described cataplexy induced by laughter. She received venlafaxine 75 mg and then pitolisant 18 mg. Daytime sleepiness and cataplexy improved but she experienced three tonic-clonic seizures on the same day during wakefulness after starting pitolisant 18 mg whereas she had been seizure-free for many years. No other explanation was found. Pitolisant was stopped and there was no other seizure. Daytime sleepiness was then treated by scheduled naps as she did not have an occupational activity. The frequency of nightmares decreased with the antidepressant drug.

## 3. Discussion

This diagnosis was challenging because of the severe insomnia presentation making interpretation of daytime sleepiness difficult. Night sleep impairment is important in patients with narcolepsy as underlined by a recent review of literature [1]. They have more stage 1 sleep and shifts from deep sleep stages to wake and stage 1 sleep compared to controls. WASO is also longer but not as much as patients with insomnia. Our patient had a long WASO probably explained by insomnia comorbid with narcolepsy. It could be secondary to sleep hygiene errors and nightmares. We did not find parasomnia characteristics nor atonia loss during REM sleep. People with narcolepsy are reported to have six-fold more nightmares than the normal population [2]. The diagnosis of post-traumatic stress disease (PTSD) could be retained for our patient. Narcolepsy and PTSD share common points. Amygdala abnormalities have been described in both conditions [3,4]. Narcolepsy could be a risk factor for PTSD due to dysfunctional amygdala. Sodium oxybate was not proposed because of epilepsy comorbidity and neither was modafinil because of drug interaction risk with antiepileptic drugs. On the contrary, pitolisant was considered to be safe in patients with epilepsy [5].

A temporal association between pitolisant introduction and our patient's seizures was possible and declared to pharmacovigilance. No other explanation was found. The mechanism of action is unclear but could be due to histaminic dysregulation of the thalamocortical pathway.

## Disclosure of interest

The authors declare that they have no competing interest.

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