



# Atypical Presentation of Dysembryoplastic Neuroepithelial Tumor

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## Abstract

Dysembryoplastic neuroepithelial tumor (DNET) is a benign glioneuronal neoplasm, usually found in children and adolescents, in the vast majority of cases associated with drug-resistant epilepsy. Typically, epileptic seizures are the main, and in most cases, their only clinical manifestation. Although DNET is a benign, biologically stable tumor with few reports of malignancy, it is one of the most common reasons for epileptic surgery. The epileptogenic potential of this tumor is so high that DNETs, along with ganglioglioma, have received the informal term “epileptomas” and are by far the leaders in the group of low-grade tumors associated with long-term epilepsy-associated tumors (LEAT). It is believed that this epileptogenicity is due to localization in the neocortex and frequent association with focal cortical dysplasias (FCD). In the world literature, there are only a few mentions of DNETs not associated with epilepsy. The article presents the experience of complex, interdisciplinary diagnosis of DNET in a child without epilepsy who complained of frequent headaches. During a comprehensive MRI examination, a cortical-subcortical pathological substrate was discovered in the left temporal lobe with radiological signs of DNET. During video-EEG monitoring of night sleep, no epileptiform signs were recorded. There was no history of epileptic seizures or other paroxysms. A control MRI revealed a slight increase in the size of the pathological substrate, which was the reason for surgical treatment. Pathological examination revealed microscopic features of DNET. This case of absence of epilepsy in a child with cortical DNET in the temporal lobe cortex suggests that the spectrum of its clinical manifestations and biological behavior is not fully understood and requires further comprehensive study.

**Keywords:** DNET; glioneuronal tumors; patient without epilepsy; neuroimaging epilepsy.

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# Атипичное клиническое проявление дизэмбриопластической нейроэпителиальной опухоли

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## Аннотация

Дизэмбриопластическая нейроэпителиальная опухоль (ДНЭО) – доброкачественное глионейрональное новообразование, как правило, встречающееся у детей и подростков, в подавляющем большинстве случаев ассоциированное с фармакорезистентной эпилепсией. Обычно эпилептические приступы являются основным и в большинстве случаев единственным клиническим проявлением ДНЭО. Несмотря на то что ДНЭО – доброкачественная, биологически стабильная опухоль с единичными упоминаниями о случаях малигнизации, она является одной из наиболее частых причин хирургического лечения эпилепсии. Эпилептогенный потенциал этой опухоли настолько высок, что ДНЭО, наряду с ганглиоглиомой, получили неофициальный термин «эпилептомы» и с большим отрывом лидируют в группе опухолей низкой степени злокачественности, ассоциированных с длительным течением эпилепсии. Есть мнение, что такая эпилептогенность обусловлена локализацией опухоли в неокортексе и частой ассоциацией её с фокальными кортикальными дисплазиями. Нами обнаружены единичные упоминания о ДНЭО, не ассоциированных с эпилепсией.

В статье представлен опыт комплексной междисциплинарной диагностики ДНЭО у ребёнка без эпилепсии, который обратился с жалобами на частые приступы головной боли. В ходе комплексного обследования при магнитно-резонансной томографии головного мозга был обнаружен кортикально-субкортикальный патологический субстрат в левой височной доле, имеющий радиологические признаки ДНЭО. При видео-ЭЭГ-мониторинге ночного сна не было зарегистрировано эпилептиформных знаков. В анамнезе не отмечалось эпилептических приступов или иных пароксизмов. При повторной магнитно-резонансной томографии головного мозга обнаружено увеличение размеров патологического субстрата, что послужило поводом для хирургического лечения. Патоморфологическое исследование выявило микроскопические признаки ДНЭО. Этот случай отсутствия эпилепсии у ребёнка с кортикальной ДНЭО в коре височной доли позволяет предположить, что спектр её клинических проявлений и течения раскрыт не полностью и требует дальнейшего всестороннего изучения.

**Ключевые слова:** дизэмбриопластическая нейроэпителиальная опухоль; глионейрональные опухоли; пациент без эпилепсии; нейровизуализация эпилепсии

**Этическое утверждение.** Исследование проводилось при добровольном информированном согласии законных представителей пациента.

**Источник финансирования.** Авторы заявляют об отсутствии внешних источников финансирования при проведении исследования.

**Конфликт интересов.** Авторы декларируют отсутствие явных и потенциальных конфликтов интересов, связанных с публикацией настоящей статьи.

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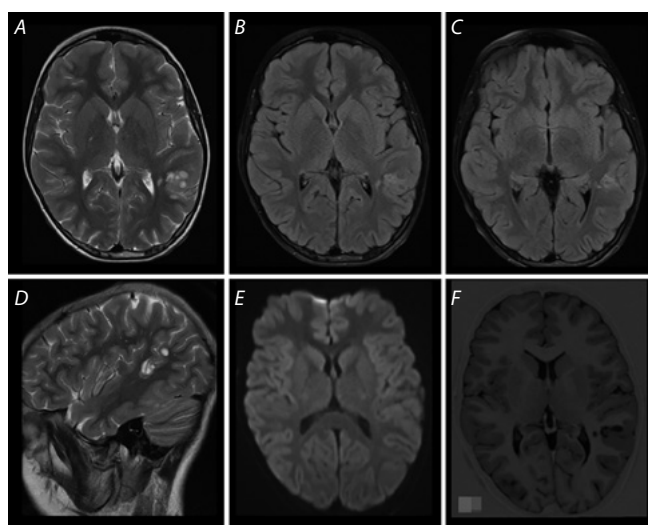
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## Introduction

The term “dysembryoplastic neuroepithelial tumor” (DNET) was first proposed in 1988 by C. Daumas-Duport et al., who identified a group of tumors with unique morphological features such as intracortical location, multinodular architecture, heterogeneity in cellular composition, and presence of a specific glioneuronal element [1]. Three forms of DNETs have been identified: simple, complex, and non-specific. Regardless of such polymorphism, DNETs are low-grade tumors with isolated reports of anaplastic transformation [2, 3]. DNETs are the second most common tumors associated with chronic intractable epilepsy and, along with gangliogliomas, are conventional representatives of low-grade long-term epilepsy-associated tumors (LEATs) [2, 4]. LEATs have common clinical, morphological, and radiological features such as association with drug-resistant or intractable epilepsy, age of onset below 20 years, frequent location in the temporal lobe, no marked neurological deficit, and extremely rare cases of anaplastic transformation [2, 3, 5]. Gangliogliomas and DNETs are by far the leaders in the LEAT group. In a very large cohort of patients who had epilepsy surgery, over 70% of all tumors were LEATs [6]. In many studies, the epileptogenicity of DNETs was up to 100%, and there is no surprise that specialists studying tumors of the LEAT group gave them an unofficial name “epileptomas” [7, 8].

In our article we described a case of DNET with atypical clinical course reported in a child without epilepsy, who underwent surgical treatment for the tumor.



**Fig. 1.** Brain MRI of patient G. at his first examination.

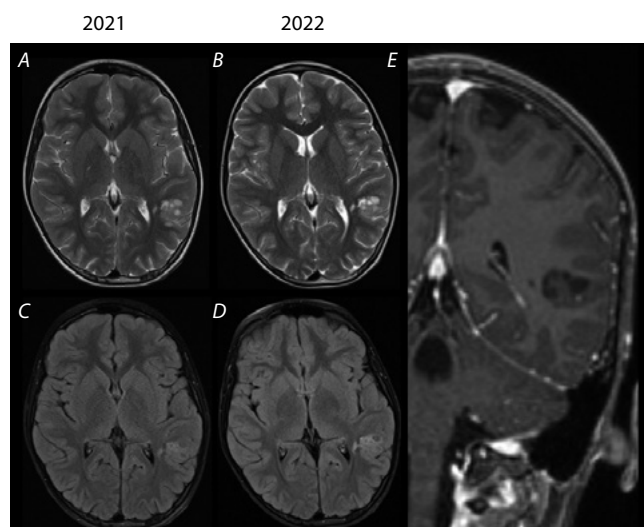
A lesion is seen in the posterolateral area of the left temporal lobe with involvement of the parietal lobe, with a multicystic structure (“soap-bubble pattern”) typical for DNET, with subcortical/cortical location and transmantle spread to the posterior horn of the homolateral lateral ventricle (A, B, C). On sagittal slices, involvement of the parietal lobe of the left hemisphere is clearly seen (D). The lesion does not demonstrate restricted diffusion or perifocal reaction (E). T1 SPC ISO images with a slice thickness of 1 mm clearly show the multicystic structure of the lesion and its predominant location in the corticomedullary area (F).

## Clinical case report

Patient G., 12 years old, consulted a neurologist with complaints of periodic headaches, which had been seen for about one year after a closed craniocerebral injury, and speech impairment, which his parents described as quiet inarticulate speech during periods of excitement. Brain magnetic resonance imaging (MRI), which was conducted as part of the patient’s comprehensive examination, showed a lesion in the left temporal lobe. Based on radiological findings and location, a tumor, presumably DNET, was suggested with a differential diagnosis of another representative of the LEAT group (Figure 1).

Considering the patient’s complaints of headaches and the polymorphism of possible atypical manifestations of temporal lobe epilepsy, he was referred to an epileptologist and administered with overnight video EEG monitoring (VEEG). VEEG monitoring showed slow and diffuse biopotentials of residual organic background with a focus of slow pathological activity in the left frontal-central area in the form of frequent bursts of high-amplitude paroxysmal theta rhythm. No clear local or interhemispheric asymmetry was identified. Typical epileptic activity was not recorded. Tolerance to hypoxia was intact. Cortical epileptogenesis was consistent with the patient’s age.

The neurological status showed no abnormalities of higher mental functions, motor domain, sensory domain, or cranial nerves.



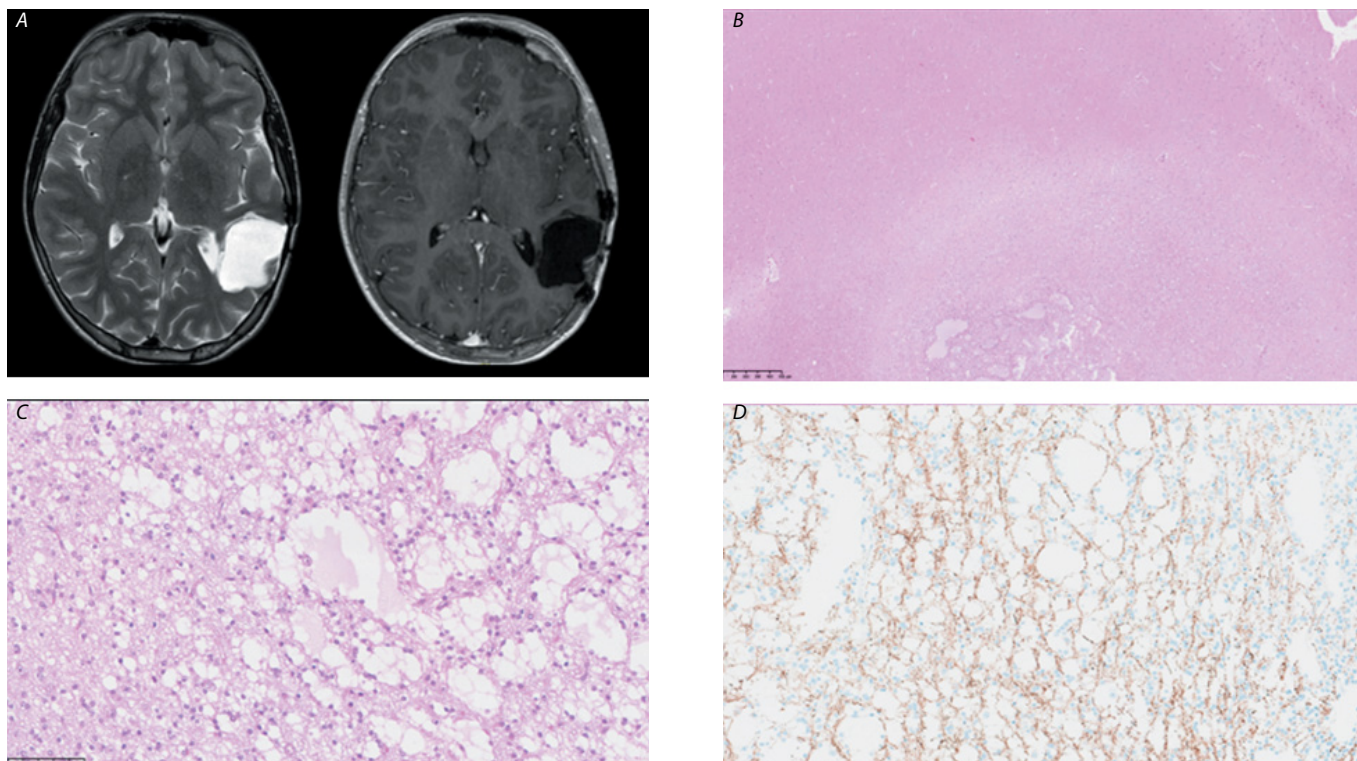
**Fig. 2.** Follow-up brain MRIs of patient G. 1 year later.

When images from 2021 and 2022 are compared, higher relaxation properties of the lesion are determined in the T2 and FLAIR MRI sequences. The multicystic structure and transmantle spread to the posterior horn of the left lateral ventricle are visualized more clearly. The retrospective comparison shows no signs of enlargement, perifocal reaction, or mass effect (A to D). No abnormal accumulation of an intravenous MRI contrast agent is seen in the structure of the lesion (E).

His personal or family history did not include any noteworthy episodes of loss of consciousness or seizures. Considering the changes in the left temporoparietal lobe, no epileptic seizures, changes in the neurological status, and VEEG monitoring findings, the interdisciplinary team discussed the patient's management, diagnosed him with "Neuroepithelial tumor of the left temporal lobe, presumably DNET", and recommended follow-up over time.

Follow-up MRI 1 year later showed non-obvious signs of biological instability of the lesion (Figure 2); therefore, the patient's neurosurgeons recommended surgical treatment.

The tumor in the left temporal lobe of the brain was removed by microsurgery with intraoperative neurophysiologic monitoring. Morphological examination of the resected tissue showed microscopic and immunohistochemical signs of simple low-grade DNET (Figure 3). A detailed examination of resected cortical fragments did not provide any evidence of an association with focal cortical dysplasia (FCD). Molecular genetic testing of the tumor tissue was performed by FISH with DNA probes; *BRAF V600E* mutations and *KIAA1549-BRAF*. At the time of the publication, the post-operative follow-up of patient G. lasted 11 months; no epileptic seizures or abnormal changes in his neurological status were found.



**Fig. 3.** Postoperative MRI and morphological findings of patient G.

MRI demonstrated total removal of the tumor from the left temporoparietal region with the transmantle track and adjacent cortex (A). At the border of the gray and white matter, tumor tissue was seen, which consisted of cells morphologically similar to oligodendrocytes, which formed unidirectional chains. Microcysts filled with mucoid substrate were observed in these chains (B). In individual microcysts, there were neurons located in the mucoid material ("floating" neurons) (C). In DNETs, neuronal axons form parallel cords that have the same direction as the columns of oligodendroglial cells. These structures are called specific glioneuronal elements, and they are typical for this tumor. Immunostaining with anti-neurofilament protein antibody (D).

## Discussion

Epileptic seizures that are difficult to control with antiepileptic therapy are the main and, in many cases, the only clinical sign of DNET. A single case of DNET in an adult patient without seizures has been described in generally available medical literature. The authors associated this atypical pattern with the absence of FCD in the peritumoral cortex [9]. However, the association with FCD is considered one of the main but far from the only factor contributing to the high epileptogenicity of DNET [6, 10]. There are three key hypotheses that explain tumor-associated epilepsy. Two of them (i.e. epileptocentric and tumor-centric) emphasize the key role of the tumor, while the third one gives preference to the role of molecular genetic aberrations found in tumor tissue [3, 6, 11, 12].

According to the epileptocentric approach, changes leading to hyperexcitability in the peritumoral cortex play a key role in the development of epilepsy. This is associated with metabolic, morphological, neurotransmitter or immunological changes in the tumor and peritumoral tissue [11, 13]. Various abnormalities of cortical development, especially FCD, are often found adjacent to pediatric brain tumors and play an important role in the development of epilepsy. These are usually type I FCDs, which have a high epileptogenic poten-

tial themselves, and the association with a tumor from the LEAT group makes this substrate superepileptogenic. A literature review suggested a wide range of detection rates for the association of neuroepithelial tumors with FCD (i. e. 20–80%) [6, 10, 14]. In patient G., no abnormalities in the architecture of the peritumoral cortex were found in the materials submitted for morphological examination. We considered this argument to be a possible factor contributing to the fact that the patient did not have epilepsy. However, this cannot be considered the only correct hypothesis because some tumors from the LEAT group associated with FCD or other cortical dysgenesias are increasingly found in patients without epilepsy [14, 15]. Therefore, the role of peritumoral FCD in the development of epileptic seizures is not fully understood, and studies in this area should be continued.

According to the tumor-centric approach, epileptic activity is triggered by the tumor itself and develops due to its direct mechanical effect. The tumor and edema cause increased intracranial pressure, leading to cerebral hypoperfusion. This results in local tissue destruction, ischemia, necrosis, neovascularization, microhemorrhages, and inflammation [11, 13]. In the case of patient G., there were no typical signs of a neoplastic process such as mass effect, perifocal edema, or neovascularization. Differences in relaxation characteristics and more distinct boundaries of the cystic component, without a significant increase in size, which were considered by the experts as progression, may be explained by the fact that different models of MRI scanners were used for the initial and follow-up examinations.

Therefore, the tumor found in the patient was relatively biologically stable, which, based on the tumor-centric theory, may have affected its epileptogenicity. However, it should be noted that the tumor-centric hypothesis was tested on gliomas and is more applicable to highly aggressive forms of these tumors with rapid progression [6].

There are increasing number of reports in the literature about a potential association of certain genetic mutations found in tumors with the occurrence of epileptic seizures. A new classification of tumors that was introduced in 2021 considers not only their histological structure but also specific genetic mutations [16]. Studies identified several genetic factors associated with the development of epilepsy in patients with brain tumors, such as *1p/19q* co-deletion and *IDH1/IDH2* mutations. They suggested that these mutations may affect the balance between inhibition and excitation in the brain, which can cause epileptic seizures [17, 18]. However, conventional LEATs are not associated with these mutations, which questions this hypothesis. Genetic changes responsible for tumor-associated epileptogenesis in LEATs are likely to be in the rat sarcoma mitogen-activated protein kinase (RAS/MAPK) and phosphoinositide 3-kinase protein kinase B mammalian target of rapamycin (PIK3-AKT/mTOR) pathways [19].

For example, *FGFR1* and *BRAF V600E* mutations, which are associated with these pathways, were detected in patients with DNET [3, 16]. Several publications indicated that the *BRAF V600E* mutation detected in tumor tissue may correlate with a worse prognosis for the postoperative outcome of epilepsy and provoke relapses of the neoplastic process [20].

Availability of molecular genetic testing of the tumor tissue in our case was limited. Testing for *BRAF V600E* mutations and *KIAA1549-BRAF* fusion was available using FISH with DNA probes, which was negative. We were unable to search for the *FGFR1* mutation, which is the most typical for DNET; however, we did not find any literature data on its role in tumor epileptogenesis, in contrast to *BRAF V600E* mutations and *KIAA1549-BRAF* fusion.

Patient's young age was yet another possible factor contributing to the absence of epilepsy. Although epilepsy occurs before the age of 20 years in over 90% of patients with DNET, there have been reports of late-onset seizures associated with this tumor [2, 21]. In one of the largest studies in patients with DNET, the mean age of epilepsy onset was 14.6 years (range, 3 months to 54 years) and age at surgery was 30.5 years (range, 6 to 65 years) [22]. The age of patient G. at the time of his surgery was 12 years, so epilepsy could develop at an older age. However, in another study in children, the mean age of onset of epileptic seizures was 8.1 years (range, 2 months to 14 years) and age at surgery was 12.4 years (range, 3.25 to 18.5 years), which also questions this hypothesis [23].

Epilepsy is known to have polymorphism of its clinical manifestations, especially if its structural cause is located in the temporal lobe. Quite often, symptoms differ from typical manifestations of epilepsy, which can be misinterpreted by specialists unfamiliar with this problem; therefore, the patients may not receive proper diagnostics and treatment. VEEG monitoring, which was conducted as part of the comprehensive examination of patient G., did not show any typical epileptic activity while awake and asleep, neither did functional tests. The patient did not have a personal or family history of epileptic seizures or other paroxysms. No abnormal symptoms were found in the patient's neurological status, and complains of headaches were the main clinical sign. Therefore, we can say with a certain degree of confidence that the patient did not have epilepsy at the time of his surgery.

## Conclusion

Although epileptic seizures are the main and, in some cases, the only clinical symptom of DNET, which justifies its unofficial term "epileptoma," this tumor may be detected in patients without seizures. Such an atypical course requires more thorough investigation of the conventional mechanisms that induce LEAT-associated epileptogenesis and possible contribution of molecular genetic aberrations.

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