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Mild malformation of cortical development with oligodendroglial hyperplasia in frontal lobe epilepsy (MOGHE): a report of the first case in Bulgaria

Dimitar Metodiev[1,2], Krassimir Minkin[3], Petia Dimova[3], Ingmar Blumcke[4], Roland Coras[4], Margarita Ruseva[5], Rumiana Ganeva[5], Dimitar Parvanov[5], Marin Penkov[6], Sevdalin Nachev[1]

[1]Neuropathological Laboratory, "Saint Ivan Rilski" University Hospital, Sofia, Bulgaria, [2]Department of Clinical Pathology, Nadezhda Women's Health Hospital, Sofia, Bulgaria, [3]Epilepsy Surgery Center, "Saint Ivan Rilski" University Hospital, Sofia, Bulgaria, [4]Department of Neuropathology, University Hospital Erlangen, Friedrich-Alexander-University Erlangen-Nürnberg, Erlangen, Germany, [5]Department of Research, Nadezhda Women's Health Hospital, Sofia, Bulgaria, [6]Department of Radiology, "Saint Ivan Rilski" University Hospital, Sofia, Bulgaria

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Abstract

Herein, we report the first case of mild malformation of cortical development with oligodendroglial hyperplasia and epilepsy (MOGHE) in Bulgaria. It is a newly recognised clinico-pathological entity with medically intractable focal epilepsy in paediatric patients.

The patient of interest is a 9-year-old boy who has been suffering from refractory epilepsy since the age of three. Positron emission tomography revealed a consistent hypometabolism with maximum in the orbitofrontal and fronto-opercular cortex, as well as in the adjacent anterior insula and the anterior temporal regions. A left frontal corticotomy anterior from the precentral sulcus, left insulectomy and temporal disconnection were performed. Pathomorphological examination of the material from the resected brain tissues demonstrated oligodendroglial hyperplasia with blurring of grey-white-matter boundaries and presence of subcortical heterotopic neurones. Eighteen months post-surgically the patient is seizure-free and drug-free.

The observed oligodendroglial hyperplasia with increased proliferative activity and heterotopic neurones in the white matter with blurring of grey-white-matter junctions are the histopathological hallmarks of MOGHE. More new cases are needed to establish further data about this distinct entity in frontal lobe epilepsy.

Key words: *cortical development, epilepsy, malformation, oligodendroglial hyperplasia, heterotopic neurones.*

Introduction

According to the revised International League Against Epilepsy classification of focal cortical dysplasia (FCD) from 2022, the presence of heterotopic neurones in the cerebral white matter (WM) in the absence of other structural lesions defines a condition known as mild malformation of cortical development (mMCD) [13]. It is noteworthy that a small number of heterotopic neurones can be found in temporal lobe biopsies even among adult patients [5,6,9,18]. The diagnosis of mMCD requires the detection of at least 30 neurones/mm^2 in the WM which can be visualised using the immunohistochemistry marker MAP2 or NeuN [13].

Communicating author:
Margarita Ruseva, Department of Research, Nadezhda Women's Health Hospital, Sofia, Bulgaria, e-mail: margarita.ruseva@gmail.com

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It has been shown that in 2-26% of patients who underwent surgery for intractable epilepsy, the pathology findings were not sufficient for definitive histological diagnosis [1,15]. In many of these patients, pre-surgical diagnostic imaging had revealed an abnormality which was not confirmed during the subsequent morphological examination.

Driven by the aforementioned, our group reviewed a series of 205 clinical cases with pharmacoresistant epilepsy operated in the period 2009-2021 at the Epilepsy Surgery Center, "Saint Ivan Rilski" University Hospital in Sofia, Bulgaria. We focused on cases with unusual histological findings that could not be attributed to any particular type of FCD.

Herein we present a 9-year-old patient with medically refractory epilepsy, initially interpreted as focal cortical dysplasia (FCD) type IIIc of the left frontal lobe, in whose brain we found: marked oligodendroglial hyperplasia predominantly in the WM of the left frontal lobe, but also affecting deep layers of the neocortex. In addition, numerous heterotopic neurones were detected in the cerebral WM. All of this led us to the new clinico-morphological entity of mild malformation of cortical development with oligodendroglial hyperplasia and epilepsy (MOGHE).

Case description

The 9-year-old boy first presented at the age of 3 years for diagnostic and treatment consideration. According to the family and medical history, he was born after an uneventful first pregnancy and delivery, without early postnatal complications and significant medical conditions. At the age of 4 months he suffered from infantile spasms with hypsarrhythmia easily controlled by short-term ACTH. Subsequently vigabatrin was used for about one year. Despite the good response to hormonal therapy with long-lasting seizure freedom, marked developmental delay became obvious without any motor deficits. At the age of 3 years, while no clinically obvious seizures were observed, he underwent brain MRI and 3-hour wake/sleep video-EEG examination. The neuroimaging revealed left-sided fronto-temporal dysplasia correlating to slowing and very prominent slow SW-epileptiform activity in the same areas, with maximum over F7-F3-T3 derivations and without overt seizure manifestation. A thorough discussion with the parents was performed and epilepsy surgery, even in the context of lacking clinical seizures, was proposed because of the presence of a clear-cut lesion with high epileptogenic potential and presumable association with the neuropsychological problems of the child. The family withheld from further pre-surgical evaluation and in the next two years the boy remained seizure-free, yet the developmental problems persisted and the behavioural issues increased. No language developed and severe hyperactivity, attention deficit, aggressive and auto-aggressive outbursts and sleep disturbances increased with time. At the age of 6 years, seizures relapsed in the form of multiple daily asymmetric axial spasms, asymmetric tonic seizures, atypical absences and few bilateral tonic-clonic seizures lateralised to the right. Anti-seizure medication attempts included valproate, levetiracetam and topiramate, which showed no efficacy and lastly, pills intake was refused by the patient also due to the severe behavioural problems. EEG was impossible to perform till the age of 7.9 years, when a very brief examination confirmed the left fronto-temporal origin of serial asymmetric epileptic spasms going into a longer asymmetric tonic seizure and ending by atypical absences with head nods only. This time, the parents agreed to proceed with new MRI and an interictal brain ^{18}F-FDG-PET, which by co-registration showed massive left-sided hypometabolism with maximum in the orbitofrontal and fronto-opercular cortex, as well as in the adjacent anterior insula and the anterior temporal regions, but going also backwards to the temporo-parieto-occipital junction with sparing of the sensory-motor areas. On voxel-based morphometric analysis of the 3D-T1-MRI by MAP18 the most dysplastic changes were seen in the frontal operculum and the mesial temporal structures.

Eighteen months post-operatively the patient is seizure-free and drug-free, with markedly improved behaviour allowing special school education, with progress in language including some spontaneous word use and better reception and communication. EEG is still impossible to perform due to fear of active opposition.

Resected brain specimens were fixed in 4% formalin and processed into paraffin according to standardised pathomorphological protocols. All sections were cut at 4 µm, mounted on positively charged slides and routinely stained with Hematoxylin and Eosin (H&E) and Luxol Fast Blue (LFB). Subsequently, immunohistochemical (IHC) stainings were performed on selected slides with the following antibodies: NeuN, Olig2, GFAP, CD34, SMI-32, Ki-67, p53, Vimentin, IDH-1, Notch1. Histopathology showed normal cortical laminar architecture. We found neither dysmorphic neurones nor balloon cells, including after additional IHC examination with SMI-32 and vimentin. Blurred grey-white-matter boundaries with subcortical heterotopic neurones were visualised (Figs. 1, 2). There was a striking increase in the density of oligodendroglial cells in the WM with signs of perivascular and perineural satelitosis (Fig. 1B). LFB did not reveal significant hypomyelination in the areas with an increased number of oligodendroglial cells (Fig. 2). Optic empty spaces with pseudo microcystic appearance, were also found in the WM, although

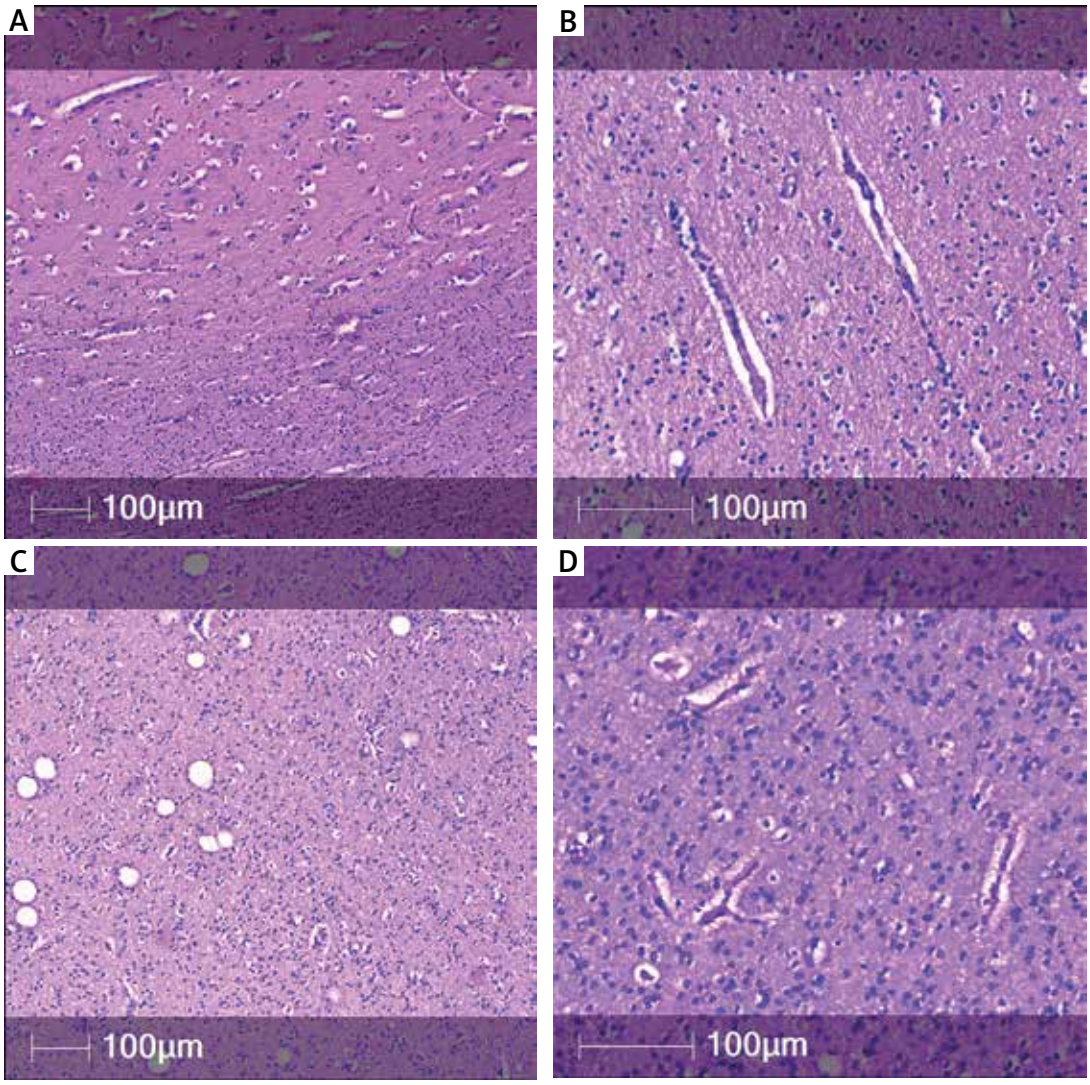

Fig. 1. Hematoxylin-eosin (H&E) staining of the resected brain specimen from the frontal lobe highlighting characteristic features of MOGHE. **A**) Dotted line demarcates the blurred grey-white matter boundary; **B**) Arrows indicate perivascular satelitosis; **C**) White matter with signs of oligodendroglial hyperplasia, heterotopic neuron (arrow) and 'pseudomicrocystic spaces' – most probably fixation artifacts; **D**) Arrows point to heterotopic neurones in the white matter.

were likely fixation artifacts (Fig. 1C). The heterotopic neurones were visualised using the NeuN marker and software quantification revealed a median density of 288 neurones/mm^2 in the WM (Fig. 3). Oligodendroglial hyperplasia was demonstrated by IHC examination with Olig2 marker revealing > 3600 Olig2-immunoreactive cells/mm^2 (Fig. 4). GFAP showed mild astrogliosis in the WM (Fig. 5A). IDH-1 (R132H), Notch1 (Fig. 5B) and p53 staining were negative. A few of the oligodendroglial cells were labelled by proliferative marker Ki-67 (Fig. 6). The described pathomorphological findings correspond to those of MOGHE.

In the specimen from the amygdala, there were a few hypertrophic cells resembling dysmorphic neurones.

Additional genetic testing was recommended to the parents of our patient.

A stop-codon mutation in the SLC35A2 gene was later identified.

Discussion

MOGHE is defined by some characteristic histopathological hallmarks – increased number of heterotopic neurones in the subcortical WM (> 30 neurones/mm^2) and increased oligodendroglial density of

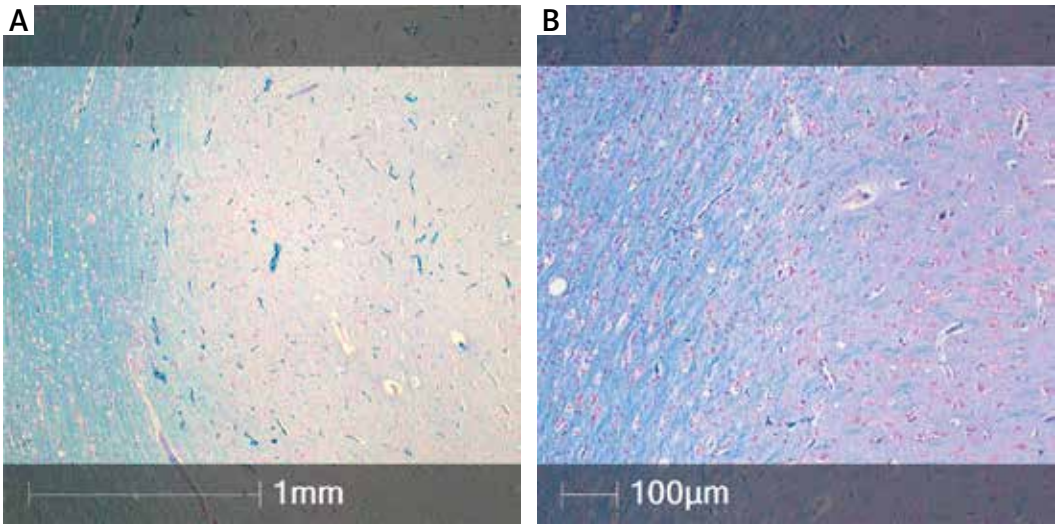

Fig. 2. Luxol Fast Blue (LFB) staining of the resected brain specimen from the frontal lobe demonstrating regular myelination and once again a blurred grey-white matter border. **A**) 40× magnification; **B**) 100× magnification.

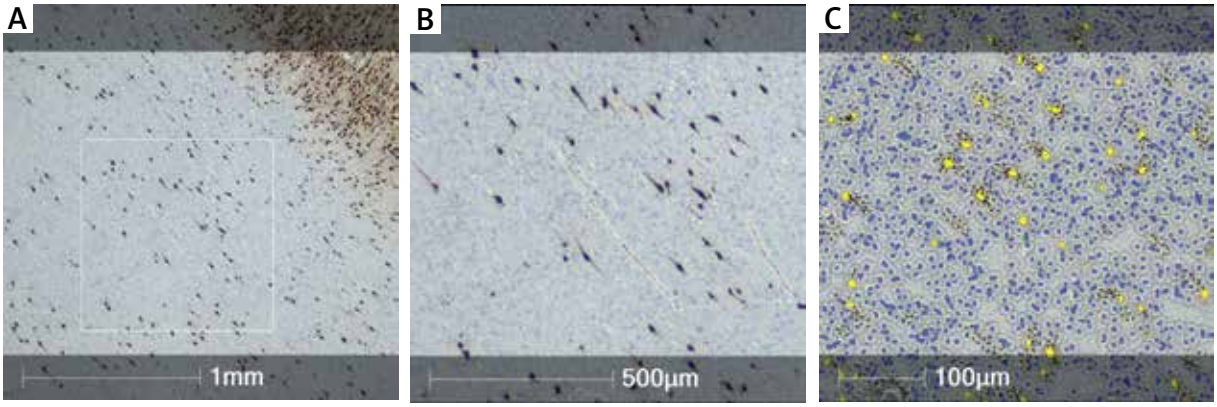

Fig. 3. NeuN IHC staining of the resected brain specimen from the frontal lobe. **A**) High density of heterotopic neurones in the white matter; grey matter visible in the upper right corner; **B**) Magnified insert of the white matter from A; **C**) Stained cell markup for spatial statistics produced by HALO® Image analysis software.

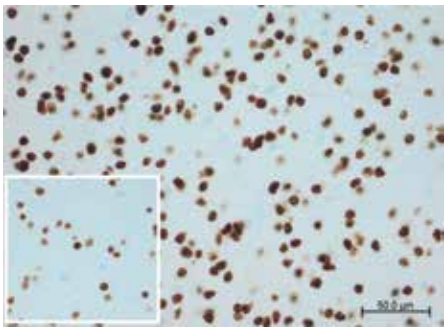

Fig. 4. Oligodendroglial hyperplasia demonstrated by IHC examination with Olig2 marker – highlighting the high density of oligodendroglial cells in contrast to regular cellularity (insert).

the WM (> 2000 Olig2-immunoreactive cells/mm^2). The diagnosis is only possible after pathomorphological examination of the material from the brain of patients with medically intractable epilepsy. It is necessary to exclude the possibility of other types of FCD structural lesions. The described cases of MOGHE predominantly affect the frontal lobe of the brain during early childhood, where the onset of epilepsy typically occurs around 2 years of age (ranging from 0.3 to 13 years) [8,15]. The age of MOGHE patients is associated with certain histological characteristics – oligodendroglial proliferative activity and signs of decreased myelination, as well as with specific seizure semiology [12,16,19]. Among paediatric MOGHE patients with frontal lesion localisation, epileptic episodes are char-

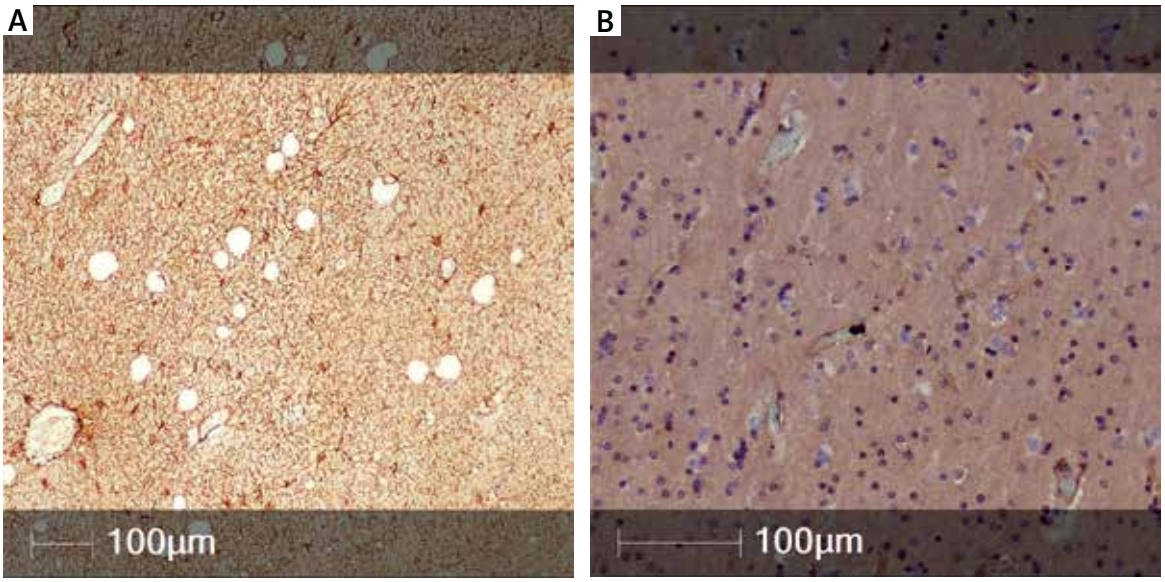

Fig. 5. IHC staining of the resected brain specimen from the frontal lobe. **A**) GFAP stained section illustrating reactive astrogliosis with highlighted activated astrocytes (arrows); **B**) NOTCH1 immunostaining revealing a negative reaction in proliferative oligodendrocytes (blue nuclei) and a single positive cell within a blood vessel lumen (arrow).

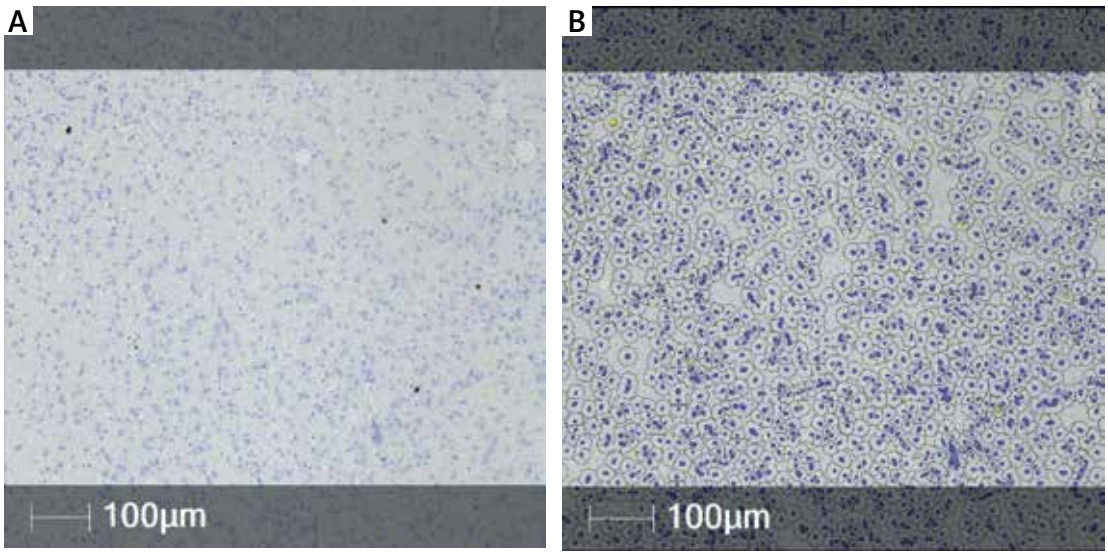

Fig. 6. Proliferative activity in the resected brain specimen. **A**) Ki-67 IHC stained cells in the white matter; **B**) Same stained cells markup (yellow nuclei) for spatial statistics produced by HALO® Image analysis software.

acterised by epileptic spasms, myoclonic seizures and focal seizures with behaviour arrest. It is worth noting that in the rare cases of adult MOGHE diagnosis, patients initially present with focal motor seizures with automatisms, hyperkinetic, and tonic-clonic seizures. In addition to the clinical manifestation, pre-surgical imaging modalities like MRI may also signify MOGHE. MRI findings, characteristic for the condition, include linear WM hyperintensities indicating reduced corticomedullar differentiation. This imaging pattern is typically seen in patients under 4 years of age [11,12,19]. Nonetheless, it is noteworthy that in a case series reported by Gaballa *et al*., in 9 out of 20 patients (45%) pre-surgical MRI did not reveal a lesion [7].

A defining pathomorphological feature of MOGHE is the increased density of heterotopic neurones in the deep WM, i.e., found in areas up to 500 µm away from the cortical surface. Heterotopic neurones located deeper than that are seen in mMCD, nevertheless, they lack signs of oligodendroglial hyperplasia [15]. There are reports of focal demyelination lesions in the regions containing heterotopic neurones; however, this was not the case in the herein described patient. Another pathognomonic sign of MOGHE that we observed in our patient is WM oligodendroglial hyperplasia affecting the grey-white matter border and the deeper layers of the neocortex.

It has been reported that proliferation activity is higher in younger patients with MOGHE [15]. The proliferative activity in MOGHE cases is generally (although not always) lower than that seen in oligodendrogliomas, comparable to that in dysembryoplastic neuroepithelial tumour and higher than that of cases with FCD types I and II. The proliferative activity in the lesion of our patient as determined by Ki-67 immunostaining was up to 1% (mean 0.65%). There was a tendency for decrease towards the cortical surface – it was most prominent among the hyperplastic oligodendrocytes in the WM, markedly lower at the grey-white matter interface, and lowest Ki-67 positivity was seen in the deeper cortical layers (V-VI) in the form of sporadic nuclear staining of oligodendroglial cells. These findings indicate that the pathologic condition likely originates in the WM, subsequently affecting the cortex-white matter border and only reaching the deeper neocortical layers. It is possible that namely the oligodendroglial proliferation engaging the neocortex gives rise to the clinical manifestation of the epilepsy.

There is perivascular and perineural satelitosis in MOGHE and we also observed signs of this process. Nonetheless, in fragmented patient biopsies, such signs may complicate the differential diagnosis and lead to confusion with oligodendroglioma. While in adult oligodendroglioma cases, 1p/19q codeletions are a frequent occurrence, that is not the case in paediatric forms of the tumour [14], which may also contribute to difficulty in differentiating this condition and MOGHE in children. In addition, the microcirculatory vessels seen in MOGHE somewhat resemble those in tumour oligodendroglial proliferation – so called 'chicken wire' vasculature. Oligodendroglioma may be diagnosed in the context of cortical dyslamination which is also associated with FCD type IIIb. Nevertheless, despite the histological similarities, MOGHE is unlikely to represent a precancerous condition preceding diffuse glial tumour such as oligodendroglioma.

Schurr *et al*. described a series of 1381 en block resected epilepsy surgery brain specimens, 51 of which were categorised as non-lesional. Upon revision, 22 of these were diagnosed as MOGHE. According to the authors, MOGHE pathogenesis involves either disruptions of intrauterine brain development or secondary regenerative mechanisms causing oligodendroglial hyperplasia with increased proliferative activity [15].

We examined the resected specimen for expression of a stem cell marker Notch1. There was no IHC reaction which reinforces the notion that MOGHE is a malformation of cortical development with possible proliferative activation of WM oligodendroglial cells. These cells demonstrate infiltrative pattern, penetrating the deep neocortex layers whereby the border with the WM becomes blurred.

The herein described microcystic spaces vaguely resemble a lesion called multinodular and vacuolating neuronal tumour, which is associated with a later onset of epilepsy, different localisation, distinct histological features and tumour cells' immunophenotype [17]. During the diagnostic process of this tumour, the use of myelin staining is of particular utility as it is histologically distinguishable from the discreet demyelination features of MOGHE. The pseudomicrocystic changes observed in our case are most probably fixation artifacts.

While the genetic basis of various types of FCD remains elusive, molecular-genetic testing provides insight into dysregulated genes which may have targeted therapeutic implications. In MOGHE patients, somatic SLC35A2 mutations are found in 45-100% of cases [3,4]. Bonduelle *et al*. reported somatic pathogenic SLC35A2 variants in 9 out of 20 genetically tested cases [4]. The gene of interest encodes a UDP-galactose transporter located in the Golgi apparatus membranes. Establishing SLC35A mutations in the cerebral tissue of patients operated due to pharmacoresistant epilepsy may create a new avenue for personalised non-invasive therapy, namely through oral galactose supplementation. This treatment strategy would be particularly valuable in cases where total surgical resection of the epileptogenic lesion is impossible, for instance when there are multiple foci (multifocal epilepsy) or when the epileptogenic zone affects extensive or critical brain regions. Galactose supplementation may also prove beneficial in patients that have undergone unsuccessful surgery, in which case it can be given in combination with other pharmacological treatments in order to manage refractory seizures.

Barba *et al*. studied brain somatic gene variants in 47 patients with MOGHE. They found two main phenotypes associated with SLC35A2 mutations [2]. The first phenotype manifested as epileptic spasms as the predominant seizure type and moderate to severe intellectual disability. It was named early epileptic encephalop-

athy. The second phenotype, so-called drug-resistant focal epilepsy, was associated with normal/borderline cognitive function and specific neuropsychological deficits. The authors concluded that more studies are needed to delineate any possible correlation between genetic variants, mutational load in the epileptic tissue and surgical outcomes [2].

In terms of treatment options and prognosis, according to the literature to date, extended surgical resection seems to be associated with the best postoperative seizure outcome [7,10,12].

Conclusions

MOGHE is a rare epileptogenic lesion with predominantly frontal localization and a typical pathomorphological pattern illustrated by blurred grey-white-matter boundaries, oligodendroglial hyperplasia, and increased density of subcortical heterotopic neurones. The proliferative activity of oligodendroglial cells is similar to that of some low-grade glial and glioneuronal tumours associated with epilepsy. The diagnosis of MOGHE is guided by the characteristic epilepsy semiology, imaging findings even prior to surgical removal, and histopathological verification. The identification of SLC35A2 mutations opens a new therapeutic approach (with oral galactose supplementation) that would be particularly beneficial in cases where complete removal of the epileptogenic areas is impossible.

Ethics consideration

Approval for publication was obtained from the institutional ethics committee (Approval No: 1/17.02.2023).

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Disclosure

The authors report no conflict of interest.

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