Epileptic Spasms at Muhimbili National Hospital Tanzania, A Retrospective Study.

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Research article

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Abstract

Background; Epileptic spasms (ES) is an epileptic encephalopathy occurring during infancy and early childhood. Early recognition and management is important to prevent severe neurological impairment. This study aimed at describing the clinical presentation, management and outcome of patients with Epileptic Spasms attending Muhimbili National Hospital (MNH), in Dar Es Salaam, Tanzania.

Methods; A retrospective cross sectional study of all patients diagnosed with epileptic spasms was conducted at MNH from July 2016 to October 2018.

Results; A total of 40 patients diagnosed with epileptic spasms were retrieved with male to female ratio of 3:2. In this study, 17 (42.5%) patients had a documented history of perinatal insult. The median age of onset of spasms was 5 months (IQR 1–12 months). In 14 (80%) out of 17 patients whom electroencephalography (EEG) findings were retrieved had abnormal EEG findings showing either generalized epileptiform discharges 7(41%), generalized slowing 5(30%), hypsarrthymia 1(6%), or abnormal focal epileptiform discharges 2 (12%). Twenty-nine (73%) received prednisolone, with a median time of spasms subsiding of 1 month with a range of 10 days to 3 months in about 90% of them.

Conclusion; The median age of onset of epileptic spasms at MNH is 5 months with the most common cause being perinatal insult in more than 40% of the patients. High dose prednisolone showed a good response in patients with epileptic spasms at MNH.

Background

Epileptic spasms (ES), formerly called infantile spasms are epileptic seizures characterized with sudden flexion or extension, of the proximal and truncal muscles. Epileptic spasms usually last longer than myoclonic seizures but shorter than tonic seizures, and last for about 2–3 seconds (1). It is one of the epileptic encephalopathies causing developmental regression and intellectual disability. The incidence of epileptic spasms has been estimated to range from 2–5/10,000 newborns. In most cases, ES is due to perinatal hypoxia, with some cases being as a result of different post-natal factors. The most common aetiologies of ES reported were hypoxic-ischemic encephalopathy, chromosomal abnormalities, malformations, stroke, tuberous sclerosis complex, and periventricular leukomalacia or haemorrhage(2–4). Poor antenatal, natal and post natal care stands out as the main underlying problem in developing countries.

This study was conducted to describe the clinical presentation, aetiology, management and outcome of patients with Epileptic Spasms attending Muhimbili National Hospital in Dar Es Salaam, Tanzania.

Methods

A retrospective cross-sectional study of all patients diagnosed with epileptic spasms was conducted at MNH. Case notes from all patients diagnosed with Epileptic Spasms from July 2016 to October 2018 were reviewed, including both inpatients and outpatients.

The data were collected using a structured questionnaire specifically designed for this study. The data were collected from patient files and electronic database of the hospital.

Results

Forty case notes of patients with Epileptic spasms were retrieved. Most of the patients were males, constituting about 60% of the studied patients. More than 80% (33 patients) included in the study were aged below 3 years.

In this study, most patients with epileptic spasms had a history of a neurological insult that may have occurred during prenatal, natal or post-natal period. Seventeen (42.5%) of patients had cerebral palsy that resulted from birth asphyxia, intrauterine infections, neonatal hyperbilirubinemia, preeclampsia and prolonged labour.

The median age of onset of patients with Epileptic spasms was 5 months (IQR 1–12 months). The nature of spasms demonstrated in large number of epileptic spasms patients in this study was flexor type 38 (97%) with the remainder having both flexor and extensor type (mixed type). Most patients in this study had variable documented number of frequencies of spasms per day with most of them about 15 (40%) having 2 to 5 clusters per day and 3 (10%) were having more than 10 clusters per day.

In 14 (80%) out of 17 patients whom EEG reports were retrieved had abnormal EEG findings showing either generalized epileptiform discharges 7(41%), generalized slowing 5(30%), hypsarrhythmia 1(6%), or abnormal focal epileptiform discharges 2 (12%). Five (71%) out of 7 patients whom magnetic resonance imaging (MRI) results were found had documented abnormal MRI findings with most of them showing features suggestive of hypoxic ischemic encephalopathy (HIE), old subdural haematoma, Megalencephally and bilateral hippocampal atrophy, severe brain atrophy and multicystic encephalomacia.

Majority of the patients 35 (85%) in this study went through a course of different medications before the symptoms subsided. Before the diagnosis of epileptic spasms was made, many patients were receiving the following medications; sodium valproate 31(78%), and phenobarbitone 9 (23%). Other medications given were carbamazepine, clonazepam and baclofen. After the diagnosis of epileptic spasms was made 29 (73%) patients were documented receiving prednisolone.

Twelve (69%) out of19 documented patients treated for epileptic spasms with prednisolone symptoms subsided within the first month of treatment with interquartile range from 10 days to 3 months.

Discussion

The study aimed at describing the aetiology, clinical presentation, management and outcome of patients with Epileptic Spasms seen at Muhimbili National Hospital. The median age at onset was 5 months and most patients have shown to respond better on high dose prednisolone.

The study showed most of the affected children had their onset of epileptic spasms before the first year of life with peak incidence of onset of almost 70% being between two and six months with males being slightly more affected thanfemales although the gender difference is non-significant. The study done by Pellock JM et al showed 50 to 77% of patients had their peak age of onset at 3 to 7 months. The results are almost similar to this study as the study population were the same as the immature central nervous system is more at risk of developing spasms.(5)

Patients in this study demonstrated two clinical types of spasms with majority of patients 38 (97%) showing flexor type and 3% had mixed flexor-extensor type involving the muscles of the neck, trunk, and extremities. This

is different from the study done by M.Stand et al using time synchronized video and polygraphic recording in 24 infants which showed most infants tend to have more than one type of spasms as 42% had mixed type, 34% flexor and 23% extensor type. The differences in results is attributed to the variations in the study designs where the retrospective method carried out in this study could have missed some important parts of the spasms event which were easily picked in the prospective study (synchronized and polygraphic recordings) in their study. (6)

As far as etiology of epileptic spasms is concerned, the study pointed out that conditions associated with perinatal hypoxia remained to be the main culprit in 17 (42.5%) of the documented patients. The most common established for epileptic spams was cerebral palsy. The risk factors for cerebral palsy were birth asphyxia, intrauterine infections, neonatal hyperbilibinemia, preeclampsia and prolonged labour. Other established causes of epileptic spasms in this study were tuberous sclerosis complex, periventricular leukomalacia or haemorrhage and Arnold Chiari brain malformation. A multicentre study done on developed countries (Canada, France and United states) by S.Semiology on ES showed hypoxic ischaemic encephalopathy (10%), genetic (8%), Tuberous Sclerosis (7%), other cerebral malformation (8%), stroke, including porencephaly (8%), periventricular leukomalacia (5%).The difference observed is contributed by improved peri natal care in developed countries compared to developing countries. (2)

In this study, 14 (80%) out of 17 patients whom EEG findings were retrieved had abnormal EEG findings, showing either generalized epileptiform discharges 7(41%), generalized slowing 5(30%), hypsarrthymia 1(6%), or abnormal focal epileptiform discharges 2 (12%). A journal on Clinical Neurophysiology by Hrachovy et al reported the prevalence of hypsarrthymia can be up to 75% among the children with epileptic spams. Little findings obtained in this study is due to the fact that many children included in this study did their EEG after undergoing a long course anti epileptic medications before the diagnosis of epileptic spasm was made. (7)

In this study, 5 (71%) out of 7 patients whom MRI results were found had documented abnormal MRI findings with most of them showing features suggestive of HIE, old subdural haematoma, Megaloencephaly and bilateral hypocampal atrophy, severe brain atrophy and multicysticencephalomacia. A retrospective study done by Khatami et al in Canada on Brain MRI findings in patients with ES where a total of 26 patients were included, 19 (73%) had abnormal MRI findings which were features of HIE sequala, tuberous sclerosis (including one with megaloencephaly), Lisencephaly, meningitis infarcts and frontal heterotopia. The simirality in results depicts that the causes of epileptic spasms in the west are the same as those in Africa as they are all due to structural causes. (8)

Majority of the patients 35 (85%) in this study went through a course of different medications before the symptoms subsided. Before the diagnosis of epileptic spasms was made, many patients were receiving the following medications; sodium valproate 31(78%), and phenobarbitone 9 (23%). Other medications given were carbamazepine, clonazepam and baclofen. After the diagnosis of epileptic spasm was made 29 (73%) patients were documented to receiveprednisolone, with 12 (63%) out of 19 patients had documented spasms cessation after 10 days of high dose steroid treatment. A randomized clinical trial study done by Lux et showed 21 (70%) had spasms cessation after being given high dose prednisolone in 14 days. The results of the studies were almost similar due to almost the same duration of treatment as prednisolone works by suppressing the corticotropin releasing hormone which has been postulated to cause epileptic spasms. (9)

Conclusion

Epileptic spasms is one the pediatric neurological conditions with the median age of onset of epileptic spasms at MNH being 5 months. The most common cause of epileptic spasms in children at MNH is perinatal insults occurring in more than 40%. Most of patients showed a good response to a high dose Prednisolone.

List Of Abbreviation

EEG- Electroencephalogram

ES- Epileptic spasm

HIE- Hypoxic ischemic encephalopathy

IQR- Inter quartile range

MRI- Magnetic resonance imaging

MUHAS- Muhimbili university of health and allied sciences

MNH- Muhimbili national hospital

SPSS- Statistical package for the social sciences

Declarations

Ethical issues

Permission for data collection was obtained from administration of Muhimbili National Hospital (MNH) and the data were kept as confidential information.

Availability of data and materials

The data set is available

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Author contributions

JM and EK assisted with study design, identifying patients for study inclusion, data collection and results analysis. JM drafted the manuscript. EK contributed to manuscript content and revised the final manuscript.

There was no conflict of interest.

References

1. Scheffer IE, Berkovic S, Capovilla G et al ILAE classification of the epilepsies: Position paper of the ILAE Commission for Classification and Terminology. Epilepsia. 2017;58(4):512.
2. Semiology S. Epileptic Spasms. 2018;(April):2010–3.
3. Shields WD. Infantile spasms: little seizures, BIG consequences. Epilepsy Curr [Internet]. 2006;6(3):63–9. Available from: http://www.ncbi.nlm.nih.gov/pubmed/16761063%5Cnhttp://www.pubmedcentral.nih.gov/articlerender.fcgi?artid=PMC1464162
4. Asano E, Juhász C, Shah A, Muzik O, Chugani DC, Shah J, et al. Origin and propagation of epileptic spasms delineated on electrocorticography. Epilepsia. 2005;46(7):1086–97.
5. Pellock JM, Hrachovy R, Shinnar S, et al. Infantile spasms: a U.S. consensus report. Epilepsia 2010; 51:2175.
6. Kellaway P, Hrachovy RA, Frost JD Jr, Zion T Precise characterization and quantification of infantile spasms.Ann Neurol. 1979;6(3):214.
7. Hrachovy, RA; Frost JD (2003). "Infantile epileptic encephalopathy with hypsarrhythmia". Journal of Clinical Neurophysiology. **20** (6): 408–425.
8. Khatami A, Sell E, Aggag M, Miller E. Brain MRI Findings in Infantile Spasm: Outcome Correlations in a Patient Cohort. Open J Med Imaging. 2016;06(03):80–92.
9. Lux AL, Edwards SW, Hancock E, Johnson AL, Kennedy CR, Newton RW, et al. The United Kingdom Infantile Spasms Study comparing vigabatrin with prednisolone or tetracosactide in a randomised trial: developmental outcome at 14 months. Epilepsia [Internet]. 2004;45 Suppl 7:273-274. Available from: https://www.cochranelibrary.com/central/doi/10.1002/central/CN-00494700/full