Catamenial epilepsy: a bizarre phenomenon!

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Catamenial Epilepsy: A Bizarre phenomenon!

Catamenial Epilepsy is an interesting phenomenon in Epileptology. Despite the variability in defining it, the general consensus is that the doubling of the baseline frequency of seizures, irrespective of type of seizure involved, its localization or the epilepsy syndrome with a pattern of clustering, related to patient’s menstrual cycle, constitutes the diagnosis of Catamenial epilepsy[1-4].

The prevalence of Catamenial epilepsy ranges anywhere between 10-78% based on the available literature. However, this may be spurious in view of variability in the criteria of diagnosis applied, population studied, and inconsistent mechanisms of recording in linking seizures with the phase of menstrual cycle phase. Having said that, in other elaborate studies it is evident that at least 33% of women with medically refractory epilepsy had Catamenial clustering of seizures. The population affected by this form of epilepsy had a median age of around 40 years and as is evident, these patients are, most vulnerable around the child bearing period of their lives[5-8].

So, what actually goes wrong during the menstrual cycle in these patients that increases the seizure frequency? Researchers feel that the hormones are the culprits! The influence the frequency of seizures through a variety of mechanisms. Some of the complicated mechanisms at the molecular level are the receptor mediated latency genomic effect, receptor mediated post-transcriptional intermediate effect and more importantly, the direct membrane-mediated short latency effect. Further details on these mechanisms are beyond the scope of this article.

Besides the general observation that an Inadequate Luteal Phase has been a common observation in epileptic females in their reproductive age, hormonal imbalances such as progesterone-estrogen ratio skewed inversely during anovulatory, premenstrual or at/during Inadequate luteal phase when the production of progesterone is low, are striking findings in many studies[9-12]. Further basis to this hypothesis is the enhancing effect of glutamate-mediated excitatory transmission and decrease gamma-amino-butyric acid (GABA) mediated inhibition by the oestrogen, which adversely affects the female with low threshold for seizures, hence predisposing for Catamenial epilepsy. On the other hand, progesterone raises the threshold for seizure by suppressing kindling and hence has a protective effect under these circumstances. Corroborating to this hypothesis, clinically, the frequency of seizures increase during the phase of transition from premenopausal to menopausal phase (progesterone levels wean with relatively stable
eizure expression in women

orenman SG. Measurement of serum LH, FSH, estradiol and progesterone levels respectively in the descending order of the menstrual cycle. The period most frequently associated with Catamenial epilepsy, followed by periovulatory phase (days 10-13) and luteal phase respectively in the descending order of frequency [20-23].

Scientific way to diagnose Catamenial epilepsy as of today has to be through meticulously recording the menstrual cycles of the patient. This helps the exact characterisation of the menstrual cycle. Maintaining seizure diaries greatly enhances the probability of diagnosing this condition. If there is a doubling of the frequency of seizures especially in the perimenstrual, periovulatory or luteal phase as against the other phases of menstrual cycle, the possibility of catamenial epilepsy is strongly considered [23-26].

Unfortunately, there is no treatment specific for catamenial epilepsy. Hence, anti-epileptic medication has to be prescribed based on the seizure type.

Amongst the agents used, Acetazolamide at a dose of 250-500mg daily for 3-7 days before the menses has been found useful. The recommended dose of this agent is around 4mg/kg in one to four divided doses, not to exceed 1 gram per day.

Clobazam is probably the only benzodiazepine to be tried successfully in catamenial epilepsy. The prescribed dose is 15-30mg/day and must be administered at least for 7-10 days in phases of menstrual cycle, where there is a high risk for seizure.

Since it is well known that physiologically, the levels of the anti-epileptic medications may decrease premenstrually, it may be wise to attempt intermittent increase in doses at times of the month, when the patient is most susceptible for having a seizure.

Lamotrigine, Hormonal therapy in the form of natural and/or medroxy progesterone, clomiphene citrate, oral contraceptive pills, Gonadotropin releasing hormone (GnRH) analogues Triptorelin have also been tried with limited success in Catamenial epilepsy [26-30].

In summary

Although a specific definition is still lacking, the consensus seems to be that the increase in seizure frequency (at least twice the baseline) during specific phase of menstrual cycle of the female, is highly suspicious of a diagnosis of Catamenial epilepsy. At least 33% of females with medically refractory seizures in their reproductive age group have this underlying condition. Women with partial epilepsy are more prone to have catamenial epilepsy, however other epilepsy syndromes associated with catamenial epilepsy is also well known. The perimenstrual followed by periovulatory phases are the phases where the patient is susceptible to have clustering of seizures as a part of catamenial epilepsy. The low progesterone to estrogen ratio is a commonly observed biochemical finding during these times. Menopause is associated with drastic decrease in the frequency of catamenial epilepsy frequency. However, Acetazolamide, clobazam, hormonal therapy, GnRH analogues along with intermittent modification in the schedule of anti-epileptic medication (based on times of month when patient is most prone to have seizures) are some of the treatment options.

Conflict of Interest

The authors have no conflict of interest.

References
