ASSESSMENT OF CHRONOBIOLOGICAL AND NUTRITIVE TRIGGERS IN PATIENTS WITH EPILEPSY AT THE PRIMARY HEALTHCARE LEVEL (CLINICAL CASE ANALYSIS)

Qurbanov Feruz Yusupovich
Student of General Medicine, Denov Abu Ali ibn Sino Public Health Technical School
feruzqurbonqulov7@gmail.com
Murtozaqulova Farogat Ergashevna
Teacher, Denov Abu Ali ibn Sino Public Health Technical School
murtazaqulovafarogat@gmail.com

ABSTRACT: This scientific article presents a retrospective analysis of the 11-year clinical evolution of epilepsy from childhood to adolescence. During clinical observation, the age-related dynamics of paroxysmal seizures, the subclinical prodromal period arising 1-3 weeks before a seizure, the visceral aura originating in the chest region, and the decrease in aura perception during serial seizures were studied. The pronounced chronobiological (seasonal) nature of the disease and latent nutritive triggers associated with sheep tail fat were also analyzed. For the first time, the day-by-day dynamics of ictal/postictal amnesia and of stepwise neuropsychological syndromes (resistant cephalgia, compulsive behavior and emotional lability) following a seizure are described. When the diet is violated, seizures occur during the sleep phase. Practical recommendations have been developed for primary healthcare workers.

Keywords: Epilepsy, prodromal phase, nocturnal paroxysms, visceral aura, postictal amnesia, affective lability, nutritive trigger, sheep tail fat, valproic acid, paramedic supervision.

1. INTRODUCTION AND RELEVANCE OF THE TOPIC

Epilepsy is a chronic neurological disease resulting from paroxysmal discharges of brain neurons, affecting approximately 1 percent of the world's population. The unexpected occurrence of seizures complicates patients' social adaptation. Anticonvulsant medications alone are not sufficient to control the disease successfully. To improve the patient's quality of life, it is important to identify individual provoking factors - exogenous

(temperature, change of seasons) and endogenous (sleep schedule, alimentary or nutritive influences) triggers - and to eliminate them. The organization of this process and continuous monitoring of patients are the direct responsibility of primary healthcare workers, in particular general practice paramedics.

2. AIM OF THE STUDY

To analyze, using the example of a patient with epilepsy, the age-related characteristics of changes in paroxysms; the prodromal and aura stages preceding a seizure; chronobiological patterns related to the seasons; the latent mechanisms of action of specific lipid (nutritive) triggers in the diet; and the mnestic and stepwise neuropsychological characteristics of the postictal period following a seizure; and to develop practical recommendations.

3. MATERIAL AND METHODS

The object of the clinical study was the 11-year disease history (the period from 8 to 19 years of age) and retrospective personal monitoring data of a 19-year-old male patient. The patient has been taking Valproic acid (Convulex) at a stable dose for a long time. The study analyzed subjective clinical signs, prodromal changes, seizure frequency, dietary factors, the postictal mnestic and neuropsychological state, and the effect of exogenous temperature.

4. RESULTS AND DISCUSSION

4.1. Age-related evolution of seizures, prodromal and aura phenomena:

In the first years of the disease (childhood), seizures were very short but occurred at a high frequency. As the patient grew older (adolescence and young adulthood), the differential interval between paroxysms became markedly longer (the number of seizures decreased), but the intensity and neurological depth of the seizures increased.

Clinical analyses showed that a long-term prodromal period (a background of low-intensity, subclinical warning signs) develops 1-3 weeks before the onset of paroxysmal seizures. At the end of this prodromal background, the patient was noted to clearly feel subjective changes - an Aura (paroxysmal neurological warning) state - shortly before the seizure begins. In this patient, the aura phenomenon has a specific character: it begins with an accumulation of somatosensory and vegetative sensations in the precardial (chest)

region (visceral aura), and in a short time reaches high intensity and passes into a paroxysm.

However, once the initial strong aura and a wave of consecutive serial seizures have begun, subjectively feeling and differentiating the auras preceding subsequent paroxysms becomes sharply more difficult (decreased aura perception). This condition is explained by the functional exhaustion of brain neurons and by inhibitory processes developing in the short interval between paroxysms.

4.2. Latent nutritive triggers and the phenomenon of nocturnal paroxysms:

The most important pattern was identified in clinical observation: animal fats, in particular sheep tail fat and yellow fat, are the strongest alimentary triggers activating the disease. Regardless of the time of day at which the patient consumes these products, a seizure does not occur immediately. Instead, the changes accumulate latently (in a hidden manner) and manifest on the night of the same day, during the sleep phase, as acute neurological malaise (nocturnal paroxysms). This condition is explained by the long digestion of long-chain lipids, the metabolic load on the liver, and the increase in nocturnal vagotonic (parasympathetic) tone, which leads to a disturbance of the balance between inhibition and excitation in the cerebral cortex. It was also found that the patient experiences vegetative crisis states even when merely smelling the pungent odor of these products.

4.3. Chronobiological (seasonal) modulation against the background of a strict diet:

The results obtained show that during periods when the patient strictly avoided the above-mentioned harmful lipid-containing products, the seizures were not eliminated completely but instead shifted to a fully chronobiological (seasonal) pattern. In this case, seizures flare up only in certain seasons: in winter hypothermia (getting chilled), during the periods of changing spring and autumn biological rhythms, and in summer hyperthermia (head overheating in the sun). This pattern proves that, even when the dietary factor is eliminated, the endogenous sensitivity of brain neurons to external climatic changes is preserved.

4.4. Phase of postictal inhibition (compensation), cognitive amnesia and stepwise neuropsychological syndromes:

After a strong wave of seizures (following up to 7-10 repetitions), the patient develops a period of prolonged deep sleep and stupor. Neurophysiologically, this state represents anticonvulsant compensation (protective inhibition) of the cerebral cortex. During this

period, cognitive-mnestic dysfunction (memory decline) and ictal/postictal amnesia become evident: the patient cannot recall at all the initial strong aura or the consecutive paroxysmal seizures themselves. In the clinical dynamics, a stepwise (day-by-day) syndromic evolution of post-seizure changes was identified:

- **Day 1 after the seizure:** The patient experiences severe cephalgia (headache) in the parietal (crown) region of the head that is absolutely resistant to conventional analgesics and non-steroidal anti-inflammatory drugs (NSAIDs) (it cannot be stopped by any medication). This stage proceeds with deep stupor and retrograde/anterograde amnesia, and the patient is subsequently unable to recall the events of the first day of the postictal period.
- **Day 2 after the seizure:** Against the background of general stupor and decreased short-term memory, the ability to self-regulate and exercise self-control declines, and a distinctive postictal compulsive behavior appears (a tendency to automatically straighten and put in order objects and items that are out of place or incorrectly positioned along the way).
- **Day 3 after the seizure:** As a result of postictal dysfunction of the limbic system and the hypothalamus, pronounced affective lability (emotional instability) develops. In this state, the patient reacts with affective crises throughout the day, directly dependent on the first visual stimulus seen after waking up (a funny or sad event/image): tears at even a slightly sad thing, uncontrollable laughter at even a slightly funny situation.

These postictal neurological, mnestic and affective deficits undergo complete regression (disappear) in the following days when the strict nutritive diet is observed and anticonvulsant medications are taken steadily; the stupor ends and the body passes into the stage of metabolic rehabilitation.

System of Triggers and Clinical-Dynamic States

Trigger / Phase type	Time of clinical manifestation	Neurological, cognitive and affective consequences	Status during diet and treatment
Prodromal phase	Latent period beginning 1-3 weeks before the seizure	Background of subclinical, low-intensity warning signs; later progresses to an acute visceral aura.	With intensified preventive control, serial seizures can be prevented.

Trigger / Phase type	Time of clinical manifestation	Neurological, cognitive and affective consequences	Status during diet and treatment
Sheep tail fat / Yellow fat	At night during sleep after consumption (after a latent period)	Severe nocturnal paroxysms, consecutive serial seizures; activation of visceral aura and decreased aura perception.	These nocturnal seizures disappear completely with a strict diet.
Change of seasons (Seasonal)	Period of spring and autumn biological rhythm changes	Seasonal malaise, diffuse headache, general stupor.	Remains stable even on a diet (endogenous rhythm).
Extreme temperatures	Winter cold (hypothermia) / strong summer sun	Increased neuronal excitability; provocation of paroxysmal seizures.	Flares up seasonally and situationally under the influence of climatic factors.
Postictal phase, days 1–2	Within the first 48 hours after the seizure	Medication-resistant parietal cephalgia; complete amnesia for the seizure and the events of day 1; compulsive automatism of straightening objects on day 2.	With regular medication, cephalgia and compulsions disappear after 2 days and memory is restored.
Postictal phase, day 3	At the beginning of the third day after the seizure	Limbic affective lability; pathological crying or laughter depending on the external visual trigger.	Ends by the end of the day, together with the stupor, under medication and diet.

5. CONCLUSION AND PRACTICAL RECOMMENDATIONS

1. In epilepsy monitoring, the effect of nutritive triggers is latent in nature: when heavy lipids (sheep tail fat) are consumed, seizures manifest not immediately but as paroxysms during the nocturnal sleep phase, against the background of increased parasympathetic tone. Completely excluding heavy animal fats from the patient's diet makes it possible to break the chain of nocturnal pathological seizures.
2. When the strict nutritive diet is maintained, the course of the disease shifts to a fully chronobiological (seasonal) modulation, which allows the paramedic to plan in advance the patient's protective measures for each season (spring, autumn, winter, summer).
3. The period following epileptic paroxysms (the postictal phase) involves not only short-term protective inhibition but also ictal/postictal amnesia and stepwise neuropsychological syndromes lasting 3 days (resistant cephalgia, compulsive automatisms and limbic affective lability). In addition, early detection of the prodromal period that arises 1-3 weeks before seizures is important for protecting the patient from serial paroxysms.
4. Primary healthcare paramedics must study each epilepsy patient's individual triggers, prodromal signs and postictal changes in the form of a chronological map, and must give the patient's family members individual dietetic and neuropsychological recommendations on proper care during the 3-day psycho-emotional and mnestic crisis period following a seizure.

REFERENCES

1. Ministry of Health of the Republic of Uzbekistan, clinical protocols: "Standards for the Diagnosis and Treatment of Epilepsy."
2. Карлов В.А. Эпилепсия у детей и взрослых, женщин и мужчин: Руководство для врачей. - Москва: Медицина, 2010. - 720 с. [Karlov V.A. Epilepsy in Children and Adults, Women and Men: A Guide for Physicians. Moscow: Meditsina, 2010. 720 pp.]
3. Shorvon S.D. Handbook of Epilepsy Treatment. - Wiley-Blackwell, 2010.
4. Devinsky O., Vezzani A., O'Brien T. J. et al. Epilepsy // Nature Reviews Disease Primers. - 2018. - Vol. 4. - P. 1-24.
5. Fisher R. S., Cross J. H., French J. A. et al. Operational classification of seizure types by the International League Against Epilepsy // Epilepsia. - 2017. - Vol. 58. - No. 4. - P. 522-530.