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CASE REPORT

Dysplastic Cerebellar Epilepsy: Complete Seizure Control Following Resection of a Ganglioglioma

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Abstract Subcortical epilepsy has been a controversial issue, partially settled by evidence showing seizure generation in hypothalamic hamartomas and also by reports of seizures caused by cerebellar lesions. We report 4-year-old girl with right hemifacial seizures and autonomic phenomena, in whom MRI showed an irregular mass in the right cerebellar peduncle. Despite several unremarkable video-EEG recordings, seizure origin in the lesion was hypothesized. Complete resection was feasible, histopathology showed a ganglioglioma, and she has been seizure free for 3 years. A fine line separates these developmental tumors from focal cortical dysplasia, and the homogeneous presentation of this entity led us to propose the terminology dysplastic cerebellar epilepsy.

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Introduction

Seizures are the clinical manifestation of sustained paroxysmal electrical neuronal discharges [1]. Traditional views posed that only cortical neurons could generate epileptic activity and subcortical structures had only indirect participation [1–4]. This notion was seriously challenged when refractory seizures in patients with hypothalamic hamartomas were shown to originate in or depend upon the deep-seated hamartomas [5, 6]. Subcortical structures have widespread connections with cerebral cortex and harbor the potential to synchronize, modulate, or disrupt normal cortical activity, suggesting they are not mere bystanders of epileptic networks and, in fact, may cause epilepsy [7]. A further step toward the existence of subcortical epilepsies was the report by Harvey and colleagues of a 6-month-old infant with a distinct epileptic syndrome associated with a developmental tumor in the cerebellum [2]. The authors hypothesized the lesion was intrinsically epileptogenic, but additional reports on the syndrome were needed to confirm their views. We report a little girl who presented at 2 months of age with intractable hemifacial seizures associated with a cerebellar ganglioglioma, in which tumor resection led to complete resolution of seizures.

Case Report

A 4-year-old girl presented with clonic twitching of the right hemiface and eyelid when she was 2 months old. Episodes were often associated with deviation of the eyes to the right, dystonic extension of the right arm, increase in respiratory rate and groaning, followed by short-lasting apnea, but without bradycardia or cyanosis (VIDEO). Episodes lasted less than 20 s and consciousness appeared to be preserved. There were no obvious precipitating factors and family history was

negative for epilepsy or febrile seizures. She was born prematurely (34 weeks) but neurological development had been unremarkable. Undernourishment was attributed to mild difficulty swallowing related to the seizures. Other than the hemifacial spasms, neurological examination was completely normal. Seizures were uncontrolled with antiepileptic drugs and occurred almost continuously. Initial scalp EEG was normal but brain CT showed a small enlargement in the right cerebellar peduncle, distorting the fourth ventricle, which, at the time, was not thought to be the cause of her seizures. When 10 months old, she had an afebrile tonic-clonic seizure and MRI (1.5T, Siemens Magnetom) showed a hyperintense irregular mass in the right cerebellar peduncle, without gadolinium enhancement (Fig. 1). Again, scalp VEEG did not reveal any discharges. The hypothesis of subcortical epilepsy caused by the cerebellar tumor was considered and led to resection of the lesion at 17 months of age. Histopathology showed dysplastic ganglion neurons associated with diffuse proliferation of astrocytes (ganglioglioma, WHO grade I) without signs of malignant degeneration (Fig. 2). Seizures were completely controlled and have never recurred. She has been without AEDs for the last 3 years and her growth has progressively recovered. Neurological examination does not disclose any signs of cerebellar dysfunction and her development had progressed uneventfully.

Discussion

Subcortical epilepsy has been a controversial issue in neurology for many years [1–3]. Since the beginning of modern epileptology, it had been accepted that only cortical neurons could lead to epilepsy, even though animal models demonstrated that seizures could arise from subcortical structures. The most remarkable clinical example is hypothalamic hamartomas, which presents with gelastic and other types of seizures, often resolved after resection of the hamartoma [5].

Hemifacial spasms in infants with a cerebellar mass were first described by Langston and Tharp in 1976, but a seizure origin was only suggested by Jayakar and colleagues in 1987. Almost 10 years later, intracerebral electrodes and single-photon emission computed tomography (SPECT) confirmed that ictal epileptic paroxysms indeed originate from developmental cerebellar tumors [2], an observation later shared by a number of other authors [8–14]. Twenty-seven cases of cerebellar lesions associated with seizures have been reported (Table 1), with onset from 1 day to 4 years, usually in the first month. Except for two patients with 95 % seizure control with AEDs [11, 15], all other were refractory to medical therapy [16, 17].

Early onset and refractoriness to AEDs strongly resemble the picture of focal cortical dysplasia (FCD), a lesion known to be intrinsically epileptogenic [18]. Interestingly, there is a growing consensus that developmental tumors share a number of features with cortical dysplasia and may even be part of a similar continuum [19, 20]. Furthermore, gangliogliomas are among the most common epileptogenic lesions in epilepsy centers [21], harboring a major propensity for spontaneous electrical discharges which probably explains the association of this lesion to this unique syndrome [22]. Not unexpectedly, histological studies of cerebellar or fourth ventricle lesions presenting with subcortical epilepsy were classified as ganglioglioma [2, 4, 9, 10, 12, 23–27], hamartoma [8, 13, 14, 28], gangliomatous hamartoma [29], ganglioneurocytoma [30–32] or low-grade dense fibrillary astrocytoma [3, 15]. Although many of these lesions may carry a developmental origin and actually represent a continuum with FCD, some are not intrinsically epileptogenic [33]. The WHO classification of brain tumors, however, considers gangliogliomas to represent a mixed neuronal-glial tumor, excluding dysplasia from

Fig. 1 a–c MRI inversion recovery weighted image sections showing the cerebellar ganglioglioma in the right superior cerebellar peduncle (*white arrows*)

Table. 1 Review of 28 cases in literature that associates a cerebellar or fourth ventricle mass to features of subcortical epilepsy (including ours)

Number	Author/year	Seizure begin	Sex	Symptoms	EEG	Invasive EEG	AEDR
1	Langston 1976	3 weeks	M	Left hemifacial twitching and eye blinking head deviation to the right, extension left elbow	Excessive theta activity and absence of a well-formed alpha rhythm	No	Yes
2	Jayakar 1987	First year	M	Left and bilateral eye blinking, left facial spasm, with eye deviation to the right, head deviation to the right, and movements of the left arm	Normal	No	Yes
3	Flueler 1990	First week	F	Right facial and eye twitching	Normal	No	Yes
4	Flueler 1990	10 months	F	Right facial and eye twitching	No information	No	Yes
5	Bills 1991	3 weeks	M	Left hemifacial and eye twitching, conjugate eyes deviation to the left, dystonic movements in the left arm	No information	No	Yes
6	Al-Shahwan 1994	First day	F	Right facial and eye twitching with flexion of the right arm, extension of the right leg	Normal	No	Yes
7	Harvey 1996	First day	F	Left hemifacial and eye twitching, head and eye deviation to the right, occasionally movements of the limbs	Normal	Epileptiform spikes at lesion site	Yes
8	McLone 1998	First day	M	Eye fluttering and upper extremities shaking	Right frontal epileptiform activity	No	Yes
9	Arzimanoglu 1999	6 weeks	M	Left hemifacial and eye twitching elevation of the left shoulder and cough-like noise	Normal	No	Yes
10	Chae 2001	First day	M	Left eye blinking and forehead contraction, abnormal vocalization, swimming like-movements of the limbs	Normal	No	Yes
11	Delalande 2001	First day	F	Left hemifacial twitching and eye blinking; sometimes bilateral eye blinking; groans and breathing abnormalities	Excessive theta activity at 3 months of age	No	Yes
12	Delalande 2001	First day	F	Left hemifacial spasm and eye blinking, head and eye deviation to the right	Occasional delta activity	High-voltage Lesion discharges	Yes
13	Mesiwala 2002	First months	M	Left or bilateral eye twitching; extremity twitching and postural arching	Bilateral cortical epileptiform activity	Epileptiform activity arising from the lesion	Yes
14	Mink 2003	22 months	M	Intermittent, non-rhythmic jerking of the distal right and left lower extremity, less frequently trunk and arm jerking	Normal	No	NA
15	Pontes-Neto 2006	Second month	F	Left orbicularis oris and left orbicularis oculi muscles, with left gaze deviation and right nystagmus, followed by breathing irregularities, profuse diaphoresis, and decreased temperature in left hemiface and arm	Bursts of rhythmic regular alpha activity over the right rolandic area and bilateral midline delta activity	No	Yes
16	Kulkami 2007	2 years	M	Right hemifacial spasms nystagmoid jerks in his right eye	Right temporal spike and wave activity	No	No
17	Dagcinar 2007	First day	F	Blinking of both eyes tonic deviation of head and eyes to the left, clonic left arm contraction	Normal	No	Yes
18	Minkin 2008	First day	F	Paroxysmal contractions involved the left orbicularis oculi, often the left forehead and lower facial muscles, sometimes accompanied by nystagmoid eye movements to the right and by tonic head deviation to the left	Normal	No	Yes
19	Gan and Connolly 2008	6 months	M	Staring, placement of his hands over his eyes, and rubbing his eyes, screaming, a scared expression, oral automatisms, often followed by vomiting or secondary generalization. Episodes of tonic stiffening of the arms and legs	Slowing, predominantly in the left occipital posterior and temporal areas, bitemporal spike and slow wave activity, and biposterior quadrant spikes	Occasional spike on the right cerebellar surface	Yes
20	Park and Oh 2009	First day	M	Left hemifacial twitching, and eye blinking, with a left-sided deviation of the eyeball and lip; irregular respiration, followed by cyanosis and bradycardia	Normal	Mass generating electrical activities related to seizures	Yes

Table 1 (continued)

Number	Author/year	Seizure begin	Sex	Symptoms	EEG	Invasive EEG	AEDR
21	Koh and Lim 2010	19 months	F	Jerking of both proximal lower extremities	Normal	Focal slow waves associated with clinical myoclonus	Yes
22	Hanai 2010	First day	M	Hemifacial spasms starting with rising of the left eyebrow and twitching of the left eyelid. Next, bilateral blinking followed; eyes were deviated to the right. Developmental delay	Normal	No	Yes
23	Zamponi 2011	Second month	M	Clusters of sudden and brief involuntary muscular jerks in proximal lower and upper extremities hemifacial twitching and eye blinking	Normal	No	Yes
24	Yagyu 2011	Soon after birth	F	Motion arrest and right eyelid contraction; intermittent closure of the right eye and right perioral contraction	Not reported	Tumor demonstrates rhythmic theta waves during eyelid twitching corresponding to electromyography	Yes
25	Lascano 2013	Second day	F	Hyperventilation and abnormal vocalization, followed by contractions of the left orbicularis oris and orbicularis oculi muscles, drooling, and staring. During most episodes, right-sided facial weakness and decreased movement of the right hemibody were observed	Interictal right posterior slow waves and ictal right posterior rhythmic delta slowing	Intralesional pseudo-periodic 2–2.5 Hz epileptiform discharges	Yes
26	Boop 2013	3 years	M	Assuming a supine position with flexing of both legs and the right arm; followed by subtle clonic jerks of the right arm. Occasional tonic-clonic seizure	Left hemisphere discharges	Rhythmic theta frequencies from the right cerebellar depth electrode	No
27	Boop 2013	4 years	M	Myoclonic seizures, accompanied by vocalizatio, occasional tonic-clonic seizures	Generalized spike and wave complexes	Continuous spikes seen on right cerebellum; associated with a generalized burst of spike and slow wave complex or bifrontal discharges	Yes
28	Martins 2015	Second month	F	Episodic clonic twitching in her right face and eye, associated with eyes' deviation to the right and rigidity in the right upper limb, with irregular breathing and apnea	Normal	No	Yes

Number	Seizure begin	Imaging	Lesion localization	Outcome	Follow-up
1	3 weeks	PEG and arteriography	Mass bulging left superior part of fourth ventricle	Initial seizure control, after seizure recurrence with lower frequency	3 years
2	First year	CT	Mass in the left cerebellar hemisphere	Complete seizure control	3 years
3	First week	CT and MRI	Right middle and superior cerebellar peduncles	Refractory seizures	12 years
4	10 months	CT and MRI	Right middle and inferior cerebellar peduncles, expanding to the medulla and pons	Refractory seizures	4 years
5	3 weeks	CT and MRI	Left superior cerebellar peduncle	Complete seizure control	3 months
6	First day	CT and MRI	Right middle and superior cerebellar peduncle	Partial seizure control (lower frequency)	Not reported
7	First day	CT and MRI	Left middle and superior cerebellar peduncles	Complete resolution after total removal	4 months
8	First day	CT and MRI	Left middle and superior cerebellar peduncles and L side of pons	Complete seizure resolution	6 months
9	6 weeks	CT and MRI	L middle cerebellar peduncle	Refractory seizures	21 years
10	First day	CT and MRI	L superior cerebellar peduncle	Partial seizure control after first resection, complete seizure control after total removal	30 months
11	First day	MRI		Complete seizure control	8 years

Table. 1 (continued)

Number	Seizure begin	Imaging	Lesion localization	Outcome	Follow-up
12	First day	MRI	Floor of the IV ventricle with mass effect over the Left sup. cerebellar peduncle	Complete seizure control	1 year
13	First months	CT and MRI	Floor of the IV ventricle with mass effect over the left inf. and middle cerebellar peduncle	Partial seizure control after partial resection and complete seizure control, after total removal	1 year
14	22 months	MRI	Left cerebellar hemisphere causing mass effect over the fourth ventricle	Complete resolution	2 years
15	Second month	MRI	Mass on the floor of the fourth ventricle, with mass effect on the left pons and cerebellar peduncles	Complete resolution	2 years
16	2 years	CT and MRI	Right cerebellar hemisphere causing mass effect over the fourth ventricle	95 % control of seizures with valproate and clobazan	Not reported
17	First day	CT and MRI	Left middle and superior cerebellar peduncle	Complete seizure control	10 months
18	First day	MRI	Left superior cerebellar peduncle	95 % reduction in seizure frequency	2 years
19	6 months	MRI	Small supracerebellar arachnoid cyst	90 % resolution, residual complex partial seizures	2 years
20	First day	MRI	Left cerebellar hemisphere adjacent to the middle cerebellar peduncle	Worsening frequency of attacks after first surgery; complete resolution after total removal	Not reported
21	19 months	MRI	Mass involving cerebellar vermis and the right middle cerebellar peduncle	Complete resolution	2 months
22	First day	CT and MRI	Left middle cerebellar peduncle	Complete resolution of seizures, recovery of developmental delay	3 years
23	Second month	MRI	Right lateral part of the fourth ventricle superior cerebellar peduncle	Complete resolution	1 year
24	Soon after birth	MRI	Mass lesion on the right side of the floor of the fourth ventricle	Complete resolution	4 months
25	Second day	MRI	Mass lesion located in the superior and medial portion of the right cerebellar hemisphere, extending to the right middle and superior cerebellar peduncle	Complete resolution	8 months
26	3 years	MRI	Hypodense, non-enhancing abnormality deep in the right cerebellum	Over 95 % control with lamotrigine and carbamazepine	4 years
27	4 years	MRI	Enhancing mass in the right cerebellum near the midline	Complete resolution	18 months
28	Second month	CT and MRI	Non-enhancing mass on the right superior cerebellar peduncle	Complete resolution	3 years

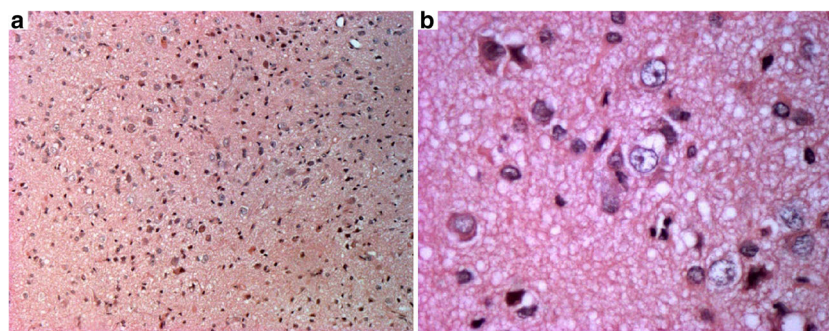**Fig. 2** Photomicrographs of the tumor histopathology. **a** H & E staining ×100—shows dysplastic ganglion neurons associated with diffuse proliferation of astrocytes. **b** Immunohistochemistry ×400—dysplastic ganglion cells displays positivity to neuronal nuclear antigen (NeuN); glial fibrillary acidic protein (GFAP) highlights astrocytic cells. Synaptophysin was not reagent

this diagnosis. Evolving on this issue, the ILAE 2010 FCD classification permits the diagnosis of FCD type IIIb, which occurs adjacent to tumors [34], particularly dysembryoplastic neuro-epithelial tumors (DNET) and gangliogliomas [19, 22]. Importantly, tumor infiltration must be previously excluded. Moreover, the origin of gangliogliomas is still unresolved and may lay in the neoplastic transformation of a previous dysplastic lesion or, in fact, represent a cortical dysplasia with abnormal cell proliferation and neoplasia [20]. Therefore, the terminology dysplastic cerebellar epilepsy still seems fit to the syndrome of hemifacial twitching, eye deviation, ipsilateral upper arm dystonia, and autonomic changes associated with developmental tumors of the cerebellum.

The cerebellum has widespread connections to the cortex, thalamus, basal nuclei, and brainstem, modulating movements and other functions [35]. The cerebellar cortex is comprised of three layers: the granular layer (excitatory), the Purkinje cell layer (inhibitory), and the molecular layer. Granule cells receive data from outside the cerebellum and modulate Purkinje cells, which provide the sole output of the cortex, through GABAergic inhibitory synapses to the cerebellar nuclei [35]. These neurons, then, send axons to many structures in the central nervous system, mainly providing excitatory input. Additionally, they supply input back to the cerebellar cortex, which forms the nucleocortical pathways, further refining the output data of cerebellar nuclei [36]. Disruption of these highly conserved pathways by cerebellar lesions may lead to irregular cerebellar output and de novo abnormal networks, building the substrate to subcortical seizures.

Differential diagnosis must include hemifacial spasms, a movement disorder that usually occurs in adults and older children, associated with a neurovascular malformation or a direct tumor compression of the facial nerve, none of which easily explains the autonomic dysregulation, eye movements, and limb dystonia. Additionally, seizure semiology imposes exclusion of a rolandic or insular epileptogenic zone. Dysplastic cerebellar epilepsy shares with temporal lobe epilepsy due to hippocampal sclerosis and some other syndromes

the clinical scenario of a high degree of medical intractability but also optimal rates of surgical control [37]. Prognosis seems to be excellent if complete tumor resection is achieved, with total seizure control in 100 % of the patients reported. However, only half of the patients control their seizures with partial resections [4, 13, 23, 26].

In summary, we report a girl with cerebellar ganglioglioma leading to subcortical epilepsy. This is a rare disorder, with marked clinical and pathological similarities among reported cases. Because of the developmental nature and intrinsic epileptogenicity of most tumors involved, this unique epileptic disorder might represent a syndrome of dysplastic cerebellar epilepsy.

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