI findings and follow-up of a 12-year-old male patient with cerebellar ganglioglioma.
Saad et al. 2008 ⁶¹	1 child	Cerebellum	1	Ganglioglioma associated with cerebral cortical dysplasia and extensive leptomeningeal involvement in a 15-year-old male patient, histopathology and diagnostic pitfalls.
Hanai et al. 2009 ⁴⁴	1 child	Cerebellum	1	MRI, ictal SPECT and FDG PET in an 18-month-old male patient with hemifacial seizures due to a cerebellar ganglioglioma.

CT, computed tomography; MRI, magnetic resonance imaging; BS/PF, brainstem/posterior fossa; FDG, 2-[¹⁸F]-fluoro-2-deoxy-D-glucose; PET, positron emission tomography; SPECT, single photon emission computed tomography; NF1, neurofibromatosis type-1.