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OROPHARYNGEAL PROLONGED DYSPHAGIA AFTER ACUTE STROKE: DIAGNOSIS AND CLINICAL PREDICTORS

By

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ABSTRACT

Background: Dysphagia following acute stroke can be a serious threat to one's health because of the risk of aspiration pneumonia, malnutrition, dehydration, weight loss, and airway obstruction. There is an evidence that early enteral feeding via percutaneous endoscopic gastrostomy (PEG) is both beneficial and safe. **Objectives:** To predict risk factors of prolonged dysphagia following acute stroke and proper management of similar cases. **Subjects and Methods:** This study was a prospective cohort study conducted on 113 patients with acute stroke admitted within 24 hours. Clinical findings and imaging results were prospectively collected, and subsequent progress was recorded. Subjects were divided into 3 groups for analysis: no dysphagia; transient dysphagia (≥14 days); or prolonged dysphagia (>14 days). Particular attention was paid to bulbar function. Stroke severity was assessed using the National Institutes of Health Stroke Scale on admission and on discharge. The water swallow test was performed to all patients who were able to attend sufficiently to follow the instructions. Modified Barium Swallow or Video-fluoroscopy test was done for some patients to detect the oropharyngeal dysphagia. **Results:** Significant associations for prolonged dysphagia were seen with large stroke, increased stroke severity, dysphasia and lesions of the frontal and insular cortex and presence of old vascular insult on brain imaging. **Conclusion:** These results indicated that it is potentially possible to identify those patients who have prolonged significant dysphagia following acute stroke at an early time point. This would allow the judicious use of early PEG to avoid aspiration pneumonia and for better nutrition.

Keywords: Acute stroke, dysphagia, risk factors, percutaneous endoscopic gastrostomy, aspiration pneumonia.

INTRODUCTION

Dysphagia is a threat to health because of the risk of aspiration pneumonia, malnutrition, dehydration, weight loss, and airway obstruction (Han et al., 2008). Stroke is the leading cause of neurologic dysphagia. Approximately, 51-73% of patients with stroke have dysphagia that can delay the patient's functional recovery (Bussell and Gonz?lez-Fern?ndez, 2011).

A number of simple clinical assessments of swallowing function have been validated as predictors of aspiration. A less invasive and cheaper example is the water swallow test of 50–150 ml, which has been demonstrated to predict risk of aspiration with high sensitivity. The sensitivity of this test can be increased by careful assessment of bulbar function with abnormalities of pharyngeal sensation (but not the gag reflex). Reduced cough reflex is particularly useful in detecting silent aspiration (Nakajoh et al., 2000).

The early identification of those at risk of significant dysphagia and requiring nutritional support permits the study of early nutritional supplementation via nasoenteric tubes (NET) or percutaneous gastrostomy (PEG) (Wanklyn et al., 1995). We have therefore, performed a prospective cohort study of patients looking specifically for identification of predictors of prolonged dysphagia following acute stroke and proper management of similar cases.

The present study was a trial to predict the risk factors of prolonged dysphagia following acute stroke and proper management.

SUBJECTS AND METHODS

This prospective cohort study included 113 patients with suspected recent cerebral stroke admitted within 24 hours to stroke unit at Ain Shams Specialized Hospital over one year. Patients were excluded if they admitted with neurological deficit due to subarachnoid hemorrhage or if they were subsequently identified as having a transient ischemic attack or a diagnosis other than stroke or those requiring neurosurgical intervention. Patients (or their guardians) gave a written informed consents after informing them about the study rationale and their right to withdraw from the study at any time without any consequences. All patients were subjected to a standard history and clinical examination within 24 hours of admission to identify clinical risk factors of stroke. Particular attention was paid to bulbar function with dysarthria, dysphonia and voluntary cough, together with symmetry and speed of tongue and palatal movements being noted. Gag reflexes were recorded as present, reduced or absent, and preservation of pharyngeal sensation were recorded. Stroke severity was assessed using the National Institutes of Health Stroke Scale (NIHSS) on admissiion and on discharge (Ver Hage, 2011).

The water swallow test performed to all patients who were able to attend sufficiently to follow the instructions. The test was considered abnormal if coughing or choking occurred or when the test time was greater than 20 seconds.

Brain computed tomography (CT) and magnetic resonance imaging (MRI) stroke protocol were also done to all patients. The date of the study, side, type, vascular territory, size and location of any acute lesions were recorded. The presence of pre-existing vascular disease, atrophy or other significant pathology were also noted.

Modified barium swallow (MBS) or video-fluoroscopy (VFS) test was done under supervision of phoniatrics unit in Ain Shams Specialized Hospital to detect the oropharyngeal dysphagia. Routine blood and biochemical tests were also done for all patients. The principal outcome measure was the presence and duration of significant dysphagia, i.e. requiring non-oral feeding or hydration. Secondary outcome measures were the development of pneumonia, feeding status at discharge and discharge destination. Patients were continually assessed clinically for the development of pneumonia, with suspected cases confirmed by chest radiography.

Statistical Analysis: Statistical presentation and analysis of the present study conducted were the mean, standard deviation, chi-square tests, and analysis of variance [ANOVA]. All statistical analyses were performed using SPSS v.20 for Windows. Results were considered significant if p-value < 0.05.

RESULTS

This study included 113 patients with suspected recent cerebral stroke admitted within 24 hours to the stroke unit. The patients in this study were divided into 3 groups; First group was declared as non-dysphagia group which included 72 patients passed the water swallow test and had no additional clinical features suggestive of dysphagia. Second group was called transient dysphagia group (30 patients) who have recovery of functional swallowing within 14 days. Third group was called prolonged dysphagia (11 patients) where significant dysphagia required non oral feeding or hydration for 14 days or more.

In the present study, in spite of there was no statistically significant difference between three groups as regard age and sex, patients with dysphagia and particularly those with prolonged dysphagia were more likely to be older and male (Table 1). There was a significant difference between the three groups as regard presence of dysphasia particularly in prolonged dysphagia group ($p < 0.001$). Accordingly, patients suffering from significant dysphasia at the onset of stroke were more liable to develop dysphagia. Analysis of the type of dysphasia showed no significant pattern for expressive versus receptive dysphasia. Also, there were significant differences between the three groups as regard the mean NIHSS score on admission ($p < 0.001$) with significant higher score in transient (11.83 ± 5.18) and prolonged dysphagia (11.6 ± 5.48) compared to non-dysphagia group (5.23 ± 3.83). Same results were found between the three groups as regard NIHSS score on discharge (Table 1).

Table (1): Clinical features of the three groups of patients.

Dysphagia / Features	Non (n = 72)	Transient (n = 30)	Prolonged (n =11)	Chi-square P-value
Age (Mean± SD)	62.35 ±10.56	64.8 ± 9.01	66.18 ± 9.8	0.388 (ns*)
Female	23 (31.9%)	9 (31.9%)	3 (27.3%)	0.944 (ns)
Male	49 (68.1%)	21 (68.1%)	8 (72.7%)	
Past history of stroke	17 (23.6%)	12 (40.0%)	6 (54.5%)	0.054 (ns)
Past history of transient Ischemic attack	6 (8.3%)	2 (6.7%)	0 (0.0%)	0.601 (ns)
Facial Weakness	42 (58.3%)	20 (66.7%)	8 (72.7%)	0.542 (ns)
Dysarthria	35 (48.6%)	18 (60.0%)	9 (81.8%)	0.096 (ns)
Dysphasia	5 (6.9%)	9 (30%)	6 (54.5%)	<0.001(hs‡)
Dysphagia / Features	**Non (n = 72)**	**Transient (n = 30)**	**Prolonged (n =11)**	**ANOVA P-value**
Nutrition Albumin (Mean±SD)	3.58 ± 0.47	3.58 ± 0.48	3.37 ± 0.78	0.438 (ns)
NIHSS† Score on admission (Mean± SD)	5.23 ± 3.83	11.83 ± 5.18	11.60 ± 5.48	<0.001(hs§)
NIHSS Score on discharge (Mean± SD)	3.88 ± 3.49	9.42 ± 5.04	9.40 ± 4.77	<0.001(hs§)

* Not significant, † National institute health stroke scale, ‡ Highly significant dysphasia in dysphagia groups particularly prolonged dysphagia group compared to non-dysphagia group, § Highly significant mean NIHSS score on admission and on discharge in patients with dysphagia, particularly prolonged dysphagia compared to non-dysphagia patients.

Regarding bulbar function assessment, abnormal palatal sensation and poor voluntary cough were the only clinical abnormal signs of bulbar function that showed significant associations with prolonged dysphagia (p = 0.44 and 0.24 respectively -Table 2). There was a highly significant larger infarctions (p < 0.001) affecting frontal and insular cortex in patients with dysphagia, particularly prolonged dysphagia compared to non-dysphagia patients. Also, presence of old vascular insult was significantly associated with prolonged dysphagia (p<0.017). In contrast, there was no significant difference in the frequency of lesions affecting parietal and occipital cortex. Also, there was no significant difference between the three groups as regard thalamic, basal ganglia and deep white matter ischemia (Table 3).

Table (2): Assessment of bulbar function in all groups.

Dysphagia / Bulbar function	Non (n =72)	Transient (n = 30)	Prolonged (n = 11)	Chi-square P*-value
Dysarthria	35 (48.6%)	18 (60.0%)	8 (72.7%)	0.453 (ns†)
Impaired palatal sensation	3 (4.2%)	14 (46.7%)	9 (81.8%)	0.044 (s‡)
Reduced or absent gag reflex	3 (4.2%)	9 (30.0%)	4 (36.4%)	0.698 (ns)
Facial Weakness	42 (58.3%)	20 (66.7%)	8 (72.7%)	0.712 (ns)
Poor voluntary cough	5 (6.9%)	10 (33.3%)	8 (72.7%)	0.024 (s§)
Failed Water swallow test	0 (0.0%)	30 (100.0%)	11 (100.0%)	> 0.05 (ns)

* P-values quoted refer to transient and prolonged groups only (x^2 test). †: not significant, ‡**:** significant impaired palatal sensation in prolonged dysphagia patients in comparison to transient dysphagia patients, **§** significant poor voluntary cough in prolonged dysphagia patients in comparison to transient dysphagia patients.

Regarding outcome measures in prolonged dysphagia group, seven patients (63.3%) needed non-oral feeding and hydration at time of discharge. Moreover, the duration of hospital stay was highly significant longer for the patients with prolonged dysphagia (26.45 days ± 5.020) in comparison to transient dysphagia (8.2 ± 2.9) and non-dysphagia groups (4.56 ± 1.12) (p < 0.001 - Table 4). There were 13(11.5%) patients had documented pneumonia, 3(27.3%) in prolonged dysphagia group, 6 (20%) in transient dysphagia group, and 4 (5.5%) in non-dysphagia group due to any cause. Also, pneumonia was significantly high in patients with dysphagia in comparison to non-dysphagia group (p = 0.026).

Table (3): Comparison of imaging findings between the three groups of dysphagia.

Dysphagia / Site of the lesion	Non (n = 72)	Transient (n = 30)	Prolonged (n = 11)	Chi-square P-value
Large Infarction*	7 (9.7%)	10 (33.3%)	6 (54.5%)	< 0.001 (hs†)
Frontal	6 (8.3%)	13 (43.3%)	5 (45.5%)	< 0.001(hs‡)
Insular	1 (1.4%)	6 (20.0%)	4 (36.4%)	< 0.001(hs§)
Capsular	10 (13.9%)	1 (3.3%)	1 (9%)	0.284 (ns‖)
Parietal	28 (38.9%)	13 (43.3%)	2 (18.2%)	0.330 (ns)
Temporal	22 (30.6%)	7 (23.3%)	5 (45.5%)	0.388 (ns)
Occipital	5 (6.9%)	2 (6.7%)	1 (9.1%)	0.962 (ns)
Thalamus	13 (18.1%)	5 (16.6%)	2 (18.2%)	0.833 (ns)
White matter/Basal Ganglia	7 (9.7%)	8 (26.7%)	3 (27.3%)	0.058 (ns)
Brainstem/ Cerebellum	13 (18.1%)	10 (33.3%)	3 (27.3%)	0.233 (ns)
Old Vascular Insult	38 (52.8%)	23 (76.7%)	10 (90.9%)	0.017 (s¶)

***** Large infarcts refer to those with a maximal diameter of greater than 3 cm on any one slice, †, ‡, § Highly significant larger infarctions affecting frontal and insular cortex in patients with dysphagia, particularly prolonged dysphagia compared to non-dysphagia patients, ‖ Not significant, ¶ significant old vascular insult in patients with dysphagia, particularly prolonged dysphagia compared to transient dysphagia.

Table (4): Comparison of outcome measures for the three groups of patients.

Dysphagia / Swallow status at discharge	Non (n=72)	Transient (n=30)	Prolonged (n=11)	Chi-square P-value
Normal diet	72 (100.0%)	22 (73.3%)	2 (18.2%)	< 0.001
Modified diet	0 (0.0%)	8 (26.6%)	2 (18.2%)	0.003
Non oral diet/hydration	0 (0.0%)	0 (0.0%)	7 (63.6%)	< 0.001†
Dysphagia / Hospital stay	**Non (n=72)**	**Transient (n=30)**	**Prolonged (n=11)**	**ANOVA P-value**
Range	3 - 7	5 - 15	20 - 35	
Mean± SD	4.56 ±1.12	8.2 ± 2.9	26.45 ± 5.02	< 0.001††

† Percentage of patients needs non oral feeding and hydration is highly significant in prolonged dysphagia group compared to transient dysphagia group, †† The duration of hospital stay was highly significant longer for the patients with prolonged dysphagia in comparison to patients with transient dysphagia or non-dysphagia.

DISCUSSION

One of the most common and earliest problems to emerge after a stroke is difficult swallowing (dysphagia). Depending on the method of assessment and how dysphagia is defined, the prevalence of dysphagia in stroke patients ranges from 30% to 67% (Edmiaston, 2010). The rate of dysphagia in acute stroke in the present study was 36.3%. This result agreed with most worldwide studies about dysphagia in stroke patients (Smithard et al., 1996, Edmiaston, 2010 and Huang et al., 2014).

A number of clinical parameters were significantly associated with prolonged dysphagia. The most significant of these were the stroke severity measured by NIHSS and dysphasia. Analysis of the type of dysphasia showed no significant pattern for expressive versus receptive dysphasia, principally due to low numbers.

Lesions of the frontal operculum (Brain Area 44 = BA 44) have been shown to substantially impair the recovery of aspiration risk. Three explanations may be considered for this finding: (1) BA 44, as part of Broca's area, is classically considered to subserve motor speech production and also non language-related motor functions and control of orofacial sensorimotor behavior (Martin et al., 2001). Depending on stimulation intensity, the frontal operculum evokes mastication at low levels and swallowing sequences at high levels of stimulation (Martin, 1997). Furthermore, there is an evidence for an overlap of BA 44 with the ventral premotor cortex (Binkofski and Buccino, 2004). Hence, a lesion of this premotor area might disrupt its access to the primary motor cortex and, thus, the descending motor pathway. (2) The frontal operculum might play a role in peri-infarct tissue recruitment after insular stroke. In motor and aphasic stroke rehabilitation, peri-infarct cortical reorganization is associated with favorable functional outcome (Thompson and den Ouden, 2008). Similarly, damage of the frontal operculum would likely compromise such a cellular and molecular repair mechanism in the transition area to the insular cortex and impair the recruitment of peri-infarct tissue after insular stroke. (3) Parts of the frontal operculum in the transition zone to the insular cortex might exhibit similar features with respect to swallowing as the insula, which has been revealed by human and animal studies (Daniels and Foundas, 1997). Damage to both areas might, therefore, cause a more severe and longer-lasting risk of aspiration. Additionally, there is an evidence that the insulo-opercular transition zone represents a primary gustatory cortex. Hence, damage to this area might diminish sensory outflow to the frontal operculum for planning and preparation of the complex movement sequence of deglutition in relation to the ingested solid food and liquid. It is notable that patients with extended risk of aspiration predominantly have lesions of this perisylvian part of the frontal operculum at the transition zone to

the insular cortex (Kobayakawa et al., 1996).

The only element of clinical assessment of bulbar function, which showed a significant association with prolonged dysphagia, was abnormal palatal sensation and a poor voluntary cough. Also, in the present study, we found that dysphagia and prolonged dysphagia in particular were significantly associated with larger lesions affecting the frontal cortex and insular cortex. Also, presence of old vascular insult was significantly associated with prolonged dysphagia.

Intracortical microstimulation study found that stimulation of the anterior sector of the dorsal insula triggered swallowing, mouthing, and chewing in monkeys (Jezzini et al., 2012). Oroalimentary behavior, including motor automatisms, has been described during epileptic seizures originating in this part of the insula (Isnard et al., 2004). Stroke patients with discrete lesions of the anterior insula have been shown to suffer from dysphagia in small case studies. Furthermore, sensory and motor signaling from and to various areas might be mediated by the insula, e.g. interacting with the oropharynx and the esophagus (Riecker et al., 2009).

A magneto-encephalographic study demonstrated consistent long-lasting activation of the insular cortex before swallowing (Watanabe et al., 2004). Taken together, we posit a major role of the insula in elevating the risk of aspiration as it may function as a key area in the regulation of both voluntary and automatic swallowing. Lesions of the caudal primary sensori-motor and premotor cortex did not prove significant as predictors of aspiration risk. These findings contrast with the results of functional imaging studies of swallowing in healthy individuals which have found marked cortical activations around the central sulcus (Michou and Hamdy, 2009). However, activation of these regions was also observed in lip pursing, tongue rolling, and tongue tapping, suggesting that activation of these areas may not be specific to swallowing per se, but might rather represent an interference and modulation of swallowing-related tasks (Foley et al., 2008).

Lesions anywhere in the motor pathways controlling swallow mechanisms may lead to dysphagia. Because of bilateral innervation and likely alternative routes of innervation, recovery is generally rapid once acute oedema and cerebral shock begin to resolve. However, when the primary motor cortex associated with swallowing is involved, particularly larger lesions which may disrupt all cortical areas involved in swallowing, dysphagia is more likely to persist (Daniels and Foundas, 1999).

This parallels the poor recovery seen after cortical strokes affecting the motor strip. Functional studies of prolonged dysphagia after stroke, using both positron emission tomography and magnetic stimulation, indicate that recovery of normal swallowing in this situation is dependent upon the development of increased cortical representation of

swallowing in the intact hemisphere (Huang et al., 2014). Thus, the finding in the present study of lesions in the frontal and insular cortex areas, which have been proposed as important in swallowing being associated with prolonged dysphagia, is entirely consistent with this model.

In contrast, there was no significant difference in the frequency of lesions affecting parietal and occipital cortex. Also, there was no significant association between dysphagia and thalamic, basal ganglia and deep white matter ischemia. This may be explained by small number of patients. Also, the small numbers of brainstem lesions made interpretation of this subgroup difficult.

CONCLUSIONS

Large stroke lesions affecting the frontal, insular cortex and thalamus on brain imaging and also presence of old vascular insult were significantly associated with prolonged dysphagia. It is potentially possible to identify those patients who will have prolonged significant dysphagia at an early time point. This would allow the judicious use of early PEG, perhaps between 7 and 10 days. This would have many potential advantages. First, the risk of pneumonia due to aspiration may be reduced, second: better nutrition in a vulnerable group can be assured.

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قسم الأمراض العصبية و النفسية - كلية الطب - جامعة عين شمس

خلفية البحث: عسر البلع ما بعد السكتة الدماغية الحادة يمكن أن يشكل تهديدا خطيرا لصحة الإنسان بسبب خطر الإلتهاب الرئوي التنفسي، وسوء التغذية، والجفاف، وفقدان الوزن، وإنسداد مجرى الهواء. وهناك أدلة على أن التغذية المعوية في وقت مبكر عن طريق المعدة أو بالمنظار عن طريق الجلد مفيدة وآمنة على حد سواء.

الهدف من البحث: دراسة عوامل خطر عسر البلع لفترات طويلة بعد السكتة الدماغية الحادة والعلاج السليم للحالات المماثلة.

المرضى وطرق البحث: أجريت الدراسة على 113 مريضا يعانون من السكتة الدماغية الحادة فى غضون 24 ساعة.و قد تم جمع مستقبلي للنتائج السريرية ونتائج التصوير وتسجيلها، ثم قسمت الحالات إلى 3 مجموعات لأغراض التحليل: لا عسر في البلع، عسر في البلع عابر (≤14 أيام)؛ أو عسر البلع الطويل لأكثر من 14يوم . وقد أولي إهتماما خاصا لوظيفة البلع، كما تم تقييم شدة السكتة الدماغية بإستخدام مقياس المعاهد الوطنية للصحة السكتة الدماغية' وإجراء إختبار إبتلاع المياه لجميع المرضى الذين تمكنوا من أداء الاختبار وإتباع التعليمات. وقد تم عمل إختبار الباريوم وإختبار فيديو التنظير لبعض المرضى للكشف عن عسر البلع في الفم والبلعوم.

النتائج: وجد أنه هناك علاقة ذات دلالة إحصائية بين عسر البلع لفترات طويلة و السكتة الدماغية الخطيرة مع وجود خلل في الكلام و الجلطات التي تصيب الفص الجبهي الأمامي و القشرة الانعزالية للمخ، وأيضاً وجود جلطات قديمة في الرنين المغناطيسي المخي.

الإستنتاج: بالإمكان تحديد المرضى الذين سوف يعانون من عسر البلع لفترات طويلة بعد السكتة الدماغية الحادة في وقت مبكر، وبالتالي التجنب السريع لعسر البلع عن طريق التغذية بالمنظار المعدي عن طريق الجلد لتجنب الإلتهابات الرئوية و ضمان حسن التغذية.