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Classification of Epilepsy Syndromes

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Abstract

People with epilepsy could also be classified into different syndromes supported specific clinical features. These features include the age at which seizures begin, the seizure types, and EEG findings, among others. Identifying an epilepsy syndrome is beneficial because it helps determine the underlying causes also as deciding what anti-seizure medication should be tried. Epilepsy syndromes are more commonly diagnosed in infants and youngsters. Some samples of epilepsy syndromes include benign rolandic epilepsy (2.8 per 100,000), childhood absence epilepsy (0.8 per 100,000) and juvenile myoclonic epilepsy (0.7 per 100,000). Severe syndromes with diffuse brain dysfunction caused, a minimum of partly, by some aspect of epilepsy, also are mentioned as epileptic encephalopathies. These are related to frequent seizures that are immune to treatment and severe cognitive dysfunction, as an example Lennox-Gastaut syndrome and West syndrome.

Keywords: Epilepsy • Neurological disorder • Mahenge

Introduction

Childhood-onset epilepsies are a complex group of illnesses with a variety of causes and traits. Some persons lack evident neurological or metabolic disorders on the inside. Variable levels of intellectual disability, aspects of autism, other mental problems, and motor issues will all be associated to them. Others have underlying chromosomal abnormalities, inherited metabolic disorders, distinctive eye, skin, and systema nervosum traits, or defects of cortical development. Many of these epilepsies fall within the umbrella of the typical epilepsy syndromes. Additionally, there are a variety of clinical syndromes that are linked to a higher risk of epilepsy but don't have epilepsy as their primary symptom [1]. For instance, 90 percent of people with Angelman syndrome and 1 to 10 percent of people with mongolism both have epilepsy. In general, genetics is thought to contribute significantly to epilepsies through a number of processes. For a number of them, straightforward and complex patterns of inheritance are discovered. However, thorough screening failed to find many single uncommon gene variations with significant effects. De novo mutagenesis seems to be a key process in the epileptic encephalopathies. De novo refers to a mutation that affects a child but not the older individuals [2]. De novo mutations can originate in sperm and egg or very early in the embryonic development process. One gene was found to be impacted by the Dravet syndrome. It can be challenging to connect syndromes with the categories in the current classification of epilepsy when the reasons are unclear. For some circumstances, categorization is created somewhat haphazardly. The 2011 classification's idiopathic (unknown cause) category comprises disorders where the general clinical characteristics and/or age-specificity strongly suggest a putative genetic aetiology. Some childhood epilepsy syndromes, such as benign rolandic epilepsy, fall under the category of unknown causes, where the reason is assumed to be genetic. Others, like Lennox-Gastaut syndrome, are included in symptomatic despite a suspected genetic basis (at least in some cases). Clinical syndromes like Angelman syndrome, when epilepsy isn't the main symptom, were classified as symptomatic but it has been claimed that they should be included in the category idiopathic. Research advancements will lead to changes in how epilepsies, and specifically epileptic syndromes, are classified [3].

Description

An inherited epileptic illness called autosomal dominant nocturnal lobe epilepsy (ADNFLE) causes seizures when you're sleeping. It is idiopathic localization-related epilepsy. Childhood is usually when it starts. These seizures have complicated motor actions such hand gripping, arm rising and lowering, and knee bending and they come from the frontal lobes. Shouting, groaning, and sobbing are other frequent vocalisations. The common incorrect diagnosis for ADNFLE is nightmares. There is a genetic basis for ADNFLE. Numerous nicotinic acetylcholine receptors are encoded by these genes [4].

Children between the ages of three and thirteen can develop benign centrotemporal lobe epilepsy, also known as benign Rolandic epilepsy, which is an idiopathic localization-related epilepsy with a peak onset in prepubertal late childhood. These patients are healthy other than having a seizure problem. Simple focal seizures that engage the facial muscles and frequently result in drooling are a hallmark of this disease. Even though most seizures are brief, they can occasionally become generalised. Most seizures only occur at night and during sleep. The EEG may show spike discharges, which are more likely to happen during drowsiness or light sleep, over the centrotemporal scalp over the Rolandic sulcus of the brain. Near puberty, seizures stop. Anticonvulsant medication may be needed to treat seizures, but occasionally they are rare enough to allow doctors to postpone therapy [5].

Conclusion

The set of disorders that make up benign occipital epilepsy of childhood (BOEC), an idiopathic localization-related epilepsy, is constantly changing. The majority of authorities list two kinds, a late subtype with an onset between seven and ten years, and an early subtype with an onset between three and five years. Visual symptoms include scotoma, fortifications (bright spots or lines), or amaurosis are frequently present after seizures in BOEC patients (blindness or impairment of vision). Hemiconvulsions, convulsions affecting only one side of the body, and forced eye or head movements are frequent. Older patients often report of greater visual problems, while younger patients typically have symptoms that are almost migraine-like with nausea and headache. Spikes recorded from the occipital (back of the head) regions can be seen on the EEG in BOEC. According to Ruben Kuzniecky et aldescription .'s of autosomal dominant transmission, the EEG and genetic pattern point to this. Recently, some have classed as BOEC a group of epilepsies known as Panayiotopoulos

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syndrome that have some clinical characteristics with BOEC but have a wider range of EEG abnormalities.

Conflict of Interest

None.

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