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Letter to the Editor

Myoclonic State should be Treated with Benzodiazepines, Levetiracetam, or Valproic Acid before Propofol or Sevofluran are Considered

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Letter to the Editor

We read with interest the article by Canasiro *et al*. about a 26-year-old woman who had apparently attempted suicide by ingesting 7.5 g of bupropion, resulting in coma and myoclonic status [1]. Since the myoclonic state did not improve despite increasing doses of midazolam and propofol and no intravenous anticonvulsants (ASM) were available, the patient was given sevoflurane (6.5 mg/hour) for inhalation [1]. Sevoflurane led to immediate complete improvement of the myoclonic state, and the patient recovered completely by day 10 [1]. The study is remarkable, but some points require discussion.

The first point is that it is not clear why no antiseizure medications (ASMs) other than midazolam and propofol were tried [1]. The argument that no other intravenous ASMs were available is not understandable, as intravenously administered ASMs are inexpensive and are usually available in every hospital for emergencies. Commonly available intravenous ASMs include phenytoin, valproic acid, levetiracetam, lacosamide, and phenobarbital. At least some of these should be available in every hospital worldwide. According to the ILAE guidelines, generalized myoclonic status should initially be treated with benzodiazepines. If these fail, valproic acid or levetiracetam should be administered intravenously [2]. There is now also a stable intravenous formulation of lamotrigine that can be tried as an alternative [3].

The second point is that it is incomprehensible why multimodal magnetic resonance imaging (MRI) and magnetic resonance angiography (MRA) of the brain were not performed [1]. Multimodal MRI is essential to determine whether the patient has suffered an embolic stroke or has diffusion-weighted imaging (DWI) abnormalities due to a myoclonic state, and to rule out causes other than bupropion intoxication. MRI may also reveal reversible signal changes (SRMAs) caused by seizures, such as cortical hyperintensities [4].

The third point is that no EEG was recorded [1]. In order to assess whether the myoclonic state was associated with EEG abnormalities that could serve as an indication for ASM treatment, it would have been crucial to record an EEG, preferably before the myoclonic state had subsided.

The fourth point is that propofol is not the ideal drug for terminating a myoclonic state. There are reports from individual cases that propofol can trigger myoclonic seizures [5, 6]. Therefore, consideration should be given to whether the resolution of the myoclonic state is due to the discontinuation of propofol rather than sevoflurane. It should also be taken into account that sevoflurane itself can trigger myoclonic seizures [7].

The fifth point concerns the discrepancy between the description of the patient as completely normal and the statement that the patient was discharged with pre-planned support [1]. Why did she need support if she was normal? What type of antidepressant treatment did the patient receive after discharge?

In summary, a myoclonic state should be treated with benzodiazepines and, if these are ineffective, with an intravenous ASM, preferably levetiracetam or valproic acid. Propofol and sevoflurane should be administered with caution, as both have the potential to trigger myoclonic seizures.

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