International Journal of Innovative Research in Medical Science (IJIRMS)
Volume 02 Issue 06 June 2017, ISSN No. – 2455-8737
Available online at - www.ijirms.in

Open Access Journal | Research Article | DOI: 10.23958/ijirms/vol02-i06/08

Influence of Sleep Deprivation in Series of Seizures Pattern in JME Patients

[1]Maniyar Roshan Zameer, [2]Doshi M.A, [3]Parveen Jahan, [4]B.N.Umarji, [5]Shivannarayan.G, [6]Parthasaradhi G, [7]Syed Muneer

[1]Research Scholar, Dept. of Anatomy, Krishna Institute of Medical Sciences, Karad, Maharastra, India
[2]Professor and Head, Department of Anatomy, Krishna Institute of Medical Sciences, Karad, Maharashtra, India
[3]Associate Professor, Department of Zoology, Moulana Azad National Urdu University, Hyderabad, Telangana, India
[4]Professor & Head, Dept. of Anatomy, Dr. N. Y. Tasgaonkar Institute of Medical Sciences, Diksal, Koshane, Karjat, Raigad, (M.H) India
[6,] Girija Neuroclinic Centre, Vijayawada, Andhra Pradesh, India
[6]SandorProteiomics Pvt. Ltd. Banjara Hill, Road No-3, Hyderabad, Telangana, India
[7]Professor, Dept. of Pathology, Viswabharati Medical College, Kurnool, Andhra Pradesh, India

Abstract:

Background: *Juvenile myoclonic epilepsy disease (JME) will disrupt the sleep and sleep deprivation can be trigger for seizures. JME is estimated around 3 in 10,000 with peak age at 14.5 to 15.5 years that affects both genders. All the JME patients identified at least one precipitating factors (PFs). The seizures usually occur after awakening and sleep deprivation (SD) was the most common precipitating factors PF (82%), stress is 64% and other factors involved 42% in younger JME patients. Seizures are precipitated by sudden awakening, sleep deprivation, emotional stress, photic stimulation, motor activity, and alcohol consumption. Juvenile Myoclonic Epilepsy (JME) is dependent on the sleep-wake cycle and SD is one of the most important factors for precipitating seizures.*

Objectives: *The aim of this study was to investigate the effect of sleep deprivation in JME seizures related with psycho-sociological life.*

Methods: *The case-control association study design was utilized to test the potential involvement of of sleep deprivation among Juvenile myoclonic epilepsy patients. In this study we evaluated the role of sleep deprivation to obtaining seizures pattern by personal interview in a series of 75 JME patients. The study has been followed up for four years in different Epilepsy centers, neuron centers and educational institutions in Andhra Pradesh and Telangana and Hyderabad-Karnatak region of Karnataka states.*

Result: *The mean age of onset was 15.20 ± 7 years and at the time of presentation was 18 ± 7 years. Out of 75 JME patients 44 (58%) were males and 31 (41%) females. Sleep deprivation found in 26 (34.6%) JME patients, awakening presents 18 (24%), psychological stress had 12 (16%), menstruation 6 (8%), TV watching 4 (5.3%) and other factors triggering 9 (12%) in all JME cases as PFs.*

Interpretation & Conclusion: *Indian patients with JME cases have difference in their clinic-electrical profile. Larger prospective studies are required to confirm these observations between them. Greater delay and avoidance of precipitation factors for treatment to the JME patients would greater the consequences of uncontrolled epilepsy.*

Keywords: ***JME, Myoclonic jerk, PFs, Sleep deprivation***

Introduction

The clinical spectrum of JME is characterized by sudden short term jerks occurring mainly on awakening and often precipitated by sleep deprivation, stress, alcohol and photic stimulation. The PF always associated with myoclonic seizures, generalized tonic-clonic seizures, and absence seizures in a variable number of cases.[1-7].

JME seizures are in the form of myoclonic jerks in 100% of the patients, generalised tonic-clonic seizures in 80%, and absence seizures in 25%. Photoparoxysmal response is seen in 40% of the patients.[8] The seizures usually occur after awakening and are precipitated by sleep deprivation. The incidence rate among JME is 0.5–6.3/100 000 per year and the vast majority of cases present between the ages of 12 and 18 years (Dhanuka et al., 2001). Precipitating factors are important in initiating and severity of epileptic attacks in the

DOI: 10.23958/ijirms/vol02-i06/08

most types of epilepsy. Sleep deprivation is the most important precipitating factors in all the JME cases,[9] Myoclonic and GTCS seizures of JME often are precipitated in adolescence stage by two main factors sleep deprivation, and psychological stress.

In experimental animal models that showed that the thalamus is essential for maintenance of rhythmic GSW discharges.[10]

Precipitation exposed by alcohol and sleep deprivation may not be recognized by the physician as part of the syndrome of JME.

Seizures are known to be triggered by conditions such as sleep deprivation, sudden awakening, fatigue, alcohol intake, flashing lights, menstruation and stres.[11,12]

Materials and Methods

We conducted personal interview and EEG reports are evaluated for 75 JME patients.

The documented diagnosis of JME followed according to criteria of the Commission on Classification and Terminology of the International League against Epilepsy (ILAE).

Written consent was obtained from all participants prior to the interview. Case notes were independently reviewed at Epilepsy society conducted by the Girija neuron centre, Viyayavada, (A.P), Neurology clinics, and Educational institutions of Telangana and Andhrapradesh States and Hyderabad-Karnataka region of Karnataka state in the following: 1) Age at onset of pattern of seizures; 2) Age at diagnosis, delay in diagnosis; 3) Factors contributing to delay in diagnosis; 4) Adverse effects of delayed diagnosis. All participants signed an informed consent form, and the investigation was approved by the Krishna Institute of Medical Sciences, Karad, (M.H), India.

We excluded those patients with psychiatric, psycho-socially deprived, intellectual, or emotional disturbances that could affect the reliability of their responses.

Result

Seizure-precipitating factors, in order of importance, were sleep deprivation, stress, alcohol, flickering lights and fatigue.

1.1. Seizure precipitation

Table1 Seizure precipitating factors (N = 75)

Precipitation	N= 75
Sleep deprivation	26 (34.6%)
Awakening	18 (24 %)
Psychological stress	12 (16%)
Menstruation	6 (8%)
TV watching	4 (5.3%)
Other factors	9 (12%)

Sleep deprivation dramatically reduces human growth hormone secretion and testosterone production. Both of these hormones are responsible for tissue healing and to burn fat and build muscle. Myoclonic seizures are sudden, quick, small jerks of the arms, shoulder, or the legs. The myoclonic seizures occur most often in the early morning just after waking up or after a nap. There is no loss of consciousness during myoclonic seizures. Awakening and sleep deprivation appeared to be the commonest precipitating factors for either MJ or GTCS (70%) in JME cases.

1.2. Electroencephalography

1. **Myoclonic EEG** - 4-6 Hz polyspikes and slow-wave generalized discharges are provoked by hyperventilation (High voltage 12 to 30 polyspikes) and photocunvulsive response.
2. **Tonic-Clonic Seizures** - EEG was obtained within five days after seizures. The epileptic form activity usually takes one or more of four main patterns.
    1. Typical 3-Hz spike and wave complexes
    2. Irregular spike and wave complexes
    3. 4- 5 Hz frequency spike and wave complexes
    4. Multifocal spike complexes.
3. **Absence EEG** - 3 Hz spike and wave pattern less than 6-10 seconds. Bilateral synchronous and symmetrical rhythmic Polyspike and waves complexes with frequency 3 Hz (2.5 to 2Hz) with focal or multifocal discharges.

Table: 2. Interictal EEG in JME cases at presentation (N = 75)

Type of Seizures	Ictal EEG	Male (N=44)	Female (N=31)
Normal	Normal EEG pattern	05 (11%)	03 (7%)
Myoclonic	4-6 Hz polyspikes and slow-wave generalized discharges	18 (41%)	16 (36%)
Generalized tonic clonic	Generalised spike-wave paroxysma (4 to 6 Hz frequency), seizures with febrile convulsions	13 (29%)	07 (16%)
Absence seizures	Bilateral synchronous and symmetrical rhythmic Polyspike and waves complexes with frequency 3 Hz with focal or multifocal discharges	05 (11%)	04 (9%)
Photo-convulsive response	Polyspike or normal	03 (7%)	01 (2%)

DOI: 10.23958/ijirms/vol02-i06/08

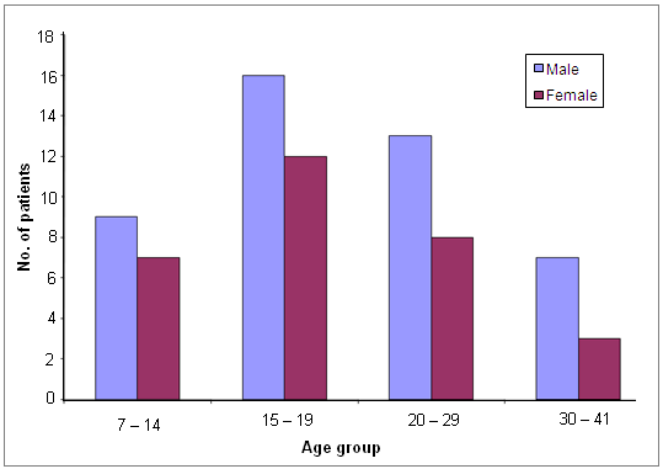
Precipitation factors such as sleep deprivation and emotional stress appears in myoclonic seizures (MS) in 60% of JME cases in both genders of adolescence stage. GTCS second most common disorder in PF by the combination of the other precipitation factors.

1.3. Demographics data

A total of 75 patients (41men, 34 women) were interviewed. The mean age at the time of interview was 25.5 + _ 8.5 (7—41 years).

The demographic data reveal that male JME patients are often visited to the epilepsy centres compare to the female JME patients due to social stigma and traditional restrictions. In our study in every age group of male JME patients are dominated M.S phenomena, while untreated JME patients between the age of 15 to 19 years has develop the generalized tonic-clonic seizures with unconsciousness.

Figure 1: Demographic data of JME (75) patients are summarized



Family history of epilepsy was present in few JME patients.

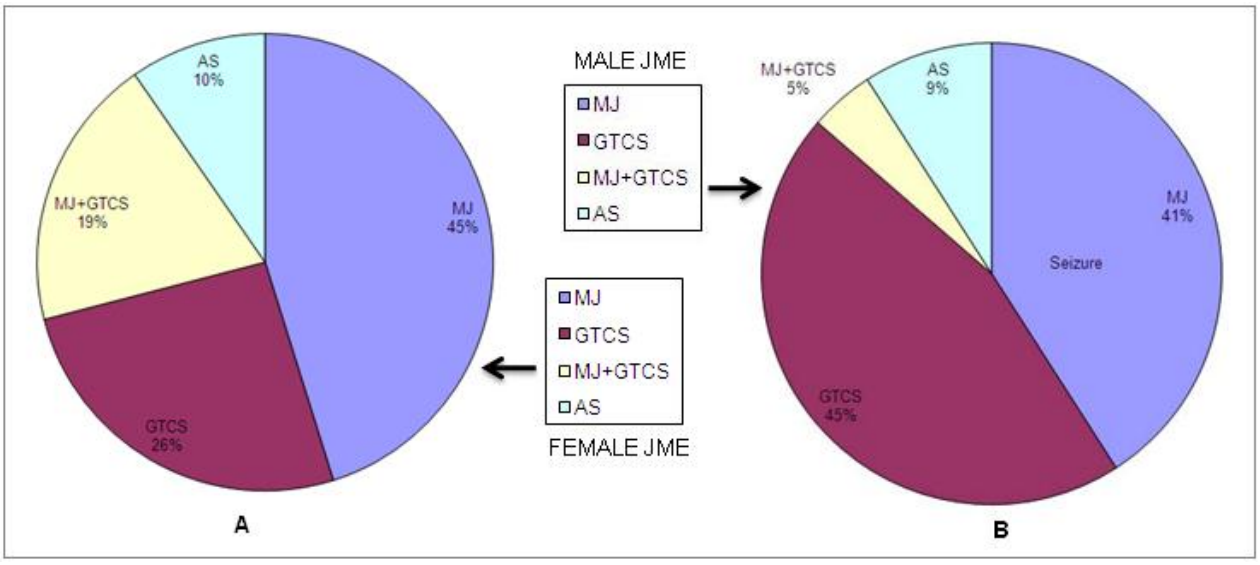
Absences began at a mean age of 8.5 years (range 7-112 years), myoclonic jerks at 14.5 years (range 11-18 years) and generalized tonic-clonic seizures at 16.5 years (range 13-22 years). Age at diagnosis of JME was 26-5 years (range 7-41 years), representing a delay in diagnosis of 14.5 years (range 5-32 years).

1.4. Seizures data

We observed in our study that myoclonic jerks (MJ) was common in both genders, however generalized tony clonic seizure (GTCS) is less prominent in female. The MS + GTCS combination phenomena of seizures often observed in both genders. Absence seizures (AS) less common in both genders and seizures more precipitate in late childhood and adolescence stage. The stop codon observed in JME patients who are precipitate by the myoclonic seizures and generalized tonic-clonic seizures.

There were 75 JME patients (44 males and 31 females) ranging in age from 7 to 41 years (mean 20 years). The age of onset of myoclonic seizures 10 to 22 (mean 15.6) years, absences seizures ranged from 5 to 11 (mean 8.0), and GTCS 12 to 24 (mean 16.4). A Myoclonic jerk on awakening by the sleep deprivation was found in 15 males of JME patients (41%), 17 females (45%). GTCS occurring predominantly in17 males (45%), and 10 females (26%) respectively, while 3 males (9%) and 4 females (10%) had absence seizures (AS), MJ+GTCS presents 2 males (5%) and 7 females (19%) in total number of 75 JME cases. JME appears around puberty and is characterized by seizures with bilateral, single or repetitive, arrhythmic, irregular myoclonic jerks, predominantly in the arms. Jerks may cause some patients to fall suddenly. No disturbance of consciousness is noticeable. The disorder may be inherited, and sex distribution is equal. The seizures usually occur shortly after awakening and precipitated by sleep deprivation.

Figure 2: Distribution of female (A) and male (B) of JME patients of seizure type and their combination



DOI: 10.23958/ijirms/vol02-i06/08

Duration JME seizures

Myoclonic seizures – 20 seconds to 2 mints or 3 mints

Tonic-Clonic seizures – 3 mints to 6 mints

Absence seizures – 5 seconds to 10 seconds / 20 seconds – 40 seconds

Discussion

Precipitation of seizures involved in several factors includes sleep deprivation (54.2%), menstruation (20%), fatigue (9.2%), stress (7.6%), concentration (6.9%), photic stimulation (1.5%), TV/video (1.5%) (murthy et. al. 1998).

In another study, fever and emotional disturbances were perceived as seizure precipitants in 29% and 16% of patients respectively.[13]

Photosensitivity on intermittent photic stimulation has long been thought to be a genetically determined EEG phenotype.[14] The role of television was established in 1997, when about 700 Japanese children developed seizures after watching a popular program called "Pocket Monster".[15] Sleep deprivation was found to be the most important precipitating factor in juvenile myoclonic epilepsy (in 54% of patients).[16]

Sleep deprivation was more frequently reported by patients who had also reported emotional stress. There was a strong correlation between sleep deprivation and stress. There has been some speculation that stress might lead to physiological changes in the corticosteroid levels as well as in the cerebral blood flow facilitating seizure occurrence.[17] In a recent study, 62% of patients cited at least one precipitant that included stress (30%), sleep deprivation (18%), fever or illness (14%), and fatigue (13%). In another study fever and emotional disturbances were perceived as seizure precipitants in 29%.[18] The (EEG) reveals a 4 to 6-Hertz polyspike and wave discharge, which in the younger child with absence seizures may be indistinguishable from that of typical absence epilepsy. In 10 to 15 percent of patients with JME, the initial EEG is normal.[19, 20] About 15% of patients with JME report that flickering lights (disco, sunlight, and videogames) are provocative.[21] Interictal activity is short bursts of polyspikes and polyspike-wave complexes after spontaneous or induced awakenings.[22,23] In general, sleep architecture is affected, with decreased quality and sleep fragmentation. The mean age of onset of the myoclonic jerks at 15 years (range, 8 to 26 years), and the generalized convulsive seizures 16 years (range, 9 to 28 years).[24]

Conclusion

A careful history could help us in identifying type of seizures among JME patients. Adequate avoidance or treatment of these factors could help in better seizure control and possibly in reduction of dosage of AEDs. Sleep-deprived EEG significantly contributed to the diagnosis in 75 JME patients. Larger prospective studies are required to confirm these observations between them. Greater delay and avoidance of precipitation factors for treatment to the JME patients would greater the consequences of uncontrolled epilepsy.

Conflict of interest statement: No conflict of interest to disclose.

Acknowledgements

We thank all of the JME patients and case subjects for participating in this study. We sincere thanks to **Dr. (Mrs.) Rajani Gaonkar Madam**, **Dr. (Mrs.) Dosi M.A**; Professor and Head, KIMS, Karad (M.H); we thankful to **Dr. (Mrs.) Parveen Jahan**, Associate Professor, Dept. of Zoology, School of sciences, Maulana Azad National Urdu University, Hyderabad (T.S) for the valuable discussion and guidelines. We thank to **Dr. Shivannarayan M.D**, D.M (Neurology), **Dr. B.N. Umarji** and **Dr. Chenana. C** Director, BRIMS, Bidar (K.A).

Reference

[1] Asconape J, Penry K. Some clinical and EEG aspects of benign juvenile myoclonic epilepsy. Epilepsia. 1984 Feb; 25(1):108-14.
[2] Delgado-Escueta AV, Enrile-Bascal F. Juvenile myoclonic epilepsy of Janz. Neurology. 1984 Mar; 34(3):285-94.
[3] Grunewald R, Panayiotopoulos CP. Juvenile myoclonic epilepsy: A review. Arch Neurol. 1993 Jun; 50(6):594-8.
[4] Janz D. Epilepsy with impulsive petit mal (juvenile myoclonic epilepsy). Acta Neurol Scand. 1985 Nov; 72(5):449-59.
[5] Panayiotpoulos CP, Obeid T, Tahan AR. Juvenile myoclonic epilepsy: a 5 year prospective study. Epilepsia March 1994; 35:285-96.
[6] Jain S, Dixit SN, Andrews PI, Radtke R, Maheshwari MC, McNamara JO. Disease expression among probands with juvenile myoclonic epilepsy and their family members in two population groups. J Epilepsy January 1996; 9:259-67.
[7] Jain S, Padma MV, Maheshwari MC. Occurrence of only myoclonic jerks in juvenile myoclonic epilepsy. Acta Neurol Scand. 1997 May; 95(5):263-7.
[8] Janz D and Durner M. Juvenile myoclonic epilepsy. In: Epilepsy, A Comprehensive Textbook Volume three (Eds J. Engel and T. A. Pedley). Philadelphia, Lippincott-Raven, Journal of Clinical

DOI: 10.23958/ijirms/vol02-i06/08

Neurophysiology: May 1998 - Volume 15 - Issue 3 - pp 279-280.

[9] Genton P, Gélisse P, Thomas P. Juvenile myoclonic epilepsy today. In: Schmitz B, Sander T, eds. Juvenile myoclonic epilepsy: the Janz syndrome. Petersfield, UK: Wrightson Biomedical, Journal of neurology, Neurosurgery and psychiatry; February 2001 - Volume 70 – 2.

[10] H.K. Meeren, J.P. Pijn, E.L. Van Luijtelaar, A.M.L. Coenen, F.H. Lopes da Silva Cortical focus drives widespread corticothalamic networks during spontaneous absence seizures in rats J Neurosci 22 (4),. 2002 Feb 15 pp. 1480–1495.

[11] Arzimanoglou A, Guerrini R, Aicardi J. Epilepsies with predominantly myoclonic seizures. In: Arzimanoglou A, Guerrini R, Aicardi J, editors. Aicardi's epilepsy in children. Philadelphia: Lippincott Williams & Wilkins; GeneReviews 2004. p. 58-80.

[12] Alfradique I, Vasconcelos MM. Juvenile myoclonic epilepsy. Arq Neuropsiquiatr. 2007 Dec; 65(4B):1266-71.

[13] Aziz H, Ali SM, Frances P, Khan MI, Hasan KZ. Epilepsy in Pakistan: a population-based epidemiologic study. Epilepsia. 1994 Sep-Oct; 35(5):950-8.

[14] Binnie CD. Simple Reflex Epilepsies. In: Engle J.Jr and Pedley TA eds: Epilepsy: A Comprehensive Textbook. Philadelphia, Lippincott-Raven Publishers, Book 1997: 2489-505.

[15] Enoki H, Akiyama T, Hattori J, Oka E. Photosensitive fits elicited by TV animation: an electroencephalographic study. Acta Paediatrica Japonica, December 1998; 40:626-630.

[16] Murthy JM, Rao CM, Meena AK. Clinical observations of juvenile myoclonic epilepsy in 131 patients: a study in South India. British epilepsy seizures, 1998 Feb; 7(1):43-7.

[17] Spector S, Cull C, Goldstein L. Seizure precipitants and perceived self-control of seizures in adults with poorly-controlled epilepsy. Epilepsy Res. 2000 Feb; 38(2-3):207-16.

[18] Frucht MM, Quigg M, Schwaner C, Fountain NB. Distribution of seizure precipitants among epilepsy syndromes. Epilepsia. 2000 Dec; 41(12):1534-9.

[19] Aliberti V, Grunewald RA, Panayiotopoulos CP, Chroni E. Focal electroencephalographic abnormalities in juvenile myoclonic epilepsy. Epilepsia. 1994 Mar-Apr; 35(2):297-301.

[20] Vijai J, Cherian PJ, Sylaja PN et al. Clinical characteristics of South Indian cohort of juvenile myoclonic epilepsy probands. Seizure. 2003 Oct; 12(7):490-6.

[21] da Silva Sousa P, Lin K, Garzon E, Sakamoto AC, Yacubian EM. Self-perception of factors that precipitate or inhibit seizures in juvenile myoclonic epilepsy. Seizure. 2005 Jul; 14(5):340-6.

[22] J. Asconape and J. K. Penry, "Some clinical and EEg aspects of benign juvenile myoclonic epilepsy," Epilepsia. 1984 Feb; 25(1), vol. 25, no. 1, pp: 108-14.

[23] Benetó, A. Santa, S. Soler et al., "La relación sueño-epilepsia," Aceptado tras revisión externa: 23-03-2007, Vigilia-Sueño, vol. 19, pp. 15–24, 2007.

[24] Martinez-Juarez IE, Alonso ME, Medina MT et al. Juvenile myoclonic epilepsy subsyndromes: family studies and long-term follow-up. Brain. 2006 May; 129(Pt 5):1269-80. Epub 2006 Mar 6.

Corresponding Author

Maniyar Roshan Zameer

Research Scholar, Dept. of Anatomy, Krishna Institute of Medical Sciences, Karad, Maharastra, India

DOI: 10.23958/ijirms/vol02-i06/08