Oropharyngeal Dysphagia: Neurogenic Etiology and Manifestation

Swapna Sebastian · Prem G. Nair · Philip Thomas ·
Amit Kumar Tyagi

Received: 18 October 2014 / Accepted: 3 November 2014 / Published online: 9 November 2014
© Association of Otolaryngologists of India 2014

Abstract To determine the type, severity and manifestation of dysphagia in patients with neurogenic etiology. Clinical documentation was done on the different etiologies, its manifestation, assessment findings and management strategies taken for patients with neurogenic oropharyngeal dysphagia who were referred for assessment and management of dysphagia over a period of three months in a tertiary care teaching hospital. Flexible endoscopic examination was done in all the patients. The severity of dysphagia in these patients were graded based on Gugging Swallowing Screen (GUSS). A total of 53 patients with neurogenic oropharyngeal dysphagia were evaluated by an otolaryngologist and a speech language pathologist over a period of three months. The grading of severity based on GUSS for these patients were done. There were 30 patients with recurrent laryngeal nerve injury due to various etiologies, one patient with Neurofibroma-vestibular schwannoma who underwent surgical excision, 16 patients with stroke, two patients with traumatic brain injury, two patients with Parkinsonism and two patients with myasthenia gravis. The manifestation of dysphagia was mainly in the form of prolonged masticatory time, oral transit time, and increased number of swallows required for each bolus, cricopharyngeal spasms and aspiration. Among the dysphagia patients with neurogenic

etiology, dysphagia is manifested with a gradual onset and is found to have a progressive course in degenerative disorders. Morbidity and mortality may be reduced with early identification and management of neurogenic dysphagia.

Keywords Dysphagia · Assessment · Management

Introduction

Normal swallowing involves the coordinated movement of the oral structures, pharynx, larynx and esophagus. The oral structure prepares the bolus for swallowing. Elevation of the soft palate and contraction of the posterior wall of the naso-pharynx prevent nasal regurgitation. The entry of the food into the airway is prevented by the upward and forward movement of the larynx, approximation of vocal cords, and movement of the epiglottis over the larynx. The bolus is pushed into the pharynx by the movement of the posterior part of the tongue against the posterior pharyngeal wall followed by pharyngeal peristaltic movement. Relaxation of the upper esophageal sphincter (UES) allows the bolus to enter the esophagus. The Oral phase of swallowing is voluntary whereas the pharyngeal and esophageal phase is involuntary.

Dysphagia is the difficulty or discomfort on swallowing, which can be of three types—Oral, Pharyngeal and esophageal depending on the stage of swallowing that is affected. In Oral dysphagia, preparation of food bolus or positioning of food in the oral cavity is affected either due to reduced strength or due to abnormal coordination of the oral muscles. Pharyngeal dysphagia occurs due to absence or delay in the triggering of swallow reflex and esophageal dysphagia is due to mechanical dysfunction of the esophagus or esophageal sphincter.

S. Sebastian (✉) · P. Thomas · A. K. Tyagi
Department of ENT, Christian Medical College, Vellore 632004,
Tamilnadu, India
e-mail: swapna_santhosh@yahoo.co.in

P. G. Nair
Department of Speech Pathology and Audiology, Amritha
Institute of Medical Sciences, Kochi, India

Cause of dysphagia can be either structural or motor. Structural causes include luminal stenosis and diverticula whereas motor causes include paresis (muscle weakness), sphincter dysfunction, and spastic disorders. Many disorders of the nervous system and muscles can cause neurogenic dysphagia like a central pathology such as stroke, head injury, previous surgery and nerve damage due to head and neck surgery, motor neuron disease, Parkinson's disease, multiple sclerosis or neurodegenerative diseases such as Alzheimer's disease or due to side effect of pharmacological agents, such as benzodiazepines, that might alter neuromuscular function.

Complications of dysphagia include weight loss, malnutrition, dehydration, aspiration pneumonia and additional morbidity and increased mortality rates.

Methodology

Clinical documentation was done on the different etiologies, its manifestation, assessment findings and management strategies taken for patients with neurogenic oropharyngeal dysphagia who were referred for assessment and management of dysphagia over a period of three months in a tertiary care teaching hospital. The medical and surgical reports were examined. Flexible endoscopic examination was done in all the patients. Gugging Swallowing Screen (GUSS) (Fig. 1) was used for dysphagia screening. GUSS is a validated reliable screening test for swallowing [1]. It has a scoring system from 0–20 points. A score of twenty points indicates normal swallowing ability without aspiration risk and a score of less than ten indicates severely reduced swallowing capabilities. It classifies degrees of dysphagia and aspiration risk into severe, moderate, mild, and no dysphagia. In patients with cerebrovascular accident (CVA), language assessment was done and type of aphasia was diagnosed based on Western Aphasia Battery [2].

Results and Discussion

The study was done on a total of 53 patients with neurogenic oropharyngeal dysphagia, evaluated by an Otolaryngologist and a speech language pathologist over a period of three months. Details regarding the assessment, findings and management strategies taken for these patients are given in Table 1.

Vocal cord palsy may be caused by diverse etiologies like neck and thoracic surgery, malignancy, trauma, intracranial causes, or may be idiopathic. Out of the 30 patients evaluated with recurrent laryngeal nerve injury, 12 of them developed vocal cord palsy followed by thyroidectomy,

two due to Direct trauma to larynx (motor vehicle accident), eight due to intubation trauma, two following Cardiac surgery (Coronary Artery Bypass Grafting [CABG]), two due to tuberculosis of lungs., one due to Cancer of lungs and three due to Cardiac disorders (Ortner syndrome/ cardio-vocal syndrome). Recurrent laryngeal nerve injury has been reported as a major complication of thyroid surgery [3–5] which can lead to alteration in the quality of voice as well as swallowing problems. Vocal cord Palsy following Cardiac surgery can result either due to intubation trauma or a direct manipulation and retraction procedure during the surgery. CC syndrome (cardio-vocal syndrome) which was first described by Ortner is characterized by hoarseness caused by compression of the left recurrent laryngeal nerve due to cardio vascular diseases like enlarged hypertensive pulmonary artery, cardiomegaly, aortic arch aneurysm, right subclavian aneurysm, ductus arteriosus aneurysm, or an enlarged left atrium which will compress the left recurrent laryngeal nerve [6–13]. Flexible endoscopic examination showed immobility of the left side vocal cord in 28 of our patients and right side palsy in two of our patients. All the patients had hoarse voice. Eventhough hoarseness of voice was found in all the 30 patients dysphagia was found only in 12/30 (40 %) patients. They showed an average score of 13 in GUSS. Dysphagia was mainly manifested in the form of aspiration for liquids. Management of dysphagia was mainly in terms of compensatory techniques like chin tuck or change in posture and diet modifications by changing the consistency of food to semi solid/pureed form.

Evaluation of a 21 years old male who had surgical excision for Neurofibroma-vestibular schwannoma showed severe involuntary coughing immediately after food intake, breathy voice, and a GUSS score of three. Flexible endoscopic examination revealed Left vocal cord palsy and aspiration. He was suggested nothing by mouth (NPO). Percutaneous endoscopic gastrostomy (PEG) tube feeding was started along with oromotor exercises and half swallow boom exercise. Neurofibromatosis Type two patients usually have vestibular schwannoma. Vagus nerve injury has been reported as a complication of vestibular schwannoma surgery [14, 15].

Stroke has been reported to be associated with dysphagia, the incidence of which range from 23 to 50 % [16–24]. Increased risk of aspiration pneumonia, dehydration and malnutrition have been reported in stroke patients as a consequence of dysphagia [25–28]. 12 of our patients had middle cerebral artery infarct showing the symptoms of aphasia (4 with Brocas aphasia and 8 with global aphasia). Eight of them had severe dysphagia and two of them had moderate dysphagia. They scored an average of eight on GUSS. Compensatory techniques like chin tuck or change in posture to reduce the likelihood of aspiration,

Fig. 1 The gugging swallow screen. available online at <http://stroke.ahajournals.org>

Name: _____
 Date: _____
 Time: _____

GUSS

(Gugging Swallowing Screen)

1. Preliminary Investigation/Indirect Swallowing Test

	YES	NO
Vigilance (The patient must be alert for at least for 15 minutes)	1 ☐	0 ☐
Cough and/or throat clearing (<i>voluntary cough</i>) (Patient should cough or clear his or her throat twice)	1 ☐	0 ☐
Saliva Swallow:	1 ☐	0 ☐
• Swallowing successful		
• Drooling	0 ☐	1 ☐
• Voice change (hoarse, gurgly, coated, weak)	0 ☐	1 ☐
SUM:	(5)	
	1 - 4= Investigate further* 5= Continue with part 2	

2. Direct Swallowing Test (Material: Aqua bi, flat teaspoon, food thickener, bread)

In the following order:

	1 → SEMISOLID*	2 → LIQUID**	3 → SOLID ***
DEGLUTITION:			
• Swallowing not possible	0 ☐	0 ☐	0 ☐
• Swallowing delayed (> 2 sec.) (Solid textures > 10 sec.)	1 ☐	1 ☐	1 ☐
• Swallowing successful	2 ☐	2 ☐	2 ☐
COUGH (involuntary): (Before, during or after swallowing – until 3 minutes later)			
• Yes	0 ☐	0 ☐	0 ☐
• No	1 ☐	1 ☐	1 ☐
DROOLING:			
• Yes	0 ☐	0 ☐	0 ☐
• No	1 ☐	1 ☐	1 ☐
VOICE CHANGE: (Listen to the voice before and after swallowing - Patient should speak „O“)			
• Yes	0 ☐	0 ☐	0 ☐
• No	1 ☐	1 ☐	1 ☐
SUM:	(5)	(5)	(5)
	1 - 4= Investigate further* 5= Continue Liquid	1 - 4= Investigate further* 5= Continue Solid	1 - 4= Investigate further* 5= Normal
SUM: (Indirect Swallowing Test AND Direct Swallowing Test) ----- (20)			

* First administer ½ up to a half teaspoon Aqua bi with food thickener (pudding like consistency). If there are no symptoms apply 3 to 5 teaspoons. Assess after the 5th spoonful.

** 3, 5, 10, 20 ml Aqua bi - if there are no symptoms continue with 50 ml Aqua bi (Daniels et al. 2000; Gottlieb et al. 1996) Assess and stop the investigation when one of the criteria is observed!

*** Clinical: dry bread; FEES: dry bread which is dipped in coloured liquid

† Use functional investigations such as Videofluoroscopic Evaluation of Swallowing (VFES), Fiberoptic Endoscopic Evaluation of Swallowing (FEES)

GUSS

(Gugging Swallowing Screen)

GUSS - EVALUATION

RESULTS	SEVERITY CODE	RECOMMENDATIONS
20 Semisolid / liquid and solid texture successful	Slight / No Dysphagia minimal risk of aspiration	<ul style="list-style-type: none"> Normal Diet Regular Liquids (First time under supervision of the SLT or a trained stroke nurse!)
15-19 Semisolid and liquid texture successful and Solid unsuccessful	Slight Dysphagia with a low risk of aspiration	<ul style="list-style-type: none"> Dysphagia Diet (pureed and soft food) Liquids very slowly - one sip at a time Functional swallowing assessments such as Fiberoptic Endoscopic Evaluation of Swallowing (FEES) or Videofluoroscopic Evaluation of Swallowing (VFES) Refer to Speech and Language Therapist (SLT)
10-14 Semisolid swallow success sful and Liquids unsuccessful	Moderate dysphagia with a risk of aspiration	Dysphagia diet beginning with : <ul style="list-style-type: none"> Semisolid textures such as baby food and additional parenteral feeding. All liquids must be thickened! Pills must be crushed and mixed with thick liquid. No liquid medication! Further functional swallowing assessments (FEES, VFES) Refer to Speech and Language Therapist (SLT)
0-9 Preliminary investigation unsuccessful or Semisolid swallow unsuccessful	Severe dysphagia with a high risk of aspiration	Supplementation with nasogastric tube or parenteral <ul style="list-style-type: none"> NPO (non per os = nothing by mouth) Further functional swallowing assessment (FEES, VFES) Refer to Speech and Language Therapist (SLT) Supplementation with nasogastric tube or parenteral

Table 1 Table showing the etiology and classification of dysphagia based on GUSS scoring

Etiology	Number of patients identified as having dysphagia	Degree of dysphagia based on GUSS scoring
Recurrent laryngeal nerve injury	12/30	Moderate dysphagia was present in 12 patients
Neurofibroma-vestibular schwannoma -surgical excision done	1/1	Severe dysphagia
Stroke	14/16	Severe dysphagia in 12 patients Moderate dysphagia in two patients
Traumatic brain injury	2/2	Severe dysphagia in both the patients
Parkinsonism	2/2	Severe dysphagia in both the patients
Myasthenia gravis	2/2	Moderate dysphagia in both the patients

diet modifications by changing the consistency of food (semi solid/pureed) in order to improve ease of chewing, bolus transit and reduce the likelihood of aspiration, physiologic technique/swallowing maneuvers like Mendelsohn maneuvers [29] and oropharyngeal exercises were recommended in these patients. Nasogastric tubes were inserted for the stroke patients with aspiration during immediate post stroke period and were removed depending on the recovery of the patient. Lateral medullary syndrome (LMS) or Wallenberg's syndrome (WS) is caused by lesion in the territory of the posterior inferior cerebellar artery or the vertebral artery [30]. Flexible endoscopy of the four of our patients with brain stem stroke (LMS) revealed cricopharyngeal spasm and pooling of saliva. These patients had to repeatedly clear pooled secretions from the throat since swallowing of even saliva was difficult due to cricopharyngeal spasm. They obtained an average GUSS scoring of two. PEG was done in all the four patients along with Shaker's exercise [31, 32] to improve opening of the cricopharyngeal sphincter.

There were two patients with traumatic brain injury who showed global aphasia with aspiration. They had an average score of six on GUSS. Nasogastric tubes were inserted during the acute period.

Two of the patients with parkinsonism revealed oral phase abnormalities which included tremors of tongue, prolonged oral phase and pharyngeal stage abnormalities like delayed swallowing reflex, and aspiration and they had an average score of eight on GUSS. Flexible endoscopic examination revealed pooling in the pyriform sinus and aspiration, Oropharyngeal stage abnormalities with abnormal lingual function and pharyngeal clearance has been reported to be as high as 50–52 % [33–39]. Nasogastric tubes were inserted for both the patients. Dysphagia assessment in Parkinson's patients is essential since it can prevent silent aspiration and the long-term sequelae of pulmonary infections, dehydration, and malnutrition.

Dysphagia in Myasthenia gravis is secondary to bulbar weakness. Two of our patients with Myasthenia gravis presented with the weakness of the oropharyngeal muscles.

Dysphagia was manifested in terms of prolonged masticatory time, oral transit time, increased number of swallows required for each bolus and aspiration and obtained an average GUSS score of 12. Compensatory techniques like chin tuck or change in head posture and diet modifications by changing the consistency of food to semi solid/pureed form was recommended for these patients.

Among the dysphagia patients with neurogenic etiology, dysphagia is manifested with a gradual onset and is found to have a progressive course in degenerative disorders. Early detection of dysphagia helps in preventing silent aspiration and to reduce morbidity and mortality. Based on the type of dysphagia exhibited by the patient, either compensatory techniques, diet modification, physiological technique, swallowing maneuvers or parenteral feeding or a combination of these are used for the management of dysphagia.

Conclusion

Neurogenic oropharyngeal dysphagia is manifested mainly in the form of prolonged masticatory time, oral transit time, increased number of swallows required for each bolus, cricopharyngeal spasms and aspiration. Early detection of dysphagia helps in preventing silent aspiration and the long-term sequelae of pulmonary infections, dehydration, and malnutrition by diet modification, change in postures, swallowing exercises or if necessary, by placement of a parenteral feeding. Morbidity and mortality may be reduced with early identification and management of neurogenic dysphagia.

References

1. Trapl M, Enderle P, Nowotny M et al (2007) Dysphagia bedside screening for acute-stroke patients: the Gugging Swallowing Screen. *Stroke* 38:2948–2952
2. Kertesz A (1982) Western aphasia battery. Harcourt Brace Jovanovich, New York

3. Ready AR, Barnes AD (1994) Complications of thyroidectomy. *Br J Surg* 81(11):1555–1556
4. Friedrich T, Steinert M, Keitel R, Sattler B, Schonfelder M (1998) The incidence of recurrent laryngeal nerve lesions after thyroid surgery; a retrospective analysis. *Zentralbl Chir* 123:25–29
5. Goncalves Filho J, Kowalski LP (2005) Surgical complications after thyroid surgery performed in a cancer hospital. *Otolaryngol Head Neck Surg* 132(3):490–494
6. Dolowitz DA, Lewis CS (1948) Left vocal cord paralysis associated with cardiac disease. *Am J Med* 4:856–862
7. Stooß K, Alkhadi H, Wildertmuth S, Lachat M, Pfammatter T (2004) Resolution of hoarseness after endovascular repair of thoracic aortic aneurysm; a case of Ortner's syndrome. *Ann Otol Rhinol Laryngol* 113(1):43–45
8. Stocker HH, Enterline HT (1958) Cardio-vocal syndrome: laryngeal paralysis in intrinsic heart disease. *Am Heart J* 56:51–59
9. Camishion RC, Gibbon JH Jr, Pierucci L Jr (1966) Paralysis of the left recurrent laryngeal nerve secondary to mitral valvular disease. Report of two cases and literature review. *Ann Surg* 163:818–828
10. Che GW, Chen J, Liu LX, Zhou QH (2008) Aortic arch aneurysm rupture into the lung misdiagnosed as lung carcinoma. *Can J Surg* 51:E91–E92
11. Bin HG, Kim MS, Kim SC, Keun JB, Lee JH, Kim SS (2005) Intrathoracic aneurysm of the right subclavian artery presenting with hoarseness: a case report. *J Korean Med Sci* 20:674–676
12. Kokotsakis J, Misthos P, Athanassiou T, Skouteli E, Rontogianni D, Lioulis A (2008) Acute Ortner's syndrome arising from ductus arteriosus aneurysm. *Tex Heart Inst J* 35:216–217
13. Gothi R, Ghonge NP (2008) Case report: spontaneous aneurysm of ductus arteriosus: a rare cause of hoarseness of voice in adults. *Indian J Radiol Imaging* 18:322–323
14. Slattery H III, Francis S, House KC (2001) Perioperative morbidity of acoustic neuroma surgery. *Otol Neurotol* 22:895–902
15. Ward BK, Francis HW, Best SR, Starmer HM, Akst LM, Gourin CG (2012) National prevalence and impact of perioperative vagus nerve injury in vestibular schwannoma. *Laryngoscope* 122(12):2824–2831
16. Singh S, Hamdy S (2006) Dysphagia in stroke patients. *Postgrad Med J* 82(968):383–391
17. Daniels SK, Ballo LA, Mahoney MC et al (2000) Clinical predictors of dysphagia and aspiration risk: outcome measures in acute stroke patients. *Arch Phys Med Rehabil* 81:1030–1033
18. Daniels SK, Brailey K, Priestly DH et al (1998) Aspiration in patients with acute stroke. *Arch Phys Med Rehabil* 79:14–19
19. Johnson ER, McKenzie SW, Sievers A (1993) Aspiration pneumonia in stroke. *Arch Phys Med Rehabil* 74:973–976
20. Kidd D, Lawson J, Nesbitt R et al (1993) Aspiration in acute stroke: a clinical study with videofluoroscopy. *Q J Med* 86:825–829
21. Gordon C, Hewer RL, Wade DT (1987) Dysphagia in acute stroke. *BMJ (Clin Res Ed)* 295:411–414
22. Mann G, Hankey GJ, Cameron D (2000) Swallowing disorders following acute stroke: prevalence and diagnostic accuracy. *Cerebrovasc Dis* 10:380–386
23. Mann G, Hankey GJ, Cameron D (1999) Swallowing function after stroke: prognosis and prognostic factors at 6 months. *Stroke* 30(744–748):748
24. Robbins J, Levin RL (1988) Swallowing after unilateral stroke of the cerebral cortex: preliminary experience. *Dysphagia* 3:11–17
25. Smithard DG, O'Neill PA, England RE, Park CL, Wyatt R, Martin DF, Morris J (1997) The natural history of dysphagia following a stroke. *Dysphagia* 12(4):188–193
26. Finestone HM, Greene-Finestone LS, Wilson ES, Teasell RW (1995) Malnutrition in stroke patients on the rehabilitation service and at follow-up: prevalence and predictors. *Arch Phys Med Rehabil* 76:310–316
27. Smithard DG, O'Neill PA, Park C, Morris J, Wyatt R, England R, Martin DF (1996) Complications and outcome after acute stroke Does dysphagia matter? *Stroke* 27(7):1200–1204
28. Daniels SK, Huckabee ML (2008) *Dysphagia following stroke*. Plural Publishing, San Diego
29. Mendelsohn MS, McConnell FM (1987) Function in the pharyngoesophageal segment. *Laryngoscope* 97(4):483–489
30. Martino R, Terrault N, Ezerzer F, Mikulis D, Diamant NE (2001) Dysphagia in a patient with lateral medullary syndrome: insight into the central control of swallowing. *Gastroenterology* 121(2):420–426 This article on PubMed
31. Shaker R, Kern M, Bardan E, Taylor A, Stewart ET, Hoffmann RG et al (1997) Augmentation of deglutitive upper esophageal sphincter opening in the elderly by exercise. *Am J Physiol* 272(6 Pt 1):G1518–G1522
32. Shaker R, Easterling C, Kern M, Nitschke T, Massey B, Daniels S et al (2002) Rehabilitation of swallowing by exercise in tube-fed patients with pharyngeal dysphagia secondary to abnormal UES opening. *Gastroenterology* 122(5):1314–1321
33. Lieberman AN (1980) Dysphagia in Parkinson's disease. *Am J Gastroenterol* 74:157–160
34. Edwards LL, Pfeiffer RF, Quigley EMM, Hofman R, Balluff M (1991) Gastrointestinal symptoms in Parkinson's disease. *Mov Disord* 6:151–156
35. Waxman MJ, Durfee D, Moore M, Morantz RA, Koller W (1990) Nutritional aspects and swallowing function of patients with Parkinson's disease. *Nutr Clin Prac* 5:196–199
36. Bushmann M, Dobmeyer SM, Leeker L, Perlmutter JS (1989) Swallowing abnormalities and their response to treatment in Parkinson's disease. *Neurology* 39:1309–1314
37. Edwards LL, Quigley EM, Harned RK, Hofman R, Pfeiffer RF (1994) Characterization of swallowing and defecation in Parkinson's disease. *Am J Gastroenterol* 89:15–25
38. Lieberman AN, Horowitz I, Redmond P, Pachter L, Lieberman I, Leibowitz M (1980) Dysphagia in Parkinson's disease. *Am J Gastroenterol* 74:157–160
39. Eadie MJ, Tyrer JH (1965) Alimentary disorder in Parkinsonism. *Aust Ann Med* 14:13–22