Rumination Syndrome in a Patient With Hashimoto's Encephalopathy

To the Editor: Hashimoto's encephalopathy (HE) is a rare, autoimmune disease commonly referred to as steroid-responsive encephalopathy associated with thyroid autoimmunity.[1] Clinical presentation is heterogeneous, with majority of cases presenting with neurological (e.g., cognitive impairment, stroke-like events, movement disorders) and psychiatric features. Progressive course, good responsiveness to corticosteroid therapy, and elevated titers of antithyroid antibodies in serum and cerebrospinal fluid (CSF) contribute to the diagnosis. We present a patient with mild cognitive and behavioral impairment associated with marked functional gastroduodenal disorder caused by HE.

Case Report

A 54-year-old, right-handed, highly-educated white woman was admitted to the psychiatric department because of eating problems that, for a year, had been diagnosed and unsuccessfully treated for bulimia nervosa, finally resulting in severe weight loss (25 kg in 2 years; 35% of her premorbid weight). Her problems were characterized by effortless regurgitation of food immediately after consumption, without nausea or abdominal pain preceding it. Also, hyperfamiliarity for unknown people was observed, with otherwise normal social functioning. Her past medical history was positive for hypertension and an episode of hyperthyroidosis in her 30s. Upon admittance to our hospital, the patient received a formal neurocognitive assessment. Except for frequent semantic paraphrasic errors during speech and naming (e.g., pig instead of rhinoceros, squirrel instead of kangaroo, piano instead of harp), the rest of the neurocognitive examination was unremarkable. Her Mini-Mental State Exam (MMSE) score was 30/30. Findings of all performed tests (CBC, regular biochemistry, thyroid hormone, vitamin B_{12}, folic acid, immunology screening, serum and CSF antibodies to syphilis, Borrelia burgdorferi and ACE, tumor markers, paraneoplastic antibodies (anti-Hu, -Yo, and -Ri), hepatitis B and C, HIV, and routine CSF analysis) were unremarkable. Serum and CSF anti-TPO titers were increased (>2,000 and 20 IJ/ml, respectively). Gastric and duodenal biopsy and antitransglutaminase antibodies were unremarkable. Brain MRI revealed a few small subcortical lesions while EEG was unremarkable. Thyroid ultrasound and scintigraphy revealed an enlarged thyroid gland, although thyroid cytology was unremarkable. She was started with 50 mg of prednisolone, with slow tapering to a dose of 10 mg during the next month. At her follow-up visit 4 weeks after, she was free of symptoms, with anti-TPO titer <10 IJ/ml. Six months later, the patient had gained 15 kg of weight, and had normal cognitive status, with an anti-TPO titer of 189 IJ/ml. At that time, the dose of prednisolone was decreased to 5 mg/day with reappearance of problems and increase of anti-TPO titer to 1,550 IJ/ml. Raising the dose of prednisolone to 10 mg again eliminated the symptoms.

Discussion

Rumination syndrome is characterized by the effortless regurgitation of food soon after intake, without nausea or heartburn.[2] It occurs mostly in infants and individuals with cognitive problems, but may also be found in cognitively healthy individuals.[3] Frequently, it is misdiagnosed as bulimia nervosa, but also other functional gastroduodenal disorders, gastroesophageal reflux, or gastroparesis. Intentional vomiting and, seldom, reswallowing, found in patients with bulimia nervosa, are the key distinguishing features between the entities. Weight loss is an additional prominent feature of rumination syndrome. It is still debatable whether it is because of rumination itself or due to depression associated to the syndrome. Although the diagnosis is based purely on clinical criteria (Rome III classification), most of the patients undergo extensive work-up to exclude gastrointestinal disorder.[2] Cognitive dysfunction associated with gastrointestinal disturbances should raise the possibilities of various etiologies (e.g., paraneoplastic syndrome, Whipple disease, celiac disease, mitochondrial disease, etc.).[4]

Cognitive deficits in our patient were detected only on formal neurocognitive examination. In contrast, functional gastroduodenal problems were pronounced, with excellent response to corticosteroid therapy. To the best of our knowledge, this is the first report to describe rumination syndrome in a patient with Hashimoto's encephalopathy.

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