Crimson Publishers
Wings to the Research

Mini Review

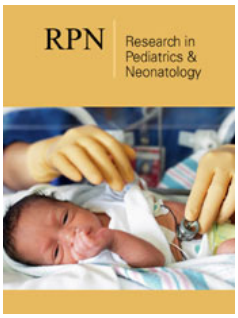
Childhood Brain Damage: Clinical and Electroencephalographic Evaluation

Aguilar Fabré Liane[1], Aguilar Fabré Kenia[1], Rodríguez Aguilar René Orlando[2] and Rodríguez Valdés René Francisco[1]*

[1]Neurodiagnostic and Rehabilitation "Dr. Moisés López González". University Health System. Autonomous University of Queretaro, Mexico

[2]University of the Valley of Mexico. Campus Queretaro, Mexico

ISSN: 2576-9200



***Corresponding author:** René Francisco Rodríguez Valdés, Neurodiagnostic and Rehabilitation "Dr. Moisés López González". University Health System. Autonomous University of Queretaro, Mexico

Submission: March 01, 2023
Published: April 05, 2023

Volume 7 - Issue 4

How to cite this article: Aguilar Fabré Liane, Aguilar Fabré Kenia, Rodríguez Aguilar René Orlando and Rodríguez Valdés René Francisco*. Childhood Brain Damage: Clinical and Electroencephalographic Evaluation. Res Pediatr Neonatol. 7(4). RPN. 000667. 2023. DOI: 10.31031/RPN.2023.07.000667

Abstract

Infant brain damage is estimated at between 2-5% of live births. Preterm newborns constitute a vulnerable population with a high risk of suffering medical problems and neurobehavioral disabilities. Perinatal asphyxia is the most representative risk factor it is a serious incident in neonates due to hypoxia and generalized ischemia that causes biochemical and functional changes of a systemic nature, particularly in the Central Nervous System (CSN). Seizures in the neonatal period are the characteristic clinical expression of a CNS dysfunction. The incidence of neonatal seizures (NS) is 0.15-1.4% of newborns. The electro-encephalogram is one of the few methods that allow the functional study of the CNS. In addition to having the advantages of safety and low economic cost. Early clinical and electroencephalographic evaluation could be useful to guide, modify, or suggest therapeutic and follow-up behaviors in newborns with risk factors for neurological damage.

Introduction

Infant brain damage is estimated at between 2-5% of live births [1] and there are several prenatal, perinatal, postnatal and social risk factors that increase the risk of developing child neurodevelopmental deviations. Damage that occurs in the perinatal period causes between 55-75% of neurological deficits. Preterm newborns constitute a vulnerable population with a high risk of suffering medical problems and neurobehavioral disabilities [2,3] including poor cognitive performance, greater learning difficulties, as well as a high risk of presenting behavioral disorders. Of the total number of premature children, up to 47% of them later present cerebral palsy, 27% show significant cognitive disorders and sensory disorders are found in 23-37% [4,5]. Perinatal asphyxia is the most representative risk factor [6] it is a serious incident in neonates due to hypoxia and generalized ischemia that causes biochemical and functional changes of a systemic nature, particularly in the Central Nervous System (CNS) [7]. Nagdyman et al. [8] state that approximately one third of newborns with asphyxia have hypoxic ischemic encephalopathy. The diagnosis of a perinatal asphyxia event implies the early onset of a neonatal neurological syndrome. Various studies have confirmed the neurological sequelae of perinatal hypoxia, which can range from mild to severe [9-12].

Neonates with perinatal asphyxia who are most at risk of death or subsequent neurological disability are those with persistently low Apgar scores, other neurological signs, and seizures in the first 48 hours of life [13]. The 5-minute Apgar score of life is the one with the highest concordance with metabolic acidosis and the best correlation with the risk of neurological sequelae, although the presence of a normal Apgar score does not exclude the possibility of future neurological sequelae [14,15]. Seizures in the neonatal period are the characteristic clinical expression of a Central Nervous System (CNS) dysfunction [16]. The incidence of neonatal seizures (NS) is 0.15-1.4% of newborns [17]. Its recognition is not always easy, and it can go unnoticed, especially in the preterm newborn, which is related

to the anatomical, biochemical and physiology of the CNS during the perinatal stage [16]. The clinical and electroencephalographic manifestations differ from those of the newborn with greater neurological maturity, reflecting functional differences due to a lower degree of myelination [18]. The occurrence of seizures in the newborn is of great importance due to its association with high rates of neurological mortality and morbidity. In addition, they require urgent diagnosis and treatment to prevent the worsening of underlying brain lesions [19,20].

The electroencephalogram is one of the few methods that allow the functional study of the CNS. In addition to having the advantages of safety and low economic cost compared to neuroimaging techniques, such as tomography and magnetic resonance imaging, which are structural methods that allow the lesion to be visualized, but do not inform us about the pathophysiological process. In relation to the etiology of the seizures and the moment of their appearance, some authors report that most of the seizures that occur before the fifth day of life are usually due to hypoxic-ischemic encephalopathy, intracranial hemorrhage, metabolic alterations, infection of the CNS or by the direct effect of drugs [17,21-23]. Ninety percent of the seizures that appear during hypoxia-ischemia do so in the first 48 hours of life. Seizures that manifest between 24-72 hours of life are due to CNS infections, drug withdrawal, hemorrhage, the onset of inborn errors of metabolism, or brain malformations [17,24-26].

Aguilar F [27] report that patients with neonatal seizures present moderate and severe electro-encephalographic alterations. The recording of a pathological EEG (critical or with serious alterations) is associated with an unfavorable evolution in most cases. A more accurate prognosis in these patients can be made based on the etiology of the neonatal seizures and the electroencephalographic patterns [19]. The persistence of pathological records beyond 72 hours after birth is invariably associated with death or severe neurological sequelae, while early recovery, before 12 or at least 36 hours, is associated with normal results or with neurological alterations minors [28]. The study by Jiménez et al. [29] describes the presence of clinical neurological alterations during the first week of life, the presence of seizures and a pathological EEG as the main prognostic factors in perinatal asphyxia. Preterm infants with a normal interictal EEG generally have a good prognosis, on the other hand, the EEG tracing of "suppression-surges" in the neonatal period translates a poor prognosis except when said tracing is of pharmacological origin [30]. Andre et al. [31] state that those asphyxia newborns who continued to present clinical and electroencephalographic alterations on the seventh day after birth, subsequently showed sequelae in up to 75% of cases.

However, the EEG is very useful to confirm suspicions, but it is not definitive for the diagnosis of neonatal seizures. Conventional electroencephalography has a series of limitations in the study of these patients, among which are:

a) Difficulties in prolonged monitoring;

b) Excessive number of electrodes;

c) Electrical interference from equipment in the environment;

d) Difficulties in interpreting the study as staff trained in clinical neurophysiology are necessary;

e) Brief recordings (45-60 minutes) that even with periodic evaluations information is lost on the evolution of the alterations of the basic activity, sleep states and sporadic seizures. The incorporation of the amplitude-integrated EEG, also known as the brain function monitor method, is a simple method of continuous recording of cortical electrical activity, allowing the prediction of final neurological evolution in as short a time as the first 6 hours of life [28,32,33].

Conclusion

Early clinical and electroencephalographic evaluation could be useful to guide, modify, or suggest therapeutic and follow-up behaviors in newborns with risk factors for neurological damage.

References

1. Ferriero DM (2005) Neonatal brain injury. N Engl J Med 351(19): 1985-1995.
2. Clark C, Woodward L (2010) Neonatal cerebral abnormalities and later verbal and visuospatial working memory abilities of children born very preterm. Dev Neuropsychol 35(6): 622-642.
3. Taylor HG, Klein N, Minich NM, Hack M (2000) Middle-school-age outcomes in children with very low birth weight. Child Dev 71 (6): 1495-1511.
4. Peterson B, Anderson AW, Ehrenkranz R, Staib LH, Tageldin M, et al. (2003) Regional brain volumes and their later neurodevelopmental correlate in term and preterm infants. Pediatrics 111(5 Pt 1): 939-948.
5. Fraello D, Maller KJ, Vohr B, Katz KH, Kesler S, et al. (2011) Consequence of preterm birth in early adolescence: The role of language on auditory short-term memory. J Child Neurol 26(6): 738-742.
6. Watanabe K, Miyazaki S, Hara K, Hakamada S (1980) Behavioral state cycles, background EEGs and prognosis of newborns with perinatal hypoxia. Electroencephalogr Clin Neurophysiol 49(5-6): 618-625.
7. Flores CJL, Cruz F, Orozco G, Vélez A (2013) Perinatal hypoxia and its impact on neurodevelopment. Rev Child Neuropsychol 8: 26-31.
8. Nagdyman N, Kömen W, Ko HK, Müller C, Obladen M (2001) Early biochemical indicators of hypoxic-ischemic encephalopathy after birth asphyxia. Pediatr Res 49(4): 502-506.
9. Azzopardi D (2010) Clinical management of the baby with hypoxic ischaemic encephalopathy. Early Human Development 86(6): 345-350.
10. Delfino A, Weinberger M, Delucchi G, Field S, Bargeño M, et al. (2010) Follow-up of newborns with perinatal asphyxia. Archives of Pediatrics of Uruguay 81: 73-77.
11. James A, Cherian S (2010) Pathophysiology of perinatal hypoxia-ischaema. Paediatrics and Child Health 20(8): 351-355.
12. Martínez BM, Diez SJ, Rutherford M, Cowan F (2010) Outcomes after central grey matter injury in term perinatal hypoxic-ischemic encephalopathy. Early Human Development 86(11): 675-682.
13. Swaiman KF, Russman BS (1999) Cerebral Palsy. In: Swaiman KF, Ashwal S (Eds.), Pediatric neurology. Principles and practice. (3rd edn), St. Louis: Mosby, pp. 312-324.

14. Manganaro R, Mami C, Gemelli M (1994) The validity of the Apgar Scores in the assessment of asphyxia at birth. Eur J Obstet Gynecol Reprod Biol 54(2): 99-102.

15. Nelson KB, Ellemberg JH (1981) Apgar score as a predictor of chronic neurologic disability. Pediatrics 68(1): 36-44.

16. Volpe JJ (1995) Neurology of the newborn. (3rd edn), WB Saunders, Philadelphia, USA.

17. Campistol J, De Jaro P, Póo P, Kraurel J, Fernández ÁE (1994) Neonatal seizures. Forms of presentation and evolution. Rev Neurol 22: 171-175.

18. Caro Castellar I (2008) Neonatal seizures. Acta Neurol Colomb 24: 25-33.

19. Lombroso CT (1996) Neonatal seizures: A clinician's overview. Brain Dev 18(1): 1-28.

20. Stafstrom CE (1195) Neonatal seizures. Pediatr Rev 16: 248-56.

21. Bergman I, Painter MJ, Hirsch RP, Crumrine PK, David R (1983) Outcome in neonates treated in an intensive care unit. Ann Neurol 14(6): 642-648.

22. Holmes G (1995) Neonatal seizures. Acta Neuro Pediatr 1: 240-251.

23. Levene MI, Traura JQ (1986) Causes of neonatal convulsions. Arch Dis Child 61: 78-79.

24. Mizrahi EM, Kellaway P (1987) Characterization and classification of neonatal seizures. Neurology 37(12): 1837-1844.

25. Andre M, Matisse N, Vert P, Debrouille CH (1988) Neonatal seizures-recent aspects. Neuro pediatrics 19(4): 201-207.

26. Rodriguez BAC, Bauzano PE, Tosina GE (1997) Practical guide to neonatal neurology. Clinical-EEG correlation. Barcelona.

27. Aguilar F (2017) Neonatal seizures: Etiology, electroencephalographic patterns and evolution. Rev Mex of Neurosciences 18: 41-47.

28. (2008) AEP Diagnostic Therapeutic Protocols: Neonatology. Spanish Association of Pediatrics.

29. Jiménez R, Figueras J, Cañadell D, Botet F, Cruz M (1989) Prognostic factors in hypoxic-ischemic encephalopathy in term newborns. An Esp Pediatr 31(3): 189-195.

30. Campistol J (2008) Neonatal Seizures. Spanish Association of Pediatrics.

31. Andre M, Debruille C, Vert P, Grunenwald O (1981) Acute fetal distress and mental deficiencies. Prospective study. Arch Fr Pediatr 38: 323-329.

32. Mizrahi EM (2004) Atlas of neonatal electroencephalography. In: Mizrahi EM, Hrachovy RA, Kellaway P (Eds.), Philadelphia, Lippincott Williams & Wilkins.

33. Rakshasbhuvankar A, Paul S, Nagarajan L, Ghosh S, Rao S (2015) Amplitude-integrated EEG for detection of neonatal seizures: A systematic review. Seizure 33: 90-98.