Acta Clin Croat (Suppl. 3) 2021; 60:25-30
doi: 10.20471/acc.2021.60.s3.03

Review

POSTERIOR CORTEX SEIZURES – PEDIATRIC CHALLENGES

Maša Malenica[1,2], Monika Kukuruzović[1,2], Iva Šeparović[1,2] and Dunja Čokolić Petrović[3]

[1]Division of Child Neurology, Department of Pediatrics, Sestre milosrdnice University Hospital Centre, Zagreb, Croatia;
[2]Faculty of Teacher Education, University of Zagreb, EpiCARE Croatia, Zagreb, Croatia;
[3]Department of Pediatrics, Osijek University Hospital Centre, Osijek, Croatia

SUMMARY – Posterior cortex seizures have a complex semiologic presentation that is especially challenging in the pediatric population. Therefore, using clinical presentation in localizing ictal involvement is not sufficient in children, thus making this type of epilepsy quite under-recognized. As most of the ictal symptoms are subjective and could well be overshadowed by symptoms arising from adjacent cortices, primarily temporal and central ones, it is necessary not to overlook this large source of pharmacoresistant epilepsies. The parietal lobe as part of an extensive synaptic network is a great imitator, thus quite often producing inaccurate localization readings on scalp electroencephalography (EEG) due to very scattered interictal discharges and uninformative ictal recordings. Using direct cortical recordings in delineating the epileptogenic zone is helpful in some cases but even highly experienced epileptologists may erroneously interpret some features as arising from other localizations, especially the frontal lobe. Epilepsy surgery from the posterior quadrant is still quite rare and relatively unsuccessful, especially in non-lesional epilepsies due to elaborate mechanisms of connectivity, misleading semiology, and non-localizing EEG recordings, possibly due to insufficiency of parietal cortex synchronicity. Applying the aforementioned to the pediatric age makes it perhaps the most difficult challenge for a pediatric epileptologist.

Key words: *Posterior cortex epilepsy; Semiology; Pediatric epilepsy; Epilepsy surgery; Connectivity*

Introduction

Posterior cortex seizures primarily originate from the parietal and occipital lobes, as well as from the posterior part of the temporal lobe[1]. They account for 5%-10% of all focal seizures, but their frequency might be underestimated especially in children due to diagnostic difficulties[2]. The challenge of identifying posterior cortex epilepsy is even greater as the seizure semiology is often a result of very fast ictal propagation and peculiar connectivity of posterior cortex that gives rise to the highly variable ictal patterns such as focal tonic or clonic seizure activity and seizures with automatisms that might mimic frontal or temporal lobe semiology. Polymorphism of ictal manifestations in addition to the inability of children to accurately describe auras and ictal sensations accounts for the potential misdiagnosis of posterior cortex seizures and potentially gives rise to the increasing number of diagnosed psychogenic non-epileptic events. As one of the rarest localizations of epilepsy surgery, the posterior cortex has been examined less extensively in terms of anatomo-electro-clinical features, thus potentially leaving many inadequately treated pharmaco-resistant children[3,4]. For this reason, profound understanding of epileptic networks, semiology propagation and treatment options is especially crucial in this group of patients.

Correspondence to: *Maša Malenica, MD, PhD*, Division of Child Neurology, Department of Pediatrics, Sestre milosrdnice University Hospital Centre, Vinogradska c. 29, HR-10000 Zagreb, Croatia
E-mail: malenicamasa96@gmail.com

Classification of Posterior Cortex Epilepsies

In addition to the conventional classification of epilepsies defined by the International League Against Epilepsy[5], posterior cortex epilepsies could be classified by the anatomic structures involved, especially for surgical series[6]. This type of classification has a more outcome oriented view as the key principle is to determine the percentage of epilepsies from a certain lobe, or beyond in reference to the postsurgical seizure freedom. In pediatric patients, the most frequently performed surgeries are multilobar resections[7]. A more difficult way of classifying posterior cortex epilepsies is based on identifying the propagation pathways by using invasive procedures such as intracranial depth electrodes. This classification aims to improve our understanding of ictal phenomena observed in posterior cortex epilepsies by differentiating ventral propagation to the temporal/mesotemporal structures and dorsal propagation to the frontal and parietal structures[8]. Even though some semiologic features are unspecific, more occipital semiology is related to dorsal propagation, whereas ventral propagation yields more temporal semiology[8]. Therefore, the presence of oroalimentary automatisms typical for children with posterior cortex seizures could mostly be linked to the secondary involvement of mesial temporal lobe structures, probably *via* the multiple anatomical connections existing among the parietal lobe and the hippocampal formation[9]. Bipedal automatisms and some other automatic motor movements seen more frequently in adult patients might point to involvement of the frontoparietal system by disinhibition of innate activities controlled by the central pattern generators[10]. Overall, the need for different classifications of posterior cortex epilepsies due to the less defined boundaries and peculiar seizure propagation underlines the complexity of this entity.

Auras

As the first ictal symptoms, auras could be a very valuable piece of information in localizing and lateralizing the lobe of seizure origin. Recognizing, describing, and recalling auras is a challenge in itself, needless to say how difficult it may be for a child, or even impossible for a non-verbal child. The polymorphic character of posterior cortex auras emphasizes diagnostic uncertainty. Somatosensory auras are highly suggestive of parietal seizures, even though they could be found in other posterior cortex epilepsies[11], but could also be elicited with stimulation of the supplementary motor area (SMA)[12]. Somatosensory auras normally include tingling, numbness, electrical shock-like feelings, thermal sensations, and pain[13]. Somatosensory illusions such as movement of body parts and sensations of altered body size are most likely elicited from the non-dominant inferior parietal lobe or temporo-parieto-occipital junction. Visual auras could be simple such as flashing lights, or moving colors arising from the primary visual cortex, as opposed to more complex ones arising from the basal temporal cortex or temporo-occipital junction involving scenes and objects. Perhaps most widely reported visual auras are blurry vision and visual motions from the mesial parieto-occipital region[14]. Like complex visual auras, complex auditory auras are produced by activation of auditory association areas in the temporo-occipital cortex. Both visual and auditory auras are often associated with vertiginous auras such as sensation of rotation or falling arising from the parietal operculum and temporo-parietal area[15].

The well-established role of the parietal cortex in the processing of polysensory information could be related to unfavorable outcome of seizures related to polymorphic auras. However, the ability to recall different details of auras may imply that the initial discharge remains within the parietal lobe, without spreading to deeper structures or to the contralateral side, which might cause loss of awareness and amnesia for the auras[16]. As expected, not many children report an aura, while parents observe behavioral arrest in more than half of the cases. Using the aura could somewhat focus the search for epileptogenic zone in cases where the patient is aware of the aura and old enough to explain it properly. However, using solely the aura and the semiology could never be specific enough to suggest the posterior cortex seizure without further investigations.

Seizure Semiology in Posterior Cortex Epilepsies

Posterior cortex epilepsy published series are scarce, especially in children. Perhaps the most common notion of posterior cortex epilepsies is the fact that they mimic epilepsies from other brain regions. Consider-

ing the large volume of the posterior cortex lacking clear anatomic boundaries, as well as its peculiar functional connectivity *via* fast propagation pathways, it is no wonder that focal seizures arising from this area will be of quite variable semiology. Most of the posterior cortex seizures are short, up to three minutes, and nearly half of them start in sleep[17]. Seizures from the parietal lobe usually start in young children around the age of 6 years, and in multilobar involvement even younger, between 1 and 2 years[18]. Seizure frequency is usually quite high (even daily), and the occurrence of status epilepticus is not uncommon.

As *per* semiologic presentations seen in posterior cortex epilepsies, the presence of manual and oral automatisms with loss of consciousness indicates a more temporal involvement, whereas tonic posturing with hypermotor behavior indicates frontal lobe involvement[15]. In the very young patients, posterior cortex epilepsies might even present as epileptic spasms which are pharmacoresistant and may require surgery[7]. Vestibular symptoms, vertiginous sensations, and visual illusions might be the most common symptoms reported in seizures of the posterior cortex. The superior parietal cortex, especially the temporal-parietal junction and the intraparietal sulcus can produce vestibular symptoms[3,19], whereas visual illusions such as object motion and blurred vision might be most likely evoked by stimulation of the basal temporal-occipital, mesial parietal-occipital, or temporal-parietal-occipital junctional region[20]. The ambiguity of certain posterior cortex symptoms, especially when not coupled with secondary generalization or clear-cut epileptic signs, underscores the misdiagnosis of posterior cortex seizures with non-epileptic events, especially in pediatric population where the incidence of parietal lobe epilepsy is very high[21-23].

Motor manifestations are mostly myoclonic (especially with the head nod) as opposed to the adult patients, tonic seizures (elevation of arms) indicating the spread to the SMA[6], and epileptic spasms, while the rest are non-motor seizures (psychomotor and hypomotor seizures) indicating a possible spread to the temporal lobe[24,25]. Oculomotor features related to posterior cortex seizures are nystagmus, eye deviation, lid myoclonia, and repetitive blinking. Ictally, these seizures might also generate vocalization, laughing, smiling, or flushing. Some of these manifestations could only be seen by very careful video-electroencephalography (EEG) monitoring rather than relying on the observer's descriptions. In pediatric patients with posterior cortex epilepsies, many motor seizures appear in axial or symmetric form, thus reducing the chance for lateralization of the epileptic zone. Reported automatisms are mostly oral and relate to the occipital lobe rather than the parietal one, whereas in adult patients, oral automatisms are very rarely described[17]. Among pediatric cases, there are almost no secondary generalized tonic-clonic seizures, which are readily reported in adult patients[26]. There have been attempts of understanding this observation and the most likely one is that there is gradual maturation of children's brains with sequential myelin formation causing poor synchronization of both hemispheres[27].

Semiology of the parietal lobe could further be characterized by positive or negative somatosensory symptoms, usually not including the sense of pain[28]. Some of the examples are kinetic such as the illusion of movement, postural or rotatory movements, or vertigo. Some resemble temporal semiology such as nausea or intra-abdominal sensation. With involvement of the non-dominant hemisphere, there could be asomatognosia or loss of awareness of a body part, whereas involvement of the dominant hemisphere could result in receptive or conductive aphasia.

With the involvement of occipital lobe, visual symptoms are most prominent, even though they could appear from the parieto-temporo-occipital junction as well. It is this junction that gives rise to the Alice in Wonderland syndrome with altered body image symptoms such as macropsia and micropsia, telopsia, pelopsia or meta-morphopsia, most commonly in children[29]. Usually, the patient describes disproportionate sizes of body parts, mainly the head and hands. Such cases should prompt investigation of the temporal lobe involvement, exclusion of the Epstein Barr virus infection, and toxic delirium. Typical occipital lobe visual symptoms affecting the visual cortex are mostly characterized by elementary visual phenomena. Patients sometimes describe or even draw flashes, colorful circles, or rotating colors as examples of positive phenomena. Less often, they notice or report negative phenomena such as scotomata, hemianopsia, or rarely amaurosis. These negative phenomena might mislead the investigation towards the ophthalmologist, or demyelinating disorder diagnostics. Fast spread to contiguous cortex might overshadow the initial symptoms

and their transient character might not be sufficient for a child to report with confidence. Localizing the hemifield with visual symptoms might point to involvement of the contralateral lobe, and recording epileptic nystagmus, or eyelid flutter could aid in confirmation of the occipital lobe involvement[30]. However, pure eye deviation (regardless of head deviation) should be considered as related to the parietal eye field, located in the lateral intraparietal subregion, but also to the presence of a second eye field in the medial parietal area[31].

Electroencephalography in Posterior Cortex Seizures

Unfortunately, like seizure semiology, encephalography in posterior cortex seizures is also far from clear and uniform. Most of the studies have shown multifocal, bilateral, or sometimes falsely localizing activity. Furthermore, studies have not even found the prognostic relevance of one or another type of EEG findings. A large diagnostic problem poses uninformative EEG, as there is often a tendency of multiple EEG spread patterns and non-localizing scalp EEG findings[32]. In fact, the presence of large association areas in the parietal lobe and its elaborate connectivity to various distant regions reasonably accounts for these EEG features, and probably accounts for a relatively higher incidence of invasive procedures in parietal lobe epilepsy compared with other focal epilepsies. Certainly, the recommendation is to use the invasive stereo-EEG method in cases where there is justified suspicion of posterior cortex epilepsy and indication for surgical treatment.

Conclusion

Childhood and adulthood posterior cortex seizures share many common clinical features but there are important distinct age-dependent differences. As a significant number of posterior cortex epilepsies occur among children with a high number of seizures and the likelihood to develop pharmacoresistant epileptic spasms, it is of utmost importance to recognize them early in the course of the disease. This phenomenon could be related to the early development and maturation of the posterior cortex but also to the presence of focal cortical dysplasia, which may delay maturation and distort the widespread networks early on. A distinct pediatric problem is the immaturity of patients and their inability (sometimes related to associated disabilities) to report auras, which may be the only symptom of a seizure arising from the posterior cortex. Poor localization of the EEG coupled with very short and frequent seizures, the lack of aura description, and heterogeneous semiology that reflects secondary spread rather than primary seizure onset makes this group of epilepsies very challenging. Detailed description of auras (by detailed history-taking) followed by video analysis of the chronology of ictal semiology and electrophysiologic features (interictal and ictal scalp EEG) together with neuroimaging (high-resolution magnetic resonance imaging) should aid in creating a complete electroclinical pattern to provide topographic orientation for posterior cortex epilepsy. In cases where these data should fail to discover the epileptogenic zone, a stereo-EEG evaluation with stereotactically placed intracerebral electrodes arranged according to a pre-defined localization could explore the areas suspected in seizure onset and early propagation towards the most effective rate of surgical success of posterior cortex epilepsy, especially in pediatric population[33,34]. In addition, neurological and neuropsychological evaluation with standardized tests applied according to the patient age should complete the work-up in these challenging epilepsies[35]. Identifying posterior cortex epilepsies by understanding the whole network upon completing the extensive diagnostic investigations should provide the best possible seizure and functional outcome.

References

1. Yu T, Wang Y, Zhang G, Cai L, Du W, Li Y. Posterior cortex epilepsy: diagnostic considerations and surgical outcome. Seizure. 2009 May;18(4):288-92. doi: 10.1016/j.seizure.2008.11.008. Epub 2009 Jan 10. PMID: 19136282.
2. Salanova V, Markand O, Worth R. Longitudinal follow-up in 145 patients with medically refractory temporal lobe epilepsy treated surgically between 1984 and 1995. Epilepsia. 1999 Oct;40(10):1417-23. doi: 10.1111/j.1528-1157.1999.tb02014.x. PMID: 10528938.
3. Bartolomei F, Gavaret M, Hewett R, Valton L, Aubert S, Régis J, Wendling F, Chauvel P. Neural networks underlying parietal lobe seizures: a quantified study from intracerebral recordings. Epilepsy Res. 2011 Feb;93(2-3):164-76. doi: 10.1016/j.eplepsyres.2010.12.005. Epub 2011 Jan 11. PMID: 21227653.

4. Binder DK, Podlogar M, Clusmann H, Bien C, Urbach H, Schramm J, Kral T. Surgical treatment of parietal lobe epilepsy. J Neurosurg. 2009 Jun;110(6):1170-8. doi: 10.3171/2008.2.17665. PMID: 19199502.
5. Scheffer IE, Berkovic S, Capovilla G, Connolly MB, French J, Guilhoto L, Hirsch E, Jain S, Mathern GW, Moshé SL, Nordli DR, Perucca E, Tomson T, Wiebe S, Zhang YH, Zuberi SM. ILAE classification of the epilepsies: Position Paper of the ILAE Commission for Classification and Terminology. Epilepsia. 2017 Apr;58(4):512-21. doi: 10.1111/epi.13709. Epub 2017 Mar 8. PMID: 28276062; PMCID: PMC5386840.
6. Baumgartner C, Flint R, Tuxhorn I, Van Ness PC, Kosalko J, Olbrich A, Almer G, Novak K, Lüders HO. Supplementary motor area seizures: propagation pathways as studied with invasive recordings. Neurology. 1996 Feb;46(2):508-14. doi: 10.1212/wnl.46.2.508. PMID: 8614523.
7. Sierra-Marcos A, Fournier-Del Castillo MC, Álvarez-Linera J, Budke M, García-Fernández M, Pérez-Jiménez MA. Functional surgery in pediatric drug-resistant posterior cortex epilepsy: electro-clinical findings, cognitive and seizure outcome. Seizure. 2017 Nov;52:46-52. doi: 0.1016/j.seizure.2017.09.013. Epub 2017 Sep 22. PMID: 28963933.
8. Craciun L, Taussig D, Ferrand-Sorbets S, Pasqualini E, Biraben A, Delalande O, Dorison N, Fohlen M, Dorfmuller G, Chipaux M. Investigation of paediatric occipital epilepsy using stereo-EEG reveals a better surgical outcome than in adults, especially when the supracalcarine area is affected. Epileptic Disord. 2018 Oct 1;20(5):346-63. doi: 10.1684/epd.2018.1000. PMID: 30378548.
9. Rushworth MF, Behrens TE, Johansen-Berg H. Connection patterns distinguish 3 regions of human parietal cortex. Cereb Cortex. 2006 Oct;16(10):1418-30. doi: 10.1093/cercor/bhj079. Epub 2005 Nov 23. PMID: 16306320.
10. Tassinari CA, Rubboli G, Gardella E, Cantalupo G, Calandra-Buonaura G, Vedovello M, Alessandria M, Gandini G, Cinotti S, Zamponi N, Meletti S. Central pattern generators for a common semiology in fronto-limbic seizures and in parasomnias. A neuroethologic approach. Neurol Sci. 2005 Dec;26 Suppl 3: s225-32. doi: 10.1007/s10072-005-0492-8. PMID: 16331401.
11. Sveinbjornsdottir S, Duncan JS. Parietal and occipital lobe epilepsy: a review. Epilepsia. 1993 May-Jun;34(3):493-521. doi: 10.1111/j.1528-1157.1993.tb02590.x. Erratum in: Epilepsia 1994 Mar-Apr;35(2):467. PMID: 8504783.
12. Guldvog B, Løyning Y, Hauglie-Hanssen E, Flood S, Bjørnaes H. Surgical treatment for partial epilepsy among Norwegian children and adolescents. Epilepsia. 1994 May-Jun;35(3): 554-65. doi: 10.1111/j.1528-1157.1994.tb02475.x. PMID: 8026401.
13. Foldvary-Schaefer N, Unnwongse K. Localizing and lateralizing features of auras and seizures. Epilepsy Behav. 2011 Feb;20(2):160-6. doi: 10.1016/j.yebeh.2010.08.034. PMID: 20926350.
14. Richer F, Martinez M, Cohen H, Saint-Hilaire JM. Visual motion perception from stimulation of the human medial parieto-occipital cortex. Exp Brain Res. 1991;87(3):649-52. doi: 10.1007/BF00227090. PMID: 1783033.
15. Fish DR, Gloor P, Quesney FL, Olivier A. Clinical responses to electrical brain stimulation of the temporal and frontal lobes in patients with epilepsy. Pathophysiological implications. Brain. 1993 Apr;116 (Pt 2):397-414. doi: 10.1093/brain/116.2.397. PMID: 8461973.
16. Abou-Khalil BW. Multiple auras: not an ominous sign for epilepsy surgery. Epilepsy Curr. 2008 Mar-Apr;8(2):39-40. doi: 10.1111/j.1535-7511.2008.00230.x. PMID: 18330465; PMCID: PMC2265812.
17. Fogarasi A, Boesebeck F, Tuxhorn I. A detailed analysis of symptomatic posterior cortex seizure semiology in children younger than seven years. Epilepsia. 2003 Jan;44(1):89-96. doi: 10.1046/j.1528-1157.2003.18302.x. PMID: 12581235.
18. Aykut-Bingol C, Bronen RA, Kim JH, Spencer DD, Spencer SS. Surgical outcome in occipital lobe epilepsy: implications for pathophysiology. Ann Neurol. 1998 Jul;44(1):60-9. doi: 10.1002/ana.410440112. PMID: 9667593.
19. Kahane P, Hoffmann D, Minotti L, Berthoz A. Reappraisal of the human vestibular cortex by cortical electrical stimulation study. Ann Neurol. 2003 Nov;54(5):615-24. doi: 10.1002/ana.10726. PMID: 14595651.
20. Ristić AJ, Alexopoulos AV, So N, Wong C, Najm IM. Parietal lobe epilepsy: the great imitator among focal epilepsies. Epileptic Disord. 2012 Mar;14(1):22-31. doi: 10.1684/epd.2012.0484. PMID: 22426412.
21. Dalmagro CL, Bianchin MM, Velasco TR, Alexandre V Jr, Walz R, Terra-Bustamante VC, Inuzuka LM, Wichert-Ana L, Araújo D Jr, Serafini LN, Carlotti CG Jr, Assirati JA Jr, Machado HR, Santos AC, Sakamoto AC. Clinical features of patients with posterior cortex epilepsies and predictors of surgical outcome. Epilepsia. 2005 Sep;46(9):1442-9. doi: 10.1111/j.1528-1167.2005.70904.x. PMID: 16146440.
22. Bartolomei F, Wendling F, Chauvel P. Le concept de réseau épileptogène dans les épilepsies partielles humaines [The concept of an epileptogenic network in human partial epilepsies]. Neurochirurgie. 2008 May;54(3):174-84. (in French) doi: 10.1016/j.neuchi.2008.02.013. Epub 2008 Apr 16. PMID: 18420227.
23. Liava A, Mai R, Tassi L, Cossu M, Sartori I, Nobili L, Lo Russo G, Francione S. Paediatric epilepsy surgery in the posterior cortex: a study of 62 cases. Epileptic Disord. 2014 Jun;16(2):141-64. doi: 10.1684/epd.2014.0648. PMID: 24853765.
24. Roger J, Dravet C, Menendez P, Bureau M. Les epilepsies partielles de l'enfant – evolution et facteurs de pronostic [The partial epilepsies in childhood – evolution and prognosis factors (author's translation)]. Rev Electroencephalogr Neurophysiol Clin. 1981 Dec;11(3-4):431-7. (in French) doi: 10.1016/s0370-4475(81)80082-2. PMID: 6808600.
25. Fois A, Malandrini F, Tomaccini D. Clinical findings in children with occipital paroxysmal discharges. Epilepsia. 1988 Sep-Oct;29(5):620-3. doi: 10.1111/j.1528-1157.1988.tb03771.x. PMID: 3409847.

26. Aykut-Bingol C, Bronen RA, Kim JH, Spencer DD, Spencer SS. Surgical outcome in occipital lobe epilepsy: implications for pathophysiology. Ann Neurol. 1998 Jul;44(1):60-9. doi: 10.1002/ana.410440112. PMID: 9667593.

27. Chugani HT, Phelps ME, Mazziotta JC. Positron emission tomography study of human brain functional development. Ann Neurol. 1987 Oct;22(4):487-97. doi: 10.1002/ana.410220408. PMID: 3501693.

28. Ho SS, Berkovic SF, Newton MR, Austin MC, McKay WJ, Bladin PF. Parietal lobe epilepsy: clinical features and seizure localization by ictal SPECT. Neurology. 1994 Dec;44(12):2277-84. doi: 10.1212/wnl.44.12.2277. PMID: 7991112.

29. Gaul C, Kraya T, Holle D, Benkel-Herrenbrück I, Schara U, Ebinger F. Migränevarianten und ungewöhnliche Manifestationen der Migräne im Kindesalter [Migraine variants and unusual types of migraine in childhood]. Schmerz. 2011 Apr; 25(2):148-56. (in German) doi: 10.1007/s00482-011-1021-8. PMID: 21431964.

30. Salanova V, Andermann F, Olivier A, Rasmussen T, Quesney LF. Occipital lobe epilepsy: electroclinical manifestations, electrocorticography, cortical stimulation and outcome in 42 patients treated between 1930 and 1991. Surgery of occipital lobe epilepsy. Brain. 1992 Dec;115 (Pt 6):1655-80. doi: 10.1093/brain/115.6.1655. PMID: 1486456.

31. Pierrot-Deseilligny C, Milea D, Müri RM. Eye movement control by the cerebral cortex. Curr Opin Neurol. 2004 Feb;17(1):17-25. doi: 10.1097/00019052-200402000-00005. PMID: 15090873.

32. Williamson PD, Boon PA, Thadani VM, Darcey TM, Spencer DD, Spencer SS, Novelly RA, Mattson RH. Parietal lobe epilepsy: diagnostic considerations and results of surgery. Ann Neurol. 1992 Feb;31(2):193-201. doi: 10.1002/ana.410310210. PMID: 1575458.

33. Ramantani G, Stathi A, Brandt A, Strobl K, Schubert-Bast S, Wiegand G, Korinthenberg R, van Velthoven V, Zentner J, Schulze-Bonhage A, Bast T. Posterior cortex epilepsy surgery in childhood and adolescence: predictors of long-term seizure outcome. Epilepsia. 2017 Mar;58(3):412-9. doi: 10.1111/epi.13654. Epub 2017 Jan 18. PMID: 28098941.

34. Francione S, Liava A, Mai R, Nobili L, Sartori I, Tassi L, Scarpa P, Cardinale F, Castana L, Cossu M, Lo Russo G. Drug-resistant parietal epilepsy: polymorphic ictal semiology does not preclude good post-surgical outcome. Epileptic Disord. 2015 Mar;17(1):32-46; quiz 46. doi: 10.1684/epd.2015.0728. PMID: 25788021.

35. Guerrini R, Scerrati M, Rubboli G, Esposito V, Colicchio G, Cossu M, Marras CE, Tassi L, Tinuper P, Paola Canevini M, Quarato P, Giordano F, Granata T, Villani F, Giulioni M, Scarpa P, Barbieri V, Bottini G, Del Sole A, Vatti G, Spreafico R, Lo Russo G; Commission for Epilepsy Surgery of the Italian League Against Epilepsy. Overview of presurgical assessment and surgical treatment of epilepsy from the Italian League Against Epilepsy. Epilepsia. 2013 Oct;54 Suppl 7:35-48. doi: 10.1111/epi.12308. PMID: 24099054.

Sažetak

NAPADAJI POSTERIORNOG KORTEKSA – IZAZOVI U PEDIJATRIJI

M. Malenica, M. Kukuruzović, I. Šeparović i *D. Čokolić Petrović*

Napadaji posteriornog korteksa imaju složemu semiologiju koja je osobito izazovna u pedijatrijskoj populaciji. Upravo zato primjena kliničke prezentacije u lokalizaciji iktalnog ishodišta nije dovoljno u djece, čime je ova vrsta epilepsije slabo prepoznata. S obzirom na to da je većina iktalnih simptoma subjektivna i može biti skrivena simptomima susjednih korteksa, primarno temporalnim i centralnim, važno je ne preskočiti ovaj važan izvor farmakorezistentnih epilepsija. Uz semiologiju, parijetalni je režanj kao dio velike sinaptičke mreže veliki imitator i često stvara netočnu lokalizaciju na elektroencefalogramu (EEG) zbog velike distribucije interiktalnih izbijanja i neinformativnih iktalnih zapisa. Korištenje direktnih kortikalnih zapisa u definiciji epileptogene zone je korisno u nekim slučajevima, no čak i vrlo iskusni epileptolozi mogu pogrešno protumačiti neke značajke s ishodom iz drugih izvora, osobito frontalnog režnja. Operacije epilepsije posteriornog kvadranta su vrlo rijetke i prilično neuspješne, osobito u ne-lezionalnim epilepsijama zbog osebujnih mehanizama povezanosti, zavaravajuće semiologije i ne-lokalizirajućeg EEG zapisa moguće zbog nedovoljne sinkroniziranosti parijetalnog korteksa. Primjena navedenog na pedijatrijsku dob čini epilepsije ovog dijela korteksa možda najvećim izazovom za pedijatrijske epileptologe.

Ključne riječi: *Epilepsije posteriornog korteksa; Semiologija; Epilepsije dječje dobi; Operacije epilepsije; Konektivnost*