

temporomandibular disorders among psychiatric cases.² Our case strongly suggests a neuroleptic-induced phenomenon because of the chronic exposure to antipsychotic drugs prior to the occurrence of bruxism and complete disappearance with clozapine. Eight earlier cases of antidopaminergic related bruxism have been reported, but even multiple drug trials have failed to provide confirmation.³ A subsequent report suggested involvement of adrenergic, serotonergic, and dopaminergic systems in the pathogenesis of bruxism, as the side effects were relieved after propranolol was added to ongoing antipsychotic treatment.⁴ Although there are rare reports of tardive dystonia induced by typical antipsychotics, clozapine, in contrast, has been reported to ameliorate tardive dystonia.⁵ To our knowledge, clozapine has not been tried in neuroleptic-induced bruxism. Positive response to clozapine, as reported in an earlier study of neuroleptic induced tardive dystonia, chronic duration, and dystonic pattern of teeth movements, further support our finding that bruxism is a focal tardive dystonia. Clozapine may be the first drug of choice in such cases even though there are few reports of tardive dystonia associated with clozapine.

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False Beliefs During Migraine Attack

To the Editor: Brain mechanism of beliefs and false beliefs remains utterly elusive. In contrast to psychiatry, where false beliefs constitute the background of mystic, cosmologic, or paranoid delusions, beliefs are seldom reported in the context of neurological disorders. A strong belief that the hemiplegic hemibody is still moving after nondominant hemispheric stroke, confabulation during Korsakoff syndrome and fluent aberrant speech production without perceiving the lack of sense during Wernicke's aphasia are classical examples of false beliefs involving specific neurological function due to brain damage.^{1–3}

Herein, we would like to report the case of a patient suffering from severe migraine attacks during which he presented with isolated false beliefs strikingly influencing his behavior.

Case Report

A 54-year-old man had suffered from migraine since early adulthood. He had no other disease and was receiving no treatment. His migraine attacks were characterized by recurrent episodes of violent headache, nausea and vomiting, and a strong need to sleep, usually

beneficial naps. The episodes lasted between 10 and 24 hours. The frequency had been diminishing over the last few years.

Last January 31st, the day after a meal with seafood and wine, the patient awoke with frontal pain, suggestive of a hangover. He left for work, as usual, but quickly returned home because of the intensity of the pain and associated nausea (he felt unable to drink his coffee and had no appetite). Once home, he slept until approximately noon. When he awoke, he was convinced that he had to go to his parents-in-law's to look for his wife, asked his daughter-in-law to close all the shutters in the house, and went to the home of his in-laws, where, when he saw that his wife was not there, he "pretended" to look for her all over the house in a somewhat mischievous fashion. When his wife arrived at her parents' home after having been called by her mother, he seemed a bit irritated at having made a mistake. He remembered all of the events. His behavior seems adapted to the conviction that he expressed, according to those present. The only thing that seemed unusual was his adherence to a false belief, vivid and long-lasting (while he was looking for his wife at her parent's house, who attempted to persuade him that she was at work, as usual, he remained convinced that she in fact was in their house). After this episode, he went to bed and again spent the entire afternoon sleeping. When his wife woke him early in the evening, he suddenly quite vehemently demanded that she cook pasta, holding his mobile phone in one hand and the alarm clock in the other. The request was remarkable given the importance granted to the time (between 7 and 8 o'clock), the imperative nature of the demand, and the annex remarks ("Does the pasta look pretty?"). In recounting

the episode, the patient remembered the importance he gave to the fact that "it should not change my self-biology." His wife prepared the pasta; he ate it, vomited, and returned to bed. During the next night, close to 4.00 a.m., he suddenly awoke "obsessed by the idea of an appointment." He quickly corrected himself and went back to sleep. The next day, he did not feel very good, but the manifestations disappeared. In recounting the events of these 20 hours, he recalled the onset of a single and isolated nighttime episode, in June 2006, where he got up close to 4.00 a.m. and set the dining room table. He remembered being convinced of an imminent family meal.

Brain CT scan, brain MRI, and prolonged EEG after a total sleep deprivation were performed and did not show any anomaly. The diagnosis of a migraine attack in a migraineur was concluded, with a recurrent false belief following each awakening during the period of the attack. This diagnosis was supported by the clinical setting, the severe frontal pain, the transient hypersomnia, and the digestive signs.

Discussion

Confusion on awakening represents a subgroup of parasomnia, possibly related to dissociated slow-wave sleep, that is, a patient with behavioral manifestations (somnambulism, sleep terror, etc.) without the EEG correlates of being awake. In contrast to this diagnosis, this patient was clearly not confused, as the witnesses reported normal behavior for the incorrect belief. In the same way, epileptic seizures were considered as a possible diagnosis, but ruled out given the lack of confusion, aphasia, memory impairment during the episodes, long duration, and normal investigations including prolonged EEG. We propose that the present patient suf-

fered from isolated false belief during the migraine attack. A possible transient dysfunction involving the right hemisphere could be suggested, according to the theory of left (so-called "dominant") interpretative brain,⁴ the lack of language disorder during the reported attack, and the potential transient impact of migraine on brain hemispheric function.

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Lithium Prophylaxis in Kleine-Levin Syndrome

To the Editor: Kleine-Levin syndrome is described as a triad of recurrent hypersomnolence, hyperphagia, and hypersexuality.¹ However, an incomplete presentation characterized by hypersomnia (which is mandatory for clinical diagnosis), with or without hyperphagia, hypersexuality, cognitive disturbances, mood symptoms, compulsive behaviors, and perceptual abnormalities has also been described. We describe a patient with a clinical diagnosis of Kleine-Levin syndrome and the favorable response to lithium.

Case Report

A 16-year-old girl presented with episodic hypersomnia for the previous 6 years. The episodes had an abrupt onset and occurred approximately once a month. The episodes lasted anywhere from 2 to 15 days. She would average about 20 hours of sleep in a day. She was observed by her family to be "dull," less interactive, and was unable to attend school. During the episodes she used to unusually crave and demand carbohydrate food. Repeated questioning and seeking reassurance were also observed, and she was unable to recollect events that occurred during episodes. She had received treatment from the third episode onward with various drugs including risperidone, olanzapine, valproate, carbamazepine, topiramate, and clobazam (a benzodiazepine) without remission from the episodes.

Biochemical and endocrine parameters including CT scan of the brain were all normal. Polysomnography was attempted, but she did not cooperate for the procedure. The treatment team considered a clinical diagnosis of Kleine-Levin syndrome. Lithium in 300 mg tablets was initiated as a prophylactic agent; the dose was increased later to 750 mg/day (serum level: 0.8meq/liter). The patient has been on lithium regularly for 6 months and has not had any recurrence of hypersomnia or behavior changes so far. She has once again returned to school and has resumed her studies.

Discussion

An incomplete presentation is more common than the complete triad of hypersomnia, hyperphagia, and hypersexuality.² Patients can appear depressed and suicidal during the episode,¹ and sometimes if the hypersexuality, social disinhibition, and behavioral dyscontrol predomi-