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Eating Behaviors and Dietary Changes in Patients With Dementia

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Abstract

Background: Eating problems and dietary changes have been reported in patients with dementia. **Objectives:** The aim of this article is to explore the generalized problems with nutrition, diet, feeding, and eating reported among patients with dementia. **Methods:** Medline and Google Scholar searches were conducted for relevant articles, chapters, and books published before 2016. Search terms used included behavioral and psychological symptoms of dementia, dementia, dietary changes, eating behavior. Publications found through this indexed search were reviewed for further relevant references. **Results:** Abnormal eating behaviors, eating problems, and dietary changes are present in most people with dementia, especially in the later stages of the condition. **Conclusion:** Individuals with dementia frequently develop serious feeding difficulties and changes in eating and dietary habits. The changes may be secondary to cognitive impairment or apraxia, or the result of insufficient caregiving, or the consequence of metabolic or neurochemical abnormalities occurring as part of the dementing process.

Keywords

behavioral and psychological symptoms of dementia, dementia, dietary changes, eating behavior

Introduction

Dementia should be conceptualized as a multifactorial process.[1] A wide range of behavioral changes, sometimes bizarre, have been reported in patients with dementia, including delusions,[2] misidentification syndromes,[3] mood changes,[4] wandering,[5] aggressive behavior,[6] and sleep disorders.[7] Among these changes are reported alterations in eating habits. Eating behavior among older adults can be seen as a complex individualistic phenomenon that is determined by physiological, pathological, and psychological components. Malnutrition or undernourishment resulting from insufficient food intake is reported in up to 85% of nursing home residents,[8] and dehydration has been documented in as many as 51% of residents.[9] Changes in dietary or eating behavior may not be evident on initial presentation of patients with dementia, whereas patients with advanced illness may resist or be indifferent to food, fail to manage the food bolus properly once it is in the mouth (oral phase dysphagia), or aspirate when swallowing (pharyngeal phase dysphagia). Such behavioral troubles are perceived as a heavy burden by family caregivers who may be stressed, depressed, and socially isolated. "Appetite" and "eating abnormalities" are assessed as one of the 12 categories in the Neuropsychiatric Inventory (NPI) scale of severity and frequency of behavioral and psychological symptoms of dementia (BPSD)[10] emphasizing the importance of these clinical features. The aim of this article is to explore the generalized problems with nutrition, diet, feeding, and eating reported among patients with dementia.

Methods

Systematic research using PubMed to search the MEDLINE database on changes in eating habits and appetite in patients with dementia was carried out (only upper time limit: January 2016). We found 142 articles using the terms "behavioral and psychological symptoms of dementia" and "vascular dementia," 36 articles using the terms "dietary changes" and "vascular dementia," 45 articles using the terms "eating behavior" and "vascular dementia," 78 articles using the terms "behavioral and psychological symptoms of dementia" and "frontotemporal dementia," 12 articles using the terms "dietary changes" and "frontotemporal dementia," 45 articles using the terms "eating behavior" and "frontotemporal dementia," 63 articles using the terms "behavioral and psychological symptoms of dementia" and "dementia with Lewy bodies," 2 articles using the terms "dietary changes" and "dementia with Lewy bodies," 9 articles using the terms "eating behavior" and "dementia with Lewy bodies," 131 articles using the terms "behavioral and psychological symptoms of dementia" and "Huntington's disease," 39 articles using the terms "dietary changes" and "Huntington's disease," 4 articles using the

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terms ''eating behavior'' and ''Huntington's disease,'' 14 articles using the terms ''behavioral and psychological symptoms of dementia'' and ''Parkinson disease associated with dementia,'' 4 articles using the terms ''dietary changes'' and ''Parkinson disease associated with dementia,'' 9 articles using the terms ''eating behavior'' and ''Parkinson disease associated with dementia,'' 3 articles using the terms ''behavioral and psychological symptoms of dementia'' and ''Progressive supranuclear palsy,'' no article using the terms ''dietary changes'' and ''Progressive supranuclear palsy,'' and 9 articles using the terms ''eating behavior'' and ''Progressive supranuclear palsy.'' Additional records were identified through other sources (ie, Google Scholar and key journals); moreover, bibliographies of selected manuscript of interest were handsearched. We manually screened the reference list of pertinent studies. Articles were excluded if they were not written in English, if no abstract was electronically available, if they were nonhuman studies, and if they did not deal with eating behavior or appetite in patients with dementia.

Neurobiology of Food Intake

From a theoretical standpoint, the brain mechanisms underlying appetite control and food preference are poorly understood. Food intake is regulated by 2 complementary drives—the homeostatic and hedonic pathways. The homeostatic control of food intake depends on long-loop[11] mechanisms involving peripheral humoral and neural signals that are processed in the hypothalamus. The hypothalamus is recognized as the main brain region regulating food intake as it relates to caloric and nutrition requirements; however, evidence is accumulating that brain circuits other than those regulating hunger and satiety are involved in food consumption and obesity. Specifically, several limbic (nucleus accumbens, amygdala, and hippocampus) and cortical brain regions (orbitofrontal cortex [OFC], cingulate gyrus, and insula) and neurotransmitter systems (dopamine, serotonin, opioids, and cannabinoids) as well as the hypothalamus are implicated in the rewarding effects of food.[12] Food intake is motivated not only by the need to restore energy homeostasis; palatable, rewarding high fat and/or sugar foods such as chocolate can motivate intake despite a state of satiety. Nucleus accumbens integrates the homeostatic, hedonic, motivational, and cognitive aspects of food intake via its connections with the prefrontal cortex, amygdala, and lateral hypothalamus.[11] Functional neuroimaging supports the interaction between homeostatic and hedonic control of food intake in humans.[13] Two appetite-modulating pathways are present—an appetite stimulating pathway, which starts with an empty stomach secreting the hormone ghrelin to target neurons of the arcuate nucleus of the hypothalamus that contain neuropeptide Y and agouti-related protein (AGRP), and an appetite-suppressing pathway that starts with adipocytes secreting the hormone leptin to target arcuate neurons containing pro-opiomelanocortin and the cocaine- and amphetamine-regulated transcript.[14] Oxytocin, which is produced in the hypothalamus, is an important component of the pathways activated by leptin and is believed to decrease food intake.[15] The neurochemical or neuropathological changes that occur in dementia result in a disruption of the central control of food intake and weight.

Results

The process of screening yielded to 89 articles to be considered in our manuscript. Due to the different neuropathological processes and clinical manifestations of dementia, appetite, and behavioral alterations will be described in each type of dementia separately, after a clustered overview.

Appetite/Eating Abnormalities in Dementia

Data from epidemiological studies suggest that older adults with dementia demonstrate problematic eating behaviors.[16] Changes in eating in dementia include a decrease or increase in amount eaten (with or without weight changes), a change in eating habits (for instance, a change in the frequency of eating, playing with food, and improper or no use of utensils), a change in food choice (most commonly a predilection toward sweet foods), and pica (persistent ingestion of nonnutritive substances).[17,18] Patients with dementia may present difficulties in recognizing that the material in front of one is food, planning of what should be transported to the mouth and in what quantity and by what means, and actual execution of that plan. A peculiar change is represented by hyperphagia that can be a serious aspect for several reasons, for example, weight gain, eating dangerous foods, and continual searching for food.[19] Without caregiver intervention, people with dementia who develop hyperphagia will continue eating until they seem physically uncomfortable and present with clinical problems. Overeating is associated with other eating changes such as changed food choice and eating inappropriate food or nonfood items.[19] Woolley et al[20] suggested that damage to a right orbitofrontal-insular-striatal circuit is associated with overeating behavior in humans and support the hypothesis that these regions integrate internal satiety signals with environmental food cues to produce adaptive eating behavior. These changes can be a serious source of stress for a carer and may precipitate the need for entering an institution.[21,22] On the other hand, various researchers have investigated links between food intake and weight loss. The causes of weight loss in this population are multifaceted and include loss of appetite secondary to the deterioration of brain regions associated with feeding behavior and functional and behavioral problems associated with dementia that make it difficult for individuals to consume adequate energy.[23] However, there is ambiguity in the link between food intake and weight loss and whether or not weight loss precedes the onset of dementia or vice versa. A logistic regression analysis showed a positive association between aversive feeding behaviors (''refusal to eat,'' ''resistive,'' and ''dysphagia'') worsening and the initial caregiver's burden after controlling for confounding factors.[24] Hsiao et al[25] aimed to explore family caregivers' experience of the problematic eating behaviors among community-dwelling

older adults with dementia. The caregivers indicated that ''forgetting'' was the first and most common eating characteristic exhibited by the patients, which contributed to overeating. However, little is known about which factors are related to eating behavior disorders and help predict the evolution of eating behavior disorders. In the study conducted by Rivière et al,[24] the authors determined that the severity of the disease and the emotional and material family burden are 2 predictors of aversive eating behaviors during Alzheimer's disease (AD).

We try to provide insights into the structure of the aforementioned behavioral disturbances in different types of dementia (Table 1).

Alzheimer's Disease

Clinical phenomenology. Alzheimer's disease is the most common causes of neurodegenerative disorder in the elderly individuals. The most distinctive ''hallmark'' lesions present within the diseased brain are the senile plaques and neurofibrillary tangles. The diagnosis of AD requires clinical evidence of memory loss and impairment of at least 1 other cognitive domain, with evidence of disturbance in social or occupational function. The impact of behavioral symptoms associated with AD is substantial.

The original description of AD, as reported by Alois Alzheimer in 1906, included malnutrition as a feature of the disease.[26] Patients with AD commonly develop difficulty in eating, especially when they become dependent in all activities of daily living. As a consequence, weight loss has been reported in many studies.[27-32] Weight loss is always a great concern for the clinical practitioner because it is an indicator of protein–energy malnutrition in the elderly patients and a predictive factor of mortality,[31,33] of morbidity,[34] and poor quality of life.[35] Day et al[36] found that individuals who lost more than 10% of their body weight between age 70 and 75 years had significantly higher mortality risk during the following 5 years compared to individuals who were relatively stable in their body weight and lost less than 5%. It is not confined to patients with severe disease who are unable or unwilling to eat, but may start earlier in the course of the disease[37-39]—a longitudinal study, involving community-dwelling elderly patients, found that participants with incident dementia or mild cognitive impairment had accelerated weight loss from as early as 6 years before diagnosis of AD.[40] Weight loss was shown to be accompanied by a greater deterioration in those aspects of dementia explored by the Mini-Mental State Examination.[41] Indeed, among patients with dementia, impaired cognition leads to a progressive loss of learned behaviors including the ability to feed oneself.[42] Problems with passivity, distraction, and refusal to eat are encountered in the moderate phase of patients with AD, whereas distraction, passivity, and inappropriate feeding velocity are predominant in more severe phases.[43] Patients with advanced dementia may display refusal of food or resist or be indifferent to food, fail to manage the food bolus properly once it is in the mouth or aspirate when swallowing. A study showed that difficulty in beginning a meal was an even stronger risk factor for hindrance of eating independence than worsening of the severity of dementia in patients with AD.[44,45] However, different nutritional states seem also to be in partnership with the demonstration of different behavioral symptoms and with a different caregiver burden.[44] Studies of AD have found sweet food preference in 5% to 39% of cases.[16,28,46,47] Weight gain due to hyperphagia has also been documented in patients with AD—hyperphagia have been reported to occur in 10% to 36% of these patients.[19,47-49]

Neurochemical and pathological correlates. Tsang et al[50] found significant decreases in 5-HT4 receptor densities in the hyperphagic, but not normophagic, patients with AD. Their data suggest that 5-HT4 receptor deficits may be a specific neurochemical correlate of hyperphagia, and point to the potential pharmacotherapeutic utility of 5-HT4 agonists for these behaviors in patients with AD. Pathological changes in the hypothalamus have been described in patients with AD, and this pathology may affect centers regulating food intake and metabolic processes.[51]

Frontotemporal Dementia

Clinical phenomenology. Frontotemporal dementia (FTD) is the term now preferred over Pick disease to describe the spectrum of non-Alzheimer's dementias characterized by focal atrophy of the frontal and anterior temporal regions of the brain. It is both clinically and pathologically heterogeneous, however, 3 main syndromic variants of FTD are generally recognized based on the most prominent clinical features at presentation—behavioral-variant FTD (bvFTD), clinically characterized by progressive behavioral changes and social interpersonal dysfunctions; semantic dementia (SD), presenting as an impairment of semantic memory; and progressive nonfluent aphasia, presenting with predominant speech production difficulties. The behavioral variant is the most common phenotype of FTD, accounting for over 50% of all cases with FTD. A wide range of behavioral changes has been reported in fv-FTD, including loss of insight, progressive impairment of decision-making, disinhibition, impulsivity, poor self-care,[52] mood changes, mental rigidity, stereotypic behaviour,[53] and apathy.[54] According to Piguet et al,[55] in patients with FTD, abnormal eating behavior is a particularly attractive target. Eating abnormalities are likely an instance of a more general pattern of behavioral disinhibition in patients with bvFTD, which is manifested as inappropriate social behavior, environmental dependency, and substance abuse.[56] More than 80% of patients with bvFTD develop feeding abnormalities during the course of their illness.[55] Eating disturbances are also observed to a lesser degree in another subtype of FTD, SD. Dramatic changes in diet may occur with high frequency at the onset of FTD and may steadily get worse as the disease progresses. In an epidemiologic study of patients with bvFTD, eating disorders, including overeating, bulimia, and food fads, were among the most common symptoms that progressed within a 4-year period.[57] Patients with FTD develop gluttony, sweets and

Table 1. Different Types of Dietary Change and Oropharyngeal Phase Alteration in Different Types of Dementia.

Types of Dementia	Dietary Intake	Oropharyngeal Phase
AD	Decrease • Apathy • Depression • Memory loss (forget to eat) • Inappropriate feeding • Diet simplification • Food refusal • Indifference to the food Increase (rare) • Memory loss • Hyperphagia (frontal variant of AD)	• Oral apraxia • Prolonged eating duration • Loss of food from the oral cavity • Difficulty chewing • Absent or continuous chewing • Multiple swallows • Reduced hypolaryngeal elevation • Silent aspiration
bvFTD	Decrease • Apathy • Loss of interests • Rigidity in eating (same food for every meal) Increase • Hyperphagia • Sweets and carbohydrate craving • Obsession with particular food • Snatching food from others • Attempting to eat inedible object	• Behavioral feeding problems (rapid eating) • Prolonged eating duration • Difficulty with bolus formation and mastication • Pharyngeal phase dysphagia • Incomplete bolus clearance with residue in pharynx • Decrease hypolaryngeal elevation • Silent aspiration
VD	Decrease • Problems related to handling food on the plate and transporting food to the mouth • Slow eating	
LBD	Autonomic dysfunction	
PDD	Decrease • Apathy • Slow eating • Difficulty in biting • Festinating type motion of tongue Increase • Preference for sweet food • Impulse control disorder (binge eating disorder)	• Difficulties with bolus manipulation control • Prolonged hold of bolus in oral cavity • Xerostomia • Abnormal airway somatosensorial function • Premature loss of bolus into pharynx • Slow bolus transfer from oral to pharyngeal cavity • Delay in initiation of pharyngeal swallow • Residue in in valleculae and pharynx • Decrease upper esophageal opening and relaxation • Silent aspiration • Aspiration
PSP	Decrease • Apathy • Slow eating • Apraxia Increase • Impulse control disorder (binge eating disorder)	
HD	Decrease • Reduce voluntary motor control • Involuntary movements • Hypokinetic state	• Poor lip closure and bolus manipulation • Difficulty with mastication • Decrease palatal elevation • Delayed initiation of pharyngeal swallow • Repetitive swallows • Pharyngeal residue • Decrease pharyngeal peristalsis • Aspiration

Abbreviations: AD, Alzheimer's Disease; bvFTD, behavioral variant of frontotemporal dementia; HD, Huntington disease; LBD, Lewy body disease; PDD, Parkinson disease with dementia; PSP, progressive supranuclear palsy; VD, vascular dementia.

carbohydrate craving, increased weight, obsessions with particular foods, and occasional alcoholism.[58] According to some authors,[59] change in food preference is a feature useful to distinguish frontal and temporal variants of FTD from AD. It is linked to ventral frontal pathology. Bozeat et al[60] include, between abnormal eating behaviors, snatching food from

others, attempting to eat inedible objects, or rigidity in eating behaviors (eg, refusal to try different foods and insisting on favorite foods for every meal). Snowden et al[61] performed a study to test predictions that bvFTD and SD give rise to distinct patterns of behavioral change. Altered food preference was in all groups almost invariably in the direction of increased preference for sweet foods, whereas oral exploration of inanimate objects was rare in all groups. However, the nature of the change differed. Gluttony, food cramming, indiscriminate eating, and continued eating while food was present were characterized in patients with bvFTD, whereas increased selectivity and food fads were characterized in patients with SD. In SD, the only commonly endorsed symptom is a tendency to eat nonedible things. It is important to note that patients with bvFTD and SD mainly experience an increase in appetite, whereas those with AD mainly show loss of appetite. According to Ikeda et al, significant weight gain occur in more than 30% of patient groups with FTD but less than 10% in those patients with AD.[47] This observation was found also in a study conducted by Mendez et al.[62]

Neuroimaging findings. A limited number of studies have investigated the neural structures responsible for eating changes in patients with bvFTD. The dissociation between appetite increase and mouthing inedible things suggests that the 2 may involve different underlying mechanisms.[60] Piguet et al[14] demonstrated that patients with bvFTD exhibit significant atrophy of the hypothalamus. The authors, using structural neuroimaging, showed that this atrophy is an early feature of the disease, being already present within 2 years of diagnosis. Their analyses further revealed that this atrophy is most pronounced posteriorly, a region containing nuclei that play a critical role in regulating feeding behavior. Combined with the postmortem investigations, their findings indicate that eating disturbance is related to neuronal loss in the posterior hypothalamus and is greater in patients with Tar DNA protein-43[20,63,64] pathology. Previous structural neuroimaging studies have highlighted that disturbance in an orbitofrontal-insular-striatal brain network underlies the emergence of eating disturbance in patients with FTD. Disease in the orbitofrontal region may impair the ability to refrain from taking food or to respond to feelings of satiety.[65] A voxel-based morphometric magnetic resonance imaging study[20] showed that pathological sweet tooth is associated with atrophy involving a distributed network of brain areas, including posterolateral OFC and lateral prefrontal cortex, anterior insula, inferior temporal lobe and temporal poles, and caudate nucleus, more extensive in the right cerebral hemisphere. In the study, hyperphagia was associated with a more restricted pattern of atrophy involving more anteromedially located OFC regions bilaterally. According to Ahmed et al,[66] caloric intake is only increased in the patients with bvFTD, whereas an increased preference for the sweetest dessert was observed in patients with both bvFTD and SD. The authors found that increased caloric intake in patients with bvFTD correlates with atrophy of bilateral anterior and posterior cingulate gyri, the thalamus, bilateral lateral occipital cortex, lingual gyri, and the right cerebellum.

Neurochemical correlates. There are profound changes in cortical serotonin levels in patients with FTD, which almost certainly contribute to the alterations in satiety and food preference.[67] It has been suggested that eating disorder in bvFTD, typically hyperphagia, relates to high concentrations of AGRP, whereas leptin elevations are responses to high body mass index (BMI).[68]

Dementia With Lewy Bodies

Clinical phenomenology in eating behaviors and dietary changes. Dementia with Lewy bodies (DLB) is a progressive, degenerative dementing disorder of unknown etiology, clinically characterized by marked fluctuations in cognitive function with varying levels of alertness and attention, visual hallucinations, extrapyramidal signs, and sensitivity to typical neuroleptics. Eating and swallowing difficulties are described as a part of autonomic dysfunction in supportive diagnostic features.[69] Swallowing depends on the complex sensory-motor mechanism regulated by the central nervous system, which includes voluntary and involuntary components. Patients with DLB have more impairment in their abilities to eat appropriately than those with AD with similar levels of global cognitive functioning.[63] Additional impairments and functional disability are mainly attributable to extrapyramidal motor symptoms. However, to our knowledge, only a few systematic studies have investigated eating/swallowing problems in patients with DLB.[70,71] In order to assess the characteristics of eating/swallowing problems in patients with DLB, Shinagawa et al[71] used a comprehensive questionnaire that comprised 40 items investigating the following 5 domains—swallowing problems, appetite change, food preferences, eating habits, and other oral behaviors. As expected, most of the problems were involved in the swallowing domain—"difficulty in swallowing foods," "difficulty in swallowing liquids," "coughing or choking when swallowing," "taking a long time to swallow," "suffering from sputum," along with some problems in other domains such as "loss of appetite," "needs watching or help," and "constipation." Except for "needs watching or help," no problems in the eating habits domain and food preference domain were noted.

Parkinson Disease With Dementia

When James Parkinson wrote his classical essay on the disease that bears his name, he described eating problems as one of the symptoms disabling the patient.[72] Parkinson disease (PD) is the second most common neurodegenerative disorder, with the prevalence estimated to be 1% of the population older than 60 years.[73] The pathologic mechanism of PD is a gradual neuronal degeneration involving the substantia nigra in the midbrain, which produces dopamine. With disease progression, nondopaminergic nuclei, such as the locus coeruleus, the nucleus basalis of Meynert, and the dorsal raphe, are affected.

The key clinical features of a decrease in dopamine include bradykinesia, resting tremor, rigidity, and impaired gait. It has become increasingly apparent that patients with PD can have impairment of certain cognitive functions and develop dementia (Parkinson disease with dementia, PDD). The typical patient with PDD has characteristic clinical features, which can be best summarized as a dysexecutive syndrome, with prominent impairment of attention, visual–spatial dysfunction, moderately impaired memory, and accompanying behavioral symptoms such as apathy[54] that can be implicated in the regulation of eating.

Clinical phenomenology in eating behaviors and dietary changes. The observed aberrant eating behavior in patients with PDD is the consequence of a complicated interaction among the patients' disease, drugs, and the actual environment, and it is not possible to isolate completely the effect of PD from the effect of dementia.[74] For what concerns eating habits, a preference for sweet food such as, for instance, cakes,[75] chocolate,[76] ice cream,[77] and carbohydrates,[78] has been documented. It has been suggested that carbohydrates and sweets, through insulin, may increase brain dopamine as a sort of compensatory mechanisms for dopamine loss associated with the disease.[79] During the meal, patients with PD show a slow eating tempo, they handle food on the plate, and have difficulty in biting off pieces of food.[74] Logemann[80] observed that the repetitive "rocking and rolling" festinating-type motion of the tongue during oral transfer is frequently evident in patients with PD. In these patients, an association between decreased cognition and weight loss or malnutrition has been reported. It may be caused by dysphagia that can be a result from dysmotility of the mouth, pharynx, and esophagus as well as sialorrhea and dry mouth. Advancing age is a risk factor for weight loss, but in the late phase of the illness, the age effect was probably surpassed by the motor and cognitive dysfunction due to the advanced widespread neurodegeneration of PD.[81] Kim et al[82] performed a study to explore the relationship between BMI changes and rate of cognitive decline in patients with PD. The decreased BMI group showed poorer cognitive function and faster rate of cognitive decline than the stable BMI group. Although effective for motor symptoms, dopamine replacement therapy is associated not only with motor side effects, such as levodopa-induced dyskinesia, but also behavioral side effects such as impulse control disorders. Impulsive and compulsive behaviors, such as binge eating, are being increasingly recognized in patients affected by PD. Binge eating disorder is defined as recurring episodes of eating significantly more food in a short period of time than most people would eat under similar circumstances, with episodes marked by feelings of lack of control.[83]

No milk today? Dairy foods have been linked to PD, and a meta-analysis of prospective cohort studies on dairy foods intake and PD risk was conducted.[84] The combined risk of PD for the highest versus the lowest level of dairy foods intake was 1.45 (1.23-1.73) for milk, 1.26 (0.99-1.60) for cheese, 0.95 (0.76-1.20) for yogurt, and 0.76 (0.511.13) for butter. A prospective study conducted in 2005 showed that drinking milk may raise the risk of PD in middle-aged men. As milk is a complex mixture of nutrients, any of its nutritional constituents could act as candidate mediators in the association between milk and PD. Whether observed effects are mediated through nutrients other than calcium or through neurotoxic contaminants warrants further study.[85] A team of American scientists, collected information from 73 175 women and 57 689 men and found that those consuming milk more frequently had a 60% greater chance of acquiring PD. A higher risk among dairy product consumers was found in both men (+80%) and women (+30%), but the association in women was nonlinear.[86] To date, the explanation for the epidemiological link between dairy products and PD remains unknown.

Progressive Supranuclear Palsy

Clinical phenomenology in eating behaviors and dietary changes. Progressive supranuclear palsy (PSP) is an atypical parkinsonian syndrome (Parkinson-plus disorder) typically characterized by progressive, early-onset postural instability, frequent (unexplained) falls, problems with eye movement, axial (involving neck or trunk) rigidity, speech/swallow difficulty, and cognitive decline.[87] Significant abnormalities are noted during the oral phase of swallowing in patients with PSP. These included slow, inadequate mastication, and delayed swallow initiation.[88] Binge eating has been described in cases of pathologically proven PSP.[89] Erro et al[90] observed 4 patients with PSP showing an eating disorder that they labeled "greed for food." The authors do not consider greed for food to be part of bulimia, but as a distinctive eating disorder, which deals more with eating "voracity" rather than with the amount of food eaten. Vidal et al[91] found that persons suffering from PSP ate meat and poultry more often and fruits less frequently than controls. To our knowledge, no previous study has been published on dietary habits in patients with PSP.

Huntington Disease

Clinical phenomenology in eating behaviors and dietary changes. Huntington disease (HD) is a fatal, autosomal dominant, neurodegenerative disease caused by expansion of a cytosine–adenine–guanosine trinucleotide repeat in the IT15 gene, codifying for huntingtin protein that leads to marked atrophy of basal ganglia structures, the caudate and putamen, as well as less marked atrophy of other brain nuclei. Classically, it is characterized by a triad of symptoms and signs, including a movement disorder, psychiatric syndromes, and dementia. Many symptoms of HD can interfere with eating—reduced voluntary motor control, involuntary movements, problems with chewing and choking, as well as changes to cognition that may cause the individual to become distracted and overwhelmed by mealtime activity. Marconi et al[92] described the case of a young woman with HD in which the eating disorder (characterized by fear of weight gain, frequent binge eating, compensatory behaviors,

and an abnormally low body weight without amenorrhea) at onset preceded motor symptoms by 6 years. Although no evidence of malabsorption has yet been found, a high proportion of patients at the later stages of the disease were reportedly malnourished despite receiving the same food intake as controls.[93] There is no compelling evidence to support the hypothesis that patients lose weight as their choreiform movements become more pronounced.[94] Weight loss is the most prominent in the final hypokinetic stages of the disease,[95] suggesting that it is an insidious event rather than secondary to hyperactivity.

Pathological correlates. In light of nonmotor symptoms and signs of disturbed functions regulated by the hypothalamus in patients with HD, it has been hypothesized that this region may also be affected by the disease, and hypothalamic atrophy was found in patients with HD by neuropathological analysis.[96]

Neuroimaging findings. Magnetic resonance imaging, positron emission tomography, and clinical data support a hypothalamic involvement in patients with early HD and premanifest HD gene carriers. Politis et al[97] demonstrated, for the first time, significant D2 receptor loss and microglia activation in the hypothalamus of HD. These changes occur very early in the course of the disease and may partly explain the development of commonly reported symptoms including progressive weight loss.

Vascular Dementia

Clinical phenomenology. Vascular dementia (VaD) is a term used to describe a particular constellation of cognitive and functional impairment. Several vascular pathologies can lead to dementia. It may be the consequences of ischemic, hemorrhagic, or ipossic lesions of the brain. Eating difficulties are a common consequence following stroke. Several aspects of eating difficulties have been identified, for example, problems related to handling food on the plate, transporting food to the mouth, chewing, swallowing, and hoarding food in the mouth. Eating difficulties can also be related to lack of energy and aberrant eating speed.[98] Swallowing function can be severely impaired in patients with VaD that show deficits in bolus formation and mastication[99] with increased risk of aspiration pneumonia.[100]

Regarding the preferences for different foods, patients with VaD show preference for relatively high-fat, sweet foods and for high-sugar, low-fat foods.[46]

Assessment and Management

Assessment. Food and care is thus a complex area. It is useful to consider problems with eating or swallowing as a clinical syndrome that triggers a medical assessment and workup. A clear understanding of the patient's functional status, stage of dementia, and baseline eating status should emerge and is essential in assessing current eating problems. Descriptions of the patient's baseline consistencies, food preferences including ethnic food, degree of independence in eating, and size and frequency of meals during the day are also essential.[101] These aspects are, typically, best captured with caregiver questionnaires since a loss of insight results in an underestimation of the presence and severity of the behaviors by the patient. Clinical assessment of individuals with eating abnormalities must always include screening for secondary causes, including medical and psychiatric conditions.

The NPI[10,102] was developed to provide a means for assessing neuropsychiatric symptoms and psychopathology of patients with AD and other neurodegenerative disorders. Appetite and eating abnormalities are assessed as 1 of the 12 categories in the NPI scale of severity and frequencies of BPSD. The Eating Behavior Scale measures the functional ability during eating in patients with AD.[103,104] The 6 items include "initiates eating," "maintains attention to meal," "locates all food," "uses utensils appropriately," "bites, chews, and swallows without choking," and "terminates meal." Ikeda et al[47] developed a questionnaire, the Appetite and Eating Habits Questionnaire, by providing an exhaustive measure of change in eating behaviors. This instrument consists of 36 questions investigating the following 5 domains—swallowing problems, appetite change, food preference (including sweet food preference and food fads), eating habits (including stereotypic eating behaviors and decline in table manners), and other oral behaviors (including food cramming and indiscriminate eating). For each abnormal behavior, the authors derived a product of frequency times severity in keeping with the method applied in the NPI.[10]

Management. As with many areas of intimate care, such as bathing and attending to toilet needs, there is relatively little research on individualized interventions about eating in persons with dementia, particularly when it is carried out at home.[105] Some of the conventional intervention methods include caregiver assistance and the use of finger foods, texture modification, and supplementation. Both food and fluid intake could follow a schedule, which ensures that eating and drinking are not forgotten.

Maintaining eating independence in patients with dementia can be facilitated by identifying factors that interfere with beginning a meal by providing multidisciplinary care, eliminating environmental factors, and providing assistance that promotes beginning a meal.[43] Within these broad environmental changes are suggestions that meals may need to be familiar, attractive, and enticing.[105] Interventions in the form of physical assistance emphasize the movement inherent in helping people to eat, movements of food to mouth, and chewing and swallowing. A less direct form of intervention is monitoring and supervision of eating and mealtimes. Caregiver assistance can vary from simply sitting and chatting with the patient during mealtimes to give specific instructions and encouragement.[23] During early stages of apraxia, serving finger foods such as hot dogs, sandwiches, and so on can make food consumption easier for patients. Treatment of eating difficulties in patients with dementia is aimed at optimizing oral intake as safely as

possible in order to preserve the pleasure of tasting and eating, while attempting to counteract malnutrition, dehydration, and aspiration to pneumonia for as long as possible.[106] Food refusal may be a symptom of depression and may respond to antidepressant treatment. The selective serotonin reuptake inhibitors fluoxetine, sertraline, paroxetine, fluvoxamine, and citalopram have all been tested to treat the behavioral symptoms of FTD (including disinhibition, depressive symptoms, carbohydrate craving, and compulsions).[107] After treatment, all these symptoms showed improvement. Topiramate is a sulfamate-substituted monosaccharide anticonvulsant that has been associated with anorexia and weight loss and been found to be of benefit in binge eating disorder[108] and bulimia nervosa.[109] A report[110] highlights a virtual resolution of severe food and alcohol binging in a patient with FTD using low-dose topiramate (50 mg twice daily). Another report[111] describes a man diagnosed with bvFTD who developed a ''sweet tooth,'' chewing 10 packets of gum per day, eating sweets to excess, and drinking liters of soft drink. Topiramate was commenced at 25 mg twice daily and the dose titrated to 200 mg twice daily but reduced to 100 mg twice daily due to nausea, and an improvement was noted in his eating behaviors within 3 weeks of the commencement of topiramate.

Conclusion

In conclusion, eating problems and dietary changes occur frequently in association with cognitive dysfunction, especially in patients with dementia in the later stages of the condition. A number of possible mechanisms have been proposed to account for changes—they may be secondary to cognitive impairment or apraxia, or the consequence of autonomic dysregulation and neuropathological and neurochemical abnormalities occurring as part of the dementing process. However, as cortical function deteriorates, with the progression of dementia, the patient's ability to obtain adequate nutrition decreases. Gluttony or hyperphagia may be one of the most discriminating clinical aspects of patients with FTD. In contrast, swallowing problems and anorexia with weight loss is more likely in patients with AD than in patients with FTD. From a clinical viewpoint, an increased awareness of specific eating behavior abnormalities may encourage early intervention and accurate therapeutic treatments.

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