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Aspiration and Swallowing in Parkinson's Disease :Two Hundred Years Later

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Abstract

In this mini-review the developments in the reesarch and treatment of swallowig disorders and the subsequent impairments in cough function in Parkinson's disease patients are discussed. Therapies for drooling and dysphagia are discussed in a disease existing this year for exactly two hundred years. Future directions to a more functional than pathogen directed approach to aspiration pneumonia, still the main cause of death in Parkinson's patients are discussed. Breaking or reversal of the cycle swallowing disorder, impaired cough reflex, aspiration pneumonia by intensifying research will be essential.

Introduction

Parkinson's Disease(PD) can lead to problems with swallowing in between 30-100% of patients at anytime during the disease [1,2] Although James Parkinson described swallowing difficulties in one of his patients in "An Essay on the Shaking Palsy" in 1817 the first publications on this subject appeared in the 80's and 90's of the last century [3-5]. It was no coincidence that at that time also the percutaneous endoscopic gastrostomy (PEG) method for feeding was introduced as well as the videofluoroscopic swallowing study (VESS) as a means of assessing the dynamics of swallowing [6,7-14]. Dysphagia in PD patients has been shown to be associated with aspiration pneumonia which is one of the potential reasons for PD hospitalization [8-10]. PD patients are believed to have a 3,8 times increased risk of aspiration compared to the general population [11]. Aspiration pneumonia has been reported as the most frequent cause of death in PD patients and probably accounts for 70% of the mortality [12]. The frequency of in-hospital aspiration was surprisingly low with 2,4% despite the low education offer in hospitals for aspiration precautions [13]. Exactly two hundred years after James Parkinson's first description dysphagia is still a major problem in PD patients.In this mini-review the developments in diagnosing PD dysphagia,prevention of aspiration and swallowing therapies are discussed.

Swallowing disorders in PD patients

In 1983 Jerry Logemann introduced the videofluoroscopic swallowing study (VESS) [14]. These studies showed specific impairments in as well the oral,pharyngeal as in the esophageal phase of swallowing in PD patients. In the oral phase these include orofacial tremor,difficulty in forming a cohesive food bolus,prolonged swallowing time,limited tongue and mandibular extension during mastication and the presence of repititive anteroposterior movements of the tongue during bolus propulsion (lingual festination). Pharyngeal impairments include delayed pharyngeal response with consequent stasis in the vallecululae and piriform sinuses with the risk of laryngeal penetration and aspiration,impairment of pharyngeal contraction and cricopharyngeal function [2].

Impairments in the esophageal phase of swallowing nclude reduced peristalsis and reduced transit time.Dysphagia in PD patients may present without symptoms making it sometimes difficult to diagnose.Some of these patients who don't complain have eliminated foods and lose weight. PD patients can have "silent aspiration"in the trachea [15]. Together with fibre optic endoscopic evaluation of swallowing (FEES) in combination with the modified barium swallow (MBS), (VESS) is the golden standard now for evaluating dysphagia in PD patients [16,17]. Esophageal dysfunction can be assessed by high-resolution manometry [18]. However all these investigations are not always feasible. In these circumstances questionnaires as the Múnich Dysphagia Test (MDT) can be used.The scores in this 26 item test correlate fairly well with FEES.The test has a sensitivity of 90% and a specificity of 86 % [19].

Dysphagia symptoms in PD patients

a. "Tongue pumping"

b. Choking when eating.

c. Coughing or gagging when eating.

d. Drooling.

e. Hoarseness,voice weakening.

f. Recurrent heartburn.

g. Food or stomach acid backing up into the throat.

h. Sensation of food getting stuck in the throat or chest or behind the breastbone.

i. Unexplained weight loss.

j. Regurgitation.

k. Difficulty initiating swallow.

l. Recurrent pneumonia.

m. Inability to control saliva in the mouth and aspirating saliva.

Of these symptoms drooling got the most attention in research probably because it is embarrassing for both the patient and the social environment.

Sialorrhea and drooling

Sialorrhea is defined as an increased amount of saliva accumulating in the mouth due to excessive production or by decreased clearance of saliva.In severe cases this may build up and result in drooling.Drooling occurs in many otolaryngeal disorders but in particular in PD patients in whom drooling may be a major poblem [20]. Hanneke Kalf et al. [21] studied diurnal and nocturnal drooling in PD patients and evaluated the association between drooling severity and the severity of facial and oral motor deficits such as reduced facial expressions,involuntary mouth opening and swallowing complaints.Diurnal drooling defined as dribbling of saliva while awake was present in 28% of PD patients.Nocturnal drooling,with or without diurnal drooling, was present in 58% of PD patients.When compared to non-droolers,droolers were older and had more severe PD,longer disease duration,worse score on dysphagia and facial expression scales and more severe involuntary mouth opening. Diurnal drooling typically appeared later in the disease course and was associated with involuntary mouth opening [21].

In a meta-analysis based on 10 studies Kalf et al.found that the prevalence rates of drooling varied between 32% and 74% depending on disease severity and definition of drooling. The mean prevalence in community-dwelling patients was 56% [21,22]. Gender was found to be a significant factor.Men were twice more likely than women to have drooling [23]. Drooling in PD patients doesn't result from excessive saliva production but rather from decreased clearance due to infrequent or impaired swallowing [24]. Although the subject of much controversy in the past the value of speech therapy has been confirmed in several studies [15,25,26]. Speech therapy for PD dysphagia include exercises to increase mobility of oropharyngeal and laryngopharyngeal structures.In particular the sphincteric action of the larynx is increased.

Other non-pharmacological treatments of drooling were approaches such as chewing gum, behavioral modification,radiotherapy and surgical treatment.Cognitive behavioral therapy is mostly used in anxiety and depression of PD patients.In a study of 6 PD patients in whom therapy was directed specific at drooling a temporary improvement was seen lasting one month [27]. Chewing gum improved swallow frequency and latency in a small PD patient study and can be tried at an individual base [28]. Radiotherapy can be used in therapy-resistant PD drooling [29]. Surgical options are salivary duct ligation or relocation,salivary gland excision and neurectomy of the chordae tympani but are exceptional procedures for drooling in PD patients and have been performed mainly in neurologically impaired children. Physical therapy consits of movement rotation therapies and the chin-tuck therapy where the chin is put down to the breast to move the food bolus anterior [30].

To date there are no studies that particularily investigated the effect of deep brain stimulation (DBS) on drooling in PD patients. A systematic review showed no effect of DBS on drooling but another study of unilateral subthalamic nucleus DBS in contrast to pallidal DBS showed a deleterious effect on drooling [31,32]. Dopaminergic drug treatment appears to have little effect on speech and swallowing compared with the major effect it has on motor symptoms in the trunk and limbs. This suggests that both dysarthrophonia and dysphagia are related to non-dopaminergic pathways [33].

The use of anticholinergics to inhibit activation at muscarinic receptors decrease the volume of drooling.The doses tolerated have not caused drooling to cease completely. Adverse effects are numerous and limit treatment.These effects include irritability,restlessness,sedation and delirium from central drug effects.Inhibition of sweat glands disturbs temperature regulation and inhibition of gastrointestinal mobility worsens already present constipation.Urinary retention is another adverse effect. Anticholinergics can worsen cognitive deficits.Transdermal scopalamine has been used with success for short periods of some 4 weeks.No data are available to illustrate its efficacy for longer times [34]. Oral glycopyrrolate was effective in drooling but there is no evidence for treating longer than one week [35]. A study using sublingual ipatropium in PD drooling showed only a benificial effect for 2 weeks compared to placebo in a 5 week study [36].

Alpha2 adrenergic receptors might be involved in drooling as the adverse effects of clozapine and yohimbine,alpha2 receptor antagonists,are drooling.Therefore the alpha 2 receptor agonist clonidine was tested in PD drooling..In a small study (n=32) of patients with PD drooling 17 received clonidine and 15 were given placebo.The assessment tool was how many times each subject had to clear their saliva in a 5-minute period at baseline,1 and 3 months after randomization.Results showed clonidine significantly

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improved the number of times of clearing saliva at both time periods [37]. Oral modafinil is an alpha1 adrenergic receptor agonist and was effective in drooling suggesting the reduced drooling might be related to the improvement of dysphagia rather than hypersalivation [20].

To date injection of the parotid or submandibular saliva glands with botulinum toxin is the most effective therapy for drooling in PD patients.Botulinum toxin has 2 serotypes, A and B. Both serotypes are effective in PD droolling. They work by inhibiting acetylcholine release, a neurotransmitter, by reducing parasympathetic and postganglionic synaptic activity reducing salivary secretion. Injection is performed at best under ultrasound guidance. The benificial effects last for 4-5 months. The adverse effect is a dry mouth which is generally mild [37-40].

Aspiration pneumonia

It is often said that people die "with" rather than "of" the disease. PD patients live generally about as long as other people in their age cohort. People often die of unrelated diseases as cancer, heart disease or stroke. But the most common cause of death in those with Parkinson's is pneumonia because the disease impairs the patient's ability to swallow putting them at risk for aspirating food or liquids into their lungs leading to aspiration pneumonia. Frequent falls and accidents due to impaired mobility triggers a cascade of problems including being bedridden and developing pneumonia.

The Campaign Study is the only prospective study that followed PD patients from time of diagnosis,mostly in their 70's, 10 years long (n=142).The researchers found that 23% were generally doing well 10 years later meaning they could maintain their balance and did not have dementia. But over half of the patients in the original group had died with the most common cause related to Parkinson's being pneumonia [41]. Aspiration pneumonia is difficult to treat. Aspiration pneumonia has two pathological conditions such as the aspiratio of "sterile" gastric content and the chemical offence of the lung affecting the alveoli. Physicians often have difficulties in identifying a causative bacterial pathogen in patients with aspiration pneumonia. Sputum cultures often contain only normal flora or oral Streptococci [42,43].

Just like PD patients many frail elderly nursing home residents with aspiration pneumonia show sporadic fever for weeks or several months suggesting that aspiration of not so virulent material occurs regularly. [44,45]. Chest CT scans show very dense consolidations in the back. The histopathology of these consolidations show dense infiltration of leucocytes in the alveolar spaces and walls. The picture is that of a chronic bronchiolar inflammation or diffuse aspiration bronchiolitis. Besides the dysphagia PD patients also have a diminished cough reflex (dystussis) with sputum suctioning. Dystussis develops probably subsequent to dysphagia. Atussis is the absence of the cough reflex [46,47].

In general PD patients with an aspiration pneumonia are treated with rehydration by i.v fluids, oxygen and empirical broad spectrum antibiotics. When they become respiratory insufficiënt the choice for mechanical ventilation is often difficult because these patients are so frail they are at risk to get rid off the ventilator machine never anymore. Therefore while realizing aspiration pneumonia is mostly not bacterial of origin,but sterile due to gastric acid or chemical the paradigm should shift from pathogen-oriented therapy to function-oriented therapy. Function therapy means focusing on the reversal of the cycle PD-(dementia), dysphagia, dystussis, atussis, silent aspiration pneumonia. Researchers should focus further on how to improve dysphagia and coughing dysfunction in PD patients rather than waiting for new antibiotics.

Conclusion

Two hundred years later after the first description of patients with Parkinson's disease by James Parkinson in 1817 progress in its trea tment has been modest which needs physicians and researchers to be humble. Levodopa is for more than 50 years the golden standard for the motor symptoms of PD patients without innovations. The non-motor symptoms as swallowing disorders, speech disorders cough dysfunction and aspiration received some attention only for the last three decades,not withstanding the fact that aspiration pneumonia is the main cause of death in PD patients. Future directions in the prevention and treatment of aspiration pneumonia should be more function directed than pathogen directed. Reversal of the cycle swallowing disorder,impaired cough reflex,aspiration pneumonia can b e a turning point in the natural course of the disease in PD patients to reach that goal research should be intensified.

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