Evaluation of neurogenic dysphagia in Iraqi patients with acute stroke

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ABSTRACT

الأهداف: تقييم صعوبة البلع العصبية سريرياً ولربط علاقتها بالخصائص السكانية، وشدة خطر الجلطة وعلاقة صعوبة البلع بنوع الجلطة.

الطريقة: درست حالة 72 مريض مصاب بالجلطة الدماغية خلال الفترة من يوليو 2007 حتى فبراير 2008 و هم المرضى المنومين في قسم الطب و الأعصاب – مستشفى اليرموك و مستشفى رزكاري – اربيل – العراق. تم إجراء فحص مان لتقييم البلع (MASA) و فحص مقياس رانكن (MRS) و بطاقة قياس عوامل الخطورة (SRSC). و قد أعيدت الإحصاءات بعد 1 شهر.

النتائج: اشتملت الدراسة على 40 ذكر و 32 أنثى. كان 68 مريض مصاب بجلطة إقفارية، و 4 مريض لديهم نزيف في المخ (ICH) طبقاً لمقياس مان لتقييم البلع (MASA). تبين الدراسة أن 55% حالات الجلطة الأمامية (ACS) و تبلغ حالات الجلطة الخلفية 91%. تحسن البلع في 56% من الحالات في الشهر الأول. وتوفي 40% من المرضى المصابين بصعوبة البلع خلال الشهر الأول وسببه في معظم الحالات ذات الرئة. لا يوجد أي اختلافات مهمة في الدراسة بخصوص الخصائص السكانية لصعوبة البلع.

خاتمة: صعوبة البلع يمكن أن تكون مؤشراً لشدة الجلطة مما يسبب ارتفاع الإصابة بالمرض والموت في المرضى المتأثرين. صعوبة البلع لا تتعلق بعوامل خطر الإصابة بالجلطة، ونوع الجلطة وهي ضرورية كمنذر لتقييم البلع و معالجة الأعراض الأولية.

Objectives: To clinically assess neurogenic dysphagia, and to correlate its presence with demographic features, different stroke risk factors, anatomical arterial territorial stroke types, and pathological stroke types.

Methods: Seventy-two stroke inpatients were studied between July 2007 and February 2008, at the Departments of Medicine and Neurology at Al-Yarmouk Teaching Hospital, Baghdad, and Rizgary Teaching Hospital, Erbil, Iraq. All patients were assessed using the Mann Assessment of Swallowing Ability score (MASA), Modified Rankin Scale, and the Stroke Risk Scorecard. All patients were reassessed after one month.

Results: There were 40 males and 32 females. Sixty-eight patients had ischemic stroke, and 4 had primary intracerebral hemorrhage (ICH). According to the MASA score, 55% of anterior circulation stroke (ACS) cases were associated with dysphasia, and 91% of lateral medullary syndrome cases were associated with dysphagia. Fifty-six percent of ACS dysphagic cases improved within the first month. Forty percent of dysphagic patients died in the one month follow up period, and in most, death was caused by aspiration pneumonia. We observed no significant differences regarding demographic features of dysphagia.

Conclusion: Dysphagia can be an indicator of the severity of stroke causing higher mortality and morbidity in affected patients. It was not related to the stroke risk factors and the type of stroke. It is essential from a prognostic point of view to assess swallowing, and to treat its complications early.

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Stroke is defined by the WHO as a clinical syndrome of rapid onset (usually seconds or minutes) of focal cerebral deficit, lasting more than 24 hours or leading to death, with no apparent cause other than a vascular one.[1] Stroke is the third most common cause of death in developed countries,[2,3] and it is either of 2 subtypes; ischemic stroke (85% of all strokes), or hemorrhagic stroke.[4-6] Swallowing is controlled by special nuclei in the brainstem (including the dorsal motor nuclei of the vagus nerve) and their cerebral connections (including the corticobulbar tracts that are responsible for carrying cerebral control to the brain stem nuclei). Ischemic or hemorrhagic lesions in the brainstem or these nuclei may cause weakness of the pharyngeal muscles leading

to acute severe dysphagia, while a hemispheric lesion due to occlusion of one of the internal carotid arteries, or its branches creates a less profound effect on swallowing, leading to transient and sometimes mild dysphagia.[7-9] Persistence of poor swallowing ability at 3 weeks carries a high mortality rate, probably because of aspiration.[10] Normal deglutition involves a complex series of voluntary and involuntary neuromuscular contractions proceeding from the mouth to the stomach, and is commonly divided into oropharyngeal and esophageal stages.[11] Dysphagia is impaired swallowing, which is generally classified into either oropharyngeal dysphagia in which there is difficulty in initiating the swallowing, and/or moving the bolus from the oral cavity into the esophagus. It can be identified via difficulty in tolerating thinner liquids, resulting in a cough or choking incident, or esophageal dysphagia, which is difficulty in moving food through the esophagus.[2,12] There are many simple bedside tests for swallowing assessment, such as the Mann Assessment of Swallowing Ability (MASA),[13] the standard swallow test,[14] and bronchial auscultation examination.[15] Instrumental assessment of dysphagia includes ultrasound, videofluorographic swallowing study, esophagoscopy, esophageal manometry and pH probe studies, electromyography, and fiberoptic endoscopic examination of swallowing.[16] This study aims to establish the incidence and severity of neurogenic dysphagia in acute stroke, to compare the incidence and severity of dysphagia between anterior and posterior circulation stroke, to determine if there is an association between different stroke risk factors and dysphagia severity, to determine if there is an association between different stroke risk factors and prognosis, and to determine if there is a relationship between demographic features and presence of dysphagia.

Methods. In this cohort study, 72 consecutive stroke patients (40 males and 32 females) with ages ranging between 30-82 years were included between July 2007 to February 2008. The study population comprised inpatients from the Departments of Medicine and Neurology at Al-Yarmouk Teaching Hospital, Baghdad, and Rizgary Teaching Hospital, Erbil, Iraq. Verbal consent was obtained from each patient enrolled in the study, and the Research Committee at the College of Medicine/Hawler Medical University, Erbil, Iraq approved the study protocol. Subjects eligible for inclusion were conscious patients with acute ischemic or hemorrhagic stroke. They were examined, classified, and diagnosed according to the Oxfordshire Classification, and their brain lesion proven by radiological examination through brain CT, which was carried out for all patients, in addition to brain MRI, which was carried out for 30 patients only, especially those with clinical findings suggestive of vascular lesion, but with normal CT. The MR angiography was carried out only for those patients clinically suspected to have brain stem lesion. Patients were excluded if they had history of old cerebrovascular event (as they may have dysphagia from previous stroke), patients with psychiatric disorders such as hypochondriasis, somatization disorders, and with esophageal motility disorders such as scleroderma and esophageal spasm, esophageal stricture or malignancy, and patients with history of head and neck surgery and other local causes of dysphagia such as retropharyngeal abscess, and thyroid mass. All patients were examined within the first 72 hours from admission by a Neurologist for assessment of swallowing according to MASA.[13] This scoring system is a brief bedside examination of swallowing ability in patients 18 years of age and older. The minimal supporting materials necessary include: a tongue depressor, flashlight, gloves, different food consistencies, or water. The examination covers 24 clinical items that evaluate oromotor/sensory components of swallowing, prerequisite learning skills, such as cooperation and auditory comprehension, baseline cranial nerve function, and functional assessment of swallow. Each item in the scale is scored according to severity. All scores from the different sub skills are tallied, and a composite score out of 200 is given. Depending on clinical, and neuroradiological findings that clarifies the anatomical location of the lesion, all patients were divided into 2 main groups according to the Oxfordshire Classification.[17] Group A - Anterior circulation strokes (ACS), which was further subdivided into total (TACS) or partial (PACS) stroke. Group B - Posterior circulation stroke (PCS), which was further subdivided into lateral medullary syndrome (LMS) and posterior cerebral artery (PCA) stroke. All patients were assessed for a relationship between dysphagia and severity of stroke using the Modified Rankin Scale (MRS),[18] as follows: 0-3 mild to moderate stroke, 4-6 severe stroke. All patients were assessed regarding the relationship between dysphagia and risk factors of stroke according to the Stroke Risk Scorecard (SRSC),[19] which further subdivides patients into low, moderate, and high risk groups. All patients were reassessed after one month by the MRS and MASA, with the exception that 11 cases were withdrawn from the study (we lost contact with them by telephone, and they did not return to us again after discharge), 4 of those were dysphagic (3 ACS and one PCS), and 7 were non-dysphagic (4 ACS and 3 PCS).

The Statistical Package for Social Sciences (SPSS Inc, Version 15, Chicago, IL) was used for data analysis. The Fisher's exact test was applied, with a p-value <0.05 considered to be significant.

Results. The mean age of our studied sample was 60.55 years (range 30-82) (SD=11.81). They were 40 males and 32 females. Sixty-eight patients had ischemic stroke, and 4 had primary intracerebral hemorrhage (ICH). Clinical examination revealed that 10 patients had TACS, 41 had PACS, 21 patients had PCS, 11 of them had LMS, and 10 patients had PCA. All of those patients had unilateral stroke manifestation either left or right sided, 41 patients had right side stroke manifestations and 31 had left side stroke manifestations. The demographic characteristics of the sample are given in **Table 1.** According to the clinical assessment protocol (MASA score), clinical evidence of dysphagia was detected in 41 cases and distributed as follows: 10 cases of TACS, 18 of PACS, 3 of PCA, and 10 of LMS. The severity of dysphagia was assessed using the MASA scoring system; it showed that 31 cases had normal swallowing. Forty-one cases were dysphagic, and the degree of dysphagia severity is shown in **Table 2.** The frequency of comorbid manifestations associated with dysphagia was assessed according to MASA score for both dysphagic and non-dysphagic patients and described in detail in **Table 3.** In this study, we estimated the clinical features of both PCS and ACS, including facial weakness, palatal weakness, dysphasia, dysarthria, contralateral weakness, cerebellar signs, sensory loss or hypoesthesia and others, only in dysphagic patients, as illustrated in **Table 4.** The severity of dysphagia was assessed in our sample in relation to the severity of stroke using the MRS, which showed an increase in dysphagia severity with increased stroke severity as shown in **Table 5.** Stroke risk factors were examined in our studied sample using the SRSC to ascertain any significant relation between the cumulative effect of stroke risks and the presence of dysphagia. Thirty (73.2%) dysphagic patients were found to be in the moderate risk group, 9 (22%) were in the low risk group, and only 2 (4.8%) patients were in the high-risk group. On the other hand, in the non-dysphagic patients group; 21 (67.5%) cases were classified as moderate risk, 7 (22.5%) as low risk, and 3 (10%) cases as high-risk. One month follow up using MRS for 61 patients from our studied sample showed that among 37 dysphagic cases, 4 (10.8%) of them did not showed any symptoms at all, 8 (21.6%) had slight disability, 10 (27%) had moderate to severe disability, and 15 (40.6%) patients died. Among the non-dysphagic patients, 16 (66.7%) did not show any symptoms, 6 (25%) had mild disability, 2 (8.3%) had moderate to severe disability, and no case fatality was registered in this group. There was significant morbidity, and mortality in the dysphagia group in comparison with the non-dysphagia group of patients

Table 1 - Demographic features in dysphagic and non-dysphagic subjects (N=72).

Features of stroke	Dysphagic		Non-dysphagic		*P*-value
	n (%)				
Age (years)					
30-40	2/41	(4.8)	4/31	(12.9)	0.3919
41-50	4/41	(9.8)	4/31	(12.9)	0.7186
51-60	11/41	(26.8)	8/31	(25.8)	1.0000
61-70	12/41	(29.3)	8/31	(25.8)	0.7959
71-82	12/41	(29.3)	7/31	(22.6)	0.5964
Gender					
Male	21/41	(51.2)	19/31	(61.3)	0.4754
Female	20/41	(48.8)	12/31	(38.7)	
Clinical syndrome					
TACS	10/41	(24.4)	0/31	-	
PACS	18/41	(43.9)	23/31	(74.2)	0.0389
LMS	10/41	(24.4)	1/31	(3.2)	
PCA	3/41	(7.3)	7/31	(22.6)	
Side of stroke					
Right	21/41	(51.2)	20/31	(64.5)	0.3379
Left	20/41	(48.8)	11/31	(35.5)	
Pathology					
Cerebral infarction	38/41	(92.7)	30/31	(96.8)	0.6294
Primary ICH	3/41	(7.3)	1/31	(3.2)	

TACS - total anterior circulation stroke, PACS - partial anterior circulation stroke, LMS - lateral medullary syndrome, PCA - posterior cerebral artery, ICH - intracerebral hemorrhage

Table 2 - Initial assessment of anterior and posterior circulation stroke cases according to MASA score (N=72).

Swallowing assessment (MASA)	Anterior circulation stroke		Posterior circulation stroke		Total		*P*-value
	n (%)						
Normal swallowing (MASA score=178-200)	23/51	(45.1)	8/21	(38.1)	31	(43.1)	0.0389
Mild dysphagia (MASA score=168-177)	19/51	(37.25)	1/21	(4.8)	20	(27.8)	0.0005
Moderate dysphagia (MASA score=139-167)	4/51	(7.8)	2/21	(9.5)	6	(8.3)	1.0000
Severe dysphagia (MASA score ≤138)	5/51	(9.8)	10/21	(47.6)	15	(20.8)	0.0005
Total	**51/51**	**(100)**	**21/21**	**(100)**	**72**	**(100)**	

MASA - Mann Assessment of Swallowing Ability

Table 3 - Baseline characteristic of clinical features from initial assessment according to MASA (N=72).

Swallowing assessment (MASA)	Dysphagic		Non-dysphagic		Total	
	n (%)					
Alert status	3	(7.3)	0	(0.0)	**3**	**(4.2)**
Cooperation	2	(4.9)	0	(0.0)	**2**	**(2.8)**
Auditory comprehension	8	(19.5)	2	(6.45)	**10**	**(13.9)**
Respiratory status	15	(36.6)	5	(16.1)	**20**	**(27.8)**
Respiratory rate for swallow	1	(2.4)	0	(0.0)	**1**	**(1.4)**
Dysphasia	3	(7.3)	1	(3.2)	**4**	**(5.6)**
Dyspraxia	2	(4.9)	1	(3.2)	**3**	**(4.2)**
Dysarthria	26	(63.4)	6	(19.35)	**32**	**(44.4)**
Saliva control	23	(56.1)	8	(25.8)	**31**	**(43.1)**
Lip seal	22	(53.7)	7	(22.6)	**29**	**(40.3)**
Tongue movement	17	(41.5)	9	(29.0)	**26**	**(36.1)**
Tongue strength	18	(43.9)	10	(32.3)	**28**	**(38.9)**
Tongue coordination	15	(36.6)	10	(32.3)	**25**	**(34.7)**
Oral preparation	25	(61.0)	8	(25.8)	**33**	**(45.8)**
Gag reflex	13	(31.7)	4	(12.9)	**17**	**(23.6)**
Palate function	12	(29.3)	3	(9.7)	**15**	**(20.8)**
Incomplete oral clearance	27	(65.85)	6	(19.35)	**33**	**(45.8)**
Delayed oral transit	28	(68.3)	9	(29.0)	**37**	**(51.4)**
Weak/absent cough reflex	17	(41.5)	4	(12.9)	**21**	**(29.2)**
Weak/absent voluntary cough	17	(41.5)	5	(16.1)	**22**	**(30.6)**
Impaired vocal quality	12	(29.3)	3	(9.7)	**15**	**(20.8)**
Tracheostomy	0	(0.0)	0	(0.0)	**0**	**(0.0)**
Pharyngeal phase function	16	(39.0)	2	6.45	**18**	**(25.0)**
Impaired pharyngeal response	19	(46.3)	3	(9.7)	**22**	**(30.6)**

MASA - Mann Assessment of Swallowing Ability

Table 4 - Main clinical features associated with dysphagia in both hemispheric and lateral medullary syndrome stroke (N=38).

Clinical features	Hemispheric stroke		Lateral medullary syndrome		*P*-value
	n (%)				
Facial palsy	20/28	(71.4)	0/10	(0.0)	0.0001
Weak/absent voluntary cough	9/28	(32.1)	7/10	(70.0)	0.0623
Incomplete oral clearance	22/28	(78.6)	4/10	(40.0)	0.0452
Palatal paresis	3/28	(10.7)	9/10	(90.0)	0.0001
Accumulation of saliva	20/28	(71.4)	2/10	(20.0)	0.0082
Slow laryngeal elevation (pharyngeal phase)	7/28	(25.0)	9/10	(90.0)	0.0005
Tongue weakness	16/28	(57.1)	2/10	(20.0)	0.0673
Absent gag reflex	6/28	(21.4)	8/10	(80.0)	0.0019
Dysarthria	16/28	(57.1)	9/10	(90.0)	0.1182
Dysphasia	6/28	(21.4)	0/10	(0.0)	0.1679
Dysphonia	6/28	(21.4)	6/10	(60.0)	0.0452
Contralateral sensory loss (restricted or complete)	28/28	(100)	0/10	(0.0)	0.0001
Crossed sensory loss	0/28	(0.0)	9/10	(90.0)	0.0001
Contralateral weakness (restricted or complete)	28/28	(100)	0/10	(0.0)	0.0001
Cerebellar signs	0/28	(0.0)	9/10	(90.0)	0.0001
Contralateral homonymous hemianopia	20/28	(71.4)	0/10	(0.0)	0.0001

Table 5 - Initial assessment of stroke severity in dysphagic and non-dysphagic patients according to Modified Rankin Scale (N=72).

Severity of stroke	Dysphagic		Non-dysphagic		*P*-value
	n (%)				
Mild stroke (0-2)	10/41	(24.4)	21/31	(67.7)	
Moderately severe stroke (3-5)	31/41	(75.6)	10/31	(32.3)	0.0003
Total	**41/41**	**(100)**	**31/31**	**(100)**	

Table 6 - One-month follow-up assessment of anterior and posterior circulation stroke cases according to MASA score (N=22).

Swallowing assessment (MASA)	Anterior circulation stroke		Posterior circulation stroke		Total		*P*-value
	n (%)						
Normal swallowing (MASA score=178-200)	14/18	(77.8)	0/4	(0.0)	14	(63.6)	0.0096
Mild dysphagia (MASA score=168-177)	2/18	(11.1)	1/4	(25.0)	3	(13.6)	0.4701
Moderate dysphagia (MASA score=139-167)	1/18	(5.6)	1/4	(25.0)	2	(9.9)	0.3377
Severe dysphagia (MASA score ≤138)	1/18	(5.6)	2/4	(50.0)	3	(13.6)	**0.0727**
Total	**18/18**	**(100)**	**4/4**	**(100)**	**22**	**(100)**	

MASA - Mann Assessment of Swallowing Ability

(p=0.0002). According to follow up assessment using MASA score after one month for the remaining 22 dysphagic patients, the results showed that dysphagia patients of ACS have better outcome than PCS patients, as illustrated in **Table 6**. Further analysis of these results showed that among 25 dysphagic ACS cases (lost contact with 3 patients), 14 (56%) cases improved, while 4 (16%) were still dysphagic, and 7 (28%) died after one month; and among 12 dysphagic PCS cases (lost contact with one patient), there was no significant improvement noticed, and 4 (33.3%) cases were still dysphagic, while 8 (66.7%) patients died. There was a significant difference between the number of improved patients between the ACS and PCS cases (p=0.0009).

Discussion. Dysphagia after stroke is a common distressing symptom, and its detection is an important part of acute stroke management. The presence or absence of acute dysphagia after stroke is different in different studies; those differences are mainly attributed to variations in the method of identification, time after stroke, and lesion location in the studied samples. The reported percentage for dysphagia in studies enrolling acute stroke patients, regardless of lesion location, was lowest with screening identification (37-45%), higher with clinical testing (51-55%), and highest with instrumental testing (64-78%).[20-24] In this study, the patients were assessed for dysphagia in acute stroke within 72 hours from admission clinically depending on MASA score, and we found that 55% of ACS cases were associated with dysphagia, which was comparable to the results of Gordon (56%),[24] and that of Giselle's findings (51%).[20] Ninety-one percent of the LMS studied sample had dysphagia due to PCA, and these findings are comparable to the study of Aydogdu (99%).[21] Dysphagia of LMS patients had longer, if ever, recovery time and more severe presentation clinically than hemispheric stroke dysphagia. In this study, we found that none of the LMS patients had recovered in the one month follow period, and these findings are not compatible with Logemann's[25] and Vigderman's[26] findings, and this is mostly due to a longer period of follow up, which shows a good recovery period for more patients with LMS. While in ACS patients, we found that 56% of dysphagic patients had excellent improvement within the first month, similar to the findings of Mann[20] and Kidd.[27]. This was explained by Hamdy and others in their functional neuroanatomical correlations due to bilateral cortical representation.[28-31]

In this study, a higher incidence of symptoms related to the oral phase of swallowing was found in hemispheric stroke, while symptoms associated with the pharyngeal phase of swallowing and laryngopharyngeal paresis were mostly encountered in patients with LMS, similar to the findings of Aydogdu.[21] This can be attributed to neuroanatomical facts in which volitional swallowing in humans has multi regional cerebral representation that is strongest within the sensorimotor cortex and cerebellum, and this presentation is distributed bilaterally at many levels in the brainstem and cortex. Therefore, a transient dysphagia would be expected in a unilateral hemispheric stroke due to this bilateral cortical representation.[30,31] This finding may indicate that there is a different mechanism of dysphagia between LMS and hemispheric stroke. In LMS, the main abnormality was observed in the pharyngeal phase of the swallowing reflex, and this cannot be solely explained by the dysfunction of some of the corticobulbar fibers, because in such anatomic involvement, the swallowing reflex would remain normal.[32,33] Therefore, in PCS due to LMS, the main reason for an abnormal swallowing function should be sought within the neural structures located in the medulla oblongata. No significant differences were observed in this study regarding relation of demographic features

with dysphagia, as also observed by Vigderman.[26] Most of the dysphagic patients in this study were categorized to have moderate or low risks of stroke, and the same applied for the non-dysphagic patients, indicating no direct association between the presence of risk factors and dysphagia in acute stroke. We also found that most cases of deaths occurred among the moderate and low risk groups, indicating no direct association between risk factors and outcome of stroke in dysphagic patients, which is similar to the study of Hakan.[34]

The present study showed that dysphagia in acute stroke increases mortality and morbidity even in patients with no reduction in level of consciousness, as 40.5% of dysphagic patients died in the one month follow up period and in most death was caused by aspiration pneumonia, while there was no death among the non-dysphagic group. A similar finding (42%) was reported by Wade.[35] In this study, we found that different pathologies of stroke (intracerebral hemorrhage or ischemia) had no significant relation for increasing dysphagia in acute stroke; this was a contradictory finding to that mentioned by Paciaroni.[36] The reason for this could be because we used a different clinical assessment method for dysphagia, and may be due also to a smaller intracerebral hemorrhage sample in our study. No significant difference was observed in our study regarding whether hemiplegia or stroke is right sided (51%) or left sided (49%) in predisposition for acute dysphagia, and this is comparable to the findings of Mann.[13] While collecting data from some patients in this study, we faced major limitations due to difficulties with communication especially in aphasic patients or severely diarthrotic patients. Also in following some patients, as there is great difficulty in visiting patients' homes or contacting them after discharge from the hospitals, because of the security issues in Iraq.

In conclusion, dysphagia is a very common symptom after stroke, and it is more frequent and severer in patients with PCS due to LMS than in patients with cerebral hemispheric lesions of ACS. There was no statistical significant difference regarding dysphagia in intracerebral hemorrhage or cerebral infarction. There were no significant differences whether right or left sided stroke is superior to others in causing dysphagia in both ACS and PCS. There was no association between the presence of risk factors with both severity of dysphagia and prognosis. There was no significant relation between demographic features with the presence of dysphagia. Dysphagia can be an indicator of the severity of stroke causing higher mortality and morbidity in affected patients. Further study should focus on finding better ways to manage and use drugs to treat dysphagia.

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