Eating disorders in dementia: binge eating in frontotemporal dementia (FTD)

Michael Kob MD, Laura Valzolgher MD

Hospital of Bolzano, Italy

Introduction

Eating disorders are listed among the behavioral and psychological symptoms of dementia, and are responsible for the high prevalence of malnutrition among these patients, especially in the later stages. However, the change in eating habits in cognitive decline can have various presentations ranging from anorexia, or lack of appetite, to hyperphagia, and overeating including dysphagia and/or selective eating behaviors. The malnutrition that follows is indeed due to undernourishment or overnutrition, and this can be associated with a high level of distress for caregivers.

Neurobiological regulatory mechanism of food intake

Although the mechanism of appetite control and food intake is not yet fully understood, two pathways are believed to be involved: homeostatic and reward pathways. In the first, the hypothalamus regulates food intake by hunger and satiety through caloric and nutritional signals. In the reward pathway food the hypothalamus, several other limbic regions are involved (nucleus accumbens, amygdala, and hippocampus) and other cortical brain regions (orbitofrontal cortex, cingulate gyrus, and insula) are involved. In particular foods high in fat and sugar (e.g. chocolate) can have a rewarding effect provoking food intake despite satiety. On a neurotransmitters level there are two regulatory mechanism of appetite: one stimulating which involves the secretion of ghrelin by an empty stomach and a negative regulatory mechanism which involves the secretion of leptin by adipocites. Leptin is believed to activate oxytocin which decreases food intake.

Eating abnormalities in a particular form of dementia: Frontotemporal Dementia (FTD)

In Frontotemporal Dementia (FTD) eating disorders constitute a core feature of the disease, especially the behavioral variant (bv-FTD), which is the most frequent FTD phenotype. In bv-FTD, eating abnormalities are seen in > 80% of patients, who present with overeating, binge eating and bulimia with significant progression in the 4 years subsequent to disease onset. Distinctive features of bv-FTD patients are gluttony, food cramming, continued eating, cravinsg for certain foods (especially sweets), sometimes obsession for candies, and sometimes food stealing. Eating abnormalities in this variant can be overwhelming and very problematic for caregivers. In the other less frequent variants of FTD, which are the nonfluent/agrammatic primary progressive aphasia (nfvPPA) and the semantic variant (svPPA), eating abnormalities tend to be less frequent. In the semantic variant patients tend to be more selective in food choices and sometimes eat non-edible foods.

From a pathogenetic point of view, neuroimaging and postmortem studies support the hypothesis that an early feature of FTD-pathology, which is atrophy in the posterior area of hypothalamus, could also be responsible for eating disorders. In laboratory studies overeating can occur despite satiety and is associated with damage in the right-sided orbitofrontal-insular-striatal circuit. The hypothesis is that lesions in the striatum could impair the response to satiety.

Management of eating disorders in FTD constitutes a real challenge. First of all, patients require a thorough assessment of severity and frequency using the NPI scale for neuropsychiatric symptoms (in which 1 of the 12 categories assesses appetite and eating habits). Conventional approaches like caregiver counseling or assistance during meals are generally insufficient and FTD patients often require a pharmacological treatment. As therapeutic option selective serotonin reuptake inhibitors (SSRI) like fluoxetine, sertraline, paroxetine, fluvoxamine and citalopram have been used to treat dishinibition, sugar craving and exessive eating in bv-FTD with moderate improvement. From a physiological perspective an interesting report describes the use of Topiramate for overeating in FTD, the same drug used for binge eating and bulimia in non-demented patients with eating disorders. Topiramate is a sulfamate-substituted monosaccharide anticonvulsant that is associated with anorexia and weight loss; its use is reported in a patient with frontotemporal dementia (bv-FTD) with abnormal eating behavior who seemed to respond to treatment. While the exact mechanism of Topiramate in binge eating disorder and bulimia is not known, Topiramate acting as an antagonist of glutaminergic receptor alpha amino-3-hydroxy-5-methylisoxozole-4propionicacid (AMPA) can reduce reward seeking behavior in animal models of substance dependance.

Conclusions

Eating disorders are frequent neuropsychological symptoms of dementia which can be assocaited with varying degrees of malnutrition. In the behavioral variant of FTD eating disorders can be severe enough to require pharmacological treatment. Some authors have suggested that the type of eating disorder could address the differential diagnosis between frontotemporal dementia (FTD) and Alzheimer disease (AD), with anorexia and hyporexia more frequent in the latter, and overeating typical of FTD.

References

Cipriani G, Carlesi C, Lucetti C, Danti S, Nuti A. Eating Behaviours and Dietary Changes in Patients With Dementia. Am J Alzheimers Dis Other Demen. 2016 Dec 1; 31 (8):706-16. doi: 10.1177/1533317516673155. 10.1177/1533317516673155.

Singam C, Walterfang M, Mocellin R, Evans A, Velakoulis D. Topiramate for abnormal eating behaviour in frontotemporal dementia. Behav Neurol. 2013 Jan 1;27(3):285-6. doi: 10.3233/BEN-120257. PMID: 23548883; PMCID: PMC5215572.

Woolley JD, Gorno-Tempini ML, Seeley WW, Rankin K, Lee SS, Matthews BR, Miller BL. Binge eating is associated with right orbitofrontal-insular-striatal atrophy in frontotemporal dementia. Neurology. 2007 Oct 2;69(14):1424-33. doi: 10.1212/01.wnl.0000277461.06713.23. PMID: 17909155.

Key highlights:

-eating disorders are part of the neuropsychological symptoms of dementia, they have presentation according to the type of dementia and increasing frequency in the later course of the disease

-the neurobiology of food intake regulation is believed to have 2 pathways: a homeostatic and a hedonistic mechanism involving different brain regions: hypothalamus, limbic system, cortical regions

-in frontotemporal dementia neurodegeneration in these areas could impair the signaling to the hypothalamus and the response to satiety

-in frontotemporal dementia, eating disorders such as binge eating and bulimia, are distinctive features of the disease (especially in the behavioral variant) that are associated with important burden and require a pharmacological approach