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Case Presentation

Unresolved Nausea during Sleep and Horrific Dreams in a 65-Year-Old Man

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Introduction

The authors report the unresolved case of severe nausea and nightmares occurring only in association with sleep (no nausea occurs until the patient enters sleep or if the patient delays sleep). The patient has had a colectomy and other abdominal surgeries in the past. One of the authors is the affected patient. Because this constellation (chronic nightmares during sleep and severe nausea upon awakening) has not been reported in published literature to the authors' knowledge, this is an index case report to stimulate the identification of additional cases, and to identify treatments in correspondence with readers. The authors also desire to advance patient care using direct patient participation in medical case literature, in which the patient's voice is an equal partner in publication.

Nausea is a perception, a symptom across a spectrum of unpleasant sensations associated with exposure to conditions or substances likely to provoke vomiting as severity increases [1]. In the nervous system, the chemotactic trigger zone (area postrema) in the medulla is involved in nausea and vomiting. Here, the blood-brain barrier is incomplete, enabling toxic substances to cause nausea and/or vomiting [2]. Nausea and vomiting can also be caused by abnormalities of the nucleus tractus solitarius in the medulla from interactions with the gut, and which has projections to the limbic system. Both the area postrema and nucleus tractus solitarius interact with the vagus nerve [3]. With respect to potential etiologies for nausea, the differential is broad and includes gastrointestinal and non-gastrointestinal disorders [4].

Case Presentation

A 65-year-old man presents with a 10-year history of severe nausea during sleep, nightmares and multiple sleep arousals followed by diminishing daytime nausea. The nausea precludes the patient from getting out of bed for the first hour after awakening. The patient describes the sensation of nausea as akin to being severely "hungover" or having a feeling of electricity running through him, with nausea always being the primary sensation. It is not accompanied by vomiting or retching.

The patient had no prior history of sleep disturbances or nausea. No major life events affecting his psychological well-being had occurred around the time the nightmares; nausea began at age 56. He first noticed nausea one or two months before vivid nightmares began. Upon falling asleep, he experiences multiple vivid nightmares which start almost immediately and last throughout the night. Themes within the nightmares range from highly troubling manifestations of everyday situations to extreme violence directed toward others or himself. The patient's sleep is interrupted spontaneously every few hours. At each waking, he notes intense nausea. Frequently, he is awake for one hour before the nausea subsides; then he returns to sleep. Such interruptions take place 2-4 times per night, every night since onset. Between 3 to 5 hours after waking for the day, the patient experiences nausea and has no appetite. He denies headache, dyspnea, sweating, light-headedness, dysphagia, or vertigo associated with the nausea. When he goes to sleep the following night, the nausea is gone, as if he were never sick in the first place.

As the patient's time since last sleep accrues, the nausea dissipates and eventually disappears by mid-afternoon. The patient had two separate instances where he did not sleep for a night and subsequently did not experience nausea the following day; the first instance was due to a city-wide flood; the second was in preparation for a sleep-deprived EEG. Furthermore, the length of sleep is directly related to how much nausea he experiences: the more he sleeps, the more intense his subsequent nausea. The only alleviating factor the patient has identified is intense aerobic exercise, during which the nausea abates, only to return within an hour.

The patient's past medical history is significant for ulcerative colitis, multiple intestinal strictures/abscesses/adhesions, osteoarthritis and hypercholesterolemia. His surgical history includes colectomy, ileoanal pouch, numerous intestinal surgeries (1987-2002), tear duct surgery and dental implants. He has had multiple surgeries to remove gastrointestinal fistulas with the last surgery taking place 7 years before the chronic nausea began. He undergoes flexible sigmoidoscopies annually which have shown no evidence of leakages or fistulas.

His medications include prazosin 1mg nightly and Crestor 5mg daily. Family and social histories are non-contributory.

On examination, the patient was alert and fully oriented, with normal mentation. His body mass index was 23.1 and neck circumference was 37.5cm. Vitals were within normal limits. His cardiovascular and respiratory examinations were unremarkable.

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Abdominal exam revealed remote laparotomy scars but was otherwise unremarkable. The neurological examination of cranial nerves, motor system, coordination, reflexes, sensation and gait were unremarkable.

The patient has undergone many investigations. Gastroscopy and gastric emptying studies were normal; abdominal ultrasound was normal aside from cholelithiasis which was associated with an ejection fraction of 2% as seen on a hepatobiliary iminodiacetic acid scan. Esophageal manometry and 24-hour esophageal pH impedance studies were normal. Abdominal and head CT scans were negative. Brain MRI (T1-weighted, fluid attenuated inversion recovery, T2-weighted) was normal.

The patient has undergone an assessment by psychiatry to investigate his symptoms. The working hypothesis was of a possible functional-somatic syndrome, such as somatic symptom disorder. Pharmacotherapy suggestions included prochlorperazine, which did not prove helpful, and for low-dose mirtazapine, which seemed to intensify the patient's nightmares.

Consultation with a respirologist at an academic sleep center noted that the patient goes to bed about 1a.m. and falls asleep without difficulty, usually within 20 minutes, without medication. He gets up between 9a.m. and noon, feeling ill. He did not have morning headache, only nausea. His Epworth Sleepiness Scale is 0/24. Further review with an outside sleep expert suggested follow-up studies of autonomic function, psychiatry review, and confirmation of history inflammatory bowel disease diagnosis. The patient's symptoms were not related to sleep disordered breathing.

Polysomnography with video monitoring showed poor sleep quality due to frequent awakenings and stage transitions, prolonged wakefulness after sleep onset, alpha intrusion and mild fragmentation from spontaneous arousals and respiratory events. Sleep-deprived electroencephalogram (EEG) done in August 2012 showed intermittent theta and some delta activities over bilateral temporal regions independently which were more prominent during hyperventilation, drowsiness and light sleep. A 3-day ambulatory EEG performed in April 2013 showed mild to moderate amounts of intermittent theta activity over both cerebral hemispheres, most pronounced at the front central midline, suggestive to mild intermittent diffuse cerebral dysfunction which is nonspecific. The patient had at least eight arousals for which he pushed the alarm button; all of these were seen in light sleep except one which was shortly after a brief REM sleep. No epileptiform discharges or interictal epileptiform discharges were observed.

Arterial blood gases were obtained in January 2011 (pH 7.44, $PaCO_2$ 42, PaO_2 70, HCO_3 28.5) and December 2011 (pH 7.42, $PaCO_2$ 45, PaO_2 72, HCO_3 29.2). There was uncertainty as to whether an acid-base abnormality is present or if these are just variants of normal.

Many therapies have been attempted so far but have been ineffective. Medications have been tried for gastroesophageal reflux disease (dexlansoprazole, esomeprazole, rabeprazole) and insomnia (zopiclone, quetiapine). Antiemetics trialed include ondansetron, domperidone, diclectin, cesamet; these worsened the patient's nausea. Anti-depressants trialed include trazodone and amitriptyline which made the nausea increase in intensity and last longer, and the nightmares also became more intense.

In 2012, transcutaneous vagal nerve stimulation was attempted before sleep, during sleep and after sleep; no effect was observed with any of these. For several months, the patient's nightmares intensified in the sense that the violence occurring was more often projected toward him than previously, and a trial of prazosin 1mg nightly coincided with a return to the previous intensity. The patient finds that he now has a higher sensitivity to psychoactive medications than before and that this has worsened over the last ten years.

The patient has conducted numerous non-pharmacological self investigations into his illness. These include elimination diets, eating early (nothing after 6pm), eating late (within 1 hour of going to bed), large portion meals, small portion meals, gluten free-diet, caffeine-free diet, changing toothpaste, drinking large volumes of water before bedtime, sleeping on an incline, sleeping on his back, replacing pillows, sheets, and mattress, sleeping with a cervical collar, sleeping in other locations and going to bed early/late. None of these has had noticeable or consistent effect.

Discussion/Conclusion

With respect to the anatomic loci associated with nausea, patient imaging has not identified any lesion. The patient's condition remains stable rather than progressive. Gastrointestinal dysmotility is considered, given impaired gallbladder motility seen on imaging; the link with the patient's nightmares remains unclear. Lastly, there is the possibility that this is a somatic disorder, as a diagnosis of exclusion.

Attempts to diagnose the patient's illness or improve his symptoms have been unsuccessful. The patient wishes to publicize his condition in seeking to identify similar scenarios and effective treatment.

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