again. He denied feeling full and said he wanted more food. Family members noted that he had larger meals and also ate more between meals. There was also evidence of searching for food—getting up in the middle of the night to eat an entire packet of biscuits or sugar—to an extent that food had to be hidden. However, on no occasion was he noted to have inappropriate behavior or hyperorality (examining or touching objects with lips). There was no history of wandering or eating inedible items. It was noted that concomitantly with marked hyperphagia there was progressive weight loss, from 130 pounds to 108 pounds over 3 years. Extensive physical examination and laboratory testing did not reveal any other abnormality.

Comment

Among the behavioral changes reported to occur in dementia are alterations in eating habits. Reduced food intake has been described in 16% to 63% of Alzheimer's disease patients and increased food intake (hyperphagia) has been described in 9% to 26% of Alzheimer's disease cases.[2] In previous studies, excessive eating has been associated with weight gain, and greater frequency of wandering, unpredictable behavior, inappropriate dressing or bodily concerns and threatening self-harm, making it difficult to pinpoint the cause of the hyperphagia.[3] As our patient was cooperative, and had no other behavioral abnormalities or language dysfunction, it was possible to make the important clinical observation that his hyperphagia was characterized by the complete absence of satiety. Meal size and termination is determined by the onset of satiety, a biological state induced by neurohumoral stimuli (such as gastric distension and the gut peptide cholecystokinin released into the circulation after a meal) that leads to meal termination.[4] Recent research points to parasympathetic afferents activated by such stimuli converging on the nucleus tractus solitarius, an area of the caudal brain stem that plays an important role in satiety and meal termination.[5] This hypothesis is supported by the finding that even in the absence of all hypothalamic influences, normal satiety is maintained.[6]

Involvement of brain stem nuclei (locus ceruleus, substantia nigra) has been described in Alzheimer's disease.[7] We speculate that damage to the nucleus tractus solitarius in the brain stem in our patient led to the loss of satiety and consequent hyperphagia. Additional damage to neurons in the cingulate gyrus or lateral hypothalamus may have led to excessive catabolic activity and weight loss in spite of markedly increased food intake. Neuropathological analysis in dementia patients with hyperphagia may further define the neuronal dysfunction underlying this curious behavioral abnormality.

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Topiramate-Induced Palinopsia

To the Editor: Topiramate is a fructopyranose derivative that has proven effective in the treatment of several psychiatric conditions, such as bulimia nervosa,[1] borderline personality disorder,[2] and alcohol abuse.[3] Although its mechanism of action is not fully understood, topiramate is known to enhance γ-aminobutyric acid (GABA) responses, impair AMPA/kainate glutamate receptors, suppress high-frequency action potential firing, and inhibit carbonic anhydrase.[4] Topiramate's most common adverse effects include somnolence, ataxia, dizziness, psychomotor slowing, speech disorders, and paresthesias.[5] We report an unusual visual disturbance in one patient taking topiramate.

Case Report

Ms. A, a 35-year-old obese woman with cyclothymic disorder, bulimia nervosa, and borderline personality disorder, sought psychiatric assessment as a part of an evaluation for intended bariatric surgery. She had a history of intermittent, 1-week episodes of depressed and irritable mood, insomnia, binging and purging behaviors, and increased sexual drive. These symptoms were regularly followed by a few days of increased self-esteem, heightened ac-

tivity, and alcohol abuse. Ms. A had a history of sexual abuse at age 6. She received combined psychotherapy and pharmacotherapy. Soon after an initial trial of topiramate and clonazepam, Ms. A reported an improvement of her insomnia, but, on the fourth day of treatment, developed affective instability and disinhibition. These side effects prompted the discontinuation of clonazepam. To keep her insomnia and other impulsive behaviors under control, topiramate was gradually increased to 100 mg/day.

Soon afterward, Ms. A reported seeing "picture in picture" images, like she was in a "discotheque," or in a place with stroboscopic lights. Those persistent "frozen pictures" faded away after a few seconds. The phenomenon repeated itself with most moving objects. Although this side effect occurred several times during a day, she did not describe the experience as anxiety-provoking and preferred not to withdraw the medication. She had a normal neurological examination and no history of seizures or substance abuse disorders other than alcohol. She was not taking any other medications at the time of the phenomenon herein described.

Palinopsia (Greek *palin*, again, and *opsis*, vision) is the symptom of persistent or recurrent visual images, following removal of the exciting stimulus.[6] There are several potential medical etiologies for palinopsia, including seizures, cerebrovascular diseases, brain neoplasms, and eye or optic nerve disease.[7] The temporal relationship between the occurrence of the palinopsia and the administration of topiramate seen in our patient suggests that this drug might have been the trigger for the observed phenomenon. In fact, there are other recent reports of topiramate-induced palinopsia.[8]

Comment

We can only speculate on the pharmacodynamic mechanisms underlying topiramate-induced palinopsia. Several drugs that have been associated with palinopsia share a 5-HT_2-receptor activity, including LSD,[9] trazodone,[10] nefazodone,[11] risperidone,[12] and, more recently, mirtazapine.[13] Although no systematic report on the serotonergic profile of topiramate has been performed to date, it has been suggested that the weight loss associated with this drug may be ascribed, directly or indirectly (via first or second messenger systems), to an action in the 5-HT_{2C} receptors.[14] Therefore, our patient's visual disturbance could well be another indirect evidence of a putative serotonergic activity associated with topiramate.

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