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Determinants of dysphagia after stroke: the role of brain injury location

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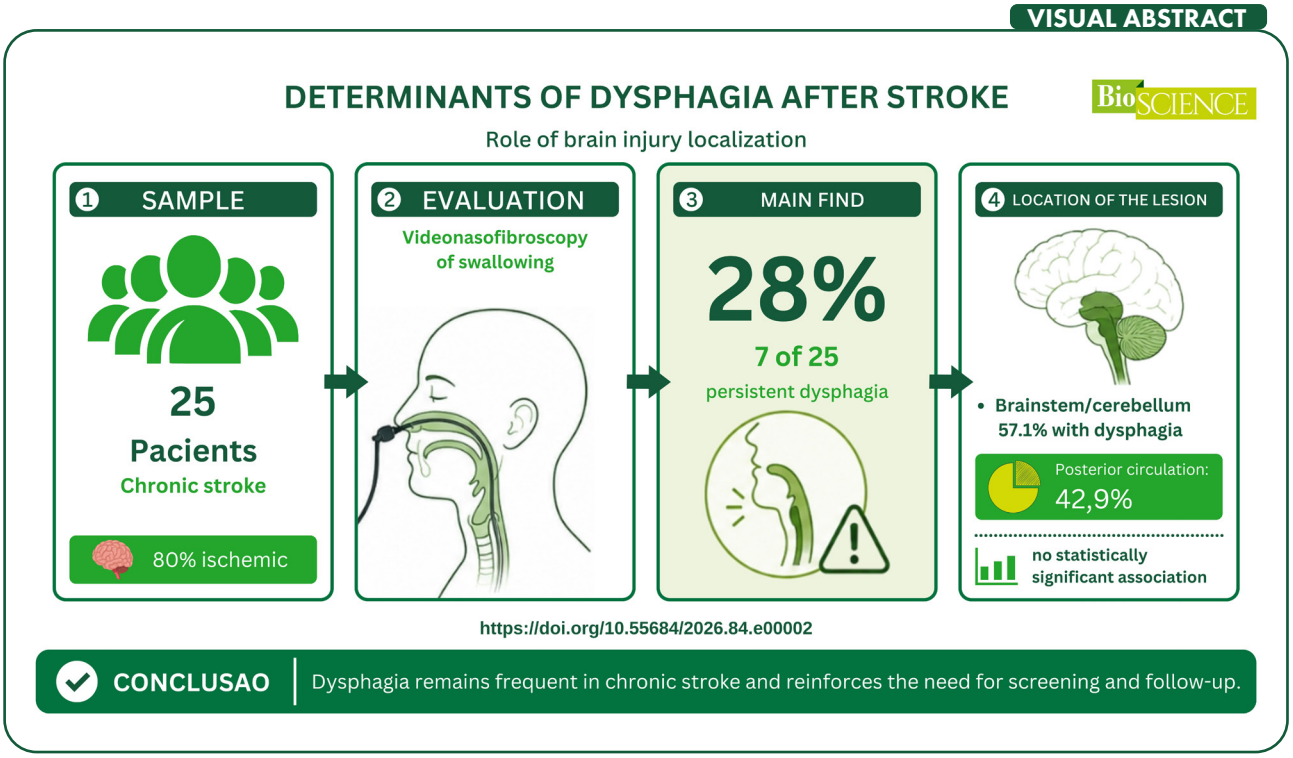
VISUAL ABSTRACT



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Author's contribution
Milton Marchioli: Conceptualization, investigation, methodology, writing.

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Central Message
Stroke is the second leading cause of death in Brazil, surpassed only by acute myocardial infarction. May present clinically as ischemic, thrombotic, embolic or hemorrhagic. Among the classic signs is dysphagia. In swallowing studies, the incidence of swallowing can vary between 41-73%. This variation is justified due to the phases when the diagnosis is made, that is, acute, subacute and/or chronic phases of stroke.

Perspective
The gold standard for diagnosing post-stroke dysphagia is swallowing videofluoroscopy. Videonasofibroscopy is considered a highly accurate test, complementary to clinical diagnosis, and often preferred in clinical settings due to its practicality, but it does not replace videofluoroscopy of swallowing as the gold standard.

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ABSTRACT

Introduction: Cerebrovascular accident (CVA) is one of the leading causes of death in Brazil and, with the increase in life expectancy of the Brazilian population, sequelae are expected to increase.

Objective: To evaluate the association between brain regions of patients diagnosed with stroke in the chronic phase and the presence of dysphagia.

Method: Clinical, cross-sectional, qualitative and quantitative, descriptive and retrospective study with data collection from 25 medical records, in which variables of association between stroke and dysphagia were analyzed, including cause of stroke, diagnosis of dysphagia by videonasofibroscopy of swallowing, compromised brain region and cerebral circulation evaluated by the Swallowing ScaleBamford/ Oxfordshire Community Stroke Project.

Results: Thirteen (52%) patients were men between 61 and 80 years old. Regarding the type of stroke, 20 (80%) were ischemic. Dysphagia remained in seven (28%) in the chronic phase of stroke. There was a predominance of brainstem/ cerebellum lesions and concomitant dysphagia in 57.1% of these patients, and even more frequent ischemia with posterior circulation involvement in 42.9%, but without a statistically significant association.

Conclusion: Patients affected by stroke, in the chronic phase, have a high percentage of dysphagia (28%) in outpatient follow-up.

KEYWORDS: Stroke. Dysphagia. Cerebrovascular circulation. Endoscopy. Encephalon.

INTRODUCTION

Cerebrovascular accident (CVA) is the second cause of death in Brazil according to data in DATASUS published in 2024 with 106,182 deaths, being surpassed only by acute myocardial infarction (AMI) which had 119,687.[1] Its incidence worldwide in 2020 was 11.71 million cases, of which 7.59 million were ischemic, 4.12 million hemorrhagic.[2,3]

Stroke can present clinically as ischemic (ICVA), thrombotic, embolic or hemorrhagic (CVA).[4] Among the classic signs is dysphagia.[5] In videofluoroscopy or videonasofibroscopy of swallowing, the incidence of swallowing can vary between 41-73%. This variation is justified due to the phases when the diagnosis is made, that is, acute, subacute and/or chronic phases of stroke.[6-13]

The use of videofluoroscopy of swallowing in the acute phase is considered the gold standard to evaluate dysphagia, and notably the penetration of food into the larynx and/or aspiration into the trachea.[14]

There is no association between the location of acute stroke in the cerebral hemispheres; however, residues in pharyngeal recesses that cause aspiration occur less frequently when the stroke is located in the cerebral hemispheres.[8,9,15] A Brazilian study demonstrated post-stroke dysphagia in the acute phase in patients with cortical and subcortical involvement, with no association of it in terms of whether the lesion is in the right or left cerebral hemisphere.[16]

When evaluated by the Oxfordshire Community Stroke Project (OCSP)[17-20] dysphagia predominates with involvement of the anterior and middle cerebral artery. However, there is no difference between dysphagia and compromised topography in either the right or left cerebral hemisphere. However, the involvement of the cerebral circulation of the carotid system predominates in relation to the vertebrobasilar system.[8:19-24]

The objective of the present study was to correlate the presence of dysphagia in those diagnosed with chronic phase stroke and/or stroke and its correlation with the lesions/regions in the brain detected by imaging in SUS users and treated at the Specialized Rehabilitation Center (CER) – Unesp Marília Auxiliary Unit, Marília Campus, in the speech therapy and neurology clinic.[25-37]

METHOD

This study was approved by the Research Ethics Committee of Unesp Marília under opinion No. 2,858,338 because it was in accordance with the Resolution of the National Health Council (CNS) 466/2012 and Resolution of the National Health Council (CNS) 510/2016

This is a cross-sectional, qualitative and quantitative, descriptive and retrospective clinical study carried out through the collection of data from medical records of the Specialized Rehabilitation Center (CER)/Unesp Marília, in a non-probabilistic sample to evaluate the presence of dysphagia in 25 patients diagnosed with stroke, in the chronic, ischemic and/or hemorrhagic phase, referred from the primary health care network and hospitals in Marília and region, who are treated at the Speech Therapy and Neurology Clinic at the CER of Unesp Marília.[26-32]

The variables collected were: age, time of stroke occurrence (years), time of follow-up at the CER outpatient clinic (years), gender, age group, education, sedentary lifestyle, smoking, alcoholism, systemic arterial hypertension, diabetes mellitus, hypercholesterolemia, atrial fibrillation, type of stroke, diagnosis of dysphagia by the use of videonasofibroscopy of swallowing, region/ lesion in the brain evidenced by imaging exam and association with dysphagia, cerebral circulation affected by the OCSP scale and association with dysphagia, and diagnosis of stroke by magnetic resonance imaging of the skull.

The speech-language pathology diagnosis of dysphagia was made through clinical evaluation of the patients attended, in which the presence or absence of dysphagia was investigated, and later confirmed by videonasofibroscopy of swallowing. In this evaluation, 180 days after the occurrence of the stroke, the presence of pharyngeal residues in epiglottic valleculae and/ or piriform recesses were considered indicative of dysphagia.[25-29.36]

In videonasofibroscopy, a Pentax® nasofibroscope, model FNL 10RP3, coupled to a PSV4000® microcamera system, was used, and a Pentax-branded® light source, model LH-150 PC, without topical anesthesia. The zscan 6.0 software was used to analyze the images of the swallowing videoendoscopy to evaluate the visualization

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of the presence of pharyngeal residues (valleculae and/or piriform sinuses) and, if present, the presence of pharyngeal residues (valleculae and/or piriform sinuses) and, if present, the presence of pharyngeal residues (valleculae and/or piriform sinuses) and, if present, the presence of pharyngeal residues (valleculae and/or piriform sinuses) and, if present, the presence of pharyngeal residues (valleculae and/or piriform sinuses) was considered positive for dysphagia.[26]

The pathophysiology and etiology of stroke was considered according to the TOAST Classification (Trial of Org 10172 in Acute Stroke Treatment) and the Manual of Routines for Stroke Care.[30,31]

The diagnosis of CVA and/or CVCH was confirmed by a neurologist at the Neurology Clinic of the CER of Unesp Marília, through analysis of imaging tests (cranial tomography and/or cranial magnetic resonance imaging) requested during the anamnesis and physical examination, and or performed in neurology outpatient clinics and/or hospitals in the city of Marília during consultation, and subsequent reassessment of imaging tests in the neurological clinic at the CER for diagnostic confirmation during the consultation. Those who had other diseases of neurological origin in addition were excluded from this study. Some referred to the CER had had a previous stroke, and for this study only the occurrence of the last stroke was considered for the analysis of the data collected in the medical records.

Statistical analysis

Quantitative variables were described as mean, standard deviation, minimum value, and maximum value. The homogeneity of the variances was analyzed by the Levene test. The comparison of means was analyzed by Student's t-test, and when the assumption of homogeneity was violated, the Welch test was used. The relationship between the qualitative variables was analyzed using the chi-square test. The level of significance was set at 5% (p-value ≤ 0.05) and the data were analyzed using the Jamovi software.

RESULT

The sample that had a stroke was characterized according to sociodemographic (age, gender, age group, and education) and clinical aspects (type of stroke, time since the occurrence of the last stroke, time of speech-language pathology follow-up at the CER/Unesp Marília/SP, cerebral circulation compromised by the OCSP scale, location of the lesion in the brain region evidenced by imaging exam, presence of dysphagia by videonasofibroscopy of swallowing, diagnosis of stroke confirmed by cranial resonance imaging, and comorbidities (sedentary lifestyle, smoking, alcoholism, systemic arterial hypertension, diabetes mellitus, hypercholesterolemia, and atrial fibrillation).

Table 1 shows the relationship between age and possible association with dysphagia predominance of older people (68.3 years, standard deviation = 5.2 years); however, there was no statistically significant difference with patients who did not present dysphagia (61.6 years, standard deviation = 11.5 years). The value of p = 0.057 suggests that age may be associated with the presence of dysphagia, although it is not statistically significant.

In addition, patients with dysphagia had a shorter time since stroke occurrence, on average 2.7 years, compared to those with stroke without dysphagia, on average 6.3 years, indicating that dysphagia occurs more in acute or subacute phases of stroke; however, p = 0.091 does not confirm a statistically significant association.

In this sample, the mean follow-up time with dysphagia and stroke at the neurology outpatient clinic was 1.[1] years (mean, standard deviation = 1.6 years), while in those without dysphagia it was four years (mean, standard deviation = 3.4 years), demonstrating a statistically significant association (p = 0.009).

TABLE 2 — Analysis of the association between the distribution of absolute (n) and relative frequency of the presence or absence of dysphagia with the variables of the sociodemographic sample and comorbidities of the patients

Variables	Categories	Dysphagia				Total		p-value
		No		Yes				
		n	%	n	%	n	%	
Gender	Women	8	44.4%	4	57.1%	12	48.0%	0.568
	Male	10	55.6%	3	42.9%	13	52.0%	
Age group	20-40 years	1	5.6%	0	0.0%	1	4.0%	0.357
	41-60 years	7	38.9%	1	14.3%	8	32.0%	
	61-80 years	10	55.6%	6	85.7%	16	64.0%	
Elementary School	Incomplete	11	61.1%	6	85.7%	17	68.0%	0.236
	Complete	7	38.9%	1	14.3%	8	32.0%	
Sedentary lifestyle	No	14	77.8%	3	42.9%	17	68.0%	0.093
	Yes	4	22.2%	4	57.1%	8	32.0%	
Smoking	No	15	83.3%	4	57.1%	19	76.0%	0.169
	Yes	3	16.7%	3	42.9%	6	24.0%	
Alcoholism	No	13	72.2%	6	85.7%	19	76.0%	0.478
	Yes	5	27.8%	1	14.3%	6	24.0%	
Hypertension systemic	No	3	16.7%	3	42.9%	6	24.0%	0.169
	Yes	15	83.3%	4	57.1%	19	76.0%	
Diabetes mellitus	No	13	72.2%	5	71.4%	18	72.0%	0.968
	Yes	5	27.8%	2	28.6%	7	28.0%	
Hypercholesterolemia	No	9	50.0%	2	28.6%	11	44.0%	0.332
	Yes	9	50.0%	5	71.4%	14	56.0%	
Atrial fibrillation	No	18	100.0%	6	85.7%	24	96.0%	0.102
	Yes	0	0,0%	1	14,3%	1	4,0%	

Note: p calculated by the chi-square test of association; #Centro Specialized in Rehabilitation (CER)/Unesp Marília.

TABLE 1 — Comparison of the mean, standard deviation, minimum and maximum value of the quantitative variables of the sample in relation to the presence of dysphagia in SUS patients with stroke in terms of age, time of stroke occurrence and follow-up time attended at the CER

Variables	Dysphagia								p-value
	No (n = 18)				Yes (n = 7)				
	Average	DP	Minimum	Maximum	Average	DP	Minimum	Maximum	
Age (years)	61.6	11.5	40.0	78.0	68.3	5.2	60.0	73.0	0.057*
Time of stroke occurrence (years)	6.3	5.2	0.0	21.0	2.7	2.4	0.0	7.0	0.091**
Follow-up time at CER/ UNESP Marília (years)#	4.0	3.4	0.0	11.0	1.1	1.6	0.0	4.0	0.009*

*p calculated by Welch's test; ** p calculated by Student's t-test; significance level of 5% (p ≤ 0.05); # Specialized Rehabilitation Center (CER)/Unesp, Marília.

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Table 2 presents an analysis of the association of dysphagia in relation to sociodemographic variables and comorbidities, as well as the respective p-values obtained in association tests (chi-square). In general, no statistically significant association was observed between the presence of dysphagia and the variables analyzed, considering the significance level of 5%.

Among women (n = 4, 57.1%), there was a higher absolute and relative frequency of dysphagia compared to men (n = 3, 42.9%). The p-value = 0.568 is compatible with this distribution, indicating the absence of a statistically significant association between gender and the presence of dysphagia. Thus, although there is numerical variation between the groups, this difference seems to be due to the small sample size and random fluctuation, and not to a systematic effect of gender on the occurrence of dysphagia.

The largest number of patients with dysphagia occurs in the 61–80 year age group, which goes hand in hand with the absolute increase in stroke cases in this age group. However, when comparing the proportion of dysphagia between the different age groups (20–40, 41–60, and 61–80 years), the association test resulted in p = 0.357, suggesting that, in the sample studied, age was not statistically significantly associated with the presence of dysphagia. This result should be interpreted with caution, as there are categories with a very small number of patients (e.g., only one individual in the 20–40 age group), which limits the statistical power of the test and increases the chance of type II error (not detecting an association that may exist in the population).

Patients with incomplete primary education represented the majority of the sample (n = 17, 68.0%) and concentrated the largest number of dysphagia cases (n = 6, 85.7%). However, when the proportions of dysphagia were analyzed between the groups with complete and incomplete primary education, no statistically significant difference was observed (p = 0.236). The coherence between the absolute and relative frequencies presented and the p-value suggests that, even with a predominance of low education among patients with dysphagia, this pattern seems to reflect the sociodemographic profile of the population served, and not an independent association between education and dysphagia.

Among sedentary patients (n = 8, 32%), a relatively higher proportion of dysphagia was detected than in non-sedentary patients (n = 17, 68%). A higher proportion of sedentary lifestyle was observed in those with dysphagia (n = 4, 57.1%) compared to those without (n = 4, 22.2%). Despite this percentage difference, the result was not statistically significant (p = 0.093), which is close to the threshold of statistical significance, but remains above the cutoff point of 5%. This result may indicate a trend of association between sedentary lifestyle and the presence of dysphagia; however, with the sample available (n = 25), the test was not able to confirm this relationship with statistical significance.

There was a higher proportion of smoking in those with dysphagia (n = 3, 42.9%) than in those without (n = 3, 16.7%). The proportion of dysphagia is numerically higher among smokers (n = 6, 24%) than among nonsmokers (n = 19, 76%); However, the value of p = 0.169 indicates that smoking and dysphagia were not significant in the sample studied. The relative frequencies presented are compatible with a scenario in which observed differences can be explained by random variation, especially considering the small number of smokers with dysphagia. Therefore, there is no robust evidence that smoking, in isolation, is associated with the occurrence of dysphagia in this group.

It was revealed that most patients did not have alcoholism (n = 19.76%), both in those without (n = 13, 72.2%) and in those with dysphagia (n = 6, 85.7%). The proportion of individuals with alcoholism was higher in those without dysphagia (n = 5, 27.8%) compared to those with (n = 1, 14.3%). In total, 24.0% of those evaluated reported alcoholism. Despite this numerical difference between the groups, the statistical analysis did not show statistical significance (p = 0.478), indicating that there was no association between alcoholism and dysphagia in the sample studied.

It was reported that the prevalence of systemic arterial hypertension was high in both with and without dysphagia, being more frequent in those without (n = 15, 83.3%) compared to those with (n = 4, 57.1%). Despite this difference, the statistical analysis did not show significance (p = 0.169), indicating the absence of a statistically significant association between hypertension and dysphagia.

It was demonstrated that the prevalence of diabetes mellitus was similar among all, with 27.8% (n = 5) without dysphagia and 28.6% (n = 2) with dysphagia. In total, 28.0% (n = 7) had diabetes. Statistical analysis showed no significant difference between those with and without dysphagia (p = 0.968), suggesting no relevant association between diabetes and dysphagia.

The analysis of the presence of hypercholesterolemia showed that nine (50.0%) of the patients without dysphagia had hypercholesterolemia, while nine (50.0%) did not. In those with dysphagia, hypercholesterolemia was observed in five (71.4%), while two (28.6%) did not have it. In the total sample, 14 individuals (56.0%) had hypercholesterolemia and 11 (44.0%) did not. There was also no statistically significant association between hypercholesterolemia and dysphagia (p = 0.332)

It was found that in the patients without dysphagia, none had atrial fibrillation (0%). In those with, one case of atrial fibrillation was identified (14.3%), while six participants (85.7%) did not. In the total sample, only one patient (4.0%) had atrial fibrillation, while 24 (96.0%) did not.

Finally, there were no statistically significant associations between dysphagia and the variables analyzed (gender, age group, education, sedentary lifestyle, smoking, alcoholism, hypertension and diabetes, hypercholesterolemia, and atrial fibrillation), although sedentary lifestyle and fibrillation presented p values close to the significance threshold, suggesting possible trends that deserve investigation in larger samples.

Table 3 presents the analysis of the association

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TABLE 3 — Analysis of the association between the distribution of absolute (n) and relative frequency of dysphagia in terms of the type of stroke, diagnosis of dysphagia by videonasofibroscopy, unilaterality of the lesion/region in the CNS##, impaired cerebral circulation and imaging tests

Variables	Categories	Dysphagia				Total		p-value
		No		Yes				
		n	%	n	%	n	%	
Type of stroke	Hemorrhagic	4	22.2%	1	14.3%	5	20.0%	0.656
	Ischemic	14	77.8%	6	85.7%	20	80.0%	
Videonasofibroscopy of swallowing	Changed	0	0.0%	7	100.0%	7	28.0%	<0.001 *
	Normal	18	100.0%	0	0.0%	18	72.0%	
Region/lesion	Right cerebral hemisphere	5	27.8%	0	0.0%	5	20.0%	0.102
	Right and left cerebral hemisphere	3	16.7%	2	28.6%	5	20.0%	
	Left cerebral hemisphere	7	38.9%	1	14.3%	8	32.0%	
	Brainstem or cerebellum	3	16.7%	4	57.1%	7	28.0%	
Cerebral circulation#	Lacunar syndrome - LACS	3	16.7%	0	0.0%	3	12.5%	0.23
	Partial anterior circulation syndrome PACS	1	5.6%	1	14.3%	2	8.3%	
	Posterior Circulation Syndrome - POCS	3	16.7%	3	42.9%	6	25.0%	
	Total anterior circulation syndrome - TACS	11	61.1%	2	28.6%	13	54.2%	
Skull resonance	No	2	11.1%	3	42.9%	5	20.0%	0.05
	Yes	16	88.9%	4	57.1%	20	80.0%	

Note: *indicates a statistically significant association between dysphagia and videonasofibroscopy of swallowing by the chi-square test for p-value ≤ 0.05; ##=CNS central nervous system; # Oxfordshire Community Stroke Project (OCSP) is a clinical classification based on neurological deficits in stroke (Bamford Scale); lacunar syndrome (LACS), total anterior circulation syndrome (TACS - total anterior circulation syndrome), partial anterior circulation syndrome (PACS), and posterior circulation syndrome (POCS). For stroke only, excluding patients with CVA.

between the presence of dysphagia and variables related to the type of stroke, diagnosis by nasovideofibroscopy of swallowing, location of the lesion/brain region in the stroke, cerebral circulation compromised by the Bamford Scale, and magnetic resonance imaging. p-values were calculated using the chi-square test, considering a significance level of 5%.

In the analysis of the type of stroke, it was found that in the patients without dysphagia, four (22.2%) had hemorrhagic stroke and 14 (77.8%) had ischemic stroke. In the patients with dysphagia, one participant (14.3%) had hemorrhagic stroke and six (85.7%) had ischemic stroke. In the total sample, five cases of hemorrhagic stroke (20.0%) and 20 cases of ischemic stroke (80.0%) were recorded. There was no significant difference between patients with and without dysphagia and the type of stroke (p = 0.656).

All patients with a diagnosis of dysphagia confirmed by videonasofibroscopy of swallowing (n = 7) had dysphagia (100%), while none with normal endoscopic evaluation (n = 18) had it. There was a statistically significant association between endoscopic findings on videonasofibroendoscopy and the presence of dysphagia (p < 0.001). These findings reinforce videonasofibroscopy of swallowing as an essential diagnostic method for the detection of dysphagia in the sample studied with high sensitivity and specificity.

Lesions in the left hemisphere were the most frequent (n = 8, 32.0%), followed by brainstem/cerebellum (n = 7, 28%), right hemisphere (n = 5, 20%), and right and left hemisphere (n = 5, 20%). Those with lesions in the brainstem or cerebellum had a higher proportion of dysphagia (n = 4, 57.1%). Although the distribution of lesions shows a trend toward a greater association between brainstem/cerebellum involvement and clinical outcome, the statistical analysis did not show significance (p = 0.102).

Regarding the classification of infarction sites evaluated by the OCSP classification, which evaluates only patients with ischemic stroke, 54.2% (n = 13) of infarctions occurring in the total anterior circulation (TACS), 8.3% (n = 2) of infarctions in the partial anterior circulation (PACS), 25% (n = 6) of infarctions in the posterior circulation (POCS), and 12.5% (n = 3) of lacunar infarctions (LACS). The presence of dysphagia diagnosed by videonasofibroscopy was more frequent among patients classified as POCS (n = 6, 42.9%). However, there was no statistically significant association between the clinically involved cerebral circulations and the occurrence of dysphagia (p = 0.23).

Regarding magnetic resonance imaging of the skull, it was found that 20 patients (80%) underwent the examination, while five (20%) did not. Among those who did not have it, three (42.9%) were associated with dysphagia; However, among those who underwent the test, four (57.1%) had dysphagia. Statistical analysis showed a trend toward an association between MRI and dysphagia, with a value of p = 0.05, indicating proximity to the significance threshold. These findings suggest that cranial MRI may contribute to the identification of dysphagia, although the results should be interpreted with caution.

DISCUSSION

The present study aimed to investigate the presence of dysphagia in stroke with definitive diagnosis by imaging tests (magnetic resonance imaging/cranial tomography), ischemic or hemorrhagic types, in the chronic phase, and association with brain lesions, in a population sample.[1,2]

According to data from the national and international literature, the diagnosis of ischemic stroke in the present study was present in 80% of the patients, in agreement with other national and international studies, and in another study by the same author.[2,3,8,9,16,20-23,31,37] The results showed a higher prevalence of stroke in those over 60 years of age, a finding that is in line with the national and international literature. Global data from 2020 indicate that the worldwide prevalence of stroke

reached 89.13 million cases, with a predominance in the most advanced age groups, reinforcing the impact of population aging on the incidence of the disease. Brazilian clinical studies also confirm this trend in the evaluation of post-stroke dysphagia in patients with cortical, subcortical and cerebellar involvement.[2,16]

In addition, studies that analyzed subcortical and internal capsule lesions identified dysphagia in 35% of the patients, predominantly in the left cerebral hemisphere, with a higher occurrence in elderly individuals.[22,23] An analysis of the OCSP classification showed that carotid circulation impairment tends to predominate in the elderly.[8,9,20,21]

In the present study, the incidence of dysphagia in the chronic phase of stroke was 28%, a result that is consistent with the literature. Previous studies have reported divergent incidence rates of dysphagia in clinical evaluation and/or by videonasofibroscopy, being 50% in clinical evaluation six months after the fact, and 2-30% in the chronic phase of stroke in endoscopic evaluation in national and international publications.[32-36]

These findings reinforce that, although dysphagia is more frequently investigated in the acute phase, a significant proportion of patients maintain the sequelae in the chronic phase, with a direct impact on quality of life and the need for prolonged multidisciplinary follow-up. The convergence between the results of the present study and the percentages described in the references confirms the clinical relevance of dysphagia as a persistent complication of stroke, highlighting the importance of screening protocols and continuous rehabilitation in specialized services.

It should be noted that in this study, patients with stroke and concomitant dysphagia at the time of care at the neurology outpatient clinic had a mean diagnosis of 2.7 years, and those without dysphagia had a mean of 6.3 years. The literature shows that dysphagia is more found in the acute and subacute phases of stroke, with a progressive reduction in chronic evaluations.[6-11,32-36]

In the present study, it was observed that patients with dysphagia had a significantly shorter mean follow-up time in the CER (1.1 years) compared to those without (4 years), a statistically significant difference ($p = 0.009$). This finding suggests that dysphagia tends to manifest and be diagnosed in earlier stages of outpatient follow-up, reflecting the need for immediate multidisciplinary intervention after the vascular event. The reduced clinical follow-up time in the present study among individuals with dysphagia may be related to the greater initial clinical severity, the need for specialized referrals, or even the interruption of follow-up due to complications. These results reinforce the importance of early screening protocols and continuous follow-up, in order to identify and treat dysphagia from the beginning of care, preventing complications such as aspiration, pneumonia, and malnutrition.[12-15,22-24,32-36]

The literature indicates that there is no clear association between the affected cerebral hemisphere and the occurrence of post-stroke dysphagia.[8,9,16,20,21] However, it is more frequent in cases of lesions of the brainstem, cerebellum, and internal capsule, due to the direct involvement of cranial nuclei and nerves related to swallowing control.[15,22,23]

The results of the present study confirm this absence of a significant association with the cerebral hemisphere involved in stroke, but reinforce the relevance of analyzing the topography of the lesion. The distribution found in this study suggests that lesions in subcortical structures, brainstem and cerebellum have a greater relationship with dysphagia, and its permanence in the chronic phase.[22-24,32-36]

The gold standard for diagnosing post-stroke dysphagia is swallowing videofluoroscopy. Videonasofibroscopy is considered a highly accurate exam, complementary to clinical diagnosis and often preferred in clinical environments due to its practicality, but it does not replace videofluoroscopy of swallowing as the gold standard. The diagnosis of certainty when using videonasofibroscopy of swallowing, after clinical evaluation, evidences a statistically significant association between the result of videonasofibroscopy of swallowing and the presence of dysphagia, in agreement with other authors who indicate it as reliable, validated by several studies, and often used as a practical alternative, especially in public services or in bedridden patients for the diagnosis of dysphagia.[12,13,27-29]

In the acute phase of stroke, studies do not show an association between the location of the stroke, right and/or left cerebral hemisphere, and the presence of dysphagia; however, the presence of aspiration occurs more frequently in individuals who have brainstem stroke, and concomitant absence in the swallowing reflex.[6,8,9,15,16] There was a predominance of lesions in the left cerebral hemisphere (n = 8, 32%), but dysphagia was more common in those with lesions in the brainstem/cerebellum (n = 7, 28%), and 57.1% of these patients (n = 4) maintained dysphagia in the chronic phase of stroke.[32-36]

The OCSP classification allows the correlation of clinical and imaging findings with the cerebral circulation involved in ischemic stroke.[17] In the present study, a predominance of events in the total anterior circulation (54.2%) was demonstrated, in line with the literature that describes a higher frequency of involvement of arteries originating from the carotid system.[18-20] However, dysphagia was more associated with posterior circulation involvement (42.9%), a finding that suggests a possible relationship between vertebrobasilar lesions and swallowing impairment, given the role of the brainstem and cerebellum in the motor coordination of swallowing. Despite this trend, no statistically significant difference was identified ($p = 0.23$), a result that is similar to previous studies that also did not demonstrate a robust association between the topography of cerebral circulation and the presence of dysphagia, suggesting that additional factors - such as lesion extent, functional impairment, and cortical-subcortical connectivity - should be

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considered in understanding the pathophysiology of post-stroke dysphagia.[18,19]

All patients evaluated were in the chronic phase of stroke, i.e., 180 days after the last ischemic or hemorrhagic event. Despite structural alterations detected in 100% of the CT scans and in 80% of the magnetic resonance imaging, no statistically significant association was observed between the presence of brain lesions on imaging studies and the occurrence of dysphagia. Among the seven patients with dysphagia, 42.9% did not undergo magnetic resonance imaging and 57.1% had lesions confirmed by the examination, suggesting that the simple detection of the lesion does not predict, in isolation, the functional impairment of swallowing.

This finding can be explained by the brain plasticity that occurs after stroke, an adaptive mechanism that allows functional reorganization and partial or total recovery of swallowing over time. Previous studies have shown that dysphagia is very frequent in the acute and subacute phases of stroke, with a significant reduction in the chronic phase due to neural reorganization and motor compensation.[32-36]

In the present study, none of the sociodemographic or clinical variables analyzed - including gender, age group, education, sedentary lifestyle, smoking, alcoholism, systemic arterial hypertension, diabetes mellitus, hypercholesterolemia, and atrial fibrillation - showed a statistically significant association with the presence of dysphagia. Only sedentary lifestyle and atrial fibrillation showed an association trend, with p-values close to the significance threshold ($p \leq 0.05$), suggesting that these factors may play some modulating role, although they were not confirmed as determinants in the sample studied.

These findings are in line with the literature, which points out that the occurrence of post-stroke dysphagia is more related to the neurological event itself - especially to the location and extent of the brain injury - than to isolated traditional risk factors.[8,9,16,20,21] Previous studies have shown that variables such as gender, age, or cardiovascular comorbidities do not have a consistent association with the presence of dysphagia, reinforcing that functional swallowing impairment is primarily due to neurological dysfunction.[22-24] In addition, investigations in patients in the chronic phase of stroke indicate that brain plasticity and neural reorganization may attenuate the impact of cardiovascular and lifestyle risk factors on swallowing function, explaining the absence of a significant association observed.[32-36]

The present study showed that dysphagia is a relevant complication in patients affected by chronic stroke, and it remains an important sequela, negatively impacting the prognosis and continuity of treatment.

Although no statistical association was identified between the location of the brain lesion and the occurrence of dysphagia, the literature points to a higher risk in lesions of the brainstem, cerebellum, subcortical structures, and internal capsule, which highlights the importance of early clinical evaluation and subsequent confirmation by videonasofibroscopy.

Thus, the results of the present study corroborate the hypothesis that post-stroke dysphagia is a direct consequence of neurological damage and the topography of the lesion, while cardiovascular and lifestyle risk factors act more as predisposing factors for the vascular event than as determinants of the presence or absence of dysphagia. Furthermore, the present study corroborates the hypothesis that, in the chronic phase of stroke, the presence of dysphagia is not directly associated with structural changes detected by imaging tests, but rather with the extent of the lesion, the involvement of critical regions and, above all, the brain plasticity that allows the return of swallowing function to normality in a significant portion of patients. The results highlight the need for systematic protocols for screening and monitoring swallowing in post-stroke patients, including those in the chronic phase, in order to prevent complications such as aspiration pneumonia, malnutrition, and early mortality.

Despite the limitations of the study, such as the small sample size and retrospective design, the findings contribute to the understanding of the relationship between chronic stroke and dysphagia in the context of the SUS and point to the importance of multicenter studies with a larger number of patients, which can deepen the analysis of the association between cardiovascular risk factors, lifestyle, and the topography of the lesion.

CONCLUSION

Patients affected by stroke in the chronic phase have a high percentage of dysphagia (28%) when they are in outpatient follow-up. Although no statistical association was identified between the location of the brain lesion and the occurrence of dysphagia, a higher risk was found in lesions of the brainstem, cerebellum, subcortical structures and internal capsule, which highlights the importance of early clinical evaluation and subsequent confirmation by videonasofibroscopy.

REFERENCES

1. Ministério da Saúde. Secretaria Executiva. Datasus. Informações de saúde. mortalidade e informações epidemiológicas. Disponível em: http://tabnet.datasus.gov.br/cgi/tabcgi.exe?sim/cnv/obt10uf.def.
2. Tsao, CW, Aday AW, Almazooq ZI, Anderson CAM, Aora P, Avery CL, et al. Heart Disease and Stroke Statistics-2023 Update: A Report from the American Heart Association. Circulation. 2023;47(8):e93-e621. https://doi.org/10.1161/cir.0000000000001123
3. BRASIL. Comissão Nacional de Incorporação de Tecnologias no Sistema Único De Saúde (CONITEC). Protocolo clínico e diretrizes terapêuticas do Acidente Vascular Cerebral Isquêmico Agudo. Brasília: Ministério da Saúde, 2021.
4. Organização Mundial da Saúde. O Manual STEPS de Acidentes Vascular Cerebrais da OMS: enfoque passo a passo para a vigilância de acidentes vascular cerebrais/doenças não transmissíveis e saúde mental. Genebra: Organização Mundial da Saúde; 2005.
5. Brasil. Ministério da Saúde. Secretaria de Atenção à Saúde. Departamento de Ações Programáticas Estratégicas. Diretrizes de atenção à reabilitação da pessoa com acidente vascular cerebral. Brasília: Ministério da Saúde; 2013.

6. Xerez, DR, Carvalho YSV, Costa MMB. Estudo clínico e videofluoroscópico da disfagia na fase subaguda do acidente vascular encefálico. Radiol Bras. 2004;37(1):9-14. https://doi.org/10.1590/S0100-39842004000100004

7. Schelp AO, Cola PC, Gatto AR, Silva RG, Carvalho LR. Incidência de disfagia orofaríngea após acidente vascular encefálico em hospital público de referência. Arquivos de Neuro-Psiquiatr. 2004;62(2B):503-6. https://doi.org/10.1590/S0004-282X2004000300023

8. Barros AFF, Fabio SRC, Furkim AM. Correlação entre os achados clínicos da deglutição e os achados da tomografia computadorizada de crânio em pacientes com acidente vascular cerebral isquêmico na fase aguda da doença. Arq Neuro-Psiquiatr. 2006;64(4):1009-14. https://doi.org/10.1590/S0004-282X2006000600024

9. Smithard DG, Smeeton NC, Wolfe CDA. Long-term outcome after stroke: does dysphagia matter? Age Ageing. 2007;36(1):90-4. https://doi.org/10.1093/ageing/afl149

10. Jacques A, Cardoso MCAF. Acidente vascular cerebral e sequelas fonoaudiológicas: atuação em área hospitalar. Rev Neurocienc. 2011;19(2):229-36. https://doi.org/10.34024/rnc.2011.v19.8371

11. Falsetti P, Acciai C, Palilla R, Bosi M, Carpinteri F, Zingarelli A, et al. Oropharyngeal dysphagia after stroke: incidence, diagnosis, and clinical predictors in patients admitted to a neurorehabilitation unit. J Stroke Cerebrovasc Dis. 2009;18(5):329-35. https://doi.org/10.1016/j.jstrokecerebrovasdis.2009.01.009

12. Bax L, McFarlane M, Green E, Miles A. Speech–language pathologist-led fiberoptic endoscopic evaluation of swallowing: functional outcomes for patients after stroke. J Stroke Cerebrovasc Dis. 2014;23(3):e195-e200. https://doi.org/10.1016/j.jstrokecerebrovasdis.2013.09.031

13. Shoji H, Nakane A, Omosu Y, Sawashima K, Teranaka S, Umeda Y, et al. The prognosis of dysphagia patients over 100 years old. Arch Gerontol Geriatr. 2014;59(2):480-4. https://doi.org/10.1016/j.archger.2014.04.009

14. East L, Nettles K, Vansant A, Daniels SK. Evaluation of oropharyngeal Dysphagia with the videofluoroscopy Swallowing Study. J Radiol Nursing. 2014;33(1): 9-13. https://doi.org/10.1016/j.jradnu.2013.08.004

15. Bassi AER, Mitre EI, Silva MAOM, Arroyo MAS, Pereira MC. Associação entre disfagia e o topodiagnóstico da lesão encefálica pós-acidente vascular encefálico. Rev CEFAC. 2004;6(2):135-42. https://doi.org/10.1590/1516-1846/200462p0135

16. Nunes MC, Jurkiewicz AL, Santos RS, Furkim AM, Massi G, Pinto GS, et al. Correlation between brain injury and dysphagia in adult patients with stroke. Int Arch Otorhinolaryngol. 2012;16(3):313-21. https://doi.org/10.7162/s1809-97772012000300003

17. Bamford J, Sandercock P, Dennis M, Burn J, Warlow C. Classification and natural history of clinically identifiable subtypes of cerebral infarction. Lancet. 1991;337(8756):1521-26. https://doi.org/10.1016/0140-6736(91)93206-o

18. Mourão AM, Almeida EO, Lemos SMA, Vicente LCC, Teixeira AL. Evolução da deglutição no pós-AVC agudo: estudo descritivo. Rev CEFAC. 2016;18 (2):417-25. https://doi.org/10.1590/1982-0216201618212315

19. Mourão AM, Lemos SMA, Almeida EO, Vicente LCC, Teixeira AL. Frequencia e fatores associado à disfagia após acidente vascular cerebral. CoDAS. 2016;28(1):66-70. https://doi.org/10.1590/2317-1782/20162015072

20. Okubo PCMI. Detecção de disfagia na fase aguda do acidente vascular cerebral isquêmico: proposição de conduta baseada na caracterização dos fatores de risco [tese]. Ribeirão Preto: Faculdade de Medicina da Universidade de São Paulo; 2008.

21. Paciaroni M, Mazzotta G, Corea F, Caso V, Venti M, Milia P, et al. Dysphagia following stroke. Eur Neurol. 2004;51(3):162-7. https://doi.org/10.1159/000077663

22. Cola MG, Daniels SK, Corey DM, Lemen LC, Romero M, Foundas AL. Relevance of subcortical stroke in dysphagia. Stroke. 2010;41(3):482-86. https://doi.org/10.1161/strokeaha.109.566133

23. Gonzalez-Fernandez M, Kleinman JT, Ky PKS, Palmer JB, Hillis AE. Supratentorial Regions of Acute Ischemia associated with Clinically important swallowing Disorders: A Pilot Study. Stroke. 2008;39(11):3022-8. https://doi.org/10.1161/strokeaha.108.518969

24. Gonzalez-Fernandez M, Ottenstein L, Atanelov L, Christian AB. Dysphagia after stroke: an overview. Curr Phys Med Rehabil Rep. 2013;1(3):187–96. https://doi.org/10.1007/s40141-013-0017-y

25. Silva RG. Disfagia orofaríngea pós-acidente vascular encefálico. In: Ferreira LP, Befi -Lopes DM, Limongi SCO, organizadores. Tratado de fonoaudiologia. São Paulo: Rocca; 2004. p. 354-69.

26. Berola, BN. Correlação entre aspiração laringotraqueal, resíduos faríngeos e escape oral posterior na disfagia orofaríngea neurogênica. [dissertação]. Marília, SP: Faculdade de Filosofia e Ciências, Universidade Estadual Paulista (UNESP); 2019.

27. Langmore S, Schatz K, Olsen N. Fiberoptic endoscopic examination of swallowing safety: a new procedure. Dysphagia. 1988; 2(4):216-19. https://doi.org/10.1007/bf02414429

28. Araújo RCP, Ferreira LMBM, Godoy CMA, Magalhães H. Fase faríngea da deglutição na disfagia pós-AVE: achados videoendoscópios e da avaliação fonoaudiológica. CoDAS. 2024;36(5):e20230242. https://doi.org/10.1590/2317-1782/20242023242pt

29. Neubauer PD, Rademaker AW, Leder SB. The yale pharyngeal residue severity rating scale: an anatomically defined and image-based tool. Dysphagia. 2015;30(5):521-8. https://doi.org/10.1007/s00455-015-9631-4

30. Adams HP Jr, Bendixen BH, Kappelle LJ, Biller J, Love BB, Gordon DL, et al. Classification of subtype of acute ischemic stroke. Definitions for use in a multicenter clinical trial. TOAST. Trial of Org 10172 in Acute Stroke Treatment. Stroke. 1993;24(1):35-41. https://doi.org/10.1161/01.str.24.1.35

31. Brasil. Ministério da Saúde. Secretaria de Atenção à Saúde. Departamento de Atenção Especializada. Manual de rotinas para atenção ao AVC / Ministério da Saúde, Secretaria de Atenção à Saúde, Departamento de Atenção Especializada. – Brasília: Editora do Ministério da Saúde, 2013. 50p.: il.

32. Mann G, Hankey GJ, Cameron D. Swallowing function after stroke: prognosis and prognostic factors at 6 months. Stroke. 1999;30(4):744-8. https://doi.org/10.1161/01.str.30.4.744

33. Nilsson H, Ekberg O, Olsson R, Hindfelt B. Dysphagia in stroke: a prospective study of quantitative aspects of swallowing in dysphagic patients. Dysphagia. 1998;13(1):32-8. https://doi.org/10.1007/pl00009547

34. Wade DT, Hewer RL. Motor loss and swallowing difficulty after stroke: frequency, recovery, and prognosis. Acta Neurol Scand. 1987;76(1):50-4. https://doi.org/10.1111/j.1600-0404.1987.tb03543.x

35. Nunes MCA. Correlação da evolução da disfagia e a recuperação clínica no acidente vascular cerebral isquêmico. [tese]. Curitiba: Universidade Federal do Paraná; 2016.

36. Meng PP, Zhang SC, Han C, Wang Q, Bai GT, Yue SW. The occurrence rate of swallowing disorders after stroke patients in Asia: a PRISMA-compliant systematic review and meta-analysis. J Stroke Cerebrovasc Dis. 2020;29(10):105113. https://doi.org/10.1016/j.jstrokecerebrovasdis.2020.105113

37. Marchioli, M. Perfil epidemiológico dos pacientes acometidos por avc com sequelas fonoaudiológicas e disfunção motora atendidos no CEES - UNESP MARÍLIA/SP. Rev. Méd. Paraná, Curitiba. 2018;76(2):9-15.