

Epilepsy and Psychiatric Disorders: Is There a Common Genetic Susceptibility?

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Editorial

Although several studies have documented a strong association between epilepsy and some psychiatric comorbidities, either before or after the diagnosis of epilepsy, depression and other disorders remain under diagnosed and undertreated in People With Epilepsy (PWE) [1].

According to epidemiological data, 25-48% of PWE suffer from mood disorders, and 14-31% from anxiety [2]. Moreover, depression represents one of the most common psychiatric disorders in PWE, with a point prevalence ranging from 12-37% in community settings [3]. In clinical studies, depressive symptoms in epilepsy have been associated with several variables such as poor seizure control, duration of epilepsy, having complex partial seizures or temporal lobe epilepsy, unemployment and the use of antiepileptic polytherapy.

Recent reports have suggested a bidirectional relationship between epilepsy and depression [4]. Both conditions are episodic and may involve common pathogenic mechanisms. Indeed, epilepsy can lead to depression through its specific psychosocial consequences such as poor patient acceptance of his epilepsy, stigma and anticipatory anxiety associated with the unpredictable occurrence of seizures. On the other hand, Depressive symptoms in epilepsy impair patients' health-related Quality of Life (QOL) and may affect the clinical course of epilepsy.

Otherwise, a high suicide rate among PWE has been documented; the percentage of deaths by suicide ranges from 1% to 33% with an overall average of about 11.5% [5].

Chang et al. have demonstrated that the relation between epilepsy and psychosis is also bidirectional; the prevalence of interictal psychosis in epilepsy being estimated at 19.4% in generalized epilepsy and 15.2% in temporal lobe epilepsy [6].

What are the mechanisms underlying these disorders?

Now, it is admitted that some psychiatric disorders share common genetic bases with epilepsy [7]. Indeed, genetic studies of copy number variants (CNVs) have shown that in some cases, the same CNV is identified in PWE and in individuals with other neuropsychiatric conditions such as autism spectrum disorders, schizophrenia, mental retardation and attention deficit hyperactivity disorders [8]. These findings suggest that a large number of candidate CNVs are not disease-specific but are involved in the expression of different

behavioural and neuropsychiatric phenotypes sharing common biological pathways, affecting glutamate and GABA neurotransmission. However, it should be stressed that the pleiotropic effect of CNVs in neuropsychiatric disorders, may be due not only to haploinsufficiency, but also to the contribution of additional genetic, epigenetic and environmental factors yet unidentified [9].

From a clinical point of view, it is essential to identify these comorbidities in PWE, using standardized scales and validated questionnaires to enhance and customize the management of these patients. Further progress in screening psychiatric comorbidities in PWE will enhance our understanding of epilepsy and could play an important role in the stratification for genetic studies.

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