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Early Transient Dysphagia in Acute Brain Stem Infarction

Akut Beyin Sapı İnfarktlarında Erken Geçici Disfaji

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ABSTRACT

Background: Electromyographical techniques are not used to investigate early transient dysphagia in stroke patients with brainstem infarction (BSI). This study aimed to employ electrophysiological methods to determine the presence of swallowing difficulties and the underlying pathophysiology of dysphagia in patients who have experienced an acute brainstem stroke.

Materials and Methods: This prospective study included 20 age-matched healthy individuals and 53 patients with BSI who experienced stroke within nine days of each other. Electrophysiological techniques, including dysphagia limit (DL) and sequential water swallowing (SWS) tests, were employed.

Results: Clinical dysphagia was observed in 21.0% of patients with BSI. Dysphagia, as determined by DL and SWS tests, was present in 52.7% of patients. Patients with cerebellar and medullary infarctions exhibited more severe dysphagia. Subclinical dysphagia was found in 54.7% of patients. Within 4 weeks, two-thirds of patients regained their ability to swallow, while one-third remained dysphagic.

Conclusion: Dysphagia is a severe symptom complex that can be fatal in a significant proportion of stroke cases. This study examined the electrical anomalies associated with swallowing in patients with acute BSI and explored the possible pathophysiological mechanisms underlying swallowing disorders caused by brainstem stroke. The electrophysiological techniques demonstrated in this study are quick, simple to use, noninvasive, and safe for patients, making them valuable for identifying subclinical dysphagia.

Keywords: Brainstem stroke, swallowing tests, electrophysiological methods, sequential water swallowing test, clinical dysphagia

ÖZ

Amaç: Bugüne kadar, beyin sapı infarktı (BSI) olan felçli hastalarda erken geçici disfajiyi araştırmak için elektromiyografik teknikler kullanılmamıştır. Bu çalışma, akut beyin sapı infarktı geçirmiş hastalarda yutma güçlüğünün varlığını ve disfajinin altta yatan patofizyolojisini belirlemek için elektrofizyolojik yöntemler kullanmayı amaçlamaktadır.

Gereç ve Yöntemler: Bu prospektif çalışmaya, yaş uyumlu 20 sağlıklı birey ve dokuz gün içinde inme geçiren beyin sapı enfarktüsü (BSI) olan 53 hasta dahil edildi. Disfaji limiti (DL) ve ardışık su yutma (SWS) testlerini içeren elektrofizyolojik teknikler kullanıldı.

Bulgular: Hastaların %21'inde klinik disfaji gözlendi. DL ve SWS testleri ile belirlenen disfaji, hastaların %52,7'sinde mevcuttu. Serebellar ve medüller enfarktüsü olanlarda daha şiddetli disfaji görüldü. Hastaların %54,7'sinde subklinik disfaji saptandı. Dört hafta içinde hastaların üçte ikisi yutma yeteneğini yeniden kazandı, üçte birinde disfaji devam etti.

Sonuç: Disfaji, inme olgularının önemli bir kısmında ölümcül olabilen ciddi bir semptom kompleksidir. Bu çalışma, akut beyin sapı infarktlı hastalarda yutmayla ilişkili elektriksel anomalileri incelemekte ve beyin sapı infarktının neden olduğu yutma bozukluğunun altında yatan olası patofizyolojik süreçleri araştırmaktadır. Bu çalışmada gösterilen elektrofizyolojik teknikler hızlıdır, kullanımı basittir, invaziv değildir ve hastalar için güvenlidir; bu da onları subklinik disfajiyi tanımlamada değerli kılar.

Anahtar Kelimeler: Beyin sapı infarktı, yutma testleri, elektrofizyolojik yöntemler, ardışık su yutma testi, klinik disfaji

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Introduction

Dysphagia is a recognized complication of both acute and hemorrhagic stroke, with 37.0-78.0% of patients with acute stroke experiencing difficulties in swallowing (1). The prevalence of dysphagia varies depending on the timing and method of evaluation (2). It is more common in patients with acute stroke than in those often presumed to have the condition, even when they do not exhibit apparent symptoms related to deglutition. Aspiration pneumonia, with an incidence ranging from 13.0% to 33.0%, is more common among stroke patients with dysphagia (3,4,5). Impaired swallowing is associated with a three-fold increase in mortality risk, primarily due to pneumonia (6). Early identification of dysphagia in patients with acute stroke significantly reduces the risk of aspiration pneumonia, a potentially fatal but preventable complication (7). Although dysphagia often resolves within a few days to a week after stroke (8), studying the early physiological changes in oropharyngeal swallowing is crucial for early diagnosis using bedside examinations and noninvasive screening methods (7,9,10). Nevertheless, 2-12% of stroke patients continue to experience dysphagia in the first month, and 7% may still struggle with swallowing 3 months after the stroke (11,12,13,14). Mild dysphagia is observed in nearly all patients with stroke (15). The stroke region plays a significant role in the prevalence of dysphagia, with dysphagia being highly prevalent in patients with carotid artery involvement (8). Approximately 25% of strokes are caused by brainstem lesions, of which dysphagia is a frequent consequence (16). In brainstem stroke (BSS) patients, dysphagia tends to be more severe and less likely to resolve spontaneously compared with those with hemispheric stroke. Although lateral medullary infarction (LMI) has been studied in relation to dysphagia, few studies have explored dysphagia in other brainstem regions (17,18). This study aimed to investigate voluntary swallowing in patients with acute BSS using electrophysiological techniques to identify the pathophysiological changes associated with neurogenic oropharyngeal dysphagia, to elucidate the spectrum and prevalence of both subclinical (acute transient dysphagia) and overt dysphagia in relation to the involvement of various brainstem regions, and to determine whether acute temporary dysphagia, as identified through clinical procedures, resolves within a week or persists beyond hospital discharge.

Materials and Methods

We examined 53 patients (34 males, 19 females) with brainstem infarction (BSI) affecting unilateral regions. The mean age of the patient sample was 64.9 years (range 37-84). This study included patients presenting to our Neurology department with an acute first stroke occurring between 1 and 9 days before presentation (mean duration five days). All patients were conscious and medically stable. We categorized patients into the following three groups:

Group A: Patients without signs or symptoms related to swallowing since stroke onset.

Group B: Patients with dysphagic complaints and symptoms from stroke onset that were completely resolved by hospital discharge.

Group C: Patients with clinical dysphagia observed at stroke onset that persisted until hospital discharge.

Groups A and B remained hospitalized for 3 weeks, whereas group C was discharged after 4 weeks. Patients who were confused or comatose were excluded. We also included 20 healthy volunteers (18 females, 2 males) without neurological, cardiovascular, cerebrovascular, or oropharyngeal disorders as a control group. The mean age of the control group was 63.9 years (range 56-75). All control subjects were selected from hospital staff and underwent normal neurogenic examinations. We performed a neurological evaluation of each patient to confirm the stroke diagnosis, clinical syndrome, pathological subtype, and functional effects using the Functional Independence Measurement score and the Level of Cognitive Functioning score.

Magnetic resonance imaging was used consistently to confirm stroke diagnosis, examine lesion location, and identify stroke type (ischemic or hemorrhagic). Enrollment was limited to patients with acute BSI. At admission, clinical and biochemical markers were assessed to evaluate dietary and hemodynamic conditions. A detailed neurological examination of the face, tongue, mouth, pharynx, and larynx was performed for each patient, and the patients were thoroughly questioned regarding dysphagia and aspiration. Within 2 days of hospitalization, a neurologist conducted a clinical bedside test to evaluate swallowing abnormalities. Dysphagia severity (DD) was ranked as follows (19):

Grade I (DD-I): No clinical or reported dysphagia.

Grade II (DD-II): The patient did not report dysphagia, but clinical examination revealed very mild dysphagia, including inappropriate voluntary cough and dysarthria.

Grade III (DD-III): The patient reported dysphagia but managed swallowing with non-oral feeding. Additional clinical indications included wet voice, cough during trial swallows, and dysphonia.

Grade IV (DD-IV): Evident clinical dysphagia requiring non-oral feeding and associated with aspiration.

Assessment of Voluntary Swallowing Electrophysiologically

Patients were instructed to sit in a chair and maintain an upright, neutral head position throughout the examination. Surface silver chloride electrodes were placed on both sides of the midline under the chin at a distance of 1 cm to record the activity of the submental muscle (SM) complex, including the mylohyoid, geniohyoid, and anterior digastricus muscles. Activities were recorded using a four-channel electromyograph (EMG) instrument (Nicolet Viking-V11.0). The analysis duration was 20 s. After filtering (100 Hz to 10 kHz), the signals were amplified, rectified, and integrated. Concurrently, a respiratory signal was acquired using a nasal cannula (Sleep Sense SLP Tel Aviv Israel) placed at the nasal entrance and linked to an airflow sensor transducer. The two EMG channels were connected to the breathing sensor output. The direction of the airflow was recorded using positive and negative polarities for expiration and inspiration, respectively. For respiratory recordings, EMG filters were set at a bandpass frequency of 0.2-30 Hz. The entire analysis time was 20 s.

Simultaneously, electrocardiogram data were captured using silver chloride cup electrodes, with one electrode positioned on the second left intercostal region and the other on the dorsum of the hand. The signals were amplified and filtered (band pass 0.2 Hz to 30 Hz).

Dysphagia Limit (DL)

The DL technique was previously described (19). This study employed the same electrophysiological methods to investigate the "DL", which is sensitive and specific for tracking neurogenic dysphagia. The DL is based on the physiological phenomenon where an oral bolus of a large liquid volume is split into two or more portions and effectively swallowed. Normally, individuals can swallow a 20 mL bolus of water in a single swallow. However, those with reduced swallowing ability may require two or more swallows for a bolus of less than 20 mL, referred to as piecemeal deglutition. Piecemeal deglutition at a volume of 20 mL is considered abnormal and indicates dysphagia. The DL is the volume of water below 20 mL at which piecemeal deglutition occurs. Electrophysiological recordings can detect swallowing difficulties of various etiologies.

Participants received 3, 5, 10, 15, and 20 mL of water in steps. The examiner instructed the subjects to immediately swallow each bolus. A graduated syringe was used to progressively deliver water into the mouth behind the incisors; swallows were initiated with water on the tongue and the tip of the tongue touching the upper incisors. Rapid rearward-directed water delivery was avoided to prevent the early escape of water flow to the pharynx and possible gag response. At the examiner's command, oscilloscopic traces were initiated for swallowing. If piecemeal deglutition or any indication of subglottic aspiration (e.g., wet voice or coughing) was observed, the examination was halted. The subjects were asked to speak between each test. If fragmented deglutition was suspected, the process was repeated and documented using the same volume of water. The term "normal DL" refers to piecemeal deglutition occurring in healthy individuals when swallowing at or above a water volume of 20 mL. Otherwise, a water volume of 20 mL was considered abnormal (21) (Figure 1).

Sequential Water-Swallowing (SWS)

All patients and control group members underwent the "SWS" test as previously described (22,23). Participants were instructed to sip 50 mL of water continuously from a cup as they would in daily life. The water temperature was 25°C. EMG traces appeared on the screen for a few seconds before the start command for SWS was given. The analysis took 20s. Each subject completed three repetitions of the SWS test, with the third trial being considered the most accurate measurement. The following variables were recorded:

Number of swallows occurring while drinking water.
Total time spent drinking 50 mL of water continuously.
Total duration of apnea during the 50 mL SWS.

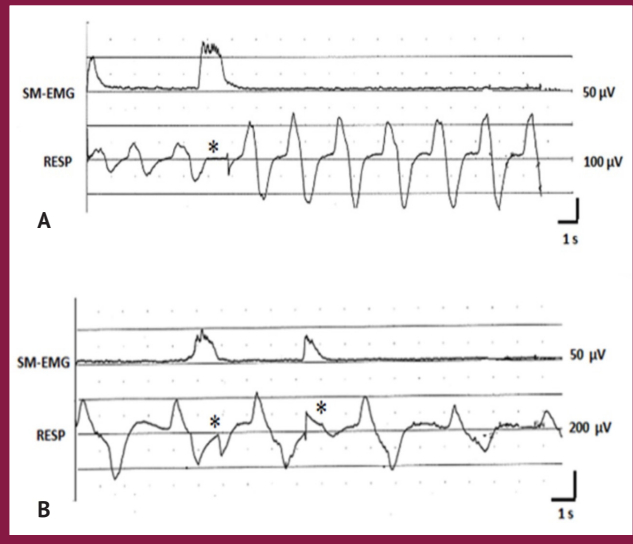

Figure 1. A) Five milliliters of water swallowed by a patient with mesencephalic infarction. B) DL of 5 mL discrete swallows in a patient with pontin infarction who swallowed water twice in a short interval
**Apnea during water swallowing, DL: Dysphagia limit, SM: Submental muscle, EMG: Electromyograph, RESP: Respiration*

During SWS, the number of swallows was determined by counting the periodic motions of the SM-EMG. The total duration of SWS was calculated from the start of the first swallow burst at the end of the final swallow burst using SM-EMG traces. The swallowing apnea duration was measured at the end of the previous respiratory cycle until the reappearance of the first respiratory cycle. Patients provided written informed consent, and the study was approved by the institutional research ethics review board. The study was approved by the İzmir Kâtip Çelebi University Faculty of Medicine Clinical Research Ethics Committee (approval number: 75, date: 07.05.2014).

Statistical Analysis

Descriptive summary statistics were reported as mean ± SD, minimum, and maximum, depending on data distribution. Categorical variables were assessed using chi-square tests and Fisher's exact test. Groups were compared using Kruskal-Wallis and Mann-Whitney U tests for continuous variables, depending on the data distribution. Post hoc comparison testing was completed using Bonferroni correction. All analyses were two-tailed, and significance was set at 5%. The statistical analysis was conducted using SPSS 15.0 for Windows.

Results

Imaging analyses confirmed that all patients had ischemic stroke and no prior cerebrovascular disease. Among the 53 patients, 30 (56.6%) had pontine infarcts, 13 (24.6%) medullary infarcts (7 lateral, 6 medial), 5 (9.4%) mesencephalic infarcts, and 5 (9.4%) cerebellar infarcts. Of the 53 BSS patients, 11 (21%) exhibited clinical dysphagia, with infarcts located in the medulla oblongata (6), pons (4), and cerebellum (1). The remaining 42 (79%) BSS patients reported no difficulty swallowing.

DL

The DL was >20 mL of water in all normal subjects, whereas it was definitively pathological and 20 mL in 5 of 11 (45%) BSS patients with Grade II clinical dysphagia (ranging from 3-15 mL of water). Forty-two patients (79%) were clinically non-dysphagic, but a pathological DL was recorded in 15 of these 42 patients (35.7%), indicating subclinical dysphagia, with DL values ranging between 1 and 20 mL. Overall, among the 53 patients with and without clinical swallowing complaints, a total of 20 patients (37.7%) demonstrated pathological DL. The highest prevalence of pathological DL was observed in patients with cerebellar infarction (3 out of 5 patients), followed by those with medullary infarction (5 out of 13 patients) and pontine infarction (10 out of 30 patients).

SWS

The total duration of swallowing ($p<0.001$), swallowing apnea ($p<0.001$), and the number of swallows ($p<0.05$) were significantly prolonged in all BSS patients compared with the normal subjects (Table 1). Figure 2 illustrate the SWS values in patients with pontine infarction and healthy subjects. The duration of swallowing was significantly longer in patients with pontine infarcts than in other brainstem locations ($p<0.05$). Similarly, the duration of swallowing apnea was significantly prolonged in patients with pontine infarction ($p<0.05$). Although the duration of swallowing apnea was longer in subjects with other brainstem involvements than in healthy subjects, the correlation did not reach a significant level. The total number of swallows did not significantly differ between patients with BSS and healthy subjects (Table 2).

During the 50 mL SWS, two healthy participants (10%) demonstrated compensatory respiration cycles (CRC), particularly between the last successive swallow intervals of SWS. CRCs were recorded in 27 patients (52.9%) and observed more than once, appearing not only during the last inter-swallow interval but also during the middle inter-swallow interval of SWS. Figure 3 illustrates CRC in a patient with medial medullary infarction (MMI). In patients with BSS, subclinical dysphagia was frequently observed in the SWS test. Increased duration of swallowing time,

Table 1. Comparison of the total duration of swallowing, swallowing apnea, and number of swallows in all BSS patients and normal subjects

		Mean± SD	Minimum	Maximum	p-value
Total swallowing duration	Normalsubjects	6.91±1.36	4.2	9.5	**p<0.001**
	Patients	10.24±3.37	4.5	17	
Swallowingapnea	Normal subjects	6.89±1.49	3.8	9.5	**p<0.001**
	Patients	8.97±3.31	3.5	16	
Number of swallows	Normal subjects	5.00±0.97	4	7	**p<0.05**
	Patients	5.86±2.16	3	12	

BSS: Brainstem stroke, SD: Standard deviation

swallow apnea, increased CRC, and to a lesser extent, an increased number of swallows were found in 21 patients (50%) without clinical dysphagia. When the pathological values of the electrophysiological tests (either DL, SWS, or both) were calculated for the clinically normal patients (42 patients), 23 out of 42 BSS patients (54.7%) presented with subclinical dysphagia (Figure 4A).

Conversely, in the 11 patients with BSS, clinical dysphagia was evident at the onset of the stroke. However, four of these patients showed clinical improvement in the end of their hospital stay. Furthermore, three patients were found to be electrophysiologically normal despite mild clinical symptoms related to oropharyngeal dysphagia. The last patient, who was electrophysiologically pathologically diagnosed with dysphagia, demonstrated recovery both clinically and electrophysiologically upon a second examination (Figure 4B).

The remaining seven patients were clinically dysphagic from onset until hospital discharge. Four of these patients were also electrophysiologically dysphagic (one with pontine, two with medullary, and one with cerebellar infarction). Three other patients with medullary infarction were clinically dysphagic, but their electrophysiological test results were within normal limits. Figure 5 illustrates the clinical course of dysphagia in these three groups of patients.

In summary, when comparing all individual pathological findings between patients with BSI and healthy subjects, clinical assessment revealed dysphagia in 21.5% of patients, pathological DL in 37.7%, and SWS abnormalities in 49%. When combining the electrophysiological findings (DL+SWS) for all patients, 52.7% of the BSI patients investigated in the first week had pathological results.

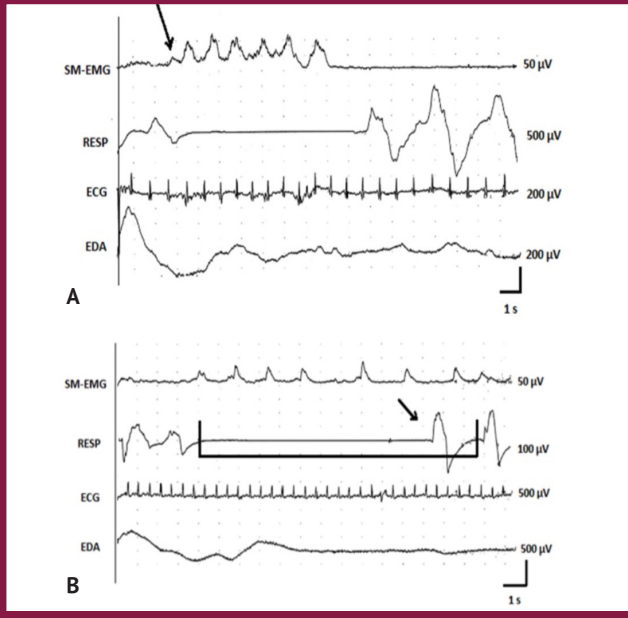

Figure 2. A) The total duration of swallowing, swallowing apnea, and the number of swallows were prolonged in a normal subject and the "foreburst" occured before the apnea interval in the same patient.B) The duration of apnea was significantly prolonged in a patient with pontine infarction who also demonstrated a compensatory respirator cycle
SM: Submental muscle, EMG: Electromyograph, ECG: Electrocardiogram, RESP: Respiration, EDA: Electrodermal activity

Table 2. Comparison of the total duration of swallowing, swallowing apnea, and number of swallows among all BSS patients

50 mL SWS	Group	Mean± SD	Minimum	Maximum	p-value
Total swallowing duration	Pons	11.27±3.49	5	17	**0.042[a]**
	Mesencephalon	8.90±2.88	6	12	
	Bulbus	8.65±2.80	4.5	13,5	
	Cerebellum	8.86±2.51	6.3	12	
Swallowing apnea	Pons	9.94±3.45	4	16	**0.031[b]**
	Mesencephalon	6.80±2.77	3.5	9.5	
	Bulbus	8.11±2.85	5	13.5	
	Cerebellum	7.20±1.96	5.5	10	
Number of swallows	Pons	6.34±2.18	4	12	**0.201[c]**
	Mesencephalon	4.80±1.79	3	7	
	Bulbus	5.36±2.50	3	11	
	Cerebellum	5.20±0.84	4	6	

[a]p (pons vs. bulbus)=0.02; [b]p (pons vs. mesencephalon)=0.046; [c]p (pons vs. mesencephalon)=0.041, BSS: Brainstem stroke, SD: Standard deviation, SWS: Sequential water swallowing

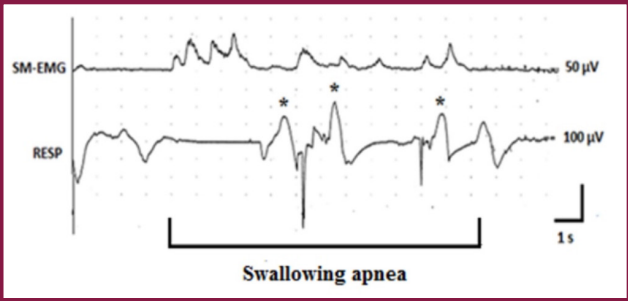

Figure 3. Compensatory respirator cycles (*) in a patient with medial medullary infarction
SM: Submental muscle, EMG: Electromyograph, RESP: Respiration

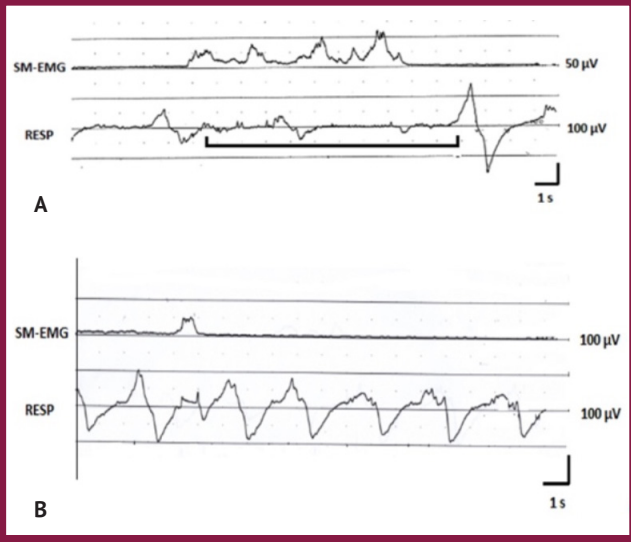

Figure 4. A) DL in 10 mL discrete swallow and prolonged apnea duration in a patient with pontine infarction. B) The second examination showed that the patient had recovered both clinically and electrophysiologically
DL: Dysphagia limit, SM: Submental muscle, EMG: Electromyograph, RESP: Respiration

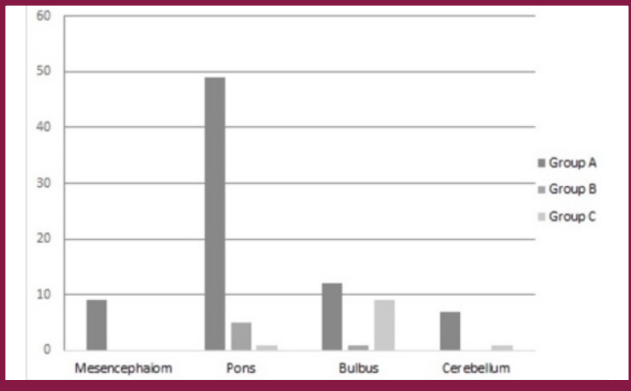

Figure 5. Clinical course of dysphagia in three groups of patients

Discussion

This study primarily aimed to investigate the incidence and characteristics of dysphagia in patients with acute BSS using electrophysiological techniques. Our findings indicate that even in patients with mild-to-moderate BSS, there is a high incidence of dysphagia that can be detected using straightforward and secure electrophysiological methods. Notably, more than half of the patients exhibited subclinical or mild dysphagia by the ninth day after stroke onset, highlighting the sensitivity of these techniques in the early detection of concealed swallowing issues.

Dysphagia is a significant concern in stroke patients, particularly because it occurs in approximately 50% of ischemic stroke cases (9). Early detection of dysphagia is crucial for reducing the risk of aspiration-related complications, such as pneumonia, which underscores the need for diligent bedside examinations and simple screening tests (6,7,8,10). In recent studies, it was stated that delayed dysphagia adversely affects the improvement of dysphagia in patients with stroke and needs to be identified early (11). Our study contributes to this body of knowledge by demonstrating that neurophysiological methods, specifically the DL and SWS tests, are effective tools for identifying both clinical and subclinical dysphagia in patients with BSS. The severity of dysphagia in patients with stroke varies depending on the location of the infarct. Our findings showed that although dysphagic symptoms are minimal in cases of mesencephalic infarction, they are significantly more severe in patients with pontobulbar involvement. This can be attributed to the impact on critical swallowing centers and networks, such as the central pattern generator (CPG), nucleus tractus solitarii, and nucleus ambigus (24,25). In contrast, neurogenic oropharyngeal dysphagia is less frequent and less severe in hemispheric strokes, likely due to early neuroplasticity in the non-affected hemisphere that aids recovery (26).

The exact nature of transient dysphagia in BSS remains unclear. However, in LMI, early-stage regional disconnection between two swallowing networks may result in severe pharyngeal dysphagia, similar to chronic Wallenberg syndrome (27,28). Dysphagia in LMI is more common in patients with rostral lesions, with cortical connections potentially aiding neuroplasticity (29). Our study found a higher incidence of dysphagia in patients with LMI (71%) than in those with MMI cases (33%). However, due to the small sample size of patients with medullary infarction, our electrophysiological findings did not show significant differences in the oropharyngeal effects between MMI and LMI.

Both LMI and MMI cases often exhibited abnormalities in DL and SWS, with only one normal case in each group (6 out of 7 vs. 5 out of 6 pathological). A high incidence of subclinical or mild dysphagia is expected given the involvement of the swallowing CPG in the lateral medullary region and the corticobulbar and corticospinal tracts in the medial medullary region. Pontine infarction was the most common in our patient group (56% of all cases), with all cases being unilateral. Severe clinical dysphagia, like quadriparesis, is observed in patients with bilateral pontine infarcts (28). In contrast, our pontine cases were clinically mild but still showed signs of dysphagia, particularly the prolonged duration of swallowing 50 mL of water during SWS. This is likely due to the involvement of the pontobulbar trigeminal system, especially if oropharyngeal input to the pontobulbar trigeminal afferent pathway is compromised. Sensory input to the pontobulbar area comes from the vagal, glossopharyngeal, and trigeminal-maxillary nerves, which innervate the oropharyngeal muscles. Any sensory impairment in this system may lead to dysphagia and aspiration (30,31). Thus, in pontine infarction, the trigeminal system may be partially damaged. Additionally, unilateral facial paresis in some pontine lesions can modestly contribute to dysphagia (32). Abnormal conduction in the afferent and efferent swallowing pathways, rather than a core anomaly in the medullary CPG, is likely responsible for early transient dysphagia, particularly in patients with pontine infarction.

It was surprising to find a high number of swallowing problems in cerebellar infarction despite the few cases. Oropharyngeal dysphagia is rarely observed in chronic neurological disorders with severe cerebellar syndrome. However, in this study, cerebellar infarction may have distended and compressed the lower brainstem structures related to deglutition, or the inferior cerebellar peduncle (corpus restiform), which is anatomically connected to the lateral medullary region, may have been involved. The early transient dysphagia observed in cerebellar infarction cases could be attributed to the indirect involvement of the CPG in the medullary region. Neuroimaging studies have shown that experimentally induced swallowing in healthy individuals can produce hyperdense fields within the cerebellum (33). Early transient dysphagia in patients with BSS can be explained by the data obtained from the DL. If a patient has overt or silent dysphagia, they cannot swallow water volumes above the 20 mL bolus and must resort to piecemeal deglutition with less than 20 mL of water. In our cohort, this type of defective swallowing was detected in approximately 39% of all patients investigated.

Piecemeal deglutition is essentially a compensatory and protective mechanism for oropharyngeal swallowing. However, it also indicates subclinical and clinical dysphagia in neurological patients, even in the absence of clinical complaints, as reported previously (20,34). Finally, DL has been recognized as a reliable, non-invasive quantitative test with high sensitivity (92%) and specificity (91%) for detecting and monitoring both clinical and subclinical dysphagia (20). Thus, in our study, early transient dysphagia appeared to be a benign process; while patients may be clinically non-dysphagic, the DL can still indicate previous dysphagia states after the "overt dysphagia" days have passed. As a more recent screening test, SWS offers a physiological approach to diagnosing dysphagia, making it a valuable tool. In our study, 49% of BSS patients exhibited clear abnormalities in the 50 mL SWS test, with a significant increase in the duration of swallowing and swallowing apnea compared with age-matched controls. Patients also showed a change in the regularity and rhythmicity of their swallowing pattern, often resulting in an irregular, arrhythmic pattern. Such abnormalities, which were previously reported in the literature, suggest that the brainstem CPG is involved in arrhythmic swallowing and compensatory respiration (23,35). The impaired regularity of the swallowing pattern, coupled with compensatory respiration, further supports the notion that early transient dysphagia in BSS patients may be linked to CPG dysfunction. Given the high incidence of early transient dysphagia, which is detected in 52.7% of BSS patients, combining electrophysiological methods for comprehensive evaluation. These non-invasive, simple-to-apply techniques are especially suited for use in acute stroke patients and can be employed in various clinical settings, including intensive care. Any detected electrophysiological abnormality should prompt close monitoring of the patient in the subsequent days and months.

This study has several limitations that should be considered when interpreting the results. First, the sample size, particularly for patients with specific types of BSIs, such as medullary infarction, was relatively small, which may limit the generalizability of our findings. Additionally, although the electrophysiological methods used in this study are non-invasive and straightforward, they may not fully capture the complexity of dysphagia, especially in patients with subtle or transient symptoms. Furthermore, the cross-sectional study design did not allow for the assessment of long-term outcomes related to dysphagia in these patients. Future studies with larger sample sizes and longitudinal follow-up are needed to confirm these findings and to provide a more comprehensive understanding of dysphagia in stroke patients.

Study Limitations

The small sample size and the small number of male patients are the limitations of our study.

Conclusion

In conclusion, this study highlights the importance of early detection and monitoring of dysphagia in patients with acute BSS. The use of electrophysiological methods, such as DL and SWS, is a sensitive and reliable method for identifying both clinical and subclinical dysphagia, even in patients with mild to moderate BSS. The high incidence of dysphagia detected in this study underscores the need for diligent screening and follow-up in this patient population to prevent complications such as aspiration pneumonia. Given the simplicity and non-invasive nature of these methods, they can be effectively employed in both acute and chronic care settings. Further research is warranted to explore the long-term impact of these findings and to refine the use of electrophysiological techniques in the management of dysphagia in stroke patients.

Ethics

Ethics Committee Approval: The study was approved by the İzmir Kâtip Çelebi University Faculty of Medicine Clinical Research Ethics Committee (approval number: 75, date: 07.05.2014).

Informed Consent: Patients provided written informed consent.

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Authorship Contributions

Surgical and Medical Practices: N.G.B., Concept: N.G.B., Y.B., Ş.A., N.G., T.K.İ., Design: N.G.B., Y.B., Ş.A., Data Collection or Processing: N.G.B., Ş.A., Analysis or Interpretation: N.G.B., N.G., T.K.İ., Literature Search: N.G.B., Writing: N.G.B.

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