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Cognitive Rehabilitation by Compensatory and Restorative Computing Training in Patients with Epilepsy and Mild Cognitive Impairment

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The study of the cognitive rehabilitation by compensatory and restorative computing training efficacy during 3 months revealed the effectiveness for epileptic patients with a significant improvement in cognitive functioning. More significant results were observed under the combined use of psychoeducation and CT, compared to the use of cognitive training only. The results of the study 12 months after CT showed that patients in study groups 1 and 2 had a level of cognitive functioning close to that at the beginning of the study, i.e. the improvement in cognitive functioning after three months of cognitive training is not sustainable without further training. There were significant differences from the indicators shown by the control group patients who did not undergo a rehabilitation program and had a more pronounced further decline in cognitive functioning, higher anxiety, and lower quality of life. Non-pharmacological correction with the use of the CT in patients with non-dementia level cognitive decline allows to slow down the progression of the latter and improve the overall mental state. The combined use of CT and psychoeducation facilitates social functioning and improves patients` quality of life, but the mechanism of psychoeducation`s impact on cognitive functioning is likely to be indirect, through the reduction of anxiety and depression symptoms. Potential areas of further research include a longer follow-up period, which may provide insight into the feasibility of using such CT regularly, and/or the search for forms of daily activity that would serve as a cognitive function training.

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Movement disorders and seizures as a manifestation of systemic lupus erythematosus

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Objective: To describe a case report of a patient with systemic lupus erythematosus (SLE) presenting with congenital hearing loss, seizures, myoclonus, and dystonia.

Background:Movement disorders and epileptic seizures are possible neurological manifestations of SLE.

Method: We report the case of a 16-year-old female with congenital hearing loss diagnosed with SLE.

Results: About two years prior to presentation, the disorder started with unexplained rises in temperature, weakness, headaches, pain in small joints and muscles, and generalized tonic-clonic seizures. The patient experienced episodes of myoclonus and abnormal dystonic posturing of the left upper arm, forearm, hand, and left toe extension three months after the onset. Brain magnetic resonance imaging revealed diffuse cortical atrophy and calcification in the globus pallidus, the right thalamus, and the temporal cortex. Electroencephalography showed temporal intermittent rhythmic delta activity; antiphospholipid antibodies (aPLs) were elevated in the serum. Steroids were prescribed for SLE; valproic acid was administered for seizures and movement disorders. During a two-year follow-up period, the patient experienced only two episodes of myoclonus and dystonia, and remained seizure-free.

Conclusion: The seizures in this patient were probably related to an autoimmune-mediated pathogenesis via aPLs, while myoclonus and dystonia might also be associated with thalamic lesions. Neurological complications of SLE are common, but the coexistence of myoclonus, dystonia, and epileptic seizures in patients with congenital hearing loss and multiple morphological changes in the brain has not been reported. Awareness of this condition can improve treatment strategies and outcomes for patients, as well as optimize resources in the healthcare setting.

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Postpartum Posterior Reversible Encephalopathy presenting as Myoclonic Seizures

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Introduction

Posterior reversible encephalopathy syndrome (PRES) can present with seizures, encephalopathy, visual disturbances, headache, or focal neurologic deficits. Possible triggers include hypertension, metabolic disturbances, toxic agents, renal impairment, septic conditions, eclampsia, and preeclampsia. PRES can also develop shortly after or several weeks after delivery. Seizures. We report a case of postpartum PRES manifesting as myoclonic seizures that persisted for several months

Case presentation

A 31-year-old female several weeks after the vaginal delivery presented to the hospital with impaired consciousness, diffuse hyperreflexia and myoclonic jerks. The symptoms first started shortly after giving birth manifesting with involuntary limb movements with subsequent motor weakness, frequent falls, recurrent episodes of altered consciousness and somnolence.

At presentation, serial myoclonic seizures were observed with a frequency of 15-16 per minute. The jerks were more prominent on the right side of the body. A comprehensive laboratory investigation including the whole panel for autoimmune encephalitis and serology for infectious disorders did not disclose any abnormality. Ictal EEG demonstrated high amplitude delta waves with superimposed beta activity and T2-weighted and flair MRI revealed characteristic images for vasogenic edema:.After the admission, the patient experienced two GTCS. Trials of several ASDs were used, but clonazepam proved to be the most effective. Subsequently, the patient progressively recovered neurologic function, however, myoclonic seizures still persisted.

Conclusion

Even though PRES is considered to be a reversible condition, rarely patients develop epilepsy. In our case, the patient developed myoclonic epilepsy, which was effectively treated with clonazepam.

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Social cognition deficits affect the quality of life in patients with drug-resistant temporal lobe epilepsy

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Objective: Numerous studies indicate a significant decline in the quality of life (QoL) among patients battling drug-resistant epilepsy. It may be associated with various factors, including health, mood, or the quality of interpersonal relations. The ability to establish close and satisfying relationships is in turn related to the individual`s social skills. This study aimed to evaluate the social cognition abilities of individuals with intractable epilepsy and understand how it affects QoL.

Methods: We gathered 80 adult individuals with drug-resistant temporal lobe epilepsy with the average duration of epilepsy equal 19±6,98 years and 80 demographically matched healthy volunteers. The neuropsychological assessment was conducted using a set of tests evaluating the quality of life (QOLIE-31-P), recognition of emotions (RMET), recognition of intentions based on observation of body movements (CID-5) and the level of depression and anxiety (HADS).

Results: Individuals with epilepsy scored significantly lower across all measures of social cognition compared to healthy controls (p0.05). Further analysis highlighted a significant positive correlation between QoL scores and RMET (r = 0.61; p0.05), CID-5 (interaction recognition: r = 0.73; p0.05; interaction naming: r = 0.38; p0.05), and a negative correlation between QoL and the depression subscale of HADS (r = -0.61; p0.05).

Conclusion: Quality of life is related with social cognition deficits and depression. Patients with epilepsy should be provided with special care including the therapy of social cognitive deficits and enhancing social competences to improve their quality of life and reduce the risk of depression.

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Objective vs subjective memory deficits in patients with mesial temporal lobe epilepsy

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Purpose: Patients with mesial temporal lobe epilepsy (MTLE) present diverse profiles of cognitive disturbances. Due to the localization of the epileptic focus, the predominant deficit observed both in objective assessments and patient-reported complaints is memory impairment. Previous studies have frequently prioritized the objective evaluation of memory, neglecting the aspect of its subjective evaluation. In this study, objective assessment (testing) and subjective assessment (questionnaire-based) were combined to obtain a comprehensive understanding of the mnestic functioning of patients. Furthermore, we aimed to investigate the relationship between subjective and objective memory impairments and the severity of depressive symptoms.

Method: Twenty-eight patients with MTLE and twenty-seven demographically matched healthy individuals were examined using Wechsler Memory Scale (WMS-IV), Questionnaire of Memory Efficiency (QME) and Beck Depression Inventory (BDI-II).

Results: Patients with MTLE achieved significantly lower scores than healthy individuals in all subtests of the WMS-IV: Logical Memory I&II (p0.001), Verbal Paired Associates I&II (p0.001), Designs I&II (p0.001), Visual Reproduction I (p=0.002), II (p=0.005), Spatial Addition (p0.001) and Symbol Span (p0.001). Furthermore, patients subjectively rated their memory lower (p=0.002) and had more severe depressive symptoms (p0.001) than healthy individuals. However, no correlation was found between QME and WMS-IV subtests, nor between QME and BDI-II.

Conclusions: Patients with MTLE exhibit impaired mnestic functions. No association was found between the severity of objective deficits and the subjective assessment of memory performance. This may be due to a reduced self-awareness regarding the extent of deficits and is not associated with a severity of depressive symptoms.

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JUVENILE ABSENCE EPILEPSY AND MYASTHENIA GRAVIS: A CASE REPORT

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Background

Juvenile absence epilepsy (JAE) is a genetic generalized epilepsy syndrome which typically starts in adolescence and is characterized by absence seizures and generalized tonic-clonic seizures (GTCS). The management and follow-up of patients affected by both JAE and myasthenia gravis (MG) may be challenging due to the potential correlation between increased MG symptoms and epileptic seizures. Anti-epileptic drugs (AEDs) which affect the neuromuscular junction`s Na+ gated channels must be administered with caution.

Case presentation

We present a 43-year-old female patient with JAE who also suffers from generalized seropositive MG diagnosed at age 22. Main symptoms of MG were ptosis, nasal speech and lower leg weakness. Symptoms were managed with pyridostigmine bromide and a low dose of prednisolone. Patient has had absence seizures, as well as GTCS from the age of 17. At age 23 she had undergone thymectomy as a mean of MG treatment. At age 35 she had a worsening of absence seizures following noncompliance to AEDs. Despite prior AEDs, control of epileptic seizures was attained following the use of levetiracetam. At age 38 she was hospitalized because of the worsening MG symptoms caused by a respiratory infection. After antibiotic and immunoglobulin treatment symptoms had regressed. Following the successful AED treatment, patient has been seizure free with MG symptoms under control.

Conclusion

This case shows that in JAE patients who also suffer from MG prudent decision making is required, which considers the best course of treatment for each condition without impacting the other. Further carefully designed studies are needed.

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Angelman syndrome - a case report

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Angelman syndrome is a rare, complex genetic disorder. Characteristic features of this condition include delayed development (noticeable by the age of 6 to 12 months), intellectual disability, severe speech impairment and problems with movement and balance which usually appear in early childhood. Most affected patients also have epileptic seizures, microcephaly and difficulty sleeping. Patients typically have a happy, excitable demeanor with frequent smiling and hand-flapping movements.

Many of the features result from the loss of function of a gene UBE3A, located on chromosome 15. Healthy individuals normally inherit one copy of the gene from each parent and both copies are active in most of the body`s tissues. However, in neurons in the brain and spinal cord, only the maternal copy is active. This parent-specific gene activation is caused by a phenomenon called genomic imprinting. If the maternal copy of the UBE3A gene is lost because of a mutation, a person will have no active copies of the gene in most parts of the brain. About 70% of cases of Angelman syndrome occur when a segment of the maternal chromosome 15 containing this gene is deleted.

We present a 28 years old patient who showed signs of delayed development at the age of 6 months with later intellectual disability, severe speech impairment, myoclonus and absence seizures, as well as happy demeanor. Extensive diagnostic workup was nonremarkable so we performed next-generation sequencing genetic testing (epilepsy panel) and found a frameshift mutation, deletion of UBE3A gene, which is the cause of Angelman syndrome.

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When is VNS the best treatment solution in pharmacoresistant epilepsy caused by grey matter heterotopia – a case report

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Grey matter heterotopias are subtype of malformations of cortical development (MCDs) characterized by accumulations of neurons in abnormal locations, mainly due to impaired migration, and present a common cause of pharmacoresistant epilepsy. A resective neurosurgical treatment, in most cases after invasive monitoring (stereo-electroencephalography), is often challenging with poor results, and for some patients alternative therapeutic methods can be offered, such as neuromodulation method - Vagus Nerve Stimulation (VNS).

We present a 21-year-old male patient who was referred to our center due to pharmacoresistant epilepsy. He had focal non-motor epileptic seizures with impaired awareness. MRI findings showed widespread bilateral grey matter heterotopia, more pronounced on the right side along the trigone and the temporal horn of the right lateral ventricle, in the hippocampal area, in insular and medial part of the right parietal lobe, as well as in the left hippocampal region. Continuous video-electroencephalography (vEEG) findings were suggestive of a complex epileptogenic network involving right temporal lobe and neighbouring structures – insula, perisylvian cortex, frontal and parietal operculum. Due to MRI findings and vEEG results, as well as patient`s preferences for minimally invasive surgery, VNS was implanted (closed-loop system). Following the procedure there was a 85% reduction in seizure frequency and the patient`s quality of life improved significantly.

Pharmacoresistant epilepsy caused by grey matter heterotopia is a complex clinical entity and each patient requires a personal approach as well as multidisciplinary treatment. Our patient presents a case with multifocal heterotopic lesions and complex epileptogenic network, with a good response to VNS stimulation.

Keywords
Grey matter heterotopia, Malformations of cortical development, Pharmacoresistant epilepsy, Vagus nerve stimulation

Declaration of patient consent
The patient has given his consent for his images and other clinical data to be reported in this case presentation.

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EEG Findings in Patients with Epileptic Encephalopathy and Continuous Spike-Wave during Slow Sleep (CSWS) among the Uzbeks.

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Epileptic Encephalopathy (EE) are severe neurodevelopmental disorders marked by refractory seizures and cognitive impairment. This study investigated EEG findings in patients with these conditions in Uzbekistan, aiming to unravel patterns and variations in the local population.

Materials and Methods:

A retrospective analysis was conducted on EEG data from diverse patients with confirmed diagnoses of Epileptic Encephalopathy. The study encompassed various age groups and genders, with EEG examinations performed at Tashkent Medical Academy between 2005 and 2021. A total of 521 children, ranging in age from 3 months to 14 years, were included in the analysis. Recordings were scrutinized for characteristic abnormalities associated with Epileptic Encephalopathy. Attention was given to identifying links between EEG change and level of cognitive decline.

Results:

Preliminary findings revealed a spectrum of EEG abnormalities with varying severity and distinct age-related patterns. Burst-suppression and hypsarrhythmia were prevalent in younger age groups with EE (34%), while multifocal epileptiform discharges (2%) and continuous spike-wave patterns during slow-wave sleep were prominent in older patients (47%). The CSWS pattern was notably linked with the most significant changes in cognitive function among the studied population.

Conclusion:

This research enhances our understanding of EEG abnormalities associated with Epileptic Encephalopathy and CSWS in Uzbekistan. The association of CSWS with pronounced cognitive changes, may have implications for refining diagnostic criteria and developing targeted treatments. Further investigations into genetic, environmental, and therapeutic factors are warranted to optimize the management of these conditions in the local population.

Epilepsy

Application of combined psychodynamic treatment of idiopathic temporal lobe epilepsy (clinical case).

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Idopathic temporal lobe epilepsy has a studied overlap with affective dysregulation.

At the same time, the use of symptomatic medications, in particular antidepressants, does not affect the course of attacks. Our experience allows us to recommend a combination of antiepileptic drugs and psychodynamic psychotherapy as the most effective method of treatment.

A clinical case: Patient K, 25 years old

The attacks began at the age of 14, when the patient was taking substances to reduce weight. The substance included stimulants. Against the background of the use of substances for weight loss, the patient developed generalized tonic-clonic seizures. The MRI data showed no pathology, the EEG pattern corresponded to the diagnosis of temporal lobe epilepsy.

Attacks continued after the weight loss substance was discontinued.

Attacks were not controlled by antiepileptic drugs for 5 years. The patient could not study or work, hardly left the house and had suicidal ideas and was also overweight.

During the treatment of antiepileptic drugs , the patient immediately received psychodynamic psychotherapy, as in the case of a psychosomatic disorder.

The K`s personal history included situations that led to tension and rigidity of affect, the development of impulsivity, and increased aggressiveness. Understanding the mechanism of formation of the attack became a key opportunity for changes in the experience of conflict situations. This led to a stabilization of attack control.Psychotherapy lasted 5 years.

The patient has no seizures at present. During this time, she got married, gave birth to a healthy child, and works successfully.

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A report on a case of late infantile neuronal ceroid lipofuscinosis (NCL) involving a CLN6 variant

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Neuronal ceroid lipofuscinoses (NCLs) represent lysosomal storage disorders marked by seizures, motor difficulties, and vision loss. The underlying cause is associated with mutations in the ceroid lipofuscinosis (CLN) genes. The CLN6 protein, essential for lysosomal function, is an endoplasmic reticulum (ER) membrane protein featuring seven transmembrane domains. In this case report, we present the clinical details of a 7 years old girl with frequent seizures, speech regress, ataxia, unstable gait since she was 3 years old.. Brain MRI scans revealed high signal intensity in parietal lobes on T2-weighted images and cerebellar atrophy in fronto-temporal areas and cerebellum. An electroencephalogram (EEG) showed picture of encephalopathy, electrical status epilepticus in sleep. Employing next-generation sequencing on a epilepsy gene panel, we identified a pathogenic homozygous missense point mutation (c.407G A; p.R136H) in CLN6.The diagnosis of late infantile neuronal ceroid lipofuscinosis (NCL) and secondary epilepsy was established, leading to the prescription of different AEDs . However, at the 6-month follow-up, the epilepsy remained poorly controlled, and other symptoms showed no improvement. This marks the initial occurrence of NCL attributed to a CLN6 mutation, broadening the spectrum of genetic possibilities associated with NCLs. Typically, NCLs are not the primary consideration in cases like this. Employing a gene sequencing panel to explore instances of unexplained seizures and development delay proves beneficial in confirming the diagnosis.