



DYSPHAGIA WITH ASPIRATION RISK IN FRONTOTEMPORAL DEMENTIA PATIENT-A CASE STUDY

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ABSTRACT

Frontotemporal dementia (FTD) is an uncommon type of dementia that mainly affects the frontal and temporal lobes which causes problems with behavior and language. Pneumonia is a common cause of death in patients with FTD. Asphyxia that can cause death is common in FTD. In these patients pneumonia is likely to be an aspiration pneumonia due to their underlying dysphagia. This article is a case study discussing about a 72-year-old male who had Frontotemporal Dementia with progressive dysphagic signs in turn affecting even salivary secretion management. Patients with AD mainly Frontotemporal Dementia exhibit oropharyngeal weakness inducing aspiration not only on oral feeding but even inducing aspiration risk on own oro-pharyngeal secretions which can induce respiratory distress with desaturations. With appropriate suctioning, postural modifications and chest physiotherapy these patients can be minimized from having episodes of salivary secretion aspiration.

Keywords: Frontotemporal dementia (FTD), Pneumonia, Dysphagia

INTRODUCTION

Frontotemporal dementia (FTD) is an uncommon type of dementia that mainly affects the frontal and temporal lobes which causes problems with behavior and language

[1]. FTD results from progressive damage to the anterior temporal, frontal, or both lobes of the brain due to a spectrum of pathological and genetic disorders [2].

Dementia mostly affects people over 65, but frontotemporal dementia tends to start at a younger age. Most cases are diagnosed in people aged 45-65, although it can also affect younger or older people. Frontotemporal dementia tends to develop slowly and get gradually worse over several years [3, 4].

The nerve cell damage caused by frontotemporal dementia leads to loss of function in these brain regions, which variably cause deterioration in behavior and personality, language disturbances, or alterations in muscle or motor functions [5-7].

FTD is divided into three types, depending on the earliest presentation of symptoms:

- **Behaviour variant frontotemporal dementia (bvFTD)** characterized by Progressive behavioral and personality decline which is marked by changes in behavior, emotions, concentration, attention, reasoning and judgment, ability to inhibit inappropriate actions, and the capacity for empathy.
- **Primary progressive aphasia (PPA)** characterized by Progressive language decline which includes inability to speak, understand spoken and written language, and write.
- **Progressive motor decline** is marked by difficulty with movement including motor planning (apraxia),

development of tremor, weakness, poor coordination, falls, gait change, and poor coordination.

Dysphagia in Frontotemporal dementia:

Pneumonia is a common cause of death in patients with FTD. Asphyxia accounted for death is also common. The pneumonia was likely to be aspiration pneumonia due to underlying dysphagia. Literature shows an article describing swallowing function in those with FTD. It reveals that FTD manifested an “apraxia of swallowing.” The problem that was visualized fluoroscopically, was difficulty in initiating the swallow, with food and liquid remaining in the patient’s mouth for long periods.

Langomore *et al* in 2007 studied swallowing characteristics in patients with FTD using Endoscopic test for swallowing. In 38% of the patient’s incomplete bolus clearance through the pharynx was noted. This could be due to the weakness of the bulbar musculature. Bolus clearance through the mouth and throat requires a fair degree of strength in the tongue and pharyngeal and laryngeal muscles. Bolus clearance is not controlled by cortical structures, but is more likely mediated by brainstem centers with facilitating input from subcortical centers and indirect pathways. Compulsive eating behavior added to dysphagia increases the risk of aspiration and its complications. Aspiration pneumonia is a common cause of death in those with FTD.

Dysphagia in early-stage FTD is characterized by delayed onset of the pharyngeal swallow and reduced lingual movement, while moderate FTD adds difficulty with oral preparation of the bolus, pharyngeal clearance, upper esophageal sphincter opening and visible aspiration on video fluoroscopy.

Silent laryngeal penetration and silent aspiration are common manifestations in FTD patients and are frequently associated with dysphagia. However, little is known about saliva aspiration in this population. FEES on patients with FTD show silent aspiration of saliva in 30%. This could be due to pooling of saliva in oral cavity along with reduced sensitivity in the epiglottis and posterior wall of the hypopharynx inducing silent aspiration and silent laryngeal penetration of saliva in patients with FTD.

Case Presentation

A 72-year-old male was admitted on 29 Dec, 2017 with SOB and cough followed by vomiting at night during sleep. The vomitus was mostly saliva mixed with gastric residue. This patient was on smashed solids and thin liquids at home. Cough reported with oral feeds since last 8-9 months but progressive form last 4 weeks with vomiting immediately post intake. The chest X ray on admission showed RT Lower lobe infiltration indicative of possible aspiration pneumonia.

Next day of admission patient was given by family water though not recommended by physician and post intake cough with vomiting noted thereafter patient was tachypneic and tachycardic. The repeat chest X-ray showed increase in RT Lower lobe infiltration indicating confirmed aspiration. Patients GCS dropped from 10 to 7 with desaturation and was started on 5 L O₂ support with saturation maintained at 98% SpO₂. NGT was inserted by medical team for nutritional support.

After 3 days of antibiotics for aspiration pneumonia patient was more alert and improvement in GCS was noted. Dysphagia rehabilitation was started and patient was tolerating few spoons of honey thick consistency but still had oropharyngeal fatigueness inducing aspiration risk after 6-7 spoons of intake.

Video fluoroscopic evaluation of swallowing was done 7th January 2018 and the results showed moderate-severe oropharyngeal dysphagia. Labial closure was better with no significant anterior spillage for thickened liquids. Lingual control for posterior propulsion of bolus was better for thickened liquids. Premature spillage noted for thin liquids. Penetration was present for nectar thick liquids and thin liquids with cough post swallow. Residue was observed in base of tongue/valleculae post swallow for all diet textures however can comparatively better clearance was noted

with 2-3 multiple effortful swallows with no secondary penetration for honey thick liquids. For pureed diet /pudding thick liquids patient was unable to clear pharyngeal residue even with multiple swallows. Pharyngeal swallow was initiated when bolus head reaches valleculae for honey thickened liquids. But for thin liquids/nectar thick liquids pharyngeal swallow was further delayed and initiated only when bolus head reached post aryepiglottic folds for nectar thick liquids and pyriform sinus for thin liquids inducing definite aspiration risk. No nasal regurgitation observed. Epiglottic inversion was incomplete but laryngeal vestibular closure was better for honey thick liquids. But was risk for secondary penetration/aspiration from pharyngeal residue and oropharyngeal fatigueness on larger volume of intake.

Patient was on trial oral feeding with small portions of honey thick liquids using safe swallow strategies. Patient tolerated small portions almost 7-8 spoons with no desaturations. However, after 7 hours of oral intake patient had desaturation with cough and increased RR at 12.00am while routine change of posture was done by nurses. Family reported that patient had gurgly breathing with pharyngeal secretions prior to positioning indicating possible aspiration of own oropharyngeal secretions. Family also reported that since last 2-3 months gurgly breathing and coughing noted while

sleeping which patient cannot self-expectorate. Similar instances of possible aspiration of salivary secretions while sleeping indicated with cough and desaturations needing oropharyngeal suctioning and chest physiotherapy happened 5-6 times during current hospital admission. Presently due to weak pharyngeal swallow patient is likely to have induced aspiration risk from own salivary secretions thereby family was educated postural management while sleeping.

Based on VFES patient did exhibit senescent related oropharyngeal dysphagia affecting oral nutritional status and was recommended to have enteral feeding for nutrition. In addition, as patient also had 3-4 instances of desaturation due to possible aspiration of own oropharyngeal secretions patient was recommended oral feeding on thickened liquids only for pleasure. Thereby patient underwent PEG tube insertion and is on full enteral feeding support.

DISCUSSION

Patients with Fronto-temporal Dementia exhibit oro-pharyngeal weakness induces aspiration not only on oral feeding but even inducing on own oropharyngeal secretions. These patients even when remained nil by mouth can have chest infections due to aspiration of own salivary secretions. With appropriate suctioning, postural modifications and chest physiotherapy these

patients can be minimized from having episodes of salivary secretion aspiration.

REFERENCES

- [1] Brun A, Gustafson L. Distribution of cerebral degeneration in Alzheimer's disease. A clinico-pathological study. *Arch PsychiatrNervenkr.* 1976;223:15–33.
- [2] Chu CC, Tranel D, Damasio AR, Van Hoesen GW. The autonomic-related cortex: pathology in Alzheimer's disease. *Cereb Cortex.* 1997;7:86–95.
- [3] Hamdy S, Mikulis DJ, Crawley A, Xue S, Lau H, Henry S, Diamant NE. Cortical activation during human volitional swallowing: an event-related fMRI study. *Am J Physiol.* 1999;277:G219–G225.
- [4] Horner J, Alberts MJ, Dawson DV, Cook GM. Swallowing in Alzheimer's disease. *Alzheimer Dis AssocDisord.* 1994;8:177–189.
- [5] Kern M, Birn R, Jaradeh S, Jesmanowicz A, Cox R, Hyde J, Shaker R. Swallow-related cerebral cortical activity maps are not specific to deglutition. *Am J PhysiolGastrointest Liver Physiol.* 2001;280:G531–G538.
- [6] RS. Cerebral cortical representation of automatic and volitional swallowing in humans. *J Neurophysiol.* 2001;85:938–950.
- [7] Susan E. Langmore, PhD; Richard K. Olney, MD; Catherine Lomen-Hoerth, MD, PhD; Bruce L. Miller, MD; *Dysphagia in Patients With Frontotemporal Lobar Dementia.* *ArchNeurol.*2007