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LETTER

What is the risk of unprovoked seizures after acute symptomatic status epilepticus?

To the Editors:

We congratulate Dr Rodrigo-Gisbert et al.[1] for providing interesting insights into the risk and predictors of long-term unprovoked seizures after status epilepticus (SE).

In a cross-sectional study of 360 consecutive participants with a first SE episode and no history of epilepsy, 30.3% presented unprovoked seizures during a median follow-up of 1.8 years.[1] Progressive symptomatic etiology, time to first-line treatment initiation longer than 1.5 h, and superrefractory SE increased, whereas older age and acute symptomatic etiology decreased the risk of unprovoked seizure recurrence. In the competing risk analysis adjusted by mortality, remote symptomatic and cryptogenic etiologies were also associated with the occurrence of unprovoked seizures.[1]

The relationship between remote and progressive causes of SE and the increased hazard of seizure recurrence during the follow-up is not unexpected. The subjects presenting with a first SE episode with underlying progressive and remote symptomatic etiologies meet the operational definition of epilepsy.[2] Conversely, the knowledge of the actual probability of developing a subsequent unprovoked seizure over time in people with SE due to acute symptomatic, as well as unknown, causes may be of paramount relevance to guide clinical practice and understand the epileptogenic potential of SE itself. The category of acute symptomatic etiology, however, encompasses a great variety of heterogeneous causes. In this regard, we have recently proposed four subcategories of acute etiologies, namely, "acute-triggering factors in epilepsy," "acute primary central nervous system (CNS) pathology" (including cerebrovascular diseases, active CNS infections, or head trauma), "secondary CNS pathology" (including metabolic disturbances or systemic infection), and "drug or alcohol intoxication and withdrawal" (Figure 1).[3] Differences in the risk of in-hospital mortality and poor functional outcome at discharge have already been identified among these subcategories.[3] It remains to be investigated whether differences also exist in the risk of post-SE epilepsy, as it looks reasonable. Although not examined individually in the study by Dr Rodrigo-Gisbert et al.,[1] the rates of remote seizures differed significantly within each subgroup of acute etiologies and were 0% in the case of alcohol abuse and sodium imbalance, 18.5% in the presence of acute CNS infection, and 23.9% in acute cerebrovascular disease. It is noteworthy that acute symptomatic SE after ischemic stroke has been shown to be a strong predictor of both the occurrence of unprovoked seizures, with a 10-year risk of epilepsy as high as 88%, and drug-resistant epilepsy.[4,5]

So far, there exists limited evidence about the long-term consequences among SE survivors, and several issues have not been addressed yet.[6] The development of scoring systems and tools to predict the risk of unprovoked seizures and epilepsy after SE could inform patients and provide guidance for clinicians. A nuanced perspective that considers distinct etiological subcategories may better suit the heterogeneity of SE and the continuum spectrum of severity and prognosis, resulting in improved stratification of the individual risk.[7,8]

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CONFLICT OF INTEREST STATEMENT

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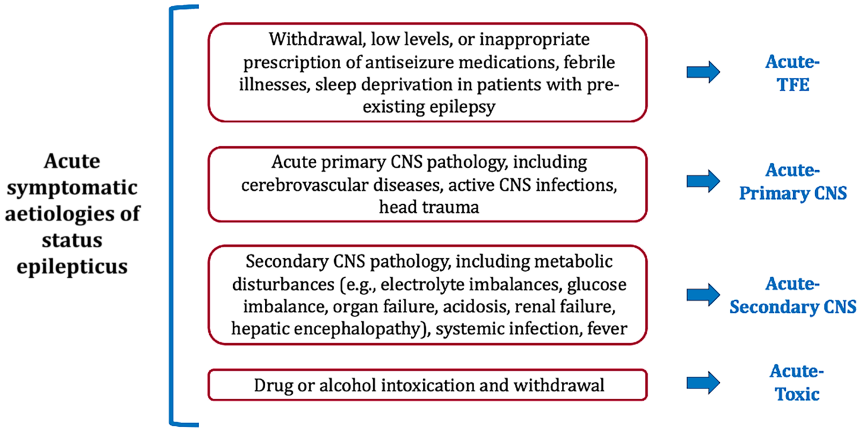

FIGURE 1 Classification of acute known causes in status epilepticus. CNS, central nervous system; TFE, triggering factors in epilepsy.

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