Alcohol dependent and GTCS patient with sleep-related hyper motor epilepsy

Dear Editor,

Sleep-related hypermotor epilepsy (SHE) is an uncommon disorder that affects people of all ages, with an estimated prevalence of 1.8 per 100,000.[1,2] There have been very few reported cases of SHE in India,[3] particularly among elderly patients. In this case, we have identified a patient previously diagnosed with epilepsy who also struggles with alcohol dependence. Over the past two years, he has been experiencing nocturnal seizures exclusively during sleep.

This 47-year-old man was hospitalized in the psychiatry department of Assam Medical College. Proper informed consent to publish his case summary was obtained from the patient and his family members. His symptoms emerged shortly after his latest seizure, involving auditory hallucinations, irrelevant speech, poor sleep, and failure to recognize family members. He also displayed physically and verbally aggressive behavior, leading to a tentative diagnosis of postictal psychosis. Further examination revealed that the patient was on treatment for generalized tonic-clonic seizures (GTCS) for two decades and was also a regular alcohol user. He had been on a daily dose of phenobarbitone 60 mg for five years. Over the past two years, his alcohol consumption has escalated throughout the day. He began experiencing episodes of abnormal repeated limb movements (mostly unilateral upper limb movements), screaming, eye-rolling, and urinary incontinence exclusively during nighttime sleep. These episodes typically occurred 3 to 4 h after falling asleep and could happen multiple times in a single night, each lasting only a few seconds. He had no recollection of these events the following day, and there were no reports of confusion or psychotic behavior during these two-year periods. Family members reported that this unusual movement differed from the jerky movements he had been experiencing for the past 20 years. Within the past 1 year, these nocturnal hypermotor movement episodes occurred 4–5 times per week. As a part of the behavioral change, only his anger increased for the last 2 years, and he started to use abusive and vulgar language socially. No family history of seizure disorder or psychiatric disorder was found. The electroencephalography (EEG) report was within normal limits, and a magnetic resonance imaging (MRI) brain showed microbleeds in the left frontal lobe with bilateral subdural hygroma and generalized atrophy shown in Figures 1-3. Other routine blood investigations were within the normal limit. An autoimmune profile could not be conducted due to financial constraints faced by the patient's family. The disease was differentiated from parasomnias using the frontal lobe epilepsy and parasomnia scale (FLEP),[4] where a score of 5 strongly indicated the existence of nocturnal frontal lobe epilepsy (NFLE). For psychotic behavior, olanzapine 20 mg was started, and valproate 1 g was added for epilepsy after stopping phenobarbitone. However, as the seizure was not controlled, Brivaracetam 100 mg was added. Thiamine was

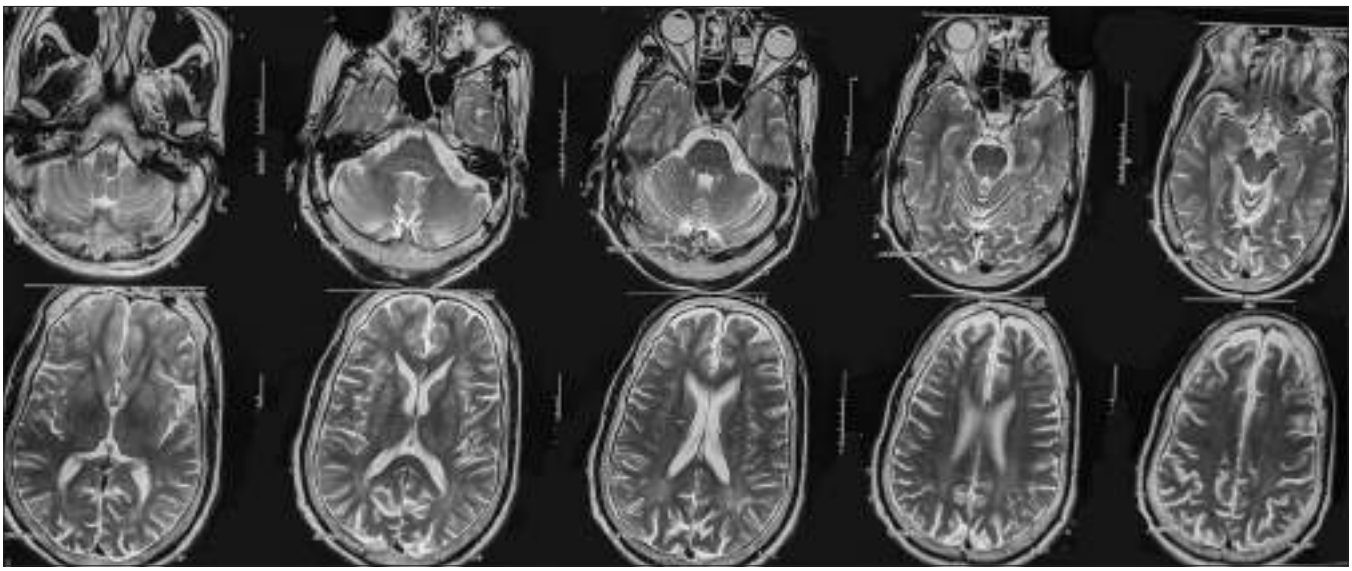


Figure 1: MRI brain Photo 1 of the patient

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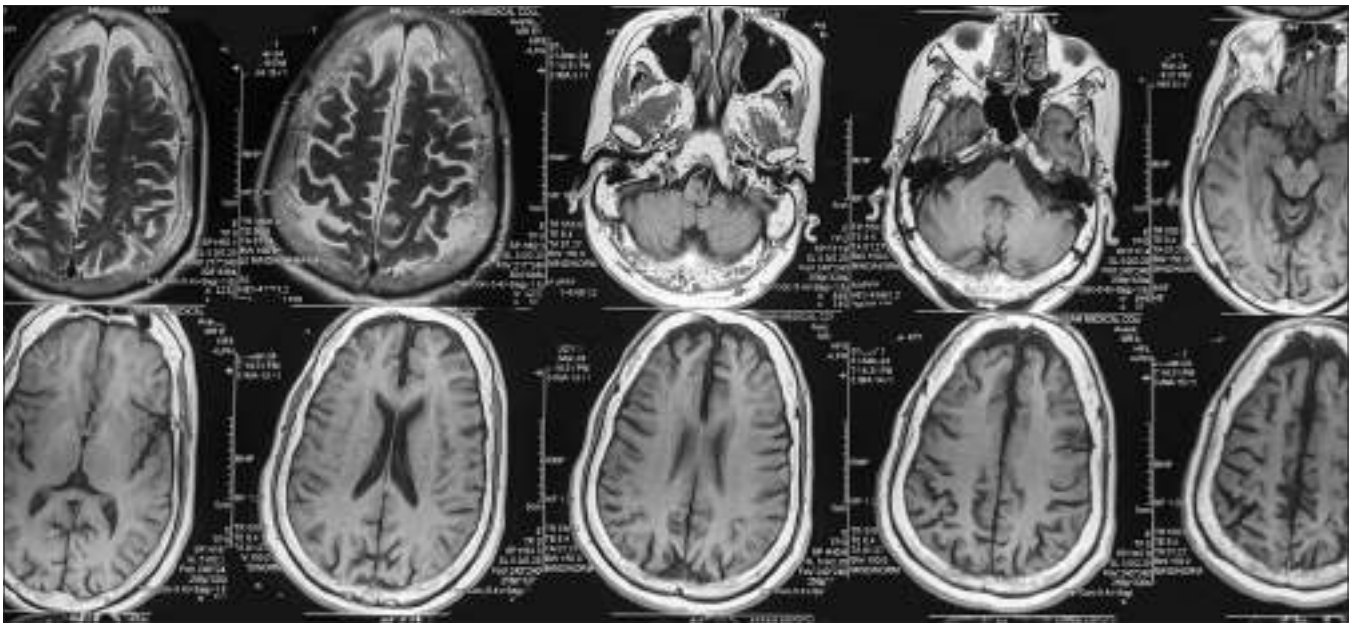


Figure 2: MRI brain Photo 2 of the patient

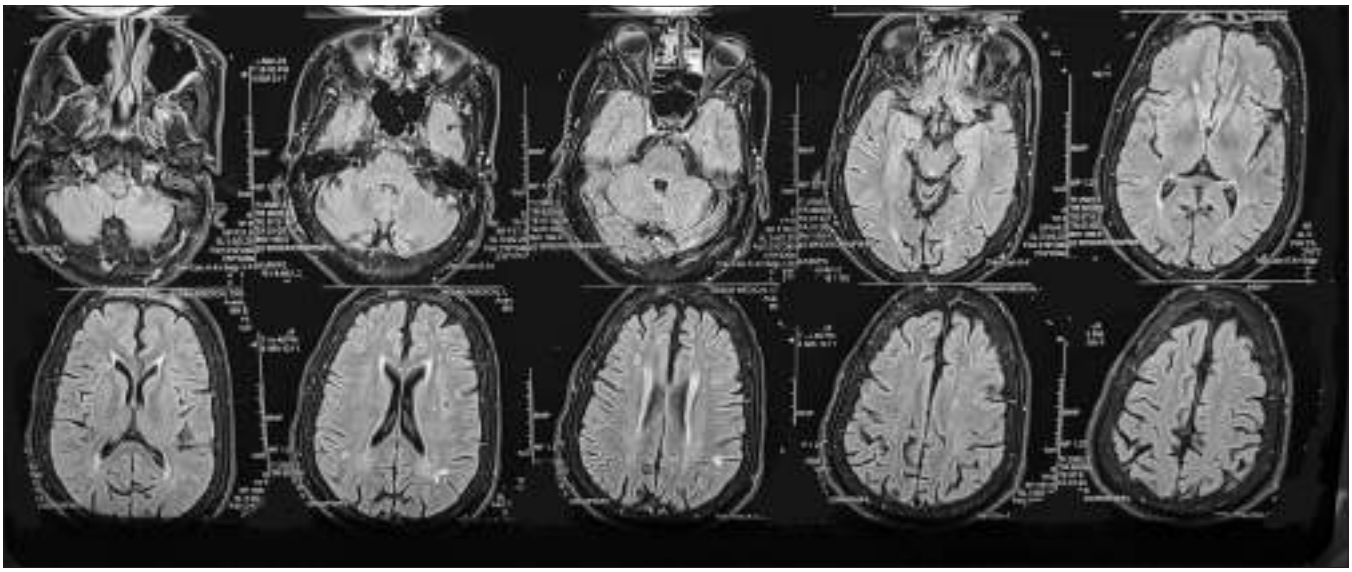


Figure 3: MRI brain Photo 3 of the patient

continued for alcohol history. Later, phenytoin 300 mg was added as the seizure episodes continued. With the medications, his seizure episodes and psychotic behavior improved within three weeks, but his perplexed behavior persisted.

The majority of cases of SHE or NFLE are familial, however, they can sometimes occur sporadically.[5] Hyperkinetic behavior (kicking) was the main feature in our case. Other minor features included unstructured vocalization like screaming, a seizure lasting less than 2 min, asymmetric tonic or dystonic seizures with or without head or eye deviation, and infrequently epileptic nocturnal wandering. These features were similar to the diagnostic criteria of SHE.[2] This occurs mostly in non-rapid eye movement (NREM) sleep and rarely during rapid eye movement (REM) sleep.[6] The interictal EEG readings may seem normal, and the EEG may not always provide clear evidence even during seizure activity, which further complicates the diagnosis of nocturnal seizures.

The patient had been experiencing intermittent unusual behavior, and hypermotor activity characterized by brief and frequent episodes over the past two years, with a rapid return to normal afterwards and without any changes in consciousness upon waking. There was no evidence of memory loss or automatic behaviors, seizure for prolonged duration (>2–3 min), or long duration of postictal confusion typically associated with temporal lobe epilepsy.

These findings strongly indicate a diagnosis of NFLE over the past two years (1). Mostly asymmetric upper and lower limb nocturnal movement during the attack over the last 2 years differentiated it from GTCS.[7] According to previous studies, a frontal lobe lesion was found in most of the patients with frontal lobe epilepsy[8] like it was in our patient.

Alcohol tends to elevate the seizure threshold, according to a few study findings.[9] Our patient, who consumed increasing amounts of alcohol regularly, would frequently drink alcohol throughout the day. This consistent alcohol intake may have maintained a higher seizure threshold during daytime hours. However, as alcohol concentrations decreased at night during sleep, his seizure threshold lowered, potentially leading to the occurrence of nocturnal seizures. While there is no direct link between alcohol consumption and SHE, disrupted sleep patterns induced by alcohol can exacerbate seizure frequency in individuals with SHE.[4]

Author contributions

SG conceived the research idea, SP conducted the research, and RG gave her valuable input. All authors contributed substantially to the write-up of the article and all take responsibility of the content of the publication.

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Nil.

Conflicts of interest

There are no conflicts of interest.

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