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Original Manuscript

Clinical EEG and Neuroscience
1–7
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DOI: 10.1177/15500594211043308
journals.sagepub.com/home/eeg
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A Rare Cause of Epilepsy: Ulegyria Revisited in a Series of 10 Patients

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Abstract

Introduction. Ulegyria results from perinatal hypoxic-ischemic brain injury in term infants. The specific mushroom-shaped configuration of ulegyria results from small atrophic circumvolutions at the bottom of a sulcus underlying an intact gyral apex. Clinically, ulegyria is generally associated with epilepsy. Here, we aimed to delineate the characteristics of patients with ulegyria and the epileptic seizures they experience. *Material and methods.* Medical records including radiology and pathology reports, video-electroencephalographic (EEG) analysis, operative notes, hospital progress and outpatient clinic notes were reviewed retrospectively in a total of 10 ulegyria patients. *Results.* Patients ages ranged between 24 and 58 years (mean, 32 ± 9.8 years). Past medical history was confirmed for neonatal asphyxia in 2 (20%). Neurological examination was remarkable for spastic hemiparesis in 1 (10%) patient with perisylvian ulegyria and for visual field deficits in 2 patients (20%) with occipital ulegyria. Ulegyria most commonly involved the temporoparietal region (n = 5, 50%) followed by the perisylvian area (n = 2, 20%). Except the one with bilateral perisylvian ulegyria, all patients had unilateral lesions (n = 9, 90%). Hippocampal sclerosis accompanied ulegyria in 2 patients (20%). All patients experienced epileptic seizures. Mean age at seizure onset was 8.8 ± 5.4 years (range, 2-20 years). Interictal scalp EEG and EEG-video monitoring records demonstrated temporoparietal and frontotemporal activities in 5 (50%) and 2 (20%) patients, respectively. The seizures were successfully controlled by antiepileptic medication in 8 patients (n = 8, 80%). The remaining 2 patients (%20) with concomitant hippocampal sclerosis required microsurgical resection of the seizure foci due to medically resistant seizures. *Discussion.* Ulegyria is easily recognized with its unique magnetic resonance imaging characteristics and clinical presentation in the majority of cases. It is highly associated with either medically resistant or medically controllable epileptic seizures. The treatment strategy depends on the age at onset and extends of the lesion that has a significant impact on the severity of the clinical picture.

Keywords

epilepsy, hypoxic-ischemic brain injury, epilepsy surgery, ulegyria

Received April 18, 2021; revised July 6, 2021; accepted August 7, 2021.

Introduction

Ulegyria, scarring of cerebral cortex, results from perinatal hypoxic-ischemic brain injury that occurs after neuronal migration finishes.[1] The scarring has a specific configuration with small atrophic circumvolutions at the bottom of a sulcus underlying an intact gyral apex, leading to mushroom-shaped gyri.[2,3] Either unilateral or bilateral lesions affect varying degrees of brain areas that may extend from small regions to extensive lesions involving the whole hemisphere.[3] An "epileptogenic" island of cortex may occur because of such a disconnection which results in seizure generation.[4] In addition to epilepsy that is generally resistant to medical treatment, most patients suffer from cerebral palsy and mental retardation.[5]

Today, clinical knowledge on ulegyria relies on case series and small-sized cohorts. In the present study, we therefore aimed to review our experience in the treatment of epilepsy in ulegyria while depicting clinical characteristics of patients as well as the epileptic seizures they experience through the data obtained by video-EEG recording and neuroimaging.

Materials and Methods

After obtaining approval from the local institutional review board, this study was carried out in the Neurology Department of Bursa Uludag University School of Medicine. All patients who were hospitalized between 2001 and 2020 at our clinics and evaluated by video-EEG imaging for the assessment of suitability for epilepsy surgery were analyzed retrospectively. The patients who received the diagnosis of

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ulegyria based on cranial magnetic resonance imaging (MRI) findings were included in the study.

Medical records including radiology and pathology reports, video-EEG analysis, operative notes, hospital progress, and outpatient clinic notes were reviewed retrospectively. A detailed anamnesis was obtained from each patient including perinatal histories for the presence of a hypoxic-ischemic insult. Next, each patient underwent a thorough neurological examination and radiological evaluation by high-resolution cranial MRI (1.5T scanner) as well as video-EEG analysis. The presence of ulegyria was examined on T1-weighted, T2-weighted, and fluid-attenuated inversion recovery (FLAIR) axial and coronal images. The patients who participated in this study were placed in the video-EEG unit in order to monitor their seizures and EEGs. Antiepileptic medications were discontinued by gradually decreasing the dosage 3 days before monitoring.[6] Superficial scalp electrodes were placed in accordance with the international 10 to 20 system. All patients were monitored 24 to 120 h continuously using GRASS-Telefactor Beehive Millennium (West Warwick) and Nicolet One VIASYS (CareFusion Corp.) long-term monitoring systems connected to a closed-circuit television system. Recordings were evaluated and analyzed by 2 senior neurologists, using GRASS-Telefactor/Nicolet reading station and Twin EEG/NicVue long-term monitoring software (CareFusion Corp.). Cerebral interictal F18-fluorodeoxyglucose-positron emission tomography (FDG-PET) was obtained in 7 of the patients. Seizure types were defined according to the Classification of the International League Against Epilepsy.[7] Similarly, neuroradiological analysis was performed by a senior neuroradiologist, who diagnosed ulegyria based on its previously described MRI findings including (I) small and atrophic gyri with relatively spared apex and broad sulci (mushroom-shaped appearance); (II) loss of volume of subcortical white matter in association with increased signal in T2-weighted and FLAIR sequences.[3,8] Neuropsychological evaluation was performed through neuropsychometric test (NPT) on all patients.

The results of clinical and radiological evaluation of each patient were discussed in the medical council consisted of 2 senior neurologists, a senior neurosurgeon, and a senior neuroradiologist in order to determine whether the patients were suitable for resective surgery for drug-resistant epilepsy. The patients whose seizures were under control with antiepileptic medication were not considered candidates for resection. Surgical results were based on Engel's classification of postoperative outcome.[9]

Results

The medical records and video-EEG recordings of a total of 1050 patients were analyzed retrospectively. Ulegyria was identified in 10 patients (6 female, 4 male) aged between 24 and 58 years (mean, 32 ± 9.8 years). Past medical history was remarkable for febrile seizures in 6 of the patients (60%) whereas there was a history of head trauma and neonatal asphyxia in 1 (10%) and 2 (20%) patients, respectively.

Neurological examination was remarkable for spastic hemiparesis in 1 (10%) of the patients contralateral to right perisylvian ulegyria. Two patients (20%) with occipital ulegyria had visual field deficits with moderate reduction of visual acuity.

Neuropsychometric test results revealed material-specific memory deficits in 2 of the patients whereas visuospatial deficits and attention deficits were noted in 1 and 4 of the patients, respectively.

According to cranial MRI results, ulegyria involved the temporoparietal region in 5 (50%) of the patients and the perisylvian area in 2 (20%) whereas frontal and occipital lobes were involved in 1 and 2 patients, respectively (Figure 1A and B). Except 1 patient with bilateral perisylvian ulegyria, all patients had unilateral lesions ($n = 9$, 90%) (Figure 1C and D). Hippocampal sclerosis accompanying ulegyria was found in 2 of the patients (20%). In all but 2 cases ($n = 2/7$) who underwent PET imaging, ulegyria was associated with hypometabolism in the cerebral regions involved.

All patients experienced epileptic seizures. A total of 23 seizures were recorded in these patients during the examination period. Mean age at seizure onset was 8.8 ± 5.4 years (range, 2-20 years). According to clinical features as well as interictal scalp EEG and EEG-video monitoring records, temporoparietal, and frontotemporal activities were noted in 5 (50%) and 2 (20%) of the patients, respectively. Generalized epileptic activity and epileptic activity caused from the occipital lobe were observed in 1 (10%) and 2 (20%) patients, respectively (Figure 2A and B). The seizures were successfully controlled by antiepileptic medication in 8 of the patients ($n = 8$, 80%). The remaining 2 patients (20%) who had accompanying hippocampal sclerosis suffered from drug-resistant epilepsy and therefore required microsurgical resection of the seizure foci. In surgically treated patients, the anterior two-thirds of the temporal lobe along with mesial structures were resected. Engel class II seizure control was achieved in both patients at the time of writing this paper. Clinical data of the patients are summarized in Table 1.

Discussion

Ulegyria is presumed to occur as a result of hypoxic-ischemic injury in term neonates and affects particularly the posterior cerebral artery distribution or the parasagittal watershed zones between the middle and posterior cerebral arteries[3] due to the higher metabolic rate in these areas in the immature brain.[10,11] The brain tissue at the bottom of gyri is vulnerable to hypoxia in newborns[12] whereas the apical parts are well-preserved. Therefore, the atrophy and thinning of the subcortical white matter at deep portions of the convolutions lead to the specific "mushroom" shape of ulegyria in the affected brain regions.[3,13,14] Consequently, this unique blood supply results in ulegyria involving primarily the posterior brain regions[2,8] although more anterior lesions may also occur.[8,15] In the

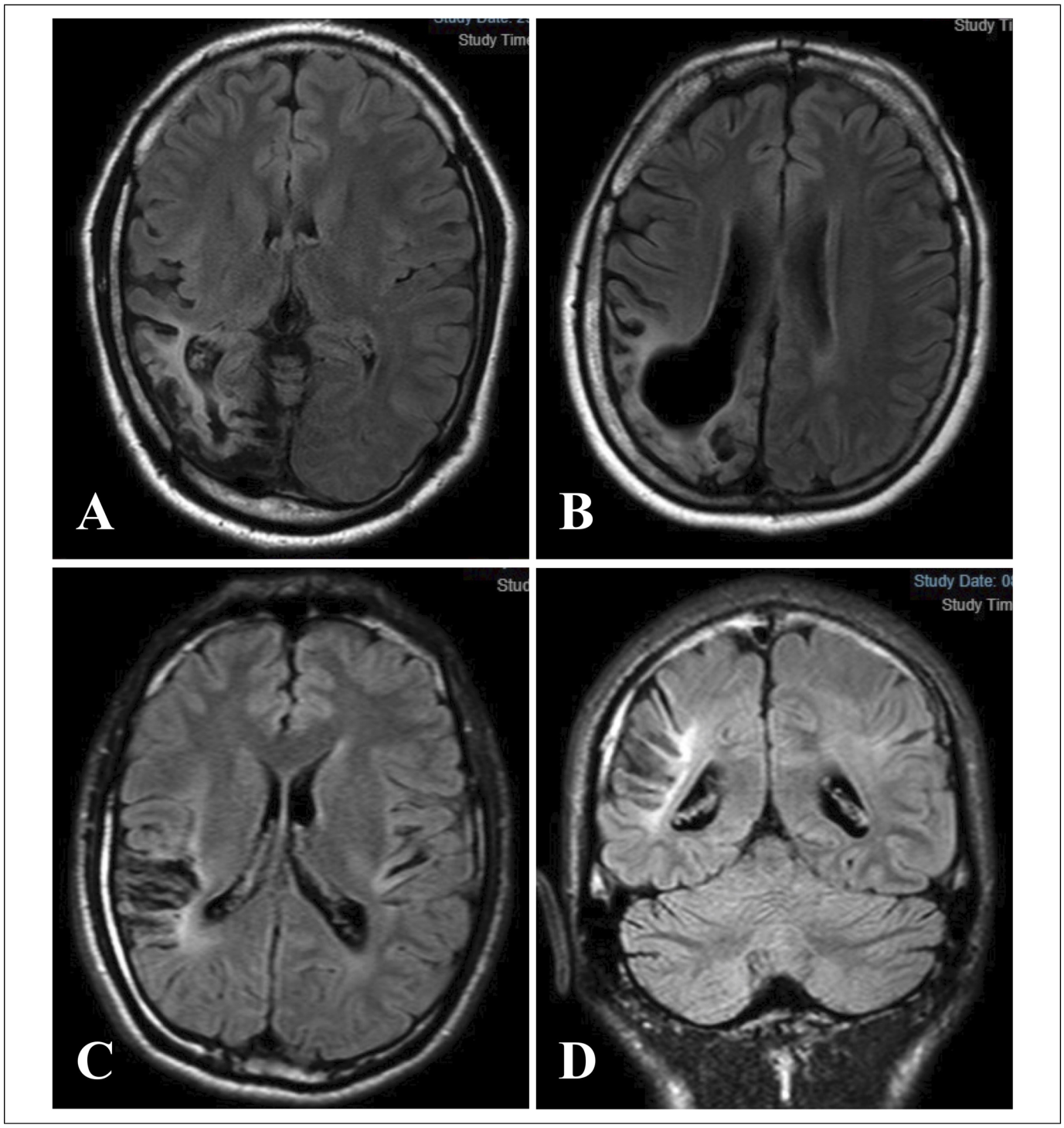


Figure 1. Magnetic resonance images demonstrating ulegyric gyri with a mushroom-like appearance. (A) Axial plane T2-fluid-attenuated inversion recovery (T2-FLAIR) image of a 26 years-old male patient with right temporoparietal ulegyria. (B) Axial plane T2-FLAIR image of a 33 years-old female patient with right temporoparietal ulegyria. (C, D) Axial and coronal planes T2-FLAIR images of a 28 years-old male patient with bilateral perisylvian ulegyria.

point of this fact, primarily the occipital or parietooccipital regions[2,8] as well as the perisylvian area has been reported as common localizations for the occurrence of ulegyric lesions in earlier studies.[4,15] In line with these, ulegyria was located in the posterior or perisylvian brain areas in all patients except the one with frontal localization in the present series.

Although first described by Bresler[16] with a clear definition more than a century ago, the clinical data regarding ulegyria

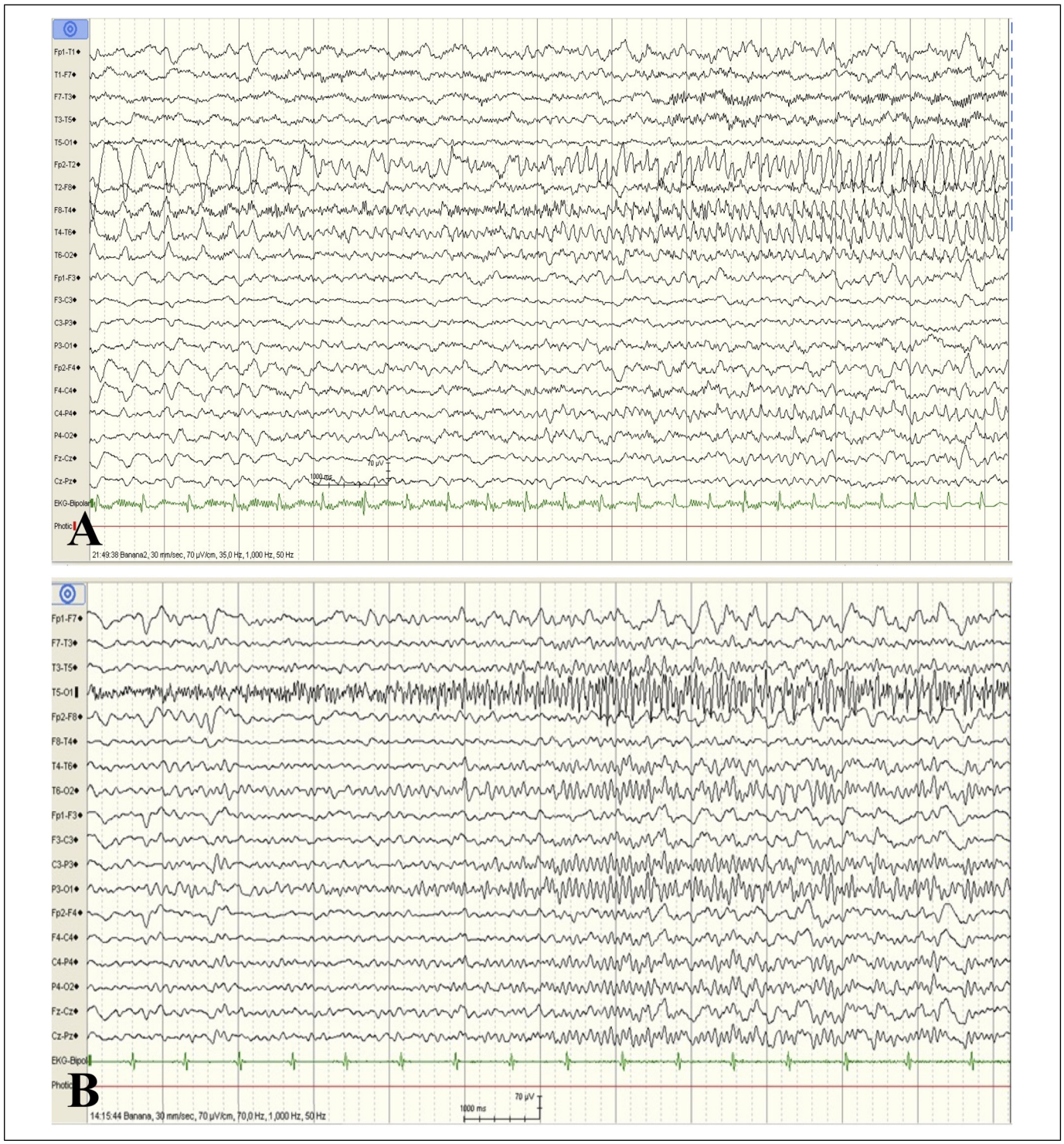


Figure 2. Representative ictal electroencephalography (EEG) findings in ulegyria. (A) Right frontotemporal ictal epileptic activity and (B) left occipital ictal epileptic activity.

remains to rely on case series and small-sized cohorts, leading to limited knowledge on clinical prognosis and the efficacy of medical and surgical treatment on seizure control. Today, with the aid of MRI, ulegyria can be readily distinguished from other cerebral lesions that can potentially cause drug-resistant epilepsy, including perinatal hypoxic-ischemic encephalopathy sequelae and developmental anomalies such as focal cortical dysplasia, polymicrogyria, multicystic encephalomalacia, and periventricular leukomalacia. For example, impaired perfusion during 20th to 24th weeks of gestation results in polymicrogyria

Table 1. Characteristics of Patients With Ulegyria.

Age/ gender	Age at onset (years)	History	Neurological examination	Seizure type	EEG findings	Cranial MRI	PET
26/M	10	Unremarkable	WNL	TLE	Right temporoparietal sharp wave and slow-wave activity	Right temporoparietal ulegyria	Right temporal hypometabolism
28/M	9	FC, FFH	Mild MR	FLE	Generalized activity	Bilateral perisylvian ulegyria	Ø
33/F	20	Unremarkable	WNL	TLE	Right temporoparietal sharp wave and slow-wave activity	Right temporoparietal ulegyria	Right temporoparietal hypometabolism
25/M	2	Neonatal asphyxia, FC	Left spastic hemiparesis	TLE	Right frontotemporoparietal slow-wave activity	Right hemisphere atrophy, Right perisylvian ulegyria	Ø
34/F	11	FC	WNL	TLE	Right temporoparietal sharp wave and slow-wave activity	Right temporoparietal ulegyria	Right temporoparietal hypometabolism
39/F	5	FC	Nystagmus, VFD	OS	Left occipital sharp wave activity	Left occipital ulegyria	Left cerebral hemisphere hypometabolism
28/M	2	Neonatal asphyxia, FC, meningitis	Mild MR	TLE	Right temporoparietal sharp wave and slow-wave activity	Right temporoparietal ulegyria	Ø
24/F	14	Unremarkable	WNL	TLE	Left frontotemporal sharp wave and slow-wave activity	Left frontal ulegyria	WNL
58/F	11	Unremarkable	VFD	TLE	Right occipital sharp wave and slow-wave activity	Right occipital ulegyria	WNL
25/F	4	FC	WNL	TLE	Left temporoparietal sharp wave and slow-wave activity	Left temporoparietal ulegyria	Left temporoparietal hypometabolism

Abbreviations: EEG, electroencephalography; FC, febrile convulsion; FFH, fall from height; FLE, frontal lobe epilepsy, F, female; M, male; MR, mental retardation; MRI, magnetic resonance imaging; PET: positron emission tomography; OS, occipital seizure, TLE, temporal lobe epilepsy; VFD, visual field defect; WNL, within normal limits.

that is consisted of microgyri coming close together and forming a thickened appearance of the cerebral cortex.[15] Although both ulegyria and polymicrogyria display white matter hyperintensities on T2-weighted images, ulegyria can be differentiated from polymicrogyria with its unique mushroom-shaped appearance that are associated with cortical lesions in the deeper parts of the sulci.[3] Similarly, focal cortical dysplasia may display white matter hyperintensities on T2-weighted images; however, no cortical atrophy is observed although cortical thickening is possible.[17]

As mentioned above, various degrees of perinatal stress, particularly hypoxic-ischemic brain injury have been underlined as the cause behind ulegyria.[1] Of note, in line with previous reports that could not document perinatal distress in all ulegyria patients, we were able to confirm a precise history of perinatal hypoxia in only 20% of the patients in this series. This was attributed to the possibility of inadequate documentation of the medical records.[4,8] On the other side, the history of febrile convulsions was present in 60% (n = 6) whereas the medical histories were unremarkable in the rest of our patients.

Ulegyria is associated with varying degrees of mental and motor disability.[2,3,8] As expected, the localization, extent of the involved brain area as well as uni- or bilaterality of ulegyria clearly defines the severity of the symptoms. For example, delayed acquisition of motor and language milestones as well as pseudobulbar signs including difficulty in sucking and swallowing were associated with perisylvian ulegyria[4] whereas occipital ulegyria resulted in visual field defects[2,5] in previous studies. Similarly, 90% of ulegyria patients had mild to moderate mental retardation in Usui et al's[2] series whereas varying severity of mental retardation was observed in 80% of patients in Kuchukhidze et al's[8] work. It should also be noted that varying proportions of bilaterality have been reported in earlier studies. Specifically, ulegyria was bilateral in 52% of the cases in Kuchukhidze et al's[8] study whereas this ratio was 56.6% and 60% in Gökçe and Çevik's[15] and Usui et al's[2] reports, respectively. In Villani et al's[3] series of 9, all cases suffered from bilateral ulegyria which was associated with severe impairment in mental and motor functions, preventing formal testing in one-third of the patients. Consistent with

former studies, we observed contralateral hemiparesis (10%) and visual impairment (20%) in ulegyria involving the parietal and occipital lobes, respectively. Additionally, although cognitive deficits including memory, visuospatial and attention deficits were noted in 7 (70%), mild mental retardation was observed only in 2 (20%) patients in the current report. In contrast with previous evidence, only 1 patient (10%) displayed bilateral ulegyria in our series.

In addition to mental and motor dysfunction, refractory epilepsy was a prominent component of the clinical picture in the majority of the published ulegyria series.[2,3,8] The accompaniment of hippocampal sclerosis, the most common pathology identified behind drug-resistant mesial temporal lobe epilepsy, to ulegyria has been reported previously in up to 48% of the cases.[3,4,8] In further detail, the seizures were refractory to medical treatment in 8 of the 9 (88.9%) patients with ulegyria in Villani et al's[3] work, 2 of which also harbored hippocampal sclerosis along with frontoparietooccipital and temporooccipital ulegyria. In Kuchukhidze et al's[8] series consisted of ulegyria primarily involving the posterior brain areas such as the occipital or parietooccipital regions, medically refractory epilepsy was observed in 17 of 25 patients. According to the same study, hippocampal sclerosis accompanied ulegyria in 48% of the cases. In Schilling et al's[4] work, hippocampal sclerosis was pathologically confirmed in 4 of the 12 cases with perisylvian ulegyria whereas the seizures were refractory to medical treatment in 5. On the other side, Gökçe and Çevik[15] have more recently reported successfully controlled epilepsy with either mono- or multidrug regimens in 26 of 30 patients with ulegyria. In this series of ulegyria, 4 patients had no seizure history. According to the same study by Gökçe and Çevik,[15] which was primarily based on radiological observations without histopathological confirmation for any of the patients, no signs of hippocampal sclerosis were noted for any patients. Interestingly, the intractability of the seizures correlated with the age at seizure onset and the extend of ulegyric lesions[2,3,8] rather than its coexistence with hippocampal sclerosis in earlier studies. On the other side, hippocampal sclerosis was concluded to result from either the same hypoxic-ischemic event responsible for the occurrence of ulegyria or result from secondary epileptogenesis.[4] Nevertheless, epilepsy severity was greatest in patients with early-onset epilepsy and the most extensive ulegyria lesions.[3] From a different perspective, ulegyria does not necessarily result in drug-resistant epilepsy in all cases.

In the present study, epilepsy was successfully controlled with a multidrug antiepileptic regimen in 80% (n = 8) of the cases whereas surgical resection of the epileptic foci was required in the remaining 2 patients who harbored concomitant hippocampal sclerosis (20%). These 2 patients with dual pathology, underwent a tailored subpial resection of the anterior two-thirds of the temporal lobe together with the mesial structures. At the time of writing this report, Engel class II seizure control was achieved in both patients. Our results were in line with the previous study of Kuchukhidze et al,[8] demonstrating medically intractable seizures in 68% of the patients with ulegyria (68%). In the same study, only 9 of 17 patients with pharmacoresistant seizures were included in the presurgical assessment program. Among these, Engel's class IA and IC seizure control were achieved in 2 patients who underwent epilepsy surgery.[8] Similarly, ulegyria was reported in one-fourth (n = 10; 25.6%) of a total of 39 medically intractable posterior cerebral cortex epilepsy cases by Usui et al,[2] who underwent surgical resection of the lesions. Seizure outcome was Engel's class I in 7 patients (Ia: 4 cases; Ic: 3 cases) and class III in 3 patients in the same study.[2] Similarly, in a multiinstitutional series of 12 patients with perisylvian ulegyria by Schilling et al,[4] resective surgery was performed in 5, resulting in Engel's class I seizure control in 1, Engel's class II seizure control in 3 and Engel's class IV seizure control in one of the patients.[4]

Potential limitations should be considered while interpreting the results of the current study. Firstly, being a retrospective observational study is a major weakness. Secondly, including only the patients who were selected from our inpatient video EEG monitoring population might have potentially led to some limitations in generalizability of the results to other patients in different settings. This also limits making more general conclusions on the incidence or prevalence of seizures in patients with ulegyria as all patients in our series have been preselected based on suspected seizures.

Conclusions

Ulegyria is a consequence of hypoxic brain injury in term neonates. Today, ulegyria is readily recognized with its unique MRI characteristics and clinical presentation including mental and motor impairment as well as seizures in the majority of the cases. Epilepsy, the major component of ulegyria, may be treated either medically or surgically in selected cases, depending on the age at onset and extent of the affected area which has a significant impact on the severity of the clinical picture.

Author Contributions

ABD conceived the study, performed data analysis, wrote the initial draft of the article, prepared the tables. PE prepared the figures, and revised the paper critically. AB, BH and IB supervised the data analysis, and critically revised the paper.

Declaration of Conflicting Interests

The authors declared no potential conflicts of interest with respect to the research, authorship, and/or publication of this article.

Funding

The authors received no financial support for the research, authorship and/or publication of this article.

Ethical Approval

Not applicable, because this article does not contain any studies with human or animal subjects.

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