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POST-INFECTIOUS EPILEPSY (LITERATURE REVIEW)

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Abstract: Recently, substantial evidence has been obtained regarding the possible involvement of inflammatory processes in epileptogenesis. Thus, several studies have established increased synthesis of specific inflammatory mediators in the brain of patients and corresponding activation of some pro-inflammatory pathways in seizure foci, as well as involvement of oxidative stress. Information has also emerged that some chronic infections, such as neurocysticercosis, HIV and herpes, without causing acute cerebral pathology, are capable of provoking epileptic seizures and even subsequent formation of refractory epilepsy. This review summarizes experimental and clinical research data on the connection between epilepsy and chronically occurring infectious diseases and neuroinflammation.

Keywords: epileptogenesis, neuroinflammation in clinic and experiment, chronic infections.

Problem relevance, role of inflammation in pathogenesis and course of epilepsy

Epilepsy is one of the most common chronic brain diseases and is characterized by persistent predisposition to epileptic seizures. Despite the development of modern drug and surgical treatment methods, in approximately one-third of cases they prove insufficiently effective. Currently, there are several theories of etiopathogenetic mechanisms of epileptic process formation leading to epileptic seizures. However, gaps still exist that prevent the development of new effective preventive treatment methods for epilepsy, including its pharmacoresistant (refractory) form.

There are numerous studies confirming the significant role of brain inflammation in epileptogenesis. Based on experimental models and clinical data, the influence of epileptic seizures on immune reaction occurrence and enhanced synthesis of inflammatory mediators in the brain has been proven, which in turn contribute to brain damage and initiation of new seizures. The significance of cytokines (IL-1β, IL-6, TNF-α), prostaglandins (PgE2) and their synthesis via the COX-2 pathway, brain infiltration by peripheral leukocytes, Toll-like receptor-4 signaling cascade and other immunity activation pathways by damaging molecular patterns has been mainly studied. These reactions lower the brain's seizure threshold, increase neuronal excitability and blood-brain barrier permeability, thereby increasing the risk of new seizure occurrence.

Under experimental conditions, it was established that immune activation in early life stages can lead to prolonged physiological disorders that manifest as epileptiform activity on EEG, more often during sleep, and correspond to certain neuropsychiatric conditions. Furthermore, some scientific works

indicate that oxidative stress arising against the background of chronic inflammation causes damage to basic cellular components and also plays an important role in epilepsy pathogenesis.

Recently, one study demonstrated that brain inflammation, by activating intracellular signaling pathways, regulates expression and/or function of molecular transporters from the ABC protein family (ABC-transporters) in brain vascular endothelial cells and gliocytes, which are components of the blood-brain barrier. Abnormal expression and/or dysfunction of molecular transporters, especially ABC transporters, may in some cases contribute to refractory epilepsy development.

Besides this cause of pharmacoresistance, some authors have suggested the existence of an interconnection between this disease course variant, chronic inflammation and hippocampal sclerosis development, which determines not only epileptic seizure occurrence but also severe, often status course of the disease.

For many years, it was believed that acute infectious diseases - meningitis, meningoencephalitis and encephalitis - play the leading role in post-infectious etiology epileptic process occurrence. However, it is precisely chronically occurring infections that are to some extent CNS-tropic, capable of persisting in the organism for extended periods and interacting with it. Prolonged antigenic stimulation in this case provokes the organism's immune response and leads to neuroinflammation that correlates with epileptogenesis. Thus, persisting infections can participate in epileptic process formation and even influence pharmacoresistance development.

Epidemiology of post-infectious epilepsy

According to ILAE data, over 60 million people worldwide suffer from epilepsy - one of the most common neuropsychiatric diseases on a global scale. Also, according to WHO data, approximately 80% of people with epilepsy live in countries with low and middle income levels. This is presumably related to increased risk of endemic diseases such as malaria and neurocysticercosis (NCC), elevated rates of traffic injuries, birth trauma, as well as differences in medical infrastructure and prevention programs and medical care accessibility.

For example, in Kenya, studying the clinical-demographic profile of children under 13 years seeking medical help for acute seizures, which subsequently are a risk factor for epilepsy development, showed that in 80% of cases the cause was precisely infections. In South Africa, when analyzing 8-year period data in children admitted to hospitals with seizure status epilepticus, the main etiological factors are meningoencephalitis and gastroenteritis (Y. Reddy, Y. Balakrishna, L. Mubaiwa, 2017).

Also, when studying statistical indicators in a Nepal hospital over a 2-year period, it was noted that among patients of all age groups, seizures are observed significantly more often in children. Generalized seizure episodes are more characteristic for them with highest prevalence in younger groups and predominantly in boys. All studied patients underwent neuroimaging and NCC was diagnosed in almost half the cases (N. Chaudhary, M. M. Gupta, S. Shrestha, 2017).

In another study in one NCC-endemic zone in Peru, serum of all residents over 2 years was evaluated for antibodies against Taenia Solium, and residents over 18 years additionally underwent brain CT. The study revealed high prevalence of this infectious disease and also confirmed that a significant portion of residents with asymptomatic epilepsy have brain calcification, which may subsequently provoke epileptic seizures. Many other infections capable of causing CNS involvement are widespread everywhere.

Neurocysticercosis

NCC is the most common helminthic infection of the nervous system and a frequent cause of reactive seizures and epilepsy. About 90% of recorded seizures in such patients are prospectively partial and do not subsequently transform into bilateral tonic-clonic seizures, indicating topographic connection of seizure semiology with NCC lesion localization (K. R. Duque, J. G. Burneo, 2017).

At the site of cysticercus penetration into brain parenchyma, a cyst forms around which gliosis occurs; subsequently it may become calcified. Nevertheless, cases exist where seizure semiology, interictal

EEG abnormalities and cysticercus location according to neuroimaging data do not correlate with each other.

Also, the connection between NCC and hippocampal sclerosis, which subsequently becomes the substrate of mesial temporal epilepsy, is not fully clear at present, as it can occur even if the cysticercus is located at a distance from the hippocampus. Accumulated data do not yet allow determining whether this is related to regular epileptic activity of a local or distant focus or to chronic inflammation due to cysticercus presence in brain tissues. However, no less attention of researchers to epilepsy occurrence is attracted by the influence of human immunodeficiency virus (HIV) and herpesvirus infections.

CONCLUSIONS

Based on the analysis of current literature on post-infectious epilepsy, several important conclusions can be drawn: **Inflammatory mechanisms play a crucial role in epileptogenesis**: The evidence strongly supports that neuroinflammation, mediated by cytokines (IL-1β, IL-6, TNF-α), prostaglandins, and other inflammatory mediators, significantly contributes to seizure susceptibility and epilepsy development. This inflammatory cascade lowers seizure thresholds, increases neuronal excitability, and compromises blood-brain barrier integrity.The relationship between chronic infections, neuroinflammation, and hippocampal sclerosis requires further investigation. Understanding these mechanisms may lead to novel therapeutic approaches combining antimicrobial, anti-inflammatory, and antiepileptic strategies.

These findings underscore the necessity for comprehensive approaches to post-infectious epilepsy prevention and treatment, incorporating infectious disease control, anti-inflammatory therapies, and targeted antiepileptic interventions, particularly in endemic regions and vulnerable populations.

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