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Letter to the Editor

When interpreting EEG abnormalities in delirium, all of its determinants and triggers must be taken into account

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Letter to the Editor

We read with interest the article by Faizal *et al*. on a cross-sectional observational study of EEG abnormalities in 120 patients with delirium admitted between April 2021 and April 2023 [1]. It was found that 80% of patients had EEG abnormalities, that EEG abnormalities increased with age, that EEG abnormalities increased with the Confusion Assessment Method (CAM), and that EEG abnormalities decreased with increasing Richmond Agitation and Sedation Scale (RASS) and with increasing Barthel Index [1]. The study is noteworthy, but some points require discussion.

The first point is that the causes of delirium in the 120 patients included were not specified [1]. Delirium is often caused by medications (e.g., new medications, higher dosages, interactions between medications, over-the-counter medications, alcohol withdrawal, illegal drugs, medications that reduce acetylcholine), electrolyte imbalances, withdrawal from long-term medications or toxins, infections, decreased sensory stimuli (e.g., hearing or vision impairments), brain disorders, urinary or bowel disorders or constipation, heart or lung disease, imbalances in brain transmitters, multimorbidity, and changes in environment or habits [2]. We should know which of these causes occurred in the cohort studied and how frequently.

The second point is that the CAM distinguishes between hyperactive and hypoactive delirium, but it was not reported how many patients had one type or the other [3]. Therefore, the analysis should also include information on how many patients had hyperactive delirium and how many had hypoactive delirium. This information is crucial, as the frequency of EEG abnormalities, treatment, and outcome may differ between these two types of delirium.

The third point is that non-convulsive or minimally convulsive status epilepticus can mimic delirium, but it was not reported how many patients had EEG findings suggestive of status epilepticus. There is also no mention of how many patients started receiving antiepileptic drugs (ASMs) after the EEG results.

The fourth point is that not only benzodiazepines and propofol have a sedative effect, but also analgesics, antiepileptic drugs, neuroleptics, hypnotics, beta-blockers, antihistamines, and muscle relaxants. How many of the included patients regularly took such medications, which could also explain a focal or generalized slowing in the EEG? Patients who were taking any of these medications at the time of EEG recording should be excluded from the study.

The fifth point concerns the discrepancy between the exclusion criteria of benzodiazepines and propofol and the need for sedation in patients with delirium. Were patients included in the study already receiving specific treatment for their delirium, or were they treatment-naive at the time of EEG recording? This point is crucial, as delirium therapy may be the reason for focal or generalized slowing in the EEG.

In summary, EEG abnormalities during delirium have multiple causes and are not only related to the delirium itself, but also to comorbidities, concomitant medication, environmental conditions, and the extent of endogenous and exogenous stressors. When assessing EEG abnormalities in patients with delirium, all determinants and triggers of delirium should be taken into account.

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